Chapter 3. Review of Literature

3.1. Review of vrana

3.1.1 Historical Review of Vrana

Ayurveda is a science of healthy life as well as diseases. Since ages, it is serving the health needs to society. Hence Shalyatantra is evolved as a special branch of Ayurveda for surgical treatments.

Ancient surgeons have done surgeries on the various diseases. They have treated from minor conditions like warts to major conditions like non-healing ulcers, plastic surgeries and grafts. The constant research for treatments of diseases are seen from Vedic period. Many references are available such as administration of Rohini Aushadi in Kshata and Vrana, and the sheetalajaladhaara to stop the bleeding in Sadhyovrana.15

A] Vedic review:

Here a descriptive information about body, diseases and treatments are available in Rigveda and Athavaveda. In Rigveda, Mantra-chikitsa, Aushadhi chikitsa and Shalya chikitsa were at its highest peak.

Ashwinikumar are “Vaidyas of god”. They learnt madhuvidy i.e transplantation surgery and pravragyavidya i.e. plastic surgery from Acharya Dadichi. They are famous plastic surgeon and organ transplant surgeon16.

Vipashyala the daughter of king Khel lost her limbs in war. However Ashwinikumar transplanted her limbs with copper/iron Metal.17

1. Athavaveda:

There are many references in Atharvaveda about Apachivedhan, removal of Garbhashay and Vranchikitsa, and ligation of artery to stop bleeding.18 In Chandogyaupanishad a reference of Madhuvidyatransplantation Surgery) is available19.

In Bruhdaaranyak and Shathpath brahmhan reference of Acharya Dadhichi is available. He was famous plastic surgeon and organ transplant surgeon. So Ashwinikumar transplanted head of horse to Dadhichi and Dadhich’s head to horse.20 Also references of Rakshoghana, Vishalyaghana, Kriminashan, Rohan and Sandhan dravyas are mentioned21.
2. **Agnipuran**: Surgical wounds are mentioned in Agnipuran.

3. **Mahabharat**: At the time of Kurukshetra yuddaha, in Bhismparva and Udyoga parva and in Anushasan parva various references regarding wound healing are mentioned. It is also stated that there were so many surgeons available for removal of Shalya.

4. **Mahavagga (Buddhist tradition)**: Vrana with pooya (pus) were treated with medicine such as kshaar. The external application of such medicines causes expulsion of pus.

5. **Koutilya Arthashastra**: In Koutilya arthashastra the reference of Dushta vrana is available. Also, the other references of Yantra, Shastra, Agad, Sneha, and Vastra are available.

6. **Jatkamala**: Dushta vranas which are painful along with pus should be carefully opened and drained. The wound becomes painful when it comes in contact with salt.

7. **Kaadambari**: Wounds are produced by constant friction. Severe injury produces disabilities in the organs. After healing, the wound the scar remains for whole life.

**B) Samhita kaal:**

A detailed description of vrana with management is mentioned in Brihattraye and Laghuttraye. Charaka has explained 36 therapeutic measures of Vrana in Dwivraniyachikitsa adhayay in Chikitsasthan. Sushrutha has mentioned 60 therapeutic measures for vrana known as Shashti-upkrama in chikitsasthan. For Vrana chikitsa, he mentioned 1st adyaya of chikitsasthana, as Dwivranachikitsa.

Information of vrana is also mentioned in Bhel-samhitha, Kashyapa samhita, Gadagnraha, Chakradatta, Yogaratnakara, and Bhaishajyaratnavali and in Madhavnidan also.
3.1.2. Review of vrana

Vrana is one of the important subject mentioned in Ayurveda. It is described in detail by Sushruta. About the Vrana various references are available in Samhitas.

A) Nirukti (Derivation):

‘vrana gata vichurnane.’

A word Vrana is derived from the root Vriya meaning to recover.

Vran + a, in the sense of ‘Gaatra Vichurnane’

Vrana is a condition, even after complete healing it leaves a scar (vranavastu) over the area. The scar remains for whole life.

Gaatra (body tissue or part of body) Vichurnane means rupture or destruction or discontinuation.

The discontinuity or destruction of body tissue is known as Vrana.

B) Definition of Vrana:

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Vrana is a condition which even after the complete healing leaves a scar over the area, which stays as long as the person is alive.

C) Specialties of Vrana:

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- Six roots of vrana i.e. mulsthan (or causes of vitiation) - Vaata, Pitta, Kapha, Rakta, Sannipataj and Agantuja
- Eight sites or vranavastu or vranaadhistan - Twak, Mamsa, Sira, Snayu, Sandhi, Asthi, Kostha, Marama.
- Five features of examination (Panchalakshan) - shape, pain, colour, odour, and discharge.
- Sixty types of treatment (Shashatividhan) - i.e. vrana treated with Shasti upkramas.
Can be treated successfully if the four necessities, i.e. Chikitsachatushaad are ideal.

D) Classification:
Vrana is classified into two categories : Nija Vrana and Aagantuj Vrana

I) Nija or Shareeraja Vrana:
Sushruta has specially mentioned Nija Vranas occurs due to Vaataja, Pittaja, Kaphaja, Raktaja and Sannipataja Dosha.
These are further classified into 15 types on the basis of vitiation of dwi-doshas and tridoshas along with Raka.
Charaka has described Nija Vrana has 3 types i.e.due to Vaata, Pitta and Kapha.

II) Aagantuja Vrana or Sadyovrana:
It is caused by external trauma from Purusha, Pashu, Pakshi, Vyaala, Prapatana, Peedana, Prahara, Teekshnaoushadha, Agni, Kshara, Visha, Kapaala, Shringa.
Sushruta has mentioned six types as Chinna, Bhinna, Vidhha, Kshata, Picchita, Ghrishta.
Ashtanga hridaya mentioned eight types as Ghrishta, Avakruta, Vicchinna, Pravilambita, Paatita, Viddha, Bhinna, Vidalith.
Ashtanga Sangraha mentiond three types as Chinna, Viddha, Picchita.Madhava Nidana has stated same as that of Sushruta.
Sharangdhara has mentioned eight types as Avkrlupta, vilambint, Chiina, Bhinna, Vidalita, Grushta, Viddha, Nipaaitit.

E) Nidana of Vrana:
Doshaj, Aaharaj and viharaj

1. Vata Dosha –
Aharaj hetu: Vaataprakopak aahaaars, i.e. Ruksha, laghu, Sheet, lavana, katu, ahaara, shushkashaaka, vallura, uddhalak, etc.
Viharaj hetu: balawat vigrah, excessive panchakarmas like Vamana, Virechana, Raktamokshana, ativyaayaama and supression of Adharanneya vega vyavaya, atiadhyanan, prapatanan, langhana, jagaran

2. Pitta Dosha --
Aharaj Hetu: Pittaprakopaka aahara i.e. amla, katu, lavana, kshaara, Ushna, teekshna, laghu, vidaahi, tila taila, pinyaaka,
Viharj Hetu: kroda, shoka, bhaya, aayas i.e. sharirpida, upavaasa, maithuna.
3. **Kapha Dosha**—

**Aharaj Hetu**: Guru, madhur, snigdha, sheeta, lavana, maasha, mahaamaasha

**Viharaj Hetu**: avyaayaam (lack of physical exercise), aalasya, diwaswapna, (sleep during day)

**F ) Vrana Lakshanas:**

There are two types of Vrana features:

**A. Samanya**: Vedana (pain.)

**B. Vishesha**: consists of signs and symptoms caused by vitiated doshas.
I) Vataja Vrana Lakshanas:

Vrana due to vaata has shyaava or aruna varna, tanu, stabdha, kathina, ruksh, having alpa sraava and vedana baahulyata and toda bhedavat Vedana.

**Table No. 1 : Vataj vrana lakshanas**

<table>
<thead>
<tr>
<th>Lakshanas</th>
<th>Sushruta⁴⁰</th>
<th>Charaka⁴¹</th>
<th>M.Ni.⁴²</th>
<th>A.H.⁴³</th>
<th>Kashyapa⁴⁵</th>
<th>A. San.⁴⁴</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Varna</strong></td>
<td>Shyaava or Aruna</td>
<td>Shyaava</td>
<td>Shyaava</td>
<td>Shyaava, Krushna, Aruna, Bhasma kapotha or Asthi Varna</td>
<td>-</td>
<td>Shyaava, Aruna, Krushna, Bhasma of Asthi</td>
</tr>
<tr>
<td><strong>Vartma</strong></td>
<td>Rooksha</td>
<td>Stabdha, Kathina</td>
<td>Stabdha, Kathina</td>
<td>---</td>
<td>Stambha, Kathina</td>
<td>-</td>
</tr>
<tr>
<td><strong>Vedana</strong></td>
<td>Todha, Bheda, Chatachata yana, etc</td>
<td>Teevra Ruk, Sphurana</td>
<td>Maharuja</td>
<td>Todha, Bheda etc.</td>
<td>Maharuja</td>
<td>Sphuruna, Todha, Bheda etc.</td>
</tr>
<tr>
<td><strong>Sraava</strong></td>
<td>Sheeta, tanu, Picchila, Alpasraava</td>
<td>Mandhasraava</td>
<td>Mandhasraava</td>
<td>Alpasraava resembling Mastu, Maamsa, Pulakaamba</td>
<td>Alpasraava resembling Mastu, dadhi, Kshaara, Maamsa Dhaavana, Pulakoda ka etc.</td>
<td></td>
</tr>
</tbody>
</table>
II) **Pittaja Vrana Lakshanas**: Vrana due to Pitta will be associated with daaha, paaka, raaga, jwara, trishna, moha and vrana has kshipra utpatti i.e sudden onset with neela, peeta varna and pootisraava i.e. associated with pus.

**Table No.2 : Pittaj Vrana lakshanas**

<table>
<thead>
<tr>
<th>Lakshanas</th>
<th>Sushruta&lt;sup&gt;46&lt;/sup&gt;</th>
<th>Charaka&lt;sup&gt;47&lt;/sup&gt;</th>
<th>M.Ni&lt;sup&gt;48&lt;/sup&gt;</th>
<th>A.H.&lt;sup&gt;49&lt;/sup&gt;</th>
<th>Kashyapa&lt;sup&gt;50&lt;/sup&gt;</th>
<th>A.San.&lt;sup&gt;51&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Varna</strong></td>
<td>Peeta, Neelabha</td>
<td>-----</td>
<td>---</td>
<td>Neela, Kapila, Pingala, Peeta,</td>
<td>----</td>
<td>Peeta, Neela, Krushna, Pingala, Haritha,</td>
</tr>
<tr>
<td><strong>Utpatti</strong></td>
<td>Kshipra, i.e. sudden onset</td>
<td>-----</td>
<td>---</td>
<td>Kshipra</td>
<td>---</td>
<td>Kshipra</td>
</tr>
<tr>
<td><strong>Anya lakshanas</strong></td>
<td>Daaha, Paaka, Raaga, with Peeta Pidaka</td>
<td>Trushna, Moha, Jwara, Sveda,</td>
<td>Trushna, Moha, Jwara, Kleda, Daaha</td>
<td>Raaga, Paaka, pain resembling Vrana caused by Kshaara</td>
<td>Jwara, Daaha, Moha, Trushna</td>
<td>Daaha, Raaga, Paaka, Jwara, Dhoomayana</td>
</tr>
<tr>
<td><strong>Sraava</strong></td>
<td>Sraava resembling Kimshuka flower, Ushna.</td>
<td>Pootisraava</td>
<td>Pootisraava</td>
<td>Sraava is warm, large in quantity. resembling Kimshuka Taila or solution of Bhasma</td>
<td>Pootisraava</td>
<td>Sraava large in quantity. resembling Gomootra, solution of Bhasma, Kimshuka or Mrudveeka or Taila</td>
</tr>
</tbody>
</table>
### III) Kaphaja Vrana Lakshanas:

Vrana due to Kapha will have paandu or shweta varna associated with ugra kandu, manda vedana, shukla, sheeta, pichhila and ghana Sraava, stabdh sirasnayujalvat.

<table>
<thead>
<tr>
<th>Lakshanas</th>
<th>Sushruta&lt;sup&gt;52&lt;/sup&gt;</th>
<th>Charaka&lt;sup&gt;53&lt;/sup&gt;</th>
<th>M.Ni.&lt;sup&gt;54&lt;/sup&gt;</th>
<th>A.H.&lt;sup&gt;55&lt;/sup&gt;</th>
<th>Ka. Sam&lt;sup&gt;56&lt;/sup&gt;</th>
<th>A.S.&lt;sup&gt;57&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Varna</strong></td>
<td>Paandu</td>
<td>Paandu</td>
<td>Paandu</td>
<td>Paandu</td>
<td>Paandu</td>
<td>Paandu</td>
</tr>
<tr>
<td><strong>Vartma</strong></td>
<td>Sthoola, covered with StabdhaSiraSna ayujaala, Katina</td>
<td>Snigdha, Guru, Bahupiccha</td>
<td>Bahupiccha, Guru, Snigdha</td>
<td>Sthoola, Katina, covered with SiraSnaay uJaala</td>
<td>Sthaimithya, Maardhava</td>
<td>Sthoolaa, Snigdha, Kathina, Sthoola</td>
</tr>
<tr>
<td><strong>Anya lakshanas</strong></td>
<td>Mandavedana, severe Kandu, feeling of heaviness</td>
<td>Manda, vedana, Sthaimithya</td>
<td>Mandavedana, Sthaimithya</td>
<td>Alparuja, Kandu, Sthaimithya</td>
<td>Manda, vedana, Sthaimithya</td>
<td>Mandha vedana, Kandu, Sthaimithya</td>
</tr>
<tr>
<td><strong>Sraava</strong></td>
<td>Shukla, Sheeta, Saandra</td>
<td>Alpasama, kleda</td>
<td>AlpaSa mkleda, Chirapa aki</td>
<td>Large qty. of Sveta Ghana sraava</td>
<td>Atisraava</td>
<td>Sraava resembling Navaneeta, Tila pishta, Naarikeloda ka</td>
</tr>
</tbody>
</table>

*Table No. 3: Kaphaj Vrana lakshanas*
IV) Raktaja Vrana Lakshanás:
This Vrana will have features similar to that of Pittaja Vrana; it has Pravaala (Rakta) Varna, Raktasraava covered with network of Krishna sphota, smells like Turanga or Vaajisthaana.

<table>
<thead>
<tr>
<th>Lakshanás</th>
<th>Sushruta$^{58}$</th>
<th>M. Ni.$^{59}$</th>
<th>A.H.$^{60}$</th>
<th>A.Sam.$^{61}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Varna</td>
<td>Pravaala Dhala Nichaya</td>
<td>Raktha</td>
<td>Pravaala (Raktha)</td>
<td>Pravaala (Raktha)</td>
</tr>
<tr>
<td>Vartma</td>
<td>Covered with network of Krushnasphota, Pidaka</td>
<td>---</td>
<td>----</td>
<td>Covered with Krushnasphota, Pidaka</td>
</tr>
<tr>
<td>Anyalakshanás</td>
<td>Smells like Turangasthaana, Vedanaayuktha, DhoomayanaSh eela and having features of Pitta</td>
<td>----</td>
<td>Smells like VaajiSthaana, has other features of Pitta</td>
<td>Smells like VaajiSthaana, has other features of Pitta</td>
</tr>
<tr>
<td>Sraava</td>
<td>RakthaSraava</td>
<td>RakthaSraava</td>
<td>SaraktapooyaSra ava</td>
<td>SaraktapooyaSra ava</td>
</tr>
</tbody>
</table>
V) Dvidoshaja Vrana lakshanas: 62
Sushruta has explained the lakshanas depending upon combination of doshas while Vagbhata and Madhavakara have the stated same.

<table>
<thead>
<tr>
<th>Lakshanas</th>
<th>V-P</th>
<th>V-K</th>
<th>P-K</th>
<th>V-R</th>
<th>P-R</th>
<th>K-R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aakruti</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Ghritamanda</td>
<td>-</td>
</tr>
<tr>
<td>Gandha</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Meenadhavantoya</td>
<td>-</td>
</tr>
<tr>
<td>Varna</td>
<td>Aruna, peeta</td>
<td>-</td>
<td>-</td>
<td>Rakta, Aruna</td>
<td>-</td>
<td>Rakta</td>
</tr>
<tr>
<td>Vedana</td>
<td>Toda, Daha, Dhoomayana</td>
<td>Toda, Kandu</td>
<td>Daaha, Ushna</td>
<td>Toda, supta</td>
<td>-</td>
<td>Kandu</td>
</tr>
<tr>
<td>Sraava</td>
<td>Peeta, Aruna</td>
<td>Sheeta, Picchila, Alpa</td>
<td>Peeta, Paandu</td>
<td>Rakta, aruna</td>
<td>Ushna, Krishna</td>
<td>Rakta, Paandu</td>
</tr>
<tr>
<td>Anya lakshanas</td>
<td>-</td>
<td>Rooksha a, Guru, Daruna</td>
<td>Guru</td>
<td>Ruksha , Tanu</td>
<td>Mridu, Visarpa</td>
<td>Guru, Picchila , Snigdha</td>
</tr>
</tbody>
</table>
VI) Tridoshaj or Sannipataj Vrana lakshanas: 63

Table No. 6: Tridoshaj or Sannipataj Vrana lakshanas

<table>
<thead>
<tr>
<th>Lakshanas</th>
<th>V-P-R</th>
<th>V-K-R</th>
<th>P-K-R</th>
<th>V-P-K</th>
<th>V-P-K-R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vedana</td>
<td>Spuran, Toda, Daha, Dhoomayan</td>
<td>Kandu, Sphuran, Chumchumayan</td>
<td>Daha, Kandu</td>
<td>has vedana of three types</td>
<td>Nirdahan, Nirmathan, Spuran, Toda, Daha, Kandu</td>
</tr>
<tr>
<td>Varna</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>has varna of three types</td>
<td>-</td>
</tr>
<tr>
<td>Sraava</td>
<td>Peeta, Tanu, Rakta</td>
<td>Paandu, Ghana, Rakta</td>
<td>Paandu, Ghana, Rakta</td>
<td>has sraava of three types</td>
<td>Nana varna</td>
</tr>
<tr>
<td>Anya lakshanas</td>
<td>-</td>
<td>-</td>
<td>Paaka, Raga</td>
<td>-</td>
<td>Paaka, Raga</td>
</tr>
</tbody>
</table>

G) Dushtavrana: 64 (Infected wound)

Dushtavrana means getting vitiated by doshas. If vrana has bad smell (foul odour), has abnormal colour with profuse discharge, severe pain intensity and takes long period to heal it is known as Dushta Vrana. The features of Dushta Vrana vary as per present dosha predominance

Lakshanas of DushtaVrana: 64

Lakshanas depending upon the discharge, consistency, shape and chronicity according to various samhitas is as follows;
<table>
<thead>
<tr>
<th><strong>Sushruta</strong>&lt;sup&gt;65&lt;/sup&gt;</th>
<th><strong>Charka</strong>&lt;sup&gt;66&lt;/sup&gt;</th>
<th><strong>M.Ni.</strong>&lt;sup&gt;67&lt;/sup&gt;</th>
<th><strong>A.H.</strong>&lt;sup&gt;68&lt;/sup&gt;</th>
<th><strong>A.S.</strong>&lt;sup&gt;69&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atisamvrutha or Ativivrutha, Ati-katina or Mrudu,</td>
<td>Mentioned 12 caracteristic features</td>
<td>Discharges Pooti Sraava, or Dushta</td>
<td>Samvrutta or Vivrutta,</td>
<td>Atisamvrutha or</td>
</tr>
<tr>
<td>Utsanna or Avasanna.</td>
<td>indicating the advanced stage of morbidity of Vrana.</td>
<td>Ashruk, has Utsangi (sinuses) inside,</td>
<td>Katina or Mrudu</td>
<td>Ativivrutha,</td>
</tr>
<tr>
<td>Atisheeta or Usna, having one of the colours Rakta, Krahsna Peeta, Shukla etc, Bhairava (fearful features), filled with Pootipooya, Mamsa, Sira, Snaayu etc. Moving in oblique track (Unmargi) having Amanoghna Darshana, Amoghnagandha, atyartVadanayukta, associated with Daaha, Paaka, Raaga, Kandu, Shopha, Pidaka etc. Discharging excessivelyDushta Shonitha,Dheer gakalaanubandha</td>
<td>Swetatva, Avasanna Vartmatva, Atisthoolaa Vartmatva, Atipinjaratva, Neelatva, Syaavatva, Atipidakatva, Rakta-Krushnatva Atipootitva, Ropyatva Kumbhi-Mukhatva, Vranas with Pootigandha, Vivarna, Bahusraava, Maharuja</td>
<td>Chirastitha, emits Pooti Gandha &amp; doesn’t possess any features of Shuddha Vrana</td>
<td>Atisheeta, Raktatwa or Paanduta, Discharges Pooti Pooya covered with Pooti Maamsa, Sira, Snaayu, Snaayu, associated with Atiruk, Daaha, Swayathu, Kandu &amp; other complications Dheerga Kaalanubandha</td>
<td>Atisheeta or UsnaRaaka, Krushna, or Paanduta, covered with Pooti Maamsa, Sira, Snaayu, etc. Discharges Pooti Pooya, Daaha, Paaka, Kandu, Swayathu, Vedana Pitaka, etc. appearing as Upadravas, Dheerga Kaalanubandha</td>
</tr>
</tbody>
</table>

Table No.7: Dustha Vrana Lakshanas
H) ShuddhaVrana: ⁷⁰ (Clean wound)

The Vrana which is not vitiated by doshas, not invaded by tridoshas, having Shyaava Oshta, developed Sama Pidaka and is not associated with Vedana and Sraava, is said to be ShuddhaVrana.

Vrana which resembles Jhvatalaabha,(i.e. edges and floor) mrudu, snigdha, slakhsna; and there is no vedana and sraava also it is good looking i.e. suvyvastith, is said to be Shuddha. Shuddha Vrana consist all these features. Sushruta and Vagbhatta have mentioned the almost similar features.

| Table No. 8: ShuddhaVrana Lakshanasy |
|---|---|---|---|---|
| Sushruta⁷¹ | Charka⁷² | M.Ni⁷³ | A.H⁷⁴ | A. S.⁷⁵ |
| Not vitiated by tridoshas having Shyaava Oshta resembles Jhvatalaabha which is Mrudu, Snigdha, free from Vedana, Sraava, good looking, and has developed Samapidika. ⁷⁶ | no Atirakta, nati- Paandu, nati Shyaava, nati Ruk, nati Utsanna nati | Resembles Jhva talaabha is Atimrudu, Slakshna, Snigdha, Suvyavasthitha, AlpaVedana, and Nirasraava. | Resembles Jhvatatalabha in redcolor, Mrudu, Slakshna with ShyaavaOshta, Samapidika, having UnnataMadhy a, not acompanied with any Upadravas. | Not vitiated by Doshas, resembles Jhva in colour, and is Slakshna having Shyaava, Oshta centre being elevated or even not having Vedana, Sraava |
I) Ruhyamaana Vrana: \(^{76}\) (Healing wound)

Lakshanas:
Vrana possesses Kapota Varna, it is free from Kleda and has Sthira Pitika is said to be Ruhyamaana Vrana. Vrana area is covered by small outgrowths (granulation tissues). Similar type of information is given by Vagbhaata and in Madhavanidaana also.

J) Samyak Roodha Vrana: \(^{77}\) (Healed Wound)

Lakshanas:
Vrana healed without eruptions (Granthi), without hardness, no pain (Vedana) and swelling, colour of the site should be similar to that of surrounding skin and the site should be flat such Vrana is known as Samyak Roodh Vrana.

K) Representation of classification of Vrana due to various reasons:

\[
\begin{align*}
\text{Vrana} & \quad \text{Kaarana} ---- \quad \text{Avastha} ---- \\
& \quad \text{Nija, Aagantuja.} \quad \text{Dushta, Shuddha, Ruhyamaana, Roodha.}
\end{align*}
\]

Vrana Aakruthi --- Aayatha, Chathurasra, Vrutta, Triputaka.
Vrana Saadhyasaadhyatha--- Sukhasaadhya, Kruchrasaadhya,
Vrana Yaapya, Asaadhya.

L) Samprapti: \(^{78}\)
Doshas being aggravated by their respective causative factors and gets lodged in any of Vrana Sthaanas. So, vranotapatti occurs. Vaata, Pitta, Kapha being aggravated by their respective causative factors and give rise to Nija Vrana.

M) Examination of Vrana: \(^{79}\)
Examination of Vrana and patient, suffering from Vralakshanas are to be carried out in 3 different ways. They are Darshana, Sparshana and Prashna.

i) Darshana:
By Darshana Pareeksha age of patient, site of Vrana, Aakruthi, Varna, condition of Vrana, Vranasraava, number of vrana, etc. can be elicited.
ii) Sparshana:
It helps in deciding the hardness or softness of Vrana, increase or decrease of local temperature, tenderness, bleeding, etc.

iii) Prashna:
By Prashna Pareeksha the cause of Vrana, type of Vedana, Agni, Bala, Saatmya etc. are to be examined. Sushruta mentioned Shadvidha Pareeksha for the diagnosis. Darshana and Sparshana should be done by Panchaindriya Pareeksha. Shushruta mentioned about Vrana examination that every surgeon examines the mulsthan of Vrana, also vrana adhisthan i.e. Panchlakshan and parigrahee i.e. vrana site. Then surgeon can decide about Shasthi upakram and with the help of chikitsa chatushpada the treatment of ulcer becomes easier.28

Table No.9: Examination Method of Vrana

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>Exam Method</th>
<th>Points to be examined</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Darshan</td>
<td>Age, Colour, body parts, Sense organ etc</td>
</tr>
<tr>
<td>2</td>
<td>Sparshana</td>
<td>Hot and cold, soft and hard, etc</td>
</tr>
<tr>
<td>3</td>
<td>Prashana</td>
<td>Cause of disease (hetu), Pains, satmya, strength of Agni</td>
</tr>
</tbody>
</table>

N) Vrana Vastu or Vrana Adhistan:80
There are eight sites of vrana known as vranavastu. They are twak, mamasa, sira, snayu, asthi, sandhi, kostha and marma. Charaka has mentioned meda instead of sandhi and antarashrya for kostha.
Madhavakara (Madhavanidana) explained Samaanya and Vishesha lakshanas in case of injury to Maamsa, Sira, Snaayu, Sandhi, Asthi, and Marma.
Table No.10: Vrana vastu and its lakshanas according to Madhavkara

<table>
<thead>
<tr>
<th>Vranasthaana</th>
<th>Lakshananas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Injury to Maamsa, Sira, Snaayu, Sandhi, Asthi</td>
<td>Saamanya Lakshanas: - Bhrama, Pralaapa, Patana, Strasthangata, Vicheshtana, Pramoha, Glani, Ushnata, Moorcha, Teevraruja, discharge of Rakta resembling Mamsodaka, loss of functions of Indriyas etc.</td>
</tr>
<tr>
<td>Injury to Sira</td>
<td>Profuse discharge like Indragopa.</td>
</tr>
<tr>
<td>Injury to Snaayu</td>
<td>Decrease in height, drooping, loss of Pain, Vrana takes long time to heal</td>
</tr>
<tr>
<td>Injury to Sandhi</td>
<td>Increase in Shopha, severe pain, loss of strength, total loss of function etc</td>
</tr>
<tr>
<td>Injury to Asthi</td>
<td>Severe pain continuously throughout the Day and night, no relief in any posture.</td>
</tr>
<tr>
<td>Injury to Maamsa Marma</td>
<td>Pallor, loss of tactile sensation.</td>
</tr>
</tbody>
</table>

O) Vranakruti or Shape of Vrana:

Vrana resembles following shapes which are curable:

- Ayata (Elongated)
- Chaturastra (square or rectangular)
- Vritta (Circular)
- Triputak (Triangular)

According to Vaghbhat the shape of vrana is depends on the shape of shalya. Shape of agantuja Vranas are Aayatha, Chaturasra, Trayasra, Mandalina, Ardhachandraakaara, Vishaala and swastika, Kutila etc. Some resembling Sharaavanirmana madhyascha, others with elevation in the centre. Aagantuja Vranas have numerous shapes, except the main four shapes others are difficult to cure.
P) Vrana Varna (colour of the Vrana): 83

The colour of the vrana site, gives an indication of the involved dosha as well as the prognosis.

**Table No.11: Vrana Varna as per involvement of Doshas.**

<table>
<thead>
<tr>
<th>Dosha</th>
<th>Colour of Vrana</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaata</td>
<td>Bhasma (ash), Kapota, Asthi, Parusha, Aruna (reddish black), Krushna.</td>
</tr>
<tr>
<td>Pitta &amp; Rakta</td>
<td>Neela, Peeta, Haritha, Shyaava, Krushna, Rakta, Kapila (brownish), Pingala (faint brown).</td>
</tr>
<tr>
<td>Kapha</td>
<td>Sweta, Paandu (dull yellowish), Snigdha (appears unctuous).</td>
</tr>
<tr>
<td>Sannipaataja</td>
<td>As tridoshas are mixed, the site acquires various colours i.e. Sarva Varna.</td>
</tr>
</tbody>
</table>

Q) Vrana Gandha: 83, 84

Vrana gandha is one of the important examinations of vrana. According to Sushruta vataj vrana has Katu gandh, Pittaj has Tikshna, Kaphaj has Visra, Rakaj has Lohagandh and Sannipatik vrana has Vyamishra gandh. Vyamishra i.e associated with all types of smells. He also mentioned that smell similar to laja, Atasi tail and Til taila (oil) are also prakrut smelles of vrana. Other smells are known as vikrut gandh. According to Charaka, Vrana has eight types of Gandha like Ghruta, Tail, and vasa i.e serum of Flesh, Puya, Rakta, Shavya, Amla and Pootik.

R) Vrana Vedana: 85

Vrana vedana are related with the doshas involvement.

i) Vrana vedana due to vata dosha:

Vrana vedana due to vaat dosha like Toda, Bheda, Tadana, Chedan, Ayamana, Manthana, Vikshepan, Chimchimayan, Nirdahana, Avabhanjana, Sphotan, Vidarana, Utpatana, Kampan. Due to vata dosh pain comes on and off. There is no consistency with respect to site, cause etc.

ii) Vrana vedana due to pitta dosha:

Oosha, Chosa, Paridaha, Dumayan, the area of vrana appears like burning amber, pain like Khsaara has been applied to the Vrana. These are the types of vedana due to pitta dosha.
iii) Vrana vedana due to Kapha dosha:

Kandu, Gaurava, Suptatva, Alpa vedana, Stambha, Saithya are the types of pain due to kapha dosha.

iv) Vrana vedana due to Rakta dosha: Similar to pitta dosha.

v) Vrana vedana due to Sannipataj vrana:

Vrana vedana due to involvement of tridosha and hence all types of vedana occurred.

S) Vrana Sraava: 86

i) Vrana sraava due to dosha

Sushrut and Vaghbhat have mentioned the list of discharges based on vrana adhisthan and involvement of doshas.

Table No.12: Vrana sraava according to doshas

<table>
<thead>
<tr>
<th>Dosha</th>
<th>Vrana Sraava</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaat</td>
<td>Parusha, Shyaava, Dadhimastu, Kshaarodak, Maamsa- dhaavana, Pulakodaka</td>
</tr>
<tr>
<td>Pitta</td>
<td>Gomeda, Gomootra, Shanka, Kashaayodaka, Maadhveeka Taila</td>
</tr>
<tr>
<td>Kapha</td>
<td>Navaneeta, Kaseesa, Majja, Naarikelodaka, Varahahavasa</td>
</tr>
<tr>
<td>Rakta</td>
<td>like lakshan as pitta but associated with more rakta srava</td>
</tr>
<tr>
<td>Sannipataj</td>
<td>Priyanguphala, Naarikelodaka, Kaanjeeka etc.</td>
</tr>
</tbody>
</table>

Features of sraava mentioned by Vagbhata are similar to Sushruta. Charaka has explained 14 types of sraava. They are Lasseka, Jala, Pooya, Asruk, Haridra, Aruna, pinjar, Kashaaya, Neela, Harita Snigdha, Ruksh, Sita, and Asita.

ii) Vrana Sraava according to Sthaana: 86

Table No.13: Vrana Sraava according to Sthaana or Vrana Adhisthan

<table>
<thead>
<tr>
<th>Sthaana</th>
<th>Sraava</th>
</tr>
</thead>
<tbody>
<tr>
<td>Twak</td>
<td>Salilaprakasha, Peetaavabaasa.</td>
</tr>
<tr>
<td>Maamsa</td>
<td>Sarpiprakasha, Sheeta, Picchila.</td>
</tr>
<tr>
<td>Sira</td>
<td>Rakta Atipavriti, Pooya comes out after Paaka.</td>
</tr>
<tr>
<td>Snaayu</td>
<td>Snigdha, Ghana, Singhanaka pratima, Sarakta.</td>
</tr>
<tr>
<td>Asthi</td>
<td>Discharge mixed with Rakta, Majja.</td>
</tr>
<tr>
<td>Sandhi</td>
<td>Picchila, Saphenarudhira.</td>
</tr>
<tr>
<td>Kostha</td>
<td>Discharges mixed with Asruk, Mootra, Pureesha, Pooya, and Udaka.</td>
</tr>
</tbody>
</table>
T) Saadhyaasadhhyatha:

i) Sukh saadhya Vrana: 87
   o Vrana arising in tarun vaya (it heals because of pratyagra dhaatus),
   o Dhruda (Body having Sthira Bhau, Mamsa, Sira, Snayus etc.)
   o Pranvannta.
   o Satwawanta (Do not suffer from Vedana caused by Darun Kriya)
   o Vrana arising in pratham dhatu i.e. Twak, Mamsa Adhistan.
   o Vrana akrutis like Ayata, Chaturasra, Tripatuk, and Vrutha.
   o Patient should be Atmvanta.
   o Vrana treated by Kushal Vaidyas.
   o Vrana sites are sphik, paayu, Prajanan (genitila), Lalata,Gandha, Oshta,
     Prushta, karna Phalakosa, Udara.
   o Vrana of recent origin and free from upadrava

ii) Kruchasadhya Vrana: 88
   o Vrana in patient having old age,
   o Patient is Krush, Alppraana, and Bheeru etc.
   o Vranas having vikrut aakruti.
   o Vranas situated in sthaan like Danth, Nassa, Apanga, Srotra, Naabhi, Jathara,
     Sevani, Nitamb, Parshwa, Kukshi, Vrana associated with people suffering
     from Kustha, Visha, Shosha, Madhumeha.
   o Vrana treated by quack
   o Vrana which emits phena, pooya, anil
   o Bhagandhara with an internal opening
   o Vrana formed over Kati and Asthi
   o Patient who is anaatmavatitha

iii) Yappya Vrana: 89
   o Vrana manifested in the following conditions can be treated but not
     completely cured Avapattika, Niruddhaprakasa(Phimosis), Sannirudh Guda,
     Sannirudh Jathara, when krimi infest vrana, Granthi, Krimi in abdomen,
     Madhumeha,sikatameha,Vaatkundalika, asthila(BPH), Dantsharkara, upakush,
     Kanthashalulak, Visarapa, etc.
iv). Asaadya Vrana:  
- Vrana with an elevated floor of excessive granulation tissue (mamsa pindavat) with excessive discharge. Containing pooya inside associated with vedana, discharge of vasa, meda, majja, mastulunga. Koshtasra vrina having discharge of peeta or asita varna, moostra, pureesha etc. and those having discharge of pooya and rakta.
  - Vrana having ostha like Ashwa apaana, protruded like goshringa,
  - Those discharging dushta rudhira, Tanu, Sheeta, Picchila srava, elevated in centre, some are Santoolavata contains Snaayu, Jala, having Durdarshana.
  - Vrana at Upanakha, over marma, over bones of leg (Janghasthi), Blind external fistula in Ano, Vrana over Median Raphae,
  - Vrana secondary to diseases of bones of pelvic region.
  - Vrana situated in all ground materials (Sarvtogath) with anumukh and mamsa budbuda
  - Vrana situated in sira and Kantha from which air escapes making of sound
  - Discharge of blood with pus in a heena mamsa person,
  - Associated with upadravas like Arochaka, Avipaak, Kaasa, Shwas
  - Bhinna vrina in Shira, Kapal followed by appearance of Mastulung.
  - Features of all the three vitiated doshas or with Kassa and Shwas are incurable
  - Vrana discharging Vasa, Majja, and Mastulung are curable if casued by aagntuj karana, otherwise it is incurable due to vitiation of three doshas.
  - Vranas with pulkodaka srava from pakwashaya, Ksharoodaka type of srava from raktashya, kalaaya type of srava from amashya and trik sandhi Pradesh
  - Vrana situated in deeper dhatu (uttarotar Dhatu)
  - If it is not cured in a particular time sadhya vrina becomes Yaapya, Yappya vrana becomes assadhya, and finally Assadhay may kills person.
  - Vrana manifested with fatal signs i.e. vrana with Arishta lakshanjas.

U) Vrana Chikitsa:  
Charak has mentioned 36 upkramas for the treatment of vrana where Sushruta has mentioned 60 upkramas. Sharangdhar has mentioned Saptakramas like pralepa, pradeha, pachan, darana, vrina shodhan, ropan. Charka has explained Samanya and Vishesh Chikitsa for Vrana.
I) Samanya Chikitsa:

Vranitasya should be given Shodhan, therapies through Vamana or Virechana.

II) Vishesh Chikitsa:

A) Vaataja vrana chikitsa

Persons suffering from Vaataja Vrana should be treated with complete Snehapana, Swedana, Upanaha, Pradeha, and Parisheka which are of unctuous nature.

B) Pittaj Vrana Chikitsa

Persons suffering from pittaj vrana should be treated with virechana, pradeha, parisheka, sarpipana, prepared by sheetala, madhura, and tikta dravyas.

C) Kaphaj Vrana chikitsa

Person suffering from Kaphaja Vrana should be treated with Pradeha, Parishechana, with drugs which are Kashaaya, Katu, Rooksha, Ushna and Laghu, Paachana etc.

D) Saptaupakrama and Shashti Upakramas

They are mentioned in treatment of Vrana Shopha.

They are Vimlapan, Avascehan, Upanaha, Paatan, Shodhan, Ropana, Vaikritaapaham and these are elaborately explained by Sushruta in the Shashti upakrama.

Table No.14: Shashti Upakramas of Vrana mentioned for vrana by Sushruta and other Acharyas

<table>
<thead>
<tr>
<th>Upakrama</th>
<th>Sushruta</th>
<th>Charak</th>
<th>Kashyap</th>
<th>A.S &amp; A.Hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apatarpana</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Aalepa</td>
<td>+</td>
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<td>maradavakarma, Aalepana.</td>
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<td>Kaathinyakara aalepa.</td>
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<td>Agnikarma</td>
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<td>+  Daha</td>
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<tr>
<td>Krishnakarma</td>
<td>+</td>
<td>Varnya</td>
<td>Savarnikaran</td>
<td></td>
</tr>
</tbody>
</table>

26
<table>
<thead>
<tr>
<th>Pandukarma</th>
<th>+</th>
<th>Varnya</th>
<th>Savarnikaran</th>
<th>Savarnikaran</th>
</tr>
</thead>
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<td>Shirovirechana</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Nasya</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Kavala dharana</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Dhoom</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Madhu-Sarpi</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Yantra</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Aaharana</td>
<td>+</td>
<td>Bhojya</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Rakshavidhana</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Shophaghna</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Shamana</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Chadana</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Shodhanalepa</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Ropanalepa</td>
<td>--</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Ropana</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Utklinnaprakshalana</td>
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<td>-</td>
<td>+</td>
<td>Prakshalan</td>
</tr>
<tr>
<td>Shodhana</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Prachhena</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Among the 36 upakramas mentioned by Charaka, Shophaghna i.e. the treatment of Vranashopha; involves Raktavsechan, Langhana, Snehan, Pralepa, Pradeha, Upanah etc. These karmas are considered consizely 11 Upakramas mentioned by Sushruta for Vranashopha.

Also, Charaka has discussed about Shastra karmas. He also mentioned Eshankarma separately & Aharankarma has been covered under six surgical measures. Charaka has describes vranopkrama as Shodhanakashaya, Taila, Ghrita, Ropanakashaya, Utsadan, Avasadana, Aalepana etc.

Sushruta has mentioned vranopkrama as Shodhana, Ropana & Vaikrutapaham etc.

**Shodhan and Ropan Definition**

In this study Shodhan and ropan concept are more important for treating chronic ulcers. As the all chronic ulcers are infected, so they need vran shodhan and ropan both to heal completely.

**A. Definition of Shodhan:**

I) शोधनम्

स्वयमेव विदिर्घ शोधन भेदितं ब्रण यानि द्रव्याणि शोधयति, ताति शोधनाणि इत्युच्चते |

शोधनम्—ृणशुद्धिकरः । (च.सू.२५/८५)

शोधनं पुनःद्विधं -----कषाय, वर्ती,कल्क, चूण, तैल, रसक्रिया, चूण,धूपन भेदेन। शोधनद्व्य विस्तरस्तु सूत्ते सूततथाने ३७ तमे अध्याये चिकित्सास्त्याने प्रथमे च दृष्टत्य:।

Shodhan can be concluded as cleaning of ulcer i.e. removal of pus and dead tissue.

**B. Definition of Ropan**

II) रोपणं

रोपणये— रोपणीय: यथाव्रणा:

‘बहिशुध्दा:’ इवाभान्ति रोप्यस्ते संप्रकिरिति:। (च.सू.२५/२५)

यथा कषायो रस:॥(च.सू, २६/४४.६)

यथा स मधुकास्तिला रोपण:॥(अं.स,सू,३९)
It can be concluded as after cleaning of ulcer it’s healing by normal tissue growth and closure of ulcer by healthy skin.

Thus these 60 procedures (shashti upkramas) can treat Vrana effectively.

V) Pathyaapathyā: 

Pathya:

Vranita should consume Jeerna Shaali, Odhana which is made warm unctuous and taken with Jaangala Maamsa. Soup prepared from Tanduliyaka, Jeevanti, Vaartaaka, Patola, Kaaravellaka, Daadima, Aamalaka etc. Vranit should avoid day sleep, excercise.

Apathya:

Vranit must avoid Navadhaanya, Maasha, Tila, Kalaaya, Kulattha, Nishpaava, Hareeta Shaaka, Katu, Amla, Lavana Rasa substances, Guda, Sushka Shaaka, eatables made from Pishta, Aaja, Anoopa, Maamsa, Sheeta Udaka, Krushara, Paayasa, Dadhi, milk etc. Vranit person should avoid Vaata, Aatapa, Raja, Dhooma, Atibhojana, Bhaya, Shoka, Krodha, Raatri Jaagarana, Vishamaashana, Vyayaama, Upavaasa, Chankramana etc.
W) **Upadravas:**
These are mainly classified as Vranasya Upadravas, Vranitasya Upadravas.

- **Vranasya Upadravas:** are five related with abnormality in Aakruthi, Vedana, Gandha, Sraava, Varna.

- **Vranitasya Upadravas:** Jwara, Atisaara, Moorcha, Hikka, Chhardi, Arochaka, Shwaasa, Kaasa, Avipaaka, Trushna.

Charaka mentioned the 16 types of Upadravas they are: Visarpa, Pakshaaghaata, Sirasthambha, Apataanaka, Moha, Unmaada, Vrana Ruk, Jwara, Trushna, Hanugraha, Kaasa, Chhardi, Atisaara, Hikka, Shwaasa and Vepathu.

X) **Vrana as upadrava:**
Vrana itself is an upadrva in diseases such as Prameha, Visarpa, Vaata rakta, shotha, Kushta, Arshas(bahya).

Y) **Vrana Arishtha lakshans (Fatal signs of Vrana):**
Sushruta has described Vrana-arishta lakshnae (fatal signs) as follows;

i) **Gandhavaikrut:** (fatal sign based on odour):
Vrana patient emits odour resembling that of Madya (alcohol), Honey, Ghee, Jasmine, Lotus, Sandal etc. It is one of important fatal sign.

ii) **Sparsh vaikrut** (fatal sign based on touch):
When patient complaints severe heat and burning sensation at vrana site but vrana itself having sheetsparsh (Cold). It is also one of the important fatal sign.

iii) **Aakruti vaikrut:** (fatal sign based on shape.)
Vrana having irregular shapes viz; flag, elephant, horse, etc.; also, Patient feels that Vrana site covered by a fine powder like substances, but actually nothing is at Vrana site; Then Vaidya can conclude about patient’s death.

iv). **Shabdvaikrut:** (Fatal sign based on sound)
Vrana produces various sounds viz Ghurgurayan and there is discharge of air or gas from Vrana. All these are indications of death.
3.2 Review of Prameha and Madhumeha

Diabetic foot ulcer is a major complication of Diabetis Mellitus. It becomes one of the medical, social and economic problems worldwide. India being termed as capital of DM. Ayush department of India also declared theme ‘Mission Madhumeha through Ayurved’ for year 2016-2017.

3.2.1 Vedic Period (4000 B.C.-1500B.C.)

The ancient references of ‘Prameha’ are found in Vedas of India. The word Prameha literally means ‘to flow’. It is derived from the Sanskrit root ‘Mih-Sechane.’

The two terms ‘Aasarava’ and ‘Prameha’ are used in Atharvveda and in Kaushiksutra, respectively. Sayan and Kesavabhatta, the well-known commentator of Vedas describe ‘Asrava’ as ‘Mutratisara.’ Whitney (1962) and Griffith interpreted this as flux and morbid flow respectively.

William Cullen (1712-1794) added the word ‘mellitus’ to Diabetes, like the Prameha-Madhumeha concept of the ancient ayurvedic classics.

3.2.2. Samhita Period (2000B.C.-800A.D.):

I) Charak Samhita:

Charak explained the etiology, symptomatology, especially samprapti, types, complications and treatment of Prameha. He explained Prameha in Charak Nidansthan 4 and chikitsa in Charak Chikitsasthan 6. In Ch. Su17, he described the samprapti of margavrodhajanya madhumeha and Sapta pramehapidaka. In Charak Indriyasthan 9, he mentioned that the madhumeha is one of the ‘ashtau maharog’ which are difficult to cure.

II) Sushruta Samhita:

Sushrut explained nidan panchak of Prameha and Pramehajanya saaptapidaka in Nidan sthan. 6. He elaborately describes the treatment in Chikitsasthana. He has explained three different Adhayay. They are Sushrut Chikitsasthana11 Prameha Chikitsitadhaya, Sushrut Chikitsasthana12, Pramehapidaka Chikitsit and Sushrut Chikitsasthana13 Madhumehachikitsa adhyaya.

He used “Kshoudrameha” synonym to Madhumeha.

III) Ashtang Hridaya:

Vagbhat describes Prameha in Ni. 10 and in chi. 12. He has explained the two types of Madhumaeha i.e. Dhatukshayajanya and Avrutpathjanya.
IV) Harita Samhita: He has explained Prameha is a papajanya vyadhi and mentioned that there are 13 types of Prameha with names like Puyameha, Ghrutmeha etc. (HA. II, Sth1/9)

V) Bhel Samhita: He has mentioned two types Narasya-swakrutam Prameha and Prakruti-prabhavam prameha.

VI) Kashyapa Samhita: He has discussed the symptoms of Pramehi Child in Vedanadahaya and explained eight pidaka in Dwivriyachikitsaadhyaya.

3.2.3) Medieval Period (800 A.D. to 900 A.D.): I) Madhavnidan: He repeated collective description of Madhumeha narrated by Charaka, Susruta and Vaghabhotta.

II) Gayadas: In ‘Nayyachandrika’ he has narrated ‘Avilmutrata’ is because of presence of dushya. (Ny.C.Su.Ni.6/6)

III) Dalhan: In his ‘Nibandha Samgraha’ tika he has mentioned that female can’t suffer from Prameha.

IV) Sharangdhara Samhita: Sharahngdhara mentioned that there are 20 types of Prameha in poorvakhandha 7th Chapter (59-62).

V) Bhavaprakasha: He described Prameha and Madhumeha along with some new herbomineral preparations.

VI) Yogratnakar: Prameha and Madhumeha with its treatment are also explained by Yogratnakar.
3.2.4 Prameha

Madhumeha is a disease mainly occurs due to the metabolic disturbances, sedentary lifestyle and sometimes due to genetic predisposition. To understand the complete Prameha with its all details; we must study the Hetu, Samprapti, Poorvarupa, Roopa.

1). Nirukti (Etymology):\(^\text{105}\)

The word ‘Prameha’ composed of two sub words i.e. Pra (Upasarg- Prefix) and ‘Meha’.

The word meha is derived from the root ‘Mih Sechane’ meaning to profuse (watering) and the Pra- means excessive quantity and frequency. Therefore, the Prameha is said to be ‘Prabhuta Mutrata’ and ‘Avil Mutrata’.

The word Madumeha consists of two words i.e Madhu and Meha.

The ‘Madhu’ word is derived from the root “Manyante Visheshen Janayati Jana Yasmin.” The root “Manjane” is applied by Dha Adesha and it shows the similarity of Urine in taste, colour and appearance etc.

In Sanskrit literature madhu word is used for Pushparasa, Jalam, Makarand, Kshir and for madhu rasa, etc.

Thus, one can conclude that if a disease in which excretion of urine is like madhu (honey) in its colour, taste, smell and consistency then it is known as Madhumeha.

Sushruta has used the term ‘Kshoudrameha’ for Madumeha\(^\text{109}\) and suggested that Prameha is not treated in a time then it is converted into Madhumeha.\(^\text{110}\)

2). Paribhasha (Definition):

‘Prakarshena prabhutam prachuram varam varammeheti mutratyagam karoti iti Prameha’ - Ma.Ni.33/1

Prameha is a syndrome which includes all those clinical conditions which are characterized by increased quantity of urine associated with or without the increased frequency of micturation. Polyuria and turbidity of the urine are two essential presenting features of the disease.
3). Synonyms:
Followings are some of the synonyms mentioned in Samhitas:

A. Ojomeha:
Ojomeha is a subtype of Vataj Prameha. Since Vata affects the Oja, so patient passes the Kashay Urine.

B. Kshoudrameha:
This synonym is used by Sushruta. This type of Pramehi patient passes urine like madhu. (Honey)

C. Paushpameha:
Pushpameha word is described in Anjan Nidana as Pushparasa resembles with madhu.

4) Classification of the Prameha:
In Brihattrayi, the Prameha is classified based on dosha dominance and the classification as follows.

A. Classification of Prameha
   I. Classification based on Dosha bheda
   II. Classification based on Sadyasadytwa

B. Classification of Madhumeha
   I). Classification based on Dosha bheda:
   Twenty types of Prameha have been described by different authors of ayurvedic Samhitas. Among these, ten are of Kaphaja type, six are of Pittaja type and four belongs to Vataja type.
### Table No.15: Classification of Prameha based on Dosh:

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>Type</th>
<th>Charaka</th>
<th>Sushruta</th>
<th>Vagbhatta</th>
<th>Madhava</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kaphaja Meha</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Udakameha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Ikshubalikarasmeha</td>
<td>+</td>
<td>+</td>
<td></td>
<td>Ikshumeha Ikshumeha</td>
</tr>
<tr>
<td></td>
<td>Sandrameha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Sandraprasadmeha</td>
<td>+</td>
<td>Surameha</td>
<td>Surameha</td>
<td>Surameha</td>
</tr>
<tr>
<td></td>
<td>Shuklameha</td>
<td>+</td>
<td>Pishtameh</td>
<td>Pishtameh</td>
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</tr>
<tr>
<td></td>
<td>Shitameha</td>
<td>+</td>
<td>Lavanameh</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Siktameha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td></td>
<td>Shanairmeha</td>
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<tr>
<td></td>
<td>Alalmeha</td>
<td>+</td>
<td>Phenameh</td>
<td>Lalameha</td>
<td>Lalameha</td>
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<tr>
<td></td>
<td>Shukrameha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Pittaja Meha</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ksharameha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td></td>
<td>Kalameha</td>
<td>+</td>
<td>Amlameha</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Nil meha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Lohitmeha</td>
<td>+</td>
<td>Shinitameh</td>
<td>Raktameh</td>
<td>Raktameh</td>
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<tr>
<td></td>
<td>Manjishthameha</td>
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<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Haridrameha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Vataja Meha</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vasasmeha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Majjameha</td>
<td>+</td>
<td>Sarpimeh</td>
<td>+</td>
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<tr>
<td></td>
<td>Hastimeha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Madhumeha</td>
<td>+</td>
<td>Kshaudrameh</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

II) Classification based on Sadyasadytwa:

Prognosis is an inevitable part of Chikitsa and the success of treatment depends on an unbiased prognosis. Therefore, we must study the classification depends on Sadyasadyatva.
Table No.16: Classification according to Sadyasadyatwa.

<table>
<thead>
<tr>
<th>Sadhya</th>
<th>Yapa</th>
<th>Asadhyya</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kaphaja</td>
<td>Pittaja</td>
<td>Vataja</td>
</tr>
<tr>
<td>Sthula(Obese)</td>
<td>Usually not much obese</td>
<td>Krisha(Asthene)</td>
</tr>
<tr>
<td>Apathyanimittaja(Acquired)</td>
<td>Acquired</td>
<td>Sahaja(hereditary)</td>
</tr>
<tr>
<td>Early Stage</td>
<td>Acute Stage</td>
<td>Advanced Stage</td>
</tr>
<tr>
<td>Without complication</td>
<td>With complication</td>
<td>with complication</td>
</tr>
</tbody>
</table>

III) Classification based on hetubhedha:
1) Sahaj and 2) Apathynamitaj

IV) Classification based on samprapti:
1). Avaranajanya
2) Dhatukshayjanya
1) Santarpanjanya
2) Apatarpanjany

V) Classification based on dehaprakruti bheda:
1) Sthula 2) Krush
Let us consider one by one.

VI) Classification based on etiological factor:
A) Sahaj : (congenital)
Sahaj Prameha occurs due to Beejadosh. Charak mentioned that the Sahaj Prameha is a Kulaj Vikar and is Asadhay. Matru pitru beeja dosha are responsible for Sahaj Prameha.

B) Apathyanimitta:j: (acquired)
The word Apathya suggests its etiology. Apathya hetusevan and ati aahar causes to Apathynamittaja Madhumeha. This type is again classified according to etiology and Samprapati.

IV. Classification based on Samprapti:
1. Avaranajanya and Dhatukshayjanya:
In Avaranajanya Madhumeha, kaphavardhaka hetusevan leads to vata avarana, due to vruda vata, oja ksharana occurs and udakavaha, medovaha srotas get affected. Patient passes madhur and kasharaja mutra. In Dhatukhashyajanya Samprapti, due to vatavardhak hetu, vataparakopa occurs and dhatukshay takes place. Dhatukshay
and ojakshaya occurs and mutravaha, udakvaha srotas affected. This is the dhatukshayjanya Prameha.

2. Santarpanjanya and Apatarpanjanya
Santarpanjanya i.e. due to excessive nutritional diet; Kaph vitiates and kaph gets aggrevated. The more intake of such diet vitiates kapha, pitta, mamsa and meda. It causes Prameha by forming avarana of vata.

On the contrarary, in Apatarpanjanya Prameha dhatu phoshan is insufficient and vata gets aggravated it leads to Apatarpanjanya Prameha.

V. Classification based on Dehaprakruti bheda: 115

1. Sthula
2. Krush (asthenic)

The sthula and krush classification is akin to obese and non-obese division.

4) Nidana of Prameha:
Let us consider one by one. According to Charak, the disease is mainly due to tridosha. However, Sushrut mentioned that it is due to Sahaj and Apathynimittaj. 116

1) Sahaj Prameha
Matru and Pitru beeja dosh are the main reason of Sahaj Prameha. If garbha gets affected from Matru and Pitru beeja dosh then by birth patient suffers from Prameha.

In conclusion, Prameha is hereditary disease.

Charaka also explained that Sahaj Madhumeha is a kulaj vikar, due to veekrut beeja (sperm and ovum). Here the patient is prone to disease because of above factor. 117

Charak stated that an excessive intake of madhura rasa by parents may get affected the kaphadhatu and it leads to veekruti (deformity) in sperm and ovum, thus during pregnancy mother and foetus gets affected by Prameha.

2) Apathynimittaj
a) General etiological factors of Prameha 118

Asyasukha i.e. sedentary lifestyle

Sawpansukha.

Excessive intake of curd or preparations from curd

Excessive intake of udaka, Aanoop mamsa i.e. meat of domestic aquatic land animals.

Payamsi i.e. excessive intake of milk and milk preparations.

Navannapanam i.e. new grains and drinks

Guda vaikrutam i.e. various preparation of sugar and jagary.

Other substance which aggravates kapha may cause Prameha.
Vagbhata said that due to ahitakar aahar and vihar, meda, mutra and kapha are vitiated; this is the main causes of Prameha\textsuperscript{119}

Sushruta mentioned that sheeta, snigdh, madhur, medya (fatty) and drava (liquid) type of food are the responsible for Prameha\textsuperscript{120}.

**B) Kaphaj Prameha Nidana\textsuperscript{121}**

The following factors which aggrivates the kapha i.e. kaphaprakopak.

Intake of tila, pisthtanna, paaysha (a type of milk preparation), krishara, vilepi and preparations of sugarcane.

Intake of milk, fresh wine, curd preparations, sheet aahar

Reduced physical activity.

Sedentary life style.

Restoring regimens which can increase mere kapha, fat and urine.

**C) Pittaj Prameha Nidana\textsuperscript{122}**

Intake of ushna, kshaar, amla, lavana, ktu dravya (i.e. Spicy food)

Ajeerna and vishaam aahar sevan,

Exposure to excessive heat, sun, emotions like anger and physical exertion.

Shushruta stated that vata, kapha, shonita and meda are involved in samprati of Pittaja Prameha\textsuperscript{122}

**D) Vataja Prameha Nidana\textsuperscript{123,124}**

Nidan of vataj prameha is as follows,

Excessive intake of katu, tikta, ruksha, sheeta veerya aahar,

Excessive physical exercise,

Excessive treatment of panchakarmas viz; vaman, virechan, aashthapan and shirovirechan.

Suppression of natural urges like mala, mutra, upavas, ratri-jagran, excessive blood loss, ati shok, udvega (worry/sorrow) and irregular posture of body are the main causes of vataj Prameha.

Stress is also found to be common cause in all types of Prameha.

Charak said that vataj prameha, due to its own properties becomes Asadhy.

Vasameha, Majjameha, Hastimeha and Madhumehaare the four types of vataj prameha.\textsuperscript{125}
Poorva roopa:
Bahumutrata is one of the important poorvaroop of prameha. It predicts the nature of disease. It is clearly stated in Sushruta samhita, Poorvaroopa transformed into roopa in association with Bahumutrata. Madhumeha is classified under the Doshaj type of Prameha; So poorvaroopa of Prameha can be taken as poorvroopa of Madhumeha.

Table No. 17: Poorvroopa of Prameha

<table>
<thead>
<tr>
<th>Poorvroopa</th>
<th>Cha</th>
<th>Su.</th>
<th>A.H.</th>
<th>A.S.</th>
<th>M.Ni</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kesheshu Jatilibhava</td>
<td>+</td>
<td>+</td>
<td>-</td>
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<td>-</td>
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<tr>
<td>Asya Madhurya</td>
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<tr>
<td>Karapadadaha</td>
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<tr>
<td>Karapada Suptata</td>
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<tr>
<td>Mukha Talu Kantha Shosha</td>
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<tr>
<td>Pipasa</td>
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<tr>
<td>Alasya</td>
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<td>-</td>
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<tr>
<td>Kaye Malam</td>
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<tr>
<td>Kaya Chhidreshu Upadeha</td>
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<tr>
<td>Paridaha Angeshu</td>
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<tr>
<td>Suptata Angeshu</td>
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<tr>
<td>Shatpada Pupilika Mutrabhisaranam</td>
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<td>-</td>
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<tr>
<td>Mutre cha Mutrasosham</td>
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<td>Visra sharer Gandhi</td>
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<td>Sarvakala Nidra</td>
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<td>Sarvakala Tandra</td>
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<tr>
<td>Snigdha Gatrata</td>
<td>-</td>
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<td>-</td>
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<tr>
<td>Pichhila &amp; Guru Gatrata</td>
<td>-</td>
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<td>-</td>
</tr>
<tr>
<td>Madhur Mutrata</td>
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<td>+</td>
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<tr>
<td>Shukla Mutrata</td>
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<tr>
<td>Sada</td>
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<tr>
<td>Shwasa</td>
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<td>-</td>
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<tr>
<td>Keshanakhativriddhi</td>
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<td>-</td>
</tr>
<tr>
<td>Sheeta Priyata</td>
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<td>-</td>
<td>+</td>
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<td>-</td>
</tr>
<tr>
<td>Hridaya Netra Jihwa Shravanopdeha</td>
<td>-</td>
<td>-</td>
<td>+</td>
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</tr>
<tr>
<td>Sweda</td>
<td>+</td>
<td>-</td>
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<td>+</td>
<td>-</td>
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<tr>
<td>Dehe Chikkanata</td>
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<td>+</td>
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</tbody>
</table>
6) **Roopaavastha of Prameha:** In Shusruta samhita it is stated that person should be diagnosed as Pramehi when poorvroopas are associated with the Bahoomutrata. Gyadasa while describing specific disease state that poorvaroopa get converted into roopa i.e. vyadhi prabhava.

1) **Specific Roopa of Madhumeha**

   **Urine Characteristics:**
   
   a) **Prabhuta Mootrata:** -
   
   This is the cardinal sign of Prameha described by all Acharyas. Urine of Madhumehi is kashaya, madhura, pandu varna and ruksha quality. As per Sushruta, the urine of Madhumehi resembles to that of honey and same description is stated by Vagbhata.

   b) **Avila Mutrata (Turbidity):** -
   
   Due to dushya, dosha and mutra patient passes turbid urine.

   c) **Picchila Mutrata:** -
   
   Patient passes the urine picchila and madhura.

2) **Associated Signs and Symptoms:**

   Sushruta has described two types of Prameha along with their manifestations as follows:

   i) **Sahaja Pramehi (Krisha-Asthenic)**
   
   - Ruksha (Dry body)
   - Alpashi (Eat less amount of food)
   - Bhrish Pipasa (Excessive thirst)
   - Parisarpansheela (Restless, always desires to wander)

   ii) **Apathyanimittaja (Sthula-Obese)**
   
   - Bahuashi (consumes excess amount of food)
   - Snigdha (Unctuous body texture)
   - Shayyasanswapnasheela (prefer sedentary life style)

D) **Kashyapa** has described the symptoms as follows: -

   Akasmata mutra Nirgaman—Child excretes urine suddenly.
   
   Akranta mutra—Flies get attracted towards urine.
   
   Shweta and Ghanamutrata. —Child passes urine turbid and Shweta.
Kashyapa has also narrated symptoms like Gaurava (Heaviness of the body), Baddhata (tightness) and Jadata (Steadiness, laziness).

7. Samprapti

Samhita describes three different sampaptis, based on Dosha dominace.

A). Samprapti of Kaphaja Prameha: 134

Charak mentioned in the NidanSthana and Chikitsasthana about the etiological factors which causes to kaph-prakopa. He also mentioned ‘Bahudrava sleshma’ is main causative factor.135

In Kaphaj Prameha, vikrita kleda is increased. The body tries to move it out. Some part of its get converted into Swed, because ‘swedasya kledavidruti.’ So it produces ‘Chikkanata dehe.’ Remaining kleda is taken to basti; therefore, prabhoota mootrata is seen. Due to vikrit Kleda various abnormalities seen in urine. Thus, due to dusit dosha-dshyhas, dhatu-shaithilya, excessive production of kapha, meda and mutra is seen and ‘Bahoodravoottra’symptom is found.136

Sushruta narrated dushyas in each doshaj type of Prameha. He stated In Kaphaj Prameha Kapha vitiates along with vata, pitta and meda. 137

B) Samprapti of Pittaj Prameha: 138

Due to hetu-sevan pitta dosha gets provoked. TheUshna Guna of Pitta, Vilyana of dravansha from dhatus takes place. This pitta along with increased kleda vitiates dhatu like meda, mamsa, Prameha occures. Shushruta mentioned that in Pittaj Prameha, Shonit also vitiates with vata and kapha.139

C) Vataj Prameha: 140

The samprapti of Vataj prameha as per Charaka, kapha, pitta and kleda are Ksheena, Vivrudha Vata with Ashayapakshagati pulls kleda and other dusyas like majja, oja rasa, meda, to Basti. In relation to Basti, kleda is increased but this is kshayatmaka Samprapti. Thus, the dusyas like vasa, majja, lasika and oja vitiates one by one and are brought to Mootraashay and eliminated in mootra form.140

Shushruta mentioned that samprati of vataj Prameha occurs due to pitta, meda, kapha, vasa and majja.141

As per Vagbhat, first kaphaj prameha is produced, then as the sampraprti progresses, the same turn to Pittaj and ultimately Vataja

As we go to kaphajmeha to vatic the gati of dosha-dushya sammurcchana can be explained as,
Table No.18: Relation between Dosha, Duysh and Madhumeha

<table>
<thead>
<tr>
<th>Dosha prakopa</th>
<th>Dushya</th>
<th>Meha produced</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kapha</td>
<td>Rasa, mamsa, meda</td>
<td>Kaphaja</td>
</tr>
<tr>
<td>Pitta</td>
<td>Rakta</td>
<td>Pittaja</td>
</tr>
<tr>
<td>Vata</td>
<td>Remaining Pradhana Dhatus</td>
<td>Vataja</td>
</tr>
</tbody>
</table>

Shusrut has mentioned that if Prameha is not treated in a particular time then it is converted into Madhumeha. So, let us consider about Madhumeha.

8. **Madhumeha:**

Madhumeha is very well explained by Charaka when Prameha not treated in particular time it gets converted into Madhumeha. Madhumeha is a vyadhi (disease).\(^{142}\) Vagbhatta explained two types of Madhumeha according to samprati.\(^{143}\)

A) Dhatukshayajanya madhumeha

B) Avranjanya Madhumeha

1. **Vataj Madhumeha:**\(^{144}\)

Charaka described detailed samprati of Vataj Prameha. Due to hetusevan vata provokes and vitiated vata with the saarbhoot dhatu like vasa, majja, oja and lasika, carries towards mootrashya resulting into Vataj Prameha.

The ruksha guna of vata carried out Ksyatamaka samprapti. So, the dusyas like mamsa, meda and oja are carried out towards mootrashaya by asyapakarsha gati and bahumootrata takes place. This condition is termed as madhumeha.

2. **Madhumeha due to Shuddha Vata:**\(^{145}\)

Due to kapha and pitta kshya (loss) and with the kshya of vasa, majja, lasika and oja, vata dosha gets aggrevated and draws oja towards basti leading to madhumeha.

A) **Dhatukshayajanya Madhumeha:**\(^{146}\)

Dhatukshaya mainly occurs due to excessive santarpan and apatrapan also. Due to apatarpan or excess santarpan, dhatupariposhankrama gets disturbed and thus the vata gets provoked.

The kshaya of vital dhatus vasa, majja, lasika and oja leads to vataparakopa. Vata makes ksharana of dhatu. Dhatus are also having Shiathilya. So, through mutravaha srotas mutra, lasika, vasa are moved out and when Kapha and Pitta gets depleted the vata provocation occurs and it leads to kshay of vasadi dhatu.\(^{147}\)
B) Avaranjanya Madhumeha: -
Vagbhata mentioned this type of madhumeha but not explained. However, Charak explained it very well. This is the unique contribution of Charaka. Due to hetusevan like guru, snigdh, amla, new grains, excessive exercise and excessive panchkarma measure Kapha and Pitta gets provoked and vitiates meda and mamsa. They in turn cause obstruction of normal pathway of vata. Thus, obstructed vata aggrivates by making avaran with dusyas and carries the vitiated doshas towards basti resulting into Madumeha.

1. Kalprabhavaja Madhumeha: 
Sushrut explained all types of Prameha, if not treated in particular time, it converted into Madhumeha. This is the later stage of disease. It is asadhya.

3.2.9. Samprapti Ghataka’s of Madhumeha: -

1. Dosha:
   - Kapha : Bahu +Abaddha in avaranjanya madhumeha
     Kshin in dhatukshayajanya madhumeha
   - Pitta : Vriddha-in avaranjanya madhumeha
     Kshina in dhatukshayajanya madhumeha
   - Vata: Avritta- in avaranjanya madhumeha

Vriddha-in dhatukshayajanya madhumeha

2. Dhatus: 
   - Rasa, Rakta, Mamsa, Meda, Majja, Vasa, Lasika, Oja, Shukra, Ambu, Sweda

3. Srotas: Medovaha, Mutravaha, Udakavaha

4. Srotodushti: Atipravritti, Sanga

5. Prakopa: Sarva sharira

6. Prasara: Rasayani

7. Sthanasamshraya.: Mutravaha Srotas

8. Agni: Dhatwagnimandya

9. Ama: Dhatugata (Aparipakwa Dhatu)

10. Udbahva: Amashaya

11. Swabhava: Chirkari
1. Dosh involvement:

1. Kapha dosh: -

Charaka described as ‘Kaphakrutch sarvum’. It indicates the importance of this dosha. Kapha is produced from Rasa; therefore Rasa and Meda are necessarily to be considered. Provakation of kledaka kapha results into sharirshaithilya; and results into atinidra, tandra, aalasya etc.

2. Pitta dosh: -

Due to dusshit pitta and its drava guna rakta, lasika, sweda and rasa these dusyas get affected. Thus swedavridhi, visragandha, paridaha, pipasa and sosha these symptoms of pittadosha vridhi appeared.

3. Vatadosha: -

This is the predominated dosha in Madhumeha samprapti. Vata aggravates due to its own etiological factors or because of avarna caused by kapha, pitta and meda. The provoked vata affects the dusya like vasa, majja, and oja, lasika. All these vital constituents are moving out. Thus, all the dhatus becomes durbale. This leads to poor vyadhikshamatava. Thus, the symptom manifests are karshya, daourbalya, angasuptata and parisa rpansheela.

In all prameha samprapties, Vyana acts as the collector of kleda and Apana as excreter.

2. Dhatu involvement: -

Dosha, dushya and nidan are the three responsible factors for the manifestation of every disease.

Charaka enumerated a special group known as dushya visesha. He mentioned dusyvisesha in chikitsasthan also. Dusyvishehas are Rasa, Rakta, mamsa, Meda, Majja, Shukra, kleda, vasa, lasika and oja.151

Sushruta explained the dushyas along with basic type and mentioned that meda is a common dushya in each type.152

All the ten dushyas along with sweda are mentioned by Vagbhatta.

2. Dhatu and Dushya involvement:

Due to combination of these dushyas 20 types of Prameha occured

I) Rasadhatu: -

As kapha gets vitiated rasa also vitiated. Vagbhata also mentioned that ‘Rasoapi shleshmavat’153So as shusrut said that vitiation of rasadhatu results into Staulya and Karshya154
So Rasdhatu plays important role in the disease. Vitiated rasa produces symptoms like aganimandya, alasya, glani, karshya, klaibya and aganimandya in madhumeha.

II) Raktadhatu: -
It gets vitiated causing complications like Pidika, Vidradhi, Alaji.

III) Mamsadhatu: -
It is involved in kaphaj Prameha and avaranjanya madhumeha. Due to vitiation of mamsa dhatu, it loses its normal consistency, develops shaithilya and provides ‘Avakash’ in the body. It turns into putimamsa pidaka.155

IV) Medadhatu: -
It is the important dushta in all types of Prameha, both quantitatively and qualitatively. Due to vitiation of doshas ‘meda evam upchiyate na tathetar dhatava.’ i.e. further dhatu develop gradually. The Dhatus prior to it also develop Kshaya. Meda itself is also having vikrit vriddh but there is prakrut medakshaya. i.e all the dhatues becomes Ksheena and shithil. Vikrit Meda can produce abnormality in Sira and produces Siradaurbalya.Here bahuabddameda is Dushyavishesha. Arundatta commented that, it is important to occur meda-kshaya, which produces. Ayushorhasa, javoparadha, krucharvyavyata, general debility, daurgandhya, Swedabadha, excessive appetite and thrust.

V) Majjadhatu: -
If Majja dhatu vitiated, Vatakshaya occurs. Vitiatied majja produces symptoms like netrang gaurava, angagaurava in Madhumeha. Due to Vataprakop with majja excessive mutra secretes leading to majjameha.

VI) Shukradhatu: -
Prameha is a kulaja vikar and occurs because of beeja dosha. Shukra possesses an important role in Sahaj Prameha. Apana and vyan are the main causative factors for shukradosha and Prameha. Due to vata aggrivation shukrakshaya occurs, and this vata causes to shukrameha also.

VII) Oja: -
Oja is the main dushta of madhumeha. Due to vataprakopa and avaran of oja it carries towards basti and excretes through urine. Due to ojakshya symptoms like gurugatrata, nidra, tandra, daurbalya are produced Ojakshaya produces rukshata156, so one can easily understand manifestations of Krushapramehi or Sahaj pramehi.
VIII) Kleda: -
Kleda is an important dushya in Prameha. The literary meaning of kleda is wetness, moisture. Kleda establishes abdhatwa in the dhatus. Increased kleda increases bahudravshleshma and when dosha and dushya gets vitiated with this bahuabddha kleda, it gets converted into mutra and results in an increased frequency of mutra. Thus, due to vrudha kleda prabhutmutrata, swedavriddhi, saithilya, daurgandhya and avilamutrata are seen.

ix) Vasa: -
Vasa is upadhatu of mamsa and is main dushya is vataj prameha. So vasmeha is a type of Vataj Prameha.

x) Lasika:-
Lasika is one type of bodyfluid. Dushti of lasika described in Hastimeha

Xii) Sweda:-
This dushya is separately mentioned by Vagbhatta, and is mainly related with kleda and meda. Sweda agrrigation results in symptoms like swedavruddhi, daurgandhya, picchilagatra and snigdhagatata etc. Only, Shushrut mentioned that in madhumeha sweda becomes sweet in nature.157

3. Srotas: -
In Madhumeha along with mutravahasrotas, there is a reference of sroto dushti only related to Mutravaha srotas but as per symptoms, we can easily understand involvement of medovaha, mamsavaha, swedavaha and udakavaha srotas too

9. Sadhyasadhytwa:
Sadyasadyta depends upon Hetu, Nidan, Rupa and Mootrapariksha

i) Sadhya
Kaphaj Prameha is sadhya as dosha and Meda also possesses same properties and dushay are of same gunas.

ii) Kruchhasadhya -
Pittaj prameha and Vataj prameha are Kruchhasadhya. Madhumeha occurs due to avaranjanya and is difficult to treat i.e. kruchhasadhya159

iii) Yapyatwa:-
Pittaj meha are said to be yapya160

iv) Asadhytwa:-
Vataj prameha is incurable because there is involvement of vital dhatus like majja, oja, and as dosha and Dushays are of Vruddha guna161
Charaka mentioned madhumeha because of bejjadosha is incurable i.e. Asadhyā. Sushruta mentioned madhumeha in association with prameh- pidaka is asadhya. Dhatukshayajanyā vataja madhumeha is Asadhaya.

10. Upadrava:
Charak has described the general complications where as Sushrut and Vaghbatha have described it according to the dosha predominance.

1. General Updrava:
Trushna, Atisara, Jwara, daha, daurbalya, Arochak, Apachana Pootimamsa, Pidaka, Alaji and vidradhi. These upadavas takes place due to long term anubandha of Prameha disease.

2. Doshaj updravas:
Sushrut mentioned twenty upadravas as per Dosha dominanace. These upadavas are as mentioned by Shushrut and Vagbhatta.

   A) Kaphaja meha Upadravas:
Makishikosarpanam, Aalsya, Mamsopachya, Pratishyaya, Shaithilya, Arochaka, Avipaka, kaphaprasek, Chhardi, Nidra, Kaasa and Shwasa.

   B) Pittaj meha Upadravas:
Vrushanayoavadaranam, Bastibheda, Medratoda, Hridshula, Amlika, Jwara, Atisara, Arochaka, Paridhumayanam, Daha, Murrchha, Pipasa, Nidranasha, Pandurog, Pittavinmutranetratva and Vibheda.

   C) Vataj meha Upadravas:
Hridgraha, Laulya, Anidra, Stambha, Kampa, Shula, Buddha purishatvaand shosha, kasa, Shvasa. Prameha and Sthaulya are closely related, therefore the complications of Sthaulya also can be observed in Prameha.

11. Prameha Pidaka:
Brihatttrayi have described Prameha pidaka is a major complication of Prameha. Charaka mentioned ‘saptapidaka’ as upadrava of madhumeha, where as Sushruta and Vaghbatha mentioned ten pidaks, and Kashayap mentioned astha pidaka. These pidaka may develop without Prameha in the person having medodushti only. Sushruta mentioned that madhumeha along with pidakais asadhya. He told that these pidaka occurs due to tridosha and vitiated meda and mamsa.
These pidaka are as follows:
Charak suggests that these pidakas are treated by sansodhan and ropan aushadhaiies, by a good surgeon. If these pidakas are not treated in particular time, can be it converted into vrana; and these pidakas requires surgical intervention.

12. Differential Diagnosis:

To differentiate Prameha, from other diseases, Charaka has explained the presence or absence of poorvaroopas; if colour of urine is haridra or rudira, and it is associated with poorvaroopas of prameha, then we can diagnose prameha. But if poorvaroopas of prameha are absent, it is only rakta and pitta vriddhi.  

If patient passes urine as like honey, madhur, Sapiccha, think about differential diagnosis in two ways, one if symptoms are due to dosha-dusya kshya, then it is
considered as upadrava of Vataj Prameh; and if the symptoms are due to Santarpanjanya hetu then it is considered as upadrava of Kaphaj Prameha.\textsuperscript{172}

Charaka mentioned that, if Medhumeha is present, it is difficult to distinguish from kaphaja mehas.

**13. Arishta Lakshana (Fatal signs):**

Charaka has mentioned arishta laksanas related to Prameha i.e. the indications of ensured death. He said that if the flies are attracted towards patient after bath patient will die due to Prameha. He also said that if he daily drinks various kinds of oil and ghees in his dream also; he may die due to Prameha.\textsuperscript{173}

In Indriyaastana, Charak stated that if the madhumehi patient losses his balmamsa; then the Vyadhi becomes Achikitsaya i.e. difficult to cure. Thus, Madhumeha is one of the “Ashtau maharog.”\textsuperscript{174,175}
3.3. Review of Ulcer and Chronic ulcer

1. History

The science of wound healing has an exciting journey over the ages. Since the caveman, man has been tending to his wounds. Wound care evolved from magical incantations, potions, and ointments, to a systematic text of wound care and surgery from Sushruta, Hippocrates and Celsus.

The Egyptians were masters in applying and arranging bandages, and they recognized the cardinal signs of infection and inflammation. Egyptian drug therapy can be regarded as having evolved from a system rooted in magic and empirical observation. Pressure sores have been found on 5,000-year-old mummies in Egypt. The ancient Egyptians used honey as a wound treatment.

The 1650 B.C. Edwin Smith Surgical Papyrus, a copy of a much older document, describes at least 48 different types of wounds. A later document (Ebers Papyrus, 1550 B.C.) relates the use of concoctions containing honey (antibacterial properties), lint (absorbent properties), and grease (barrier) for treating wounds. These same properties are still considered essential in contemporary daily wound management. Yet another very early account of wound healing dating back to about 2000 B.C. suggests that the Sumerians employed two modes of treatment: a spiritual method consisting of incantations and a physical method of applying poultice-like materials to the wound.

Hippocrates, a Greek physician and surgeon, 460-377 B.C., known as the father of medicine, used vinegar to irrigate open wounds and wrapped dressings around wounds to prevent further injury. He washed ulcers with wine and after having softened them by oil, he dressed them with fig leaves. Galen, a notable Roman surgeon, was first to recognize that pus from wounds inflicted by the gladiators preceded wound healing.

Plinio used mineral remedies as lead and silver, Galen used spice ointments. These advances achieved in wound care and surgery for healing wounds by Hippocrates and Celsus were lost after the fall of the Roman Empire. In Europe, the middle ages were a regression of wound care back to potions and charms. It was not until the time when
large armies started using muskets and cannons, that surgical wound care emerged again.

The 19th century brought significant advances in wound treatment. Joseph Lister, a Professor of Surgery in London, recognized that antisepsis could prevent infection. Lister placed carbolic acid into open fractures to sterilize the wound and prevent sepsis. Changes were also made to sterilize the surroundings of a wounded patient. Hand washing prior to care along with sterilization of instruments as well as wearing of gowns, masks and gloves began in 1880s.

The scale of wound infections was most evident in times of war. During the American Civil War, infected wounds accounted for some 17,000 deaths. World War I brought new types of wounds from advanced weaponry and contamination from the trenches. A Belgian military surgeon, Depage, introduced wound debridement and delayed wound closure and would use microbiological assessment to determine if wound was safe for closure.

The use of antibiotics ushered in a new era in wound care. Penicillin was first used clinically in 1940. However, the use of antibiotics did not end wound infections as resistant bacteria and new surgical interventions has risen. The primary method for wound management is prevention. The use of systemic antibiotics and surgical drainage and excision of damaged tissue are primary methods of wound management in present day.

2. Definition of Ulcer: 177,178

A lesion on the surface of the skin or mucous surface caused by superficial loss of tissue usually with inflammation.

An open sore or lesion of skin or mucous membrane accompanied by sloughing of inflamed necrotic tissue

An injury especially one in which in skin or another external surface is torn pierced, cut or otherwise broken, it is the discontinuity or break of the surface. Though both ulcer and wound have some similar and dissimilar features, they mean the same–disruption in epithelial surface. A wound with superficial loss of tissue from trauma is not primarily an ulcer, but may become ulcerated if infection occurs.
Chronic ulcers: Chronic ulcers are the wounds that fail to heal; in general, they have a fibrotic margin and a bed of granulation tissue which may include areas of slough (necrotic tissue).

3. Physiological and Pathological consideration of wound healing: 189

Wound healing is a dynamic, interactive process involving soluble mediators, blood cells, extracellular matrix and parenchymal cells. Wound healing has three phases’ inflammation, tissue formation, and tissue remodeling that overlap in time.

A) Inflammation:

Tissue injury causes the disruption of blood vessels and extravasation of blood constituents. The blood clot reestablishes hemostasis and provides a provisional extracellular matrix for cell migration. Platelets not only facilitate the formation of a hemostatic plug but also secrete several mediators of wound healing, such as platelet-derived growth factor, that attract and activate macrophages and fibroblasts. However, in the absence of hemorrhage, platelets are not essential to wound healing. Numerous vasoactive mediators and chemotactic factors are generated by the coagulation and activated-complement pathways and by injured or activated parenchymal cells. These substances recruit inflammatory leukocytes to the site of injury. Infiltrating neutrophils cleanse the wounded area of foreign particles and bacteria and are then extruded with the eschar or phagocytosed by macrophages.

In response to specific chemo attractants, such as fragments of extracellular-matrix protein, transforming growth factor β, and monocyte chemo attractant protein 1, monocytes also infiltrate the wound site and become activated macrophages that release growth factors such as platelet-derived growth factor and vascular endothelial growth factor, which initiate the formation of granulation tissue. Macrophages bind to specific proteins of the extracellular matrix by their integrin receptors, an action that stimulates phagocytosis of microorganisms and fragments of extracellular matrix by the macrophages.

Adherence to the extracellular matrix also stimulates monocytes to undergo metamorphosis into inflammatory or reparative macrophages. Adherence induces monocytes and macrophages to express colony-stimulating factor 1, a cytokine necessary for the survival of monocytes and macrophages; tumor necrosis factor α, a
potent inflammatory cytokine; and platelet-derived growth factor, a potent chemoattractant and mitogen for fibroblasts. Other important cytokines expressed by monocytes and macrophages are transforming growth factorα, interleukin-1, transforming growth factor β, and insulin like growth factor I. The monocyte- and macrophage-derived growth factors are almost certainly necessary for the initiation and propagation of new tissue formation in wounds; because macrophage depleted animals have defective wound repair. Thus macrophages appear to have a pivotal role in the transition between inflammation and repair.

B) Epithelialization:

Epithelialization of wounds begins within hours after injury. Epidermal cells from skin appendages such as hair follicles quickly remove clotted blood and damaged stroma from the wound space. At the same time, the cells undergo marked phenotypic alteration that includes retraction of intracellular tonofilaments dissolution of most inter-cellular desmosomes, which provide physical connections between the cells; and formation of peripheral cytoplasmic actin filaments, which allow cell movement. Furthermore, epidermal and dermal cells no longer adhere to one another, because of the dissolution of hemidesmosomal links between the epidermis and the basement membrane, which allows the lateral movement of epidermal cells. The expression of integrin receptors on epidermal cells allows them to interact with a variety of extracellular-matrix proteins (e.g., fibronectin and vitronectin) that are interspersed with stromal type I collagen at the margin of the wound and interwoven with the fibrin clot in the wound space. The migrating epidermal cells dissect the wound, separating desiccated eschar from viable tissue. The path of dissection appears to be determined by the array of integrins that the migrating epidermal cells express on their cell membranes.

The degradation of the extracellular matrix, which is required if the epidermal cells are to migrate between the collagenous dermis and the fibrin eschar, depends on the production of collagenase by epidermal cells, as well as the activation of plasmin by plasminogen activator produced by the epidermal cells. Plasminogen activator also activates collagenase (matrixmetalloproteinase1) and therefore facilitates the degradation of collagen and extracellular-matrix proteins.
One to two days after injury, epidermal cells at the wound margin begin to proliferate behind the actively migrating cells. The stimuli for the migration and proliferation of epidermal cells during reepithelialization have not been determined, but several possibilities exist. The absence of neighbour cells at the margin of the wound (the “free edge” effect) may signal both migration and proliferation of epidermal cells. Local release of growth factors and increased expression of growth-factor receptors may also stimulate these processes. Leading contenders include epidermal growth factor, transforming growth factor α, and keratinocyte growth factor. As reepithelialization ensures, basement-membrane proteins reappear in a very ordered sequence from the margin of the wound inward, in a zipper like fashion. Epidermal cells revert to their normal phenotype, once again firmly attaching to the reestablished basement membrane and underlying dermis.

C) Formation of Granulation Tissue:

New stroma, often called granulation tissue, begins to invade the wound space approximately four days after injury. Numerous new capillaries endow the new stroma with its granular appearance. Macrophages, fibroblasts, and blood vessels move into the wound space at the same time. The macrophages provide a continuing source of growth factors necessary to stimulate fibroplasia and angiogenesis; the fibroblasts produce the new extracellular matrix necessary to support cell in growth; and blood vessels carry oxygen and nutrients necessary to sustain cell metabolism. Growth factors, especially platelet-derived growth factor 4 and transforming growth factor β1, in concert with the extracellular-matrix molecules, presumably stimulate fibroblasts of the tissue around the wound to proliferate, express appropriate integrin receptors, and migrate into the wound space. Indeed, platelet-derived growth factor accelerates the healing of chronic pressure sores and diabetic ulcers, and basic fibroblast growth factor has been used with some success to treat chronic pressure sores.

The structural molecules of newly formed extracellular matrix, termed the provisional matrix, contribute to the formation of granulation tissue by providing a scaffold or conduit for cell migration. These molecules include fibrin, fibronectin, and hyaluronic acid. In fact, the appearance of fibronectin and the appropriate integrin receptors that bind fibronectin, fibrin, or both on fibroblasts appears to be
the rate-limiting step in the formation of granulation tissue. The fibroblasts are responsible for the synthesis, deposition, and remodeling of the extracellular matrix. Conversely, the extracellular matrix can have a positive or negative effect on the ability of fibroblasts to synthesize, deposit, remodel, and generally interact with the extracellular matrix. Cell movement into a blood clot of cross-linked fibrin or into tightly woven extracellular matrix may require an active proteolytic system that can cleave a path for cell migration. A variety of fibroblast-derived enzymes, in addition to serum-derived plasmin, are potential candidates for this task, including plasminogen activator, collagenases, gelatinase A, and stromelysin. After migrating into wounds, fibroblasts commence the synthesis of extracellular matrix. The provisional extracellular matrix is gradually replaced with a collagenous matrix, perhaps as a result of the action of transforming growth factor β1. Once an abundant collagen matrix has been deposited in the wound, the fibroblasts stop producing collagen, and the fibroblast-rich granulation tissue is replaced by a relatively acellular scar. Cells in the wound undergo apoptosis 35 triggered by unknown signals. Dysregulation of these processes occurs in fibrotic disorders such as keloid formation, morphea, and scleroderma.

D) Neovascularization:

The formation of new blood vessels is necessary to sustain the newly formed granulation tissue. Angiogenesis is a complex process that relies on extracellular matrix in the wound bed as well as migration and mitogenic stimulation of endothelial cells.

The induction of angiogenesis was initially attributed to acidic or basic fibroblast growth factor. Subsequently, many other molecules have also been found to have angiogenic activity, including vascular endothelial growth factor, transforming growth factor β, angiogenin, angiopoitropin, angiopoietin 1, and thrombospondin, to name but a few. Low oxygen tension and elevated lactic acid may also stimulate angiogenesis. Many of the molecules mentioned above appear to induce angiogenesis by stimulating the production of basic fibroblast growth factor and vascular endothelial growth factor by macrophages and endothelial cells. Activated epidermal cells of the wound secrete large quantities of vascular endothelial cell growth factor. Basic fibroblast growth factor may set the stage for angiogenesis during the first three
days of wound repair, whereas vascular endothelial-cell growth factor is critical for angiogenesis during the formation of granulation tissue on days 4 through 7.

In addition to angiogenesis factors, appropriate extracellular matrix and endothelial receptors for the provisional matrix are necessary for angiogenesis. Proliferating microvascular endothelial cells adjacent to and within wounds transiently deposit increased amounts of fibronectin within the vessel wall. Since angiogenesis appears to require the expression of functional fibronectin receptors by endothelial cells, the perivascular fibronectin may act as a conduit for the movement of endothelial cells into the wound. Protease expression and activity are also necessary for angiogenesis.

The series of events leading to angiogenesis may be as follows. Injury causes destruction of tissue and hypoxia. Angiogenesis factors such as acidic and basic fibroblast growth factor are immediately released from macrophages after cell disruption, and the production of vascular endothelial-cell growth factor by epidermal cells is stimulated by hypoxia. Proteolytic enzymes released into the connective tissue degrade extracellular-matrix proteins. Fragments of these proteins recruit peripheral-blood monocytes to the site of injury, where they become activated macrophages and release angiogenesis factors. Certain macrophage angiogenesis factors, such as basic fibroblast growth factor, stimulate endothelial cells to release plasminogen activator and procollagenase. Plasminogen activator converts plasminogen to plasmin and procollagenase to active collagenase, and in concert these two proteases digest basement membranes. The fragmentation of the basement membrane allows endothelial cells stimulated by angiogenesis factors to migrate and form new blood vessels at the injured site. Once the wound is filled with new granulation tissue, angiogenesis ceases and many of the new blood vessels disintegrate because of apoptosis 46. This programmed cell death probably is regulated by a variety of matrix molecules, such as thrombospondins 1 and 2, and antiangiogenesis factors, such as angiostatin, endostatin, and angiopoietin 2.

4) Wound Contraction and Extracellular-Matrix Reorganization:

Wound contraction involves a complex and superbly orchestrated interaction of cells, extracellular matrix, and cytokines. During the second week of healing, fibroblasts assume a myofibroblast phenotype characterized by large bundles of actin-
containing microfilaments disposed along the cytoplasmic face of the plasma membrane of the cells and by cell–cell and cell–matrix linkages. The appearance of the myofibroblasts corresponds to the commencement of connective-tissue compaction and the contraction of the wound.

The contraction probably requires stimulation by transforming growth factor β1 or β2 and platelet-derived growth factor, attachment of fibroblasts to the collagen matrix through integrin receptors, and cross-links between individual bundles of collagen. Collagen remodeling during the transition from granulation tissue to scar is dependent on continued synthesis and catabolism of collagen at a low rate. The degradation of collagen in the wound is controlled by several proteolytic enzymes termed matrix metalloproteinases, which are secreted by macrophages, epidermal cells, and endothelial cells, as well as fibroblasts17. The various phases of wound repair rely on distinct combinations of matrix metalloproteinases and tissue inhibitors of metalloproteinases.

5) **Tensile strength:**

Wounds gain only about 20 percent of their final strength in the first three weeks, during which time fibrillar collagen has accumulated relatively rapidly and has been remodeled by contraction of the wound. Thereafter the rate at which wounds gain tensile strength is slow, reflecting a much slower rate of accumulation of collagen and, more important, collagen remodeling with the formation of larger collagen bundles and an increase in the number of intermolecular cross-links. Nevertheless, wounds never attain the same breaking strength (the tension at which skin breaks) as uninjured skin. At maximal strength, a scar is only 70 percent as strong as normal skin.

6. **Clinical signs of infection:**

Although a detailed discussion of the many conditions associated with abnormal ulcer healing is beyond the scope of this review, several examples will illustrate the multifactorial nature of these conditions. Diabetic ulcers are an excellent example of how multiple physiologic and biochemical defects can lead to impaired healing. They usually occur in patients who are unable to sense and relieve cutaneous pressure because of neuropathy. Ischemia secondary to vascular disease impedes
healing by reducing the supply of oxygen and other nutrients. Diabetic ulcers are also prone to infection because of impaired granulocytic function and chemotaxis. Other abnormalities associated with diabetic ulcers include prolonged inflammation, impaired neovascularization, decreased synthesis of collagen, increased levels of proteinases, and defective macrophage function. Keloids and hypertrophic scars that are characterized by excess accumulation of collagen within the wound are examples of fibroproliferative disorders. In these conditions, abnormalities in cell migration and proliferation, inflammation, synthesis and secretion of extracellular-matrix proteins and cytokines, and remodeling of the wound matrix have all been described. Increased activity of fibrogenic cytokines (e.g., transforming growth factor β1, insulin-like growth factor 1, and interleukin-1) and exaggerated responses to these cytokines have also been noted. In addition, abnormal epidermal–mesenchymal interactions and mutations in regulatory genes have recently been proposed to help explain abnormal healing.

7. Clinical experience with growth factors:

The overall clinical experience with growth factors and other mediators to accelerate wound healing has been discouraging. This is not surprising, considering that wound repair is the result of a complex set of interactions among soluble cytokines, formed blood elements, extracellular matrix, and cells. It is possible that combinations of various growth factors given at precisely timed intervals would be more effective in promoting healing. Indeed, synergistic effects on wound repair have been demonstrated for several growth-factor combinations65. Among these factors, only recombinant platelet-derived growth factor has been approved by the Food and Drug Administration (FDA) for the treatment of wounds.

8. Insights from fetal wound healing:

Fetal wounds reepithelialize rapidly. Unlike adult epidermal cells, which resurface the wound by “crawling” across it, embryonic epidermal cells are pulled forward by the contraction of actin fibers that draw the wound edges together as the opening of a purse is closed by a purse string. Fetal wounds also heal without scarring. One reason for this may be the small amount of transforming growth factor β1, a scarpromoting cytokine, in fetal skin. The addition of transforming growth factor β1 to fetal wounds results in scarring. Furthermore, fetal skin is rich in
metalloprotein that may promote scar less healing. Scarring is reduced in adult rats given neutralizing antibodies to transforming growth factors β1 and β2 and those given transforming growth factor β3, which down-regulates the other transforming growth factor β isoforms. This result supports the central role of transforming growth factor β1 in scar formation.

9. Cytokines important in ulcer healing:

Cytokine- major source- target cells and major effects:

1) Epidermal growth factor- family- Epidermal and mesenchymal regeneration
2) Epidermal growth factor- Platelets- Pleiotropic-cell motility and proliferation
3) Transforming growth factor α- Macrophages, epidermal cells -Pleiotropic-cell motility and proliferation Heparin-binding epidermal growth factor- Macrophages Pleiotropic-cell- motility and proliferation
4) Fibroblast growth factor-family- Wound vascularization
5) Basic fibroblast growth factor-Macrophages-endothelial cells Angiogenesis and fibroblast proliferation
6) Acidic fibroblast growth factor -Macrophages, endothelial cells- Angiogenesis and fibroblast proliferation
7) Keratinocyte growth factor- Fibroblasts Epidermal-cell motility and proliferation
8) Transforming growth factor β-family -Transforming growth factors β 1 and β2- Platelets, macrophages - Fibrosis and increased tensile strength, Epidermal-cell motility, chemotaxis of macrophages and fibroblasts, extracellular-matrix synthesis and remodeling.
9) Transforming growth factor β 3-Macrophages - Antiscarring effects.
10) Other Platelet derived growth factor- Platelets- macrophages, epidermal cells Fibroblast proliferation and chemo attraction, macrophage chemo attraction and activation.
11) Vascular endothelial growth factor- Epidermal cells, macrophages- Angiogenesis and increased vascular permeability.
12) Tumor necrosis factor α-Neutrophils- Pleiotropic expression of growth factors.
13) Interleukin-1- Neutrophils- Pleiotropic expression of growth factors.
14) Insulin-like growth factor I- Fibroblasts, epidermal cells- Re-epithelialization and granulation-tissue formation.
15) Colony stimulating factor 1 - Multiple cells - Macrophage activation and granulation tissue formation.

10. Classification of ulcers: 180

A. Non-specific ulcer

B. Specific ulcers

C. Malignant ulcers

A. Non-specific ulcer:

1) Traumatic ulcer: Any trauma to the skin. Bony proneness are more prone for the ulcers. Trauma may be physical, chemical, mechanical. They are mostly irregular in shape, painful.

2) Infective: The primary ulcers are infected by various microorganisms. These are small or multiple, with discharge.

3) Arterial ulcer/ Ischemic ulcer: These are due poor peripheral circulation. This may see in various arterial diseases like emboli, atherosclerosis, Diabetes, vasculitis, etc. These ulcers mostly seen on the anterior and outer aspects of the leg, dorsum of the foot, on the toes or the heel. Pain are the main symptom with punched out edge.

4) Venous ulcers: These ulcers are due to hypertension in vein mostly seen in varicose vein, Thrombosis and Phlebitis.

Venous ulcers are most common on inner side just above medial malleolus of leg. The shape is usually ovoid, single in number with irregular, thin blue margin, pale granulation tissue, Pigmentation and eczema is seen in the vicinity of ulcer. These ulcers are usually shallow. Pain is minimal mostly in initial period.

5) Neurogenic ulcers: Occurs due to, impairment of the nutrition of tissues, inadequate blood supply and neurological deficit or repeated trauma to the insensitive part of the body. These ulcers mostly seen on heel when patient is non-ambulatory. Edge is punched out, base is slightly indurated and floor is covered with slough. Surrounding skin has no sensation and these are painless. E.g. Bed sore, perforating ulcers.

6) Martorells ulcer: Mostly seen in hypertensive/ atherosclerotic peoples.

7) Cryopathic ulcer: These result from cold trauma.
8) **Tropical ulcer**: Characteristic feature of this ulcer is callousness towards healing. Edge is slightly raised and exudes copious serosanguineous discharge. Pain is an important symptom. In some cases, it destroys surrounding tissue and spreads widely.

9) **Diabetic ulcer**: The small injury to the diabetic patients’ tissue may cause chronic infection and ulcer formation. Ulceration is worsening by ischemia due to diabetic atherosclerosis, infection or peripheral neuritis. The sensation is reduced in chronic cases. Toes and feet are normally affected.

10) **Miscellaneous ulcers**: Ulceration mostly seen in anemia, leukemia, polycythemia, systemic sclerosis, Rheumatoid arthritis, ulcerative colitis, poliomyelitis, arteriovenous fistula, various collagen disorders, chronic lymph edema, and cortisone ulcers etc.

(B) **Specific ulcers**: These are seen in T.B, syphilis, soft chancre, leprosy, Actinomycosis and Meleny’s ulcer

1) **Tuberculous ulcer**: Seen in bursting of cold abscess; may form tuberculosis lymphnode, TB of bone or joint. It is oval generally with irregular crescent border, often multiple in number.

2) **Syphilitic ulcer**: Ulcers due to syphilis are seen in all 3 stages. Single, painless, indurated base, oval to round and deep saucer shape.

3) **Soft chancre**: Mostly seen on genital part. These are often painful, gradually become pustular and ulcerate to form soft sores. These are multiple, soft, rounded painful, and readily bleed and edges are undermined.

4) **Actinomycosis**: This condition causes multiple ulcers. At first area becomes indurated, nodules appear, which soften and later ulcerates in various places. Surrounding skin often looks bluish in colour. Discharges yellow colour sulphur granules.

5) **Leprosy ulcer**: Mainly seen in the extremities of foot and hand. It is due to peripheral neuropathy and pressure induced parenchyma

6) **Meleny’s ulcer**: These are in post-operative wounds commonly seen over abdomen, thorax, etc., it is very painful with signs of toxemia. It has undermined edges, foul smelling, granulation tissue with seropurulent discharge.

C) **Malignant Ulcer**: Rodent ulcer/Basal cell carcinoma:

It is locally invasive carcinoma of basal layer of epidermis. It is of low grade malignancy. It is commonly seen on the face, above line from corner of mouth to ear, inner canthus of eye, nose on and around nasolabial fold, has risen and pearly white
beded edge, irregular in shape and floor covered with coat of dried serum, epithelial cells.

i) Epithelioma (squamous cell carcinoma): This occurs commonly, in the dorsum of hands, in the face, limbs, lips, vulva, penis etc. It has normal temperature and usually not tender, oval or circular in shape with raised and everted edge, indurated base and floor is covered by necrotic tumor, serum & blood.

ii) Marjolins ulcer: It is the name given to a squamous cell carcinoma which arises in a chronic benign ulcer or scar. It is slow growing malignant lesion, painless and edge is not always raised and everted.

11. Classification of wound: 181,182

Wounds can be classified into 5 types

1). Incised wounds- Caused by sharp objects, edges of the wound are sharp. Tends to gape and bleed freely.

2). Lacerated wounds- Caused by blunt objects, edges of the wound are jagged. Causes minimal bleeding because of crushing.

3). Penetrating wounds- (variation of punctured wound) - Stab injuries of abdomen are notorious, depth is more.

4). Crushed or contused wounds- Caused by blunt trauma.

5). Abrasion- Caused by scraping away of superficial skin layer and is very painful.

12. Causes of ulcer: 183

1) Vascular: venous, arterial, lymphatic, vasculitis

2) Neuropathic: diabetes, spina bifida, leprosy

3) Metabolic: diabetes, gout

4) Connective tissue disease: Rheumatoid arthritis, Scleroderma, Systemic lupus erythematosus, Pyoderma gangrenosum (often reflection of systemic disorder)

5) Haematological disease: red blood cell disorders; sickle cell disease, white blood cell disorders: Leukaemia, platelet disorders: Thrombocytosis

6) Dysproteinaemias: cryoglobulinaemia, amyloidosis

7) Immunodeficiency: HIV, immunosuppressive therapy

8) Neoplastic: basal cell carcinoma, squamous cell carcinoma, metastatic disease

9) Infectious: bacterial, fungal, viral.

10) Traumatic: pressure ulcer, radiation damage.

11) Iatrogenic: drugs induced.
13. Factors Influencing Ulcer Healing:

A) Local factors: ¹⁸³

- Inadequate blood supply
- Increased skin tension
- Poor surgical apposition
- Wound dehiscence
- Poor venous drainage
- Presence of foreign body and foreign body reactions
- Continued presence of microorganisms
- Infection
- Excess local mobility, such as over a joint

B) Systemic factors: ¹⁸³

- Advancing age and general immobility
- Obesity
- Smoking, Alcohol, Tobaco chiwing
- Malnutrition
- Deficiency of vitamins and trace elements
- Systemic malignancy and terminal illness
- Chemotherapy and radiotherapy
- Immunosuppressant drugs,
- corticosteroids, anticoagulants
- Connective tissue disorders
- Impaired macrophage activity
- (Malacoplakia)

14. Forms of Ulcer Healing: ¹⁸⁴

I) Healing By 1<sup>st</sup> Intension:
In simple and non-infective ulcer of healthy person healing by minimum scar formation.

II) Healing By 2<sup>nd</sup> Intension:
In marked tissue destruction ulcer can heal without edges approximation by any surgical procedure.
III) Healing By 3rd Intension:
Frist wound can heal naturally, when the ulcer is free of infection then it is surgically closed.

15. Abnormal wound healing: 183

- Sinus and Fistula formation
- Malignancy
- Osteomyelitis
- Contractures and deformity in surrounding joints
- Systemic amyloidosis
- Heterotopic calcification
- Colonization by multiple drug
- Resistant pathogens, leading to antibiotic resistance
- Anemia
- Septicemia
- Excess scar and Keloid formation due to excess deposition of extracellular matrix at ulcer site.
- Deficient Scar Formation: found I ulcer in which the granulation tissue is inadequate
- Dehiscence of ulcer Herniation of wound: Seen due to increased pressure within ulcer. Mostly seen in abdominal ulcer.
- Ulceration: Due to inadequate blood supply and perfusion as in Varicose ulcer, Atherosclerosis, Neuropathic ulcers

16. Laboratory investigations before treating a wound: 183

- Hemoglobin- Anemia may delay healing
- White cell count- Infection
- Platelet count- Thrombocytopenia
- Erythrocyte sedimentation rate (E.S.R.) and C-reactive protein - Non-specific markers of infection and inflammation; useful in diagnosis and monitoring treatment of infectious or inflammatory ulceration.
- Urea and creatinine–High urea impairs wound healing. Renal function important when using antibiotics.
- Albumin Protein loss- delays healing
- **Glucose, hemoglobin A1C**- Diabetes mellitus
- **Markers of autoimmune disease**- Such as rheumatoid factor, antinuclear antibodies, anticardiolipin antibodies, lupus anticoagulant. Indicative of rheumatoid disease, SLE and other connective tissue disorders. Cryoglobulins, cryofibrinogens, prothrombin time, partial thromboplastin time - Haematological diseases.
- **Deficiency or defect of**: Antithrombin III, protein C, protein S, factor V Leiden Vascular Thrombosis.
- **Haemoglobinopathy screen** Sickle cell anemia, Thalassemia
- **HIV status**- Kaposi’s sarcoma
- **Serum protein electrophoresis**- Bence-Jones proteins Myeloma
- **Urine analysis** - Protein loss, Urinary tract infection, connective tissue disease
- **Kidney function test**: BUN, serum Creatinine, Serum Electrolytes
- **Liver function test**: Liver cirrhosis, Jaundice, Liver parenchymal disease.
- **Wound swab**: culture and sensitivity of infective organism.

17. **Non-healing Ulcers**: 185-188

A wound or ulcers which does not reduce in area (simple length x width) by at least 50% in 4 weeks, has greater than 90% likelihood of non-healing at 12 weeks, should an ulcer progressing too slowly, it is appropriate to alter the modality to restart healing.

**Non-healing Ulcers**: have traditionally been defined as those that fail to progress through an orderly sequence of repair in a timely fashion. Such wounds are sometimes thought of as being caused by neglect, incompetence, misdiagnosis, or inappropriate treatment strategies. However, some wounds are resistant to all efforts of treatment aimed at healing, and alternative end points should be considered; measures aimed at improving the quality of life will be paramount in these instances.

**A chronic ulcer** is defined as a wound that does not heal within an expected time frame (i.e. 6 weeks) despite optimal correction of any underlying pathological processes interfering with the body’s normal process of wound healing. Most of these wounds fall into three types: Venous ulcers; Pressure ulcers; and Diabetic ulcers
18. Clinical features of Non-Healing Ulcer: 189

- Absence of healthy granulation tissue
- Presence of necrotic and unhealthy tissue in the wound bed
- Excess exudate and slough
- Lack of adequate blood supply
- Failure of re-epithelialisation
- Cyclical or persistent pain
- Recurrent breakdown of wound
- Clinical or subclinical infection

19. Prevalence of Chronic Ulcers: 190-194

The study from India shows that etiology of chronic ulcers included systemic conditions such as diabetes, atherosclerosis, tuberculosis, and leprosy. Other major causes included venous ulcers, pressure ulcers, vasculitis, and trauma. The study report stated that inappropriate treatment of acute traumatic wounds was the most common cause of the chronic ulcer.190 Chinese study shows that the principle etiology (67%) of ulceration is trauma or traumatic wounds compounded by infection. Diabetic ulcers, venous ulcers, and pressure ulcers accounted for 4.9%, 6.5%, and 9.2%, respectively. Most of these wounds were seen in farmers and other agricultural workers.191-192

It has been reported that ulcers related to venous insufficiency constitute 70%, arterial disease 10%, and ulcers of mixed etiology 15% of leg ulcer presentations.193 The remaining 5% of leg ulcers result from less common pathophysiological causes, and this latter group comprise considerable challenges in diagnosis, assessment, and management.194

One of major cause of non-healing ulcers is Diabetes. For the treatment of chronic Diabetic ulcer, one should know the pathophysiological understanding very well.

20. Pathophysiology of Diabetic Ulcer: 195

The major contributing factors in Pathophysiology are Prolonged Hyperglycemia, Neuropathy, Ischemia, and Infection.
1. Diabetic Neuropathy:

The changes of Neuropathy in Diabetes are developing in Sensory, Motor and Autonomic nervous system. The prolonged Hyperglycemia increases enzyme action of Aldose Reductase and Sorbitol Dehydrogenase. These enzymes convert intracellular glucose to Sorbitol and Fructose. The accumulation of these sugar molecules in nerve result in decrease synthesis of Myonositol in nerve this enzyme is essential in normal nerve conduction signals. The enzymatic and metabolic changes in Diabetes get accumulate in peripheral nerve which reduces sensations.

In sensory neuropathy, there is loss of sensations of sole and peripheral skin. This result in repeated trauma. In Motor Neuropathy, the muscles of feet are affected more. This result in reduce control on pedal muscles. The unequal distribution of force while walking result in pressure point in feet. The pressure point skin is thickened and results in formation of Callus on feet. In Autonomic Neuropathy, there is loss of control on peripheral blood vessels. This result in Ischemic changes in local circulation. The Autonomic Neuropathy also results in dryness of skin and hair fall. The ultimate effect of this result in fissuring of skin. The thick callus formation in feet acts as a foreign body for feet. The pressure created by callus causes brushing and extravasation of blood and serum from micro capillary. The dry skin and fissuring skin creates readymade culture media for bacteria result in ulcer formation.

2) Vasculopathy:

The chronic Diabetes affects small as well as large blood vessels.

A) Microangiopathy: The occlusion of Arterioles and Capillaries result in patchy ischemic changes which result in gangrene formation. The acid Schiff positive causes thickening of small vessels. Also, the muscular control of vessels is losses due to Autonomic Neuropathy.

B) Macroangiopathy: Formation of Atheroma plaques in vessel wall is predisposing for thrombus formation.

C) Monckeberg’s sclerosis: The thickening of blood vessel due to calcification of muscular coat of arteries.
D) **Intimal fibrosis:** This type of thickening of arterial vessel is due to normal aging phenomenon.

3) **Infection:**

In chronic Diabetes patient, the prolonged Hyperglycemic media is available at fissuring or crack site. The defense mechanism in chronic cases is also low. This form the nedus place for bacteria to grow. The underlying abces and cellulitis also causes poor glycemic control of body. This creates viscous cycle of hyperglycemia and infection. Infection are due to surface organisms like Staphylococcus, Streptococcus. Other organism are aerobic gram positive and gram negative like Ecoli, Klebsella, and Proteusecetra.

21. **Ulcer Assessment guidelines:**

1. **History:** The chronicity of Diabetes and poor control of Diabetes result in complication of Diabetes like Neuropathy, Vasculopathy, Nephropathy, Retinopathy etc. The history of Hypertension, habits like Alcohol, tobacco, Smoking, Obesity is also important in Vasculopathy and defense mechanism. Family history is also important in disease prognosis. Occupation history is important for pressure distribution on ulcer and Glycemic control.

2. **Occupation:** Pressure phenomenon, work culture-Venous ulcer in long standing people, Neuropathy in drivers etc.

3. **Ulcer examination guidelines:**

A. **General examination:**

- The Nutrition, Gait, Psychological, Edema, Anemia, pigmentation
- Systemic examination: CVS, RS, PA, CNS
- Feet Examination: Shape, Size, Deformity, Thickening, Callosity

B. **Ulcer examination:**

- Shape-Oval, Circular, Irregular (Arterial, Venous, Diabetic, Pressure, Other)
- Size-Length, Depth, Breadth of Fistula.
- Skin-Healthy, Dry, Ischemic, Pigmentation, Maceration, Nails.
- Location-Medial malleolus- Venous ulcer,
- Lateral malleolus-Arterial ulcer,
- Plantar surface- Diabetic ulcer,
- Sacrum-Pressure ulcer.
- Edges: Slopping-Venous, Punched-Arterial, Rolled-Basel cell Carcinoma, Everted-Squamous cell Carcinoma, Purple-Vasculitis, Undermining-Tuberculosis, Syphilis ulcer.
- Bed-Necrotic, Slough, Black
- Secretion: Serous, Pus, Hemosangio
- Granulation: Pink Colour-Healthy Granulation,
- Red Ischemic-Unhealthy infected,
- Black-Necrotic tissue, absent,
- Over granulation-Non-healing tendency
- Odour: Mild, Moderate, Sever, Foul
- Vascular Assessment:
  1) Pallor
  2) Ischemic Changes
  3) Peripheral pulse
  4) Capillary refilling time.
- Neurological Assessment:
  2. Motor Assessment: A) leg deformity- Claw, Charcot
     B) Ulcer pressure points
     C) Tendon reflux
     D) Muscle power.
  3. Autonomic Assessment-Dryskin, hairloss, hyperpigmentation, local temperature.
- Infection: Signs of Inflammation, Fever, pain, redness, edema etc. pus discharge
- Osteomyelitis: X-ray examination.
TABLE. No. 20: Table of ulcers signs and symptoms with their differential diagnosis

<table>
<thead>
<tr>
<th>Signs ↓</th>
<th>Type →</th>
<th>Arterial</th>
<th>Venous</th>
<th>Diabetic</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site</td>
<td></td>
<td>Lateral malleolus</td>
<td>Medial malleolus</td>
<td>Planter area</td>
<td>Variable</td>
</tr>
<tr>
<td>Size</td>
<td></td>
<td>Small</td>
<td>Small-Large</td>
<td>Small-Large</td>
<td>Variable</td>
</tr>
<tr>
<td>Skin</td>
<td></td>
<td>Pale</td>
<td>Variable</td>
<td>Necrotic</td>
<td>Variable</td>
</tr>
<tr>
<td>Bed</td>
<td></td>
<td>Pale</td>
<td>Variable</td>
<td>Necrotic</td>
<td>Variable</td>
</tr>
<tr>
<td>Shape</td>
<td></td>
<td>Round</td>
<td>Irregular</td>
<td>Round</td>
<td>Round</td>
</tr>
<tr>
<td>Edge</td>
<td></td>
<td>Smooth</td>
<td>Irregular</td>
<td>Smooth</td>
<td>Variable</td>
</tr>
<tr>
<td>Oder</td>
<td></td>
<td>No</td>
<td>?</td>
<td>++</td>
<td>Few</td>
</tr>
<tr>
<td>Pain</td>
<td></td>
<td>+</td>
<td>?</td>
<td>?</td>
<td>+</td>
</tr>
</tbody>
</table>

22. Diabetic Ulcer Grades: Diabetic Ulcer Grades Classification According to Wagner’s & Armstrong University of Texas:

Table No.21: Diabetic ulcer grades classification

<table>
<thead>
<tr>
<th>Grades</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>A)</td>
<td>Callus or Scar</td>
<td>Superficial Infection</td>
<td>Wound up to Tendon/Capsule</td>
<td>Penetrating to Bone /Joint</td>
<td>Necrotic foot</td>
<td>Entire foot Necrosis</td>
</tr>
<tr>
<td>B)</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
</tr>
<tr>
<td>C)</td>
<td>Infection</td>
<td>Infection</td>
<td>Infection</td>
<td>Infection</td>
<td>Infection</td>
<td>Infection</td>
</tr>
<tr>
<td>D)</td>
<td>Ischemia + Infection</td>
<td>Ischemia + Infection</td>
<td>Ischemia + Infection</td>
<td>Ischemia + Infection</td>
<td>Ischemia + Infection</td>
<td>Ischemia + Infection</td>
</tr>
</tbody>
</table>

23. Management of Diabetic Ulcers: The aim of any ulcer treatment is to assure quick healing with durability form of healing and with minimal scar formation.
A) General medical and surgical treatment aspects:

- Careful inspection
- Vascular assessment
- Neurological assessment
- Describes size, shape, edges, surrounding skin, pain, discharge, ulcerbed, and granulation tissue.
- Routine investigation and special investigations.
- Internal Antibiotics, Analgesics, Medical management of Diabetes, and associated disease.
- Multivitamins, proper Nutrition
- Rest with offloading techniques, use of walker.
- Surgical debridement as per requirement, restore artery supply, vascular reconstruction, drainage, Amputation as per requirement.
- Daily dressing, regular assessment
- Proper footwear.

B) Local management:\textsuperscript{201,202}

1. Drainage: The proper drainage of pus, debries, and gases from hidden pockets by probing and debridement techniques is called drainage.

2. Debridement: The process of removal of dead necrotic tissue or foreign material from and around a wound to expose healthy tissue is called debridement.

A. Types of debridement are:\textsuperscript{201,202}

1. Surgical debridement: done in wounds with a large amount of necrotic tissue.

2. Mechanical debridement: this technique has been used for decades in wound care. By allowing a dressing to proceed from moist to wet, and manually removing the dressing, is a form of non-selective debridement. It is done in wounds with moderate amount of necrotic debris, hydro therapy, saline irrigation and saline moistened gauze comes under this.
3. Debriment Devices:  

a) Vacuum assisted closure (VAC): Intermittent negative pressure at 125mm of Hg at ulcer surface promotes the healing process. By improving blood supply, growth factor, nutrients flow.

b) Radiant heat bandages: The heat improves local perfusion, subcutaneous oxygen tension, which enhances healing process.

c) Tropical hyperbaric oxygen therapy: By achieving atmospheric pressure of 1.02 to 1.03 atm at ulcer site stimulates collagen formation, fibroblast growth. Hyperbaric oxygen is lethal to anaerobes. The hyperbaric oxygen is administered by using sealed polythene bag over ulcer area. The 100% oxygen is delivered at ulcer area with pressure of 20 to 30 mm of Hg.

d) Whirlpool hydrotherapy: Water under high pressure is used to debride ulcer area.

e) Autolytic: It uses the body’s own enzymes and moisture to rehydrate, soften and liquefy the slough. It is achieved with hydro colloids, hydrogels, and transparent films.

4) Chemical debridement: These are chemical desloughing agents.

A) Hydrogen peroxide (H₂O₂): it releases nascent oxygen, allows aerobic bacteria to grow they eat away the slough. Usually it is mixed with acriflavine. Acriflavine is a mild antiseptic and irritant it promotes the growth of granulation tissue.

B) Eusol: Edinbergh University Solution. It is a sodium hypochlorite (NaOCl). It acts by releasing nascent chlorine, which combines with slough to form water soluble complexes. In dilute concentrations it kills fibroblasts, neutrophils and endothelial cells in tissue culture.

Eusol delays the appearance of Hydroxyproline (the amino acid marker of wound collagen content) and prolongs the acute inflammatory response. It has no role in the treatment of open wounds that are clean and healing well with no signs of invasive infection.

C) Povidone iodine: Strong bactericidal for gram positive and negative organisms (it has a broad spectrum of activity but its anti-bacterial effect is reduced by contact with pus or exudate). It should not be used in patients who are sensitive to iodine.

D) Chlorhexidine: It is the topical antiseptic which is effective against a wide range of gram positive and negative organisms and some fungi.
5) **Biological**: maggot debridement therapy is an old remedy that has been revised and proven to be invaluable in cleansing non-healing wounds. This simple procedure involves the placement using restrictive dressing of live disinfected maggots into non-healing wounds to promote for cleansing of necrotic tissue and initiation of the healing process.

6) **Topical agents**:

A wide variety of topical wound cleaning agents being available and bacteriostatic agents being promoted for local wound application. Some of them are:
- Povidine iodine 2.5%-It is Bactericidal
- Chlorhexidine solution: Bactericidal
- Bactracin: Antibacterial
- Hydrogen peroxide (H2O2)
- Dankin’s solution (Chlorazene) 0.25%Bacteriocidal
- Sucralfate: Its antimicrobial activity is by its macrophage activity. It prevents the release of cytokines from damaged skin cells there by exerting anti-inflammatory and smoothening effect.
  - PDGF: Helps to rapidly heal chronic non-healing Ulcers. PDGF derived from patients own blood. (Platelet derived growth factor i.e.PGDF)
  - Collagen dressings: The Collagen is obtained from bovine extract. Available in the form of gel granules and incorporated with alginate dressing. Collagen provides additional proteins for healing.
  - Hyaluronic acid: Enhances the structural organization of extracellular matrix which increases meiotic activity.
  - Oxandrolone: This is anabolic steroid, it is anticatabolic and protein sparing properties which enhances protein synthesis.
  - Silver Arglaes: Powerful antibacterial agent like Silver Nitrate, Silver Oxide, Silver Chloride.
  - Growth factor: Use of Becapteargin, Vecombinant platelet derived growth factor.
  - Human skin: By tissue culture technique as a derma graft.
C) **Dressing:**

**Dressing:** Covering the ulcer area with various materials

Feeling dead spaces with medicated gauze to avoid anaerobic organism growth.

Applying the moisture and medicated solution and packing the Ulcer.

Providing proper cushioning and covering ulcer area.

Applying bandaging and sticking for wrapping.

Applying cast or splint to protect and support the ulcer.

**Dressing materials:**

1) Regular dressing: Cotton and cotton fibers gauze.

2) Conventional dressing: The regular dressing material allows evaporating moisture and entry of microorganisms. Also, these materials have tendency to bind ulcer tissue which causes pain and damage. This lead to develop composite dressing materials like Tule grass fibers, these materials incorporated with Vaseline, antibacterial agent.

3) Synthetic dressing: Films these are polymer sheet coated adhesive on one side. Used in superficial wound.

4) Foam and sprays: These dressing sheets of foamed solution of polymer such as polyvinyl alcohol. These materials have advantage of thermal insulation and thus maintain moisture in ulcer surface. Further they are gas permeable, nonadherent, light and comfortable.
3.4 Review of Diabetes Mellitus:

1. Definition:
Diabetes Mellitus is a clinical syndrome, due to impaired metabolism and characterized by chronic hyperglycemia. There is a greater or lesser impairment in the metabolism of carbohydrates, lipids and proteins.\(^{207}\) Hyperglycemia is due to deficiency in insulin secretion, or decreased glucose utilization and increased glucose production.\(^{208}\) The term diabetes was originally introduced to describe the clinical symptom associated with high glucose levels.

2. Prevalence
The prevalence of Diabetes is continued increasing dramatically. It is undoubtedly one of the most challenging health problems in the 21\textsuperscript{st} Century. According the latest 2016 data from WHO, globally nearly about 422 million adults are living with Diabetes.\(^{209}\) The global prevalence of diabetes has nearly doubled since 1980, rising from 4.7\% to 8.5\% in the adult population.\(^{210}\) The International Diabetic Federation estimates in 2013 that 381 million peoples having Diabetes.\(^{211}\) The number is projected to almost double by 2030.\(^{212}\) Diabetes occurs through the world, but it is most common (type 2) in more developed countries. The increase in diabetes in developing countries is due to the trend of urbanization and life style changes, increasing sedentary lifestyle, less physically demanding work and the global nutrition transition.

The number of people with type 2DM is increasing in every country. Almost half of deaths attributable to high blood glucose occur before the age of 70 years.\(^{213}\) WHO reports that diabetes will be the 7\textsuperscript{th} leading cause of death in 2030.\(^{214}\) India have 69.2 million people living with Diabetes (8.7\%) as per the data.\(^{215}\) India is projected to be capital to 109 million individuals with diabetes by 2035.\(^{216}\) According to International diabetic Federation, India had more diabetic than any other country in the World. 80\% of people with DM living in low and middle-income countries. Presently as many as 50\% of people with diabetes are undiagnosed. So, there is need to detect Diabetes early in its course. Considering the importance of the disease we must review some important features of the Diabetes Mellitus.
3. Types: There are three main types of diabetes mellitus

1) Type 1 Diabetes Mellitus (T1DM): Insulin dependent Diabetes Mellitus (IDDM) or “Juvenile diabetes”

2) Type 2 Diabetes Mellitus (T2DM): Non-insulin dependent Diabetes mellitus (NIDDM) or “Adult” Onset of diabetes is primarily due to lifestyle factors and genetics.

3) Gestational Diabetes (GD): Resembles type 2 diabetes, but occurs during pregnancy and may improve or disappear after delivery.


IDDM and NIDDM these types were proposed by WHO in 1980 and 1985 have disappeared.

The new classification system identifies four types diabetes mellitus; type 1, type 2, other specific and gestational. The etiological classification is as below.

| Table no. 22: Etiological Classification of Diabetes Mellitus. Adapted from WHO |
|-----------------------------------------------|-----------------|
| I. Typ1 Diabetes mellitus                     | 3. Nicotinicacid |
| A. Autoimmune                                 | 4. Glucocorticoids|
| B. Idiopathic                                 | 5. Thyroid hormones|
| II. Type2 Diabetes mellitus                   | 6. Diazoxide    |
| Ranges from relative insulin deficiency to    | 7. β-adrenergic agonists |
| disorders                                      | 8. Tiazides     |
| of insulin secretion and insulin resistance   | 9. Dilantin     |
| III. Other specific types of diabetes mellitus|
| A. Genetic defects in β-cell function         | 10. α interferon|
| 1. Chromosome 12, HNF-1α (MODY 3)             | ii. Infections  |
| 2. Chromosome 7, glycosidase (MODY 2)         | 1. Congenital rubeola |
| 3. Chromosome 20, HNF-4α (MODY 1)            | 2. Cytomegalovirus|
| 4. Mitochondrial DNA                          | iii. Infrequent forms of autoimmune diabetes |
| 5. Monogenic diabetes                         | 1. Stiff-man syndrome) |
| B. Genetic defects in insulin action          | 2. Antibodies against insulin receptors |
| 1. Type A insulin resistance                  | iv. Other syndromes occasionally |
| 2. Leprechaunism                              |                  |
3. Rabson-Mendenhall syndrome
4. Lipotrophic diabetes
C. Disease of the exocrine pancreas
1. Pancreatitis
2. Pancreatectomy/trauma
3. Neoplasia
4. Cystic fibrosis
5. Hemochromatosis
6. Fibrocalcific pancreatitis
D. Endocrinopathies
1. Acromegaly
2. Cushing syndrome
3. Glucagonoma
4. Pheochromocytoma
5. Hyperthyroidism
6. Somatostatinoma
7. Aldosteronoma
i. Pharmacologically or chemically induced
1. Vacor
2. Pentamidine

4. Rabson-Mendenhall Syndrome

5. Clinical Features of Diabetes Mellitus: 219

Most of the symptoms are similar in both types of diabetes but develop more rapidly in type 1 diabetes and more typical. These are

- 3P’s; polyuria (increased urination)
- Polydipsia (excessive thirst)
- Polyphagia (excessive appetite)
- Burning/tingling/pricking sensation in the hand or feet.
- Fatigue, feeling of tiredness, lethargy
- Blurred vision
- Pruritis vulvae, Balanitis (genital Candidiasis)
- Unexplained weight loss
- Nocturia

associated with diabetes
1. Down syndrome
2. Klinefelter syndrome
3. Turner syndrome
4. Wolfram syndrome
5. Friedreich ataxia
6. Huntington’s chorea
7. Lawrence-Moon-Biedel syndrome
8. Myotonic dystrophy
9. Porphyria
10. Prader-Willi syndrome

IV. Gestational diabetes mellitus

Occurs in mostly in women during gestation.
6. **Criteria for diagnosis.**

The 1997 ADA recommendations for diagnosis of DM focus on (FPG) Fasting Plasma Glucose, while WHO focuses on (OGTT) Oral Glucose Tolerance Test. Here, we are considering about FPG.

i) **Random Blood Sugar:**

Should be less than 200 mg/dl

Random is defined as any time of day without regard to time since last meal.

ii) **Fasting Blood Sugar:**

Should be less than 126 mg/dl.

Fasting is defined as no caloric intake per at least 8 hrs.

iii) **PPBS (2 hrs.) (Post prandial blood sugar):**

Should be less than 140 mg/dl

IN a patient with characteristic sign and symptoms of diabetes, a fasting venous plasma glucose >126 mg/dl, or a random venous plasma glucose >200 mg% confirmed on repeat testing. Diagnostic –ve test for diabetes does not mean that the person will never get DM.

7. **Investigations:**

- Blood glucose Tests:
- Fasting blood sugar (FBS)
- Post Parndial blood sugar (PPBS)
- Random blood sugar
- Oral glucose Tolerance tests(OGTT)
- Hb A1C (glycosylated haemoglobin)
- Urine tests-glucose, albumin. Ketones, etc.
- Insulin tests
- S. Insulin
- Insulin sensitivity test

**Other complimentary tests**

- Glycated serum protein (GSP)
- S. Fructosamine
- C. Peptide--It is a simple, cost effective, non-invasive method of assessment of beta cell capacity.
- Blood Urea
- Lipid profile
- E.C.G.
- HbA1C—it is a form of haemoglobin that is measured primarily to identify the three-month average plasma glucose concentration. A normal non-diabetic HbA1C is 3.5-5.5%. For non-diabetics, the usual reading is 4-5.9%.
  For people with diabetes, an HbA1C level of 6.5% is considered good control.

8. Etiology and Pathogenesis of DM

In both types of DM, environmental factor and genetic susceptibility is important.

A) DM I

Type 1 DM (IDDM) is a T-Cell mediated autoimmune disease. It involves destruction of the insulin-secreting β cells in the pancreatic islets and this process takes many years. Features of diabetes do not become evident until a majority of β cells (70-90%) are destroyed. This type is a result of complete or near total insulin deficiency.

When 70-90% β cells have been destructed, Hyperglycemia, with associated classical syndrome is occurs.

I) Pathology: 

Approximately 85% of patients have circulating islet cell antibodies, and the majorities also have detectable anti-insulin antibodies before receiving insulin therapy. Most islet cell antibodies are directed against glutamic acid decarboxylase (GAD) within pancreatic β cells.

Due to autoimmune destruction of pancreatic β-cells, deficiency of insulin secretion occurs. It results into metabolic changes related with T1DM. Due to reduced insulin secretion, the function of pancreatic α-cells is becoming abnormal and excessive secretion of glucagon occurs. Normally, glucagon secretion reduced by hyperglycemia but in type 1 DM, glucagon secretion is not suppressed by hyperglycemia. Elevated glucagon levels aggravates metabolic defects due to insulin deficiency. Insulin deficiency is the basic defect in T1DM, but a defect in the administration of insulin is also present. Deficiency in insulin leads to uncontrolled lipolysis and elevated levels of free fatty acids in the plasma. It suppresses glucose metabolism in peripheral tissues as skeletal muscle. This reduces glucose utilization. Also, insulin deficiency in T1DM causes impaired glucose, lipid and protein metabolism.
DM 1 is associated with other autoimmune disorders. Such as thyroid disease, Addison’s disease, pernicious anemia and vitiligo also.\(^{226}\)

**ii) Genetic Predisposition:** \(^{227}\)

Genetic factors affect about one–third of susceptibility of type 1 DM. HLA (Human –Lymphocytes Antigen); DR3 or DR4 are associated with increased susceptibility to Type, candidate gene and genome-wide association have implicated other genes in type 1 diabetes, e.g. CD25, IL2RA. The genes associated type’s diabetes overlap with autoimmune disorders.

**iii) Environmental Factors**

Environmental factors have an important role in promoting clinical expressions of the disease. Due to reduced exposure to micro organism in early childhood suppress maturation of immune system and increases susceptibility to autoimmune disease.

**iv) Viral:**

Viral infections in the pancreas affect function of β cells e.g. mumps, coxackie B4, rubella, etc.\(^{228}\) Stress may precipitate type 1DM by stimulating counter – regulatory hormones.\(^{229}\) Various nitrosamines, coffee and (BSA) Bovine serum albumin i.e. (a constitute of cow’s milk) have been proposed as dibetogenic.\(^{230}\) Thus, due to all these factors and due to β cells destruction adequate insulin secretion inhabits and normal glucose level cannot be maintained for long time. Eventually, all type 1 diabetic patients will require insulin therapy to maintain Normoglycemia.

**B) DM II**

**i) Pathology** \(^{231}\)

It is characterized by impaired insulin secretion, insulin resistance, excessive hepatic glucose production and abnormal fat metabolism.

Type 2 is more complex condition than type 1 DM, because there is a combination of two main pathological defects one is reduced insulin secretion through insulin resistance to the actions of insulin in liver and muscles together. Second is there is reduced insulin secretion due to impaired pancreatic β Cell functions. It causes insulin deficiency.\(^{232}\)

- **a) Insulin Resistance:**

Type 2 DM, often associated with other disorders, particularly central obesity, hypertension and dyslipidaemia (i.e. elevated level of LDL Cholesterol and decreased level of HDL cholesterol.) \(^{233}\)
Primary cause of insulin resistance is unclear but. Intra–abdominal ‘central’ adipose tissue releases large quantities of FFAs. These FFAs is known cause of peripheral insulin resistance and hepatic insulin resistance. Elevated plasma FFAS levels impairs insulin stimulated glucose uptake into muscles. Also, adipose tissue releases several hormones like adipokines which influence sensitivity to insulin in other tissues.\textsuperscript{234}

b) Pancreatic $\beta$ Cell failure:\textsuperscript{235}

In type 2 DM, FFAS and elevated plasma glucose creates toxic effects on pancreatic $\beta$ Cell, hence insulin secretion reduced.

Due to reduction in $\beta$ Cell nos. $\alpha$Cell mass is unchanged and glucagon secretion is increased; which may contribute to the hyperglycemia.

ii) Genetic Predisposition:\textsuperscript{236}

In monozygotic twin’s concordance rates for type 2 diabetes is 100%

iii) Environmental factors

DM II is associated with overeating especially combined with obesity. It is also common in the middle–aged and elderly people. Thus, this is the most common form of Diabetes mellitus. It is highly associated with a family history of diabetes, older age, obesity and lack of exercise and sedentary life style.\textsuperscript{237}

C) Other type:

MODY\textsuperscript{238} (maturity onset diabetes of the young):

It is defined as hyperglycemia diagnosed before the age of twenty–five years and treatable five years without insulin, in cases where islet cell antibodies (ICA) are negative.

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**Fig. No. 1 Flow chart of Pathology of Diabetes Mellitus II**
9. Complications of Diabetes Mellitus: 239

Complications of Diabetes mellitus fall into two major divisions i.e. Acute Complications & Chronic Complications. The complications resulting from the disease are associated with the damage or failure of various organs such as the eyes, kidneys & nerves.

**Acute Complications:**
- Hypoglycemia
- Diabetic Ketoacidosis (DKA)
- Non Ketoic hyperosmolar state

**Chronic Complications:**
- Macrovascular Complications:
  - Coronary artery disease.
  - Peripheral Vascular disease.
  - Cerebro vascular disease.
- Microvascular Complications:
  - Diabetic Eye disease
  - Retinopathy (non-proliferative/proliferative)
  - Macular edema
  - Glaucoma
  - Cataracts
  - Diabetic Neuropathy
  - Poly neuropathy /mono neuropathy
  - Autonomic neuropathy.
  - Diabetic Nephropathy
  - Other
  - Gastro intestinal [gastroparesis, diarrhoea]
  - Genito urinary [uropathy /sexual dysfunction]
  - Dermatologic infections.
  - Diabetic foot.

10. Treatment

The goals of therapy for type 1 or type 2 DM are
- Eliminate symptoms related to hyperglycemia.
• Reduce or eliminate long term microvascular complications of DM.
• Allow the patient to achieve as normal life style as possible. So, the management can be planned as under
• Education of patient about DM, nutrition and exercise.
• Monitoring the level of Glycemic control.
• Assessment of glycemic control
• Oral hypoglycemic agents.
• Insulin

1. Education of Patient: 240

Diabetes self management education is an important part of treatment. A variety of strategies and techniques should be used to provide adequate education and problem management, let us consider.

• Nutrition241

Medical Nutrition Therapy (MNT) is an integral component of DM.
Nutritional recommendation for Dm

• Carbohydrate:
Patient, especially type1 DM has been advised to maintain a regular intake of carbohydrates through the day. But by using insulin analogues or (CSII) continuous subcutaneous insulin injections, allowed greater flexibility in the timing and choice of carbohydrate intake.
People with type 2 diabetes, limitations of retimed carbohydrate and restriction of total caloric intake is important.

• Fat: The intake of total fat should be restricted to less than 35% of energy intake.

• Diabetic foods and sweeteners: Low –calorie and sugar–free drinks are useful for patients with diabetes.

• Weight Management:242
Patients having DMII are overweight or obese, and many anti-diabetic drugs including insulin encourage weight – gain. So, weight loss reduction can be achieved by reduction in energy intake and an increase in energy consumption through physical activity.

83
1. **Exercise:**
   Diabetic patient should advise to regular physical activity for approximately 30 minutes daily, as this improves insulin sensitivity and lipid profile and lowers blood pressure. Programs for the treatment & prevention of Diabetes mellitus. It reduces the risk of cardiovascular disease, hyperlipidemia, hypertension & obesity.

2. **Self Monitoring of Blood Glucose (SMBG)**
   It permits the well informed and motivated patient to make appropriate adjustments in treatment (i.e. particularly insulin dose). BG monitoring is most useful in patients having risk of hypoglycemia, during undercurrent illness and prescription of corticosteroids and during changes in therapy.

3. **Assessment of Glycemic Control:**
   Oral drugs are used to lower blood glucose level by achieving following goals.
   - Drugs that primarily stimulate insulin secretion.
   - Drugs that alter insulin action.
   - Drugs that principally affect absorption of glucose.
   Glycated HB or HbA1C should be measured in all individuals with DM. Postprandial and nocturnal hyperglycemia may not be detected by SMBG of fasting pp but will be reflected in HbA1C.

4. **Insulin Administration:**
   Insulin is obtained from pork pancreas or is made chemically identical to human insulin by recombinant DNA technology or chemical modification of pork insulin.
   - Type of insulin depending on action.
     - **Rapid – acting** - (Insulin analogues, lispro, aspart, glulisine)
     - **Short Acting – Soluble**
     - **Intermediate – Isophone (NPH) Lente**
     - **Long acting – (Bovine alternate)**
   - **Insulin dose regimen**
     The choice of regimen depends on the patient’s degree of glycemic control, the severity of underlying insulin deficiency, the patient’s lifestyle. Twice daily administration of a short-acting and intermediate acting insulin given in combination before breakfast and the evening meal, is the simplest
regimen and still used commonly. Hypoglycemia, weight gain, peripheral edema, insulin antibodies these are side effects of insulin therapy. The prevalence of diabetes is high at the population level. So, there is an economical, psychological, social burden on our healthcare system and the individuals living with the disease. The impact of diabetes is reaches in a wide state, it is essential to each country for implementation of preventive and curative measures. Lifestyle modification will undoubtedly play a key role in the ultimate solution to the problem of diabetes.
3.5 Review of Drug

I. Daruharidra *(Berberis aristata)*

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern Botany.

1. Vedakalin review:

   The World’s first ever literature is said to be Veda. Vedic era is from 6000 B.C. to 600 B.C. The Vedic literature is very vast. It contains the knowledge about various sciences. It is dispersed in Samhita, Brahman, Upanishad and Vedanga. Samhita have many branches and each has its own characteristics. Daruharidra is mentioned in Vedic literature with various names as follows-

   1. **Dharuharidra**: In Keshav Paddhati 3/10 Kwatha of Daruharidra and Haridra is used for Avasinchanarth in the management of Khalitya.
   2. **Putudaru-Putadru**: as a Devdar according to Mujumdar. According to Acharya P.V. Sharma Daruharidra which is used as a Samindha in yagya. In ShathpathBramanyak it is mentioned as aromatic and inflammable. Siddha Ghruta for Anjjan and Abhyangarth.

2. Samhitakalin Review:

   I. **Charak Samhita**:  
   - It is included in Aragwadhadi Varga. (Ch.Su.3/10)  
   - It is constituent of Pittaj Pramehahar Churna. (Ch. Vi.6/32)  
   - It is constituent of Rakttapittanashakyog Churna.  
   - It is constituent of Dahashamakchurna in Madatyayachikitsa. (Ch.Chi 4/73)  
   - It is constituent of Svarnakar lepa in Vrana Chikitsa. (Ch.Chi 25/116)  
   - It is constituent of Dahashamak lepa in Aragvadhadi Adhyaya. (Ch.Su.3/26)  
   - It is constituent of Mustadichurna in Kushta Chikitsa. (Ch.Chi 7/65)  
   - It is constituent of Vatpittajanya Shothahar Churna.  
   - It is constituent of Shothahartaila in Shotha Chikitsa. (Ch.Chi 12/25)  
   - It is constituent of Anjjan used inVisha Chikitsa. (Ch.Chi 23/69)  
   - It is constituent of Amruta Ghruta in Visha Chikitsa. (Ch.Chi 24/5)  
   - It is constituent of Phalatrikadi Kwatha in Prameha Chikitsa. (Ch.Chi 6/40)  
   - It is constituent of Kushthanashak Lepa in Kushatha Chikitsa. (Ch.Chi7 /84)  
   - It is constituent of DarviGhruta Kushatha Chikitsa. (Ch.Chi 7/135)  
   - It is constituent of VranaShodhak Kashaya in Vrana Chikitsa. (Ch.Chi 25/84)
- It is constituent of VranaRopak Taila in Vrana Chikitsa. (Ch.Chi 25/93)
- It is constituent of Visarpa Avchurnan in Visarpa Vrana Chikitsa. (Ch.Chi 20/94)

II. Sushrut Samhita: 

- It is constituent of Siddha Ghruta in Kusht harog Chikitsa. (Su.Chi.9/31)
- It is constituent of Lepa in Kshudra roga Chikitsa. (Su.Chi.9/35)
- It is constituent of Abandhya Vrana Rasakriya Dwivraniya roga Chikitsa.
- It is constituent of Siddhataila in Sadya Vrana roga Chikitsa. (Su.Chi.1/73)
- It is constituent of Nadi Vrana Shodhanarth kafka in Bhagandarroga Chikitsa. (Su.Chi.2/75)
- It is constituent of Vajrak tile in Nadirog Chikitsa.
- Lepa in Kshudraroga Chikitsa (Su.Chi.9/55)
- It is constituent of siddha taila in Maha Kushtaroga Chikitsa. (Su.Chi.10/15)
- It is constituent of Vajraktaile in Maha Kushtaroga Chikitsa. (Su.Chi.10/16)
- It is constituent of Lavanmeha sevanarth dravya. (Su.Chi.11/9)
- It is constituent of Vajraktaile in Maha Kusht haroga Chikitsa. (Su.Chi.9/57)
- It is constituent of RopantaIa in SadyaVrana roga Chikitsa. (Su.Chi.2/75)
- It is constituent of Siddha Ghruta Jwararoga Chikitsa (Su.Ut.39/227)

III) Ashtanga Hriday: 

- It is constituent of Avachurnan in VisarpaRogaChikitsa. (A.Hr.Chi.19/50)
- It is constituent of Chaurna in Shotharoga Chikitsa.
- It is constituent of Kashya in Shotharoga Chikitsa. (A.Hr.Chi.17/2, 32)
- It is constituent of Churna in KushtharogaChikitsa. (A.Hr.Chi.19/50)
- It is constituent of Bala Taila in VatarogaChikitsa.
- It is constituent of Kashya in Balarog Sarvadoshhara Churna. (A.Hr.Chi. 21/75)
- It is constituent of Bhutrao Ghruta in Bhootpratisshedroga Chikitsa. (A.Hr.Ut. 5/19)
- It is constituent of Siddha kafka pan in Granthi Arbud Nadi Vrana roga Chikitsa. (A.Hr.Ut.30/32)
It is constituent of Lepa in Ajgallika Vrana in Kshudraroga Chikitsa. (A.Hr.Ut.32/2)
It is constituent of Churna in Kaphajmeharoga Chikitsa. (A.Hr. Chi121/6, 7)
It is constituent of Kashya in Shotharoga Chikitsa. (A.Hr. Chi 17/32)
It is constituent of Kashaya Churna in Kushtharoga Chikitsa. (A.Hr. Chi19/37)
It is constituent of Kawal Churna in Mukharoga Chikitsa. (A.Hr.Ut.22/56)
It is constituent of Siddha Ghruta in Vranaroga Chikitsa. (A.Hr.Ut.25/67)

IV.Nighantu Kaal: 254

It is mentioned in Haritkyadi Varga of Ashtanga Nighantu.
It is mentioned in Guduchyadi Varga of Dhanvantari Nighantu.
It is mentioned in Guduchyadi Varga of Shodhal Nighantu.
It is mentioned in Abhayadi Varga of Madanpal Nighantu.
It is mentioned in Aushadhi Varga of Raj Nighantu.
It is mentioned in Mishraparakaran Haritkyadi Varga of Bhavprkash Nighantu.
It is mentioned in Daruharidradi Varga of Nighantu Adarsha.

3) Morphology of Daruharidra: 255

Daruharidra consists of dried stem of Berberis aristata DC. (Fam. Berberidaceae); an erect, spinous, deciduous shrub, usually 1.8-3.6 m in height found in Himalayan ranges at an elevation of 1000-3000 m, and in the Nilgiri hills in South India.

a) Macroscopic structure:

Drug available in spices of variable length and thicken. Bark is about 0.4 - 0.8 cm thick, pale yellowish-brown, soft, closely and rather deeply furrowed, rough and brittle.

Xylem portion is yellow, more or less hard, and radiate with xylem rays, pith mostly absent, when present it appears small. Yellowish-brown when dried, fracture short in bark region, splintery in xylem; taste, bitter.

b) Microscopic structure:

Stem- Shows rhytidoma with cork consisting of 3-45 rectangular and squares, yellow coloured, thin-walled cells, arranged radially, sieve elements irregular in shape. Thin walled, a few cells containing yellowish-brown contents. Phloem fibers arranged in Tangential rows, consisting of 1-4 cells. Each fiber short thick-walled,
spindle-shaped, lignified having wide lumen; half inner portion of rhytidoma traversed by secondary phloem rays. Phloem rays run obliquely consisting of radially elongated parenchymatous cells. Almost all phloem ray cells having single prismatic crystals of calcium oxalate, a few cells of rhytidoma also contain prismatic crystals of calcium oxalate. Stone cells also found cattered in phloem ray cells in groups, rarely single, mostly elongated. A few rounded, arranged radially, some of which contain a single prism of calcium oxalate crystals. Secondary phloem is a broad zone, consisting of sieve elements and phloem fibers, traversed by multi seriate phloem rays. Sieve elements arranged in tangential bands and tangentially compressed cells alternating with single to five rows of phloem fibers, phloem fibers short, lignified, thick-walled having pointed ends. Secondary xylem consisting of xylem vessels, tracheid’s, xylem fibers and traversed by multi seriate xylem rays; xylem vessels numerous, small to medium sized, distributed throughout xylem region in groups or in singles, groups of vessels usually arranged radially; isolated vessels cylindrical with rounded or projected at one or both ends with spiral thickening; xylem fibers numerous, lignified, large, thick-walled with wide lumen, and pointed tips; xylem rays quite distinct, straight, multiseriate, consisting of radially arranged rectangular cells, each ray 30-53 cells high, 8-12 cells wide, a few ray cells containing brown content.

C) Powder:

Colour—Yellow; shows mostly fragments of cork cells, sieve elements, yellow colored phloem fibers entire or in pieces, stone cells in singles or in groups, numerous prismatic crystals of calcium oxalate, xylem vessels having spiral thickening, thick-walled, lignified xylem fibers and ray cells.

D) Identity, Purity And Strength:

Foreign matter not more than 2 per cent, Appendix 2.2.2.
Total Ash not more than 14 per cent, Appendix 2.2.3.
Acid-insoluble ash not more than 5 per cent, Appendix 2.2.4.
Alcohol-soluble extractive not less than 6 per cent, Appendix 2.2.6.
Water-soluble extractive not less than 8 per cent, Appendix 2.2.7.

E) Part in use: Bark, fruit, root, stem, wood.
F) Chemical composition: 256
It contains various chemical components. Berberaine, Oxyberberaine, Berbamine, Aromoline, Karachine, Palmatie, Oxycanthine, Taxilamiene these are the main chemical compounds.

G) Kalpas of Daruharidra: 255
BhringarajTaila, Ashwagandharishta, Khadiradi Gulika, Khadirarishta, JatyadiTaila, Triphaladi Ghruta.

H) Properties and Action: 255
1. Rasa: Tikta
2. Vipaka: Katu
3. Virya: Ushna
4. Guna: Rukshya, Ushna

I) Therapeutic uses: 255
Kandu, Kushty-aroga, Medoroga, Mukharoga, Vrana, Shotha, Atisar, Urustambha, Kapharoga, Mukha, Karnaroga, Netraroga, Meha, Vishamjwara, Rakttapitta.

J) Dose – Kwath 5-10 ml, Churna 1-3 gm

4) Classification:
1) Classification in Samhita: 256

<table>
<thead>
<tr>
<th>CharakaSamhita</th>
<th>SushrutaSamhita</th>
<th>AshtangaHridaya</th>
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<tbody>
<tr>
<td>Lekhaniya Gana</td>
<td>Haridradi Gana</td>
<td>ShoroveerechaniyaGana</td>
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<td>Arshoghana Gana</td>
<td>Mustadi Gana</td>
<td>HaridradiVarga</td>
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<td>MustadiVarga</td>
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<td>Ropan Gana</td>
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<td>Nabhipakhar Varga</td>
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2) Classification According to Nighantu: 254

Table No.24: Classification of Daruharidra according to Nighantu.

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<thead>
<tr>
<th>Nighantu</th>
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<tr>
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<td>Bhavprakash Nighantu</td>
<td>Mishraprakaran Haritkyadi Varga</td>
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<tr>
<td>Nighantu Adarsha</td>
<td>Daruharidradi Varga</td>
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3) Taxonomical Classification: 257

Table No.25: Taxonomical Classification of Daruharidra

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<th>Kingdom</th>
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L) Paryayi Nama and Nirukati of Daruharidra: 254, 258, 259

- **Daruharidra**: Yellow colour bark and flowers.
- **Katankareri**: Spinous leaves.
- **Kantakini**: Spinous plant.
- **Kaleyak**: It removes doshas.
- **Kusumbhala**: It gives yellow colour dye.
- **Krumihara**: It has anti helmentic and antibiotic activity.
- **Darvi**: Its bark is important part.
- **Pachampacha**: Improves digestion and liver function.
- **Parjanya**: Its fruiting season is rainy season.
- **Parjani**: This prevents from diseases.
- **Pitadaru, Pitadru**: Its bark is in yellow colour.
- **Vishodhani**: Its action is as body purifier.
M) Parayayi Nama of Daruharidra: 254

Table No. 26: Parayayi Nama of Daruharidra according to Nighantu

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<thead>
<tr>
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</table>

N) Vernacular names: 255
- **Sanskrit**: Katamkateri, Darvi
- **Bengali**: Daruharidra
- **English**: Indian Berberry
- **Guajarati**: Daruharidra, Daruhuladur
- **Hindi**: Daruhaldi, Darhald
- Kashmiri: Kannadarishana, Maradarishina, Daruhaladi
- Malayalam: Maramannal, Maramanjal
- Marathi: Daruhalad
- Oriya: Daruharidra, Daruhalidi
- Punjabi: Sumalu
- Tamil: Gangeti, Varatiumanjal
- Telugu: Manupasupu
- Urdu: Darhald

O) GunakarmatmakaVivechana: 254

Table No.27: Gunapanchak of Druharidra according to Nighantu

<table>
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<tr>
<th>Properties</th>
<th>A.N.</th>
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Table No. 28: Dosha, karma and Rogaghanata of Daruharidra: 254

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<th>Activity</th>
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<td>Dosha karma</td>
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<td>Pittanuta,Ka phaanuta</td>
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<td>Pramukha Karma</td>
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<td>Urdhwajatr uroghara, Rujahar,Vis hodhini, Vishodhini,</td>
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<td>Varnya,Twak adosh-hhara</td>
</tr>
<tr>
<td>Rog-ghnata</td>
<td>Vrana,Meh a,Kandu,Kn umi,Pinas, Aruchi</td>
<td>Krumihar, Hemakanta</td>
<td>Netrya, karnya rogieta, AsyaRog ajeeta, Meh a, Kushta, Visha, Vrana</td>
<td>Vrana,Meha, Kandu,Veesa rpa,Twakdos ha,Visha,Karna, Akshiroga</td>
<td>Karnarog Netrarog</td>
<td>Netraroga,M ukharoga,Me ha,Pandu,Vra na,Shothha, Astra</td>
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</tbody>
</table>
P) Shloka references of Various Nighantu for Paryayinam, Gunapanchak, Dosh Karma and Rogghanta of Daruharidra:

1. अष्टाङ्गलिखण्डः–२०. हरिद्रादिगण
हरिद्रादिगणवक्षणरीशयामाचनिविर्णा

2.धन्वन्तरिनिघण्डः–१. गुनयादिगर्गदारुहरिद्रा
विशेषधन्तकृमिहरापीतसारुचिचिनाशिनी॥६०॥

3. शोदलिनिघण्डः–नामसंज्ञाहः (प्रथमभाग)– १. गुनयादिजर्गदारुहरिद्रा

4. मदनपालिनिघण्डः– १. अभयादिगर्गदारुहरिद्रा
5. राजनिघण्डु - ६. पिप्पल्यादिवर्गदास हरिद्रा
अन्यादास हरिद्रा च दार्वी पीठदु पीतिका
कालेयकंपीतादासियरागालाकामिली ||२०० ||
कट्टकटेरीपरज्ञापतादासिनशासमृता
कालीयकांकायतीजाठीपतचांभन्ना
स्यात्कर्नेकिनेरायारकाशदायरमा ||२०१ ||
तिकादास हरिद्रातुकदारणादमेंहली२
कण्डविशपत्त्यदाविश्वकर्मकण्डविश्वनुता ||२०२ ||
6. कैयदेवनिघण्डु - १. ओषधिवर्गदास हरिद्रा
कट्टकटेरी परज्ञा दार्वी दार्तिती निशा ||
पीठा दास हरिद्रा स्यात् पीठदु: पीठचन्दनम ||१९१६ ||
पचामपचा हेमकान्ति: पीठदार: कट्टकटी ||
तददद दार्वी विशेषण कर्णनेतास्थरोगजित ||१९१७ ||
7. भावप्रकाश-पूर्वधर-मिर्शकपकरण - २. हरीतक्यादिवर्गदास हरिद्रा
दार्वीहरिद्रा च परज्ञापरज्ञानीतिच
कट्टकटेरीपीठदुसहवेत्सैवपचामपचा ||१७५ ||
सैवकालीयक: प्रोकस्तथाकालेयकोपिच
पीठदु भहरिदु ८ पीठदासुचपीठकम ||१७६ ||
II) Nimba (*Azadirachta indica*)

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern Botany.

1) **Vedic review of Nimba:**

In Vedic literature Nimba is described. The Samindha of Nimba is not allowed in Yagya. It is used for Dantadhavan.


Pichumanda is describe in PainiyaGanapath: 3/10/24

2) **Samhitakalin Review:**

I. **CharakSamhita:**

- As a constituent of KhadiradiGhruta in KushthaRog Chikitsa. (Ch.Chi.7/152)
- As a constituent of MahatikkataGhruta in Krumi Chikitsa. (Ch.Chi.7/153)
- As a constituent of AragvadhadiAdhya for TwakRogChikitsa. (Ch.Su.3/38)
- As a constituent of VamakDravya In Apamargatanduliya Chapter. (Ch.Su.2/7)
- As a constituent of Churna in PittajPramehaRogChikitsa. (Ch.Chi.4/38)
- As a constituent of MustadiChurna in KushtaRogChikitsa. (Ch.Chi.7/157)
- As a constituent of VranadaChurna for Bath Kushta RogChikitsa. (Ch.Chi.7/129)

II. **SushruraSamhita:**

- As a constituent of DhoopanDravya in VranitaupasanaChikitsa. (Su.Chi.19/28)
- As a constituent of Siddha Ghruta in KushthaRogaChikitsa. (Su.Chi.9/46)
- As a constituent of Siddha Ghruta in VidradhiChikitsa. (Su.Chi.16/17)
- As a constituent of Siddha Ghruta in JwaraChikitsa. (Su.Ut.39/226)
- As a constituent of DhoopanDravya in VranitopasanaChikitsa. (Su.Su.19/28)
- As a constituent of Mahavajarak Tail in KushtaRogaChikitsa. (Su.Chi.9/58)
- As a constituent of DhoopanDravya in Agrohopkranik Adhyaya Chikitsa. (Su.Su5/18)
As a constituent of Shodhan Tail in Mishrak Adhyaya. (Su.Su. 37/17)
As a constituent of Churana for Snan, Lepa in Kushta Chikitsa. (Su.Chi. 9/14)
As a constituent of Kwatha in Kushta and Krumi in Kushtha Chikitsa. (Su.Chi 9/51)
As a constituent of Kwatha in Prameha Chikitsa. (Su.Chi. 11/8,9)
As a constituent of Siddha Tail in Granthi Apachi Arbuda Chikitsa. (Su.Chi. 18/47)
As a constituent of Lepa in Kshudra Roga Chikitsa. (Su.Chi. 20/21)

III. Ashtanga Hriday: 266

As a constituent of Rasayan Churna in Rajyakshma Chikitsa. (A.Hr.Chi.5/28)
As a constituent of Shodhanadi Gana for Vaman. (A.Hr.Su.15/1)
As a constituent of Shodhanadi Varga for Pitta shamanarth. (A.Hr.Su.15/6)
As a constituent of Churna in Sannipatic Jwara Chikitsa. (A.Hr.Chi.1/65)
As a constituent of Churna in Pittaj Prameha Chikitsa. (A.Hr.Chi.12/8)
As a constituent of Ghruta in Vidradi, Gulma Chikitsa. (A.Hr.Chi.13/11)
As a constituent of Kashayam in Vranashodhan Chikitsa. (A.Hr.Chi.13/35)
As a constituent of Mahatikttak Ghruta in Kshtharoga Chikitsa (A.Hr.Chi.19/9)
As a constituent of Dhoopan Dravya in Vranadhoopanarth. (A.Hr.Su.29/26)
As a constituent of Kwatha in Dushta Varana Dhavan in Vrana Chikitsa. (A.Hr.Ut.25/42)
As a constituent of Churana in Shodhan and Ropan in Vrana Chikitsa. (A.Hr.Ut.25/43,55)

IV. Nighantu Kala: 267

It is mentioned in Guduchyadi Varga of Ashtanga Nighantu.
It is mentioned in Guduchyadi Varga of Dhanvantari Nighantu.
It is mentioned in Guduchyadi Varga of Shodhal Nighantu.
It is mentioned in Abhayadi Varga of Madanpal Nighantu.
It is mentioned in Prabharadi Varga of Raj Nighantu.
It is mentioned in Mishraprakaran Guduchyadi Varga of Bhavprakash Nighantu.
It is mentioned in Nimbadi Varga of Nighantu Adarsha.
3. Morphology of Nimb: 268

Nimba (Leaf): Azadirachta indica A. Juss Syn. Melia Azadirachta Linn. (Fam. Meliaceae); a moderate sized to fairly large evergreen tree. Attaining a height of 12-15 m with stout trunk and spreading branches, occurring throughout the country up to an elevation of 900 m.

Description: 268

a) Macroscopic:
Leaves-Compound, alternate, rachis 15-25 cm long, 0.1 cm thick; leaflets with oblique base, opposite, exstipulate, lanceolate, acute, serrate, 7-8.5 cm long and 1.0-1.7 cm wide,
Colour - slightly yellowish-green;
Odour - indistinct
Taste - bitter

b) Microscopic:
Leaf-Midrib -leaflet through midrib shows a biconvex outline; epidermis on either side covered externally with thick cuticle; below epidermis 4-5 layered collenchyma present; stele composed of one crescent-shaped vascular bundle towards lower and two to three smaller bundle towards upper surface; rest of tissues composed of thin-walled, parenchymatous cells having secretory cells and rosette crystals of calcium oxalate; phloem surrounded by non-lignified fibre strand; crystals also present in phloem region.
Lamina - shows dorsiventral structure; epidermis on either surface, composed of thin-walled, tangentially elongated cells, covered externally with thick cuticle; anomocytic stomata present on lower surface only; palisade single layered; spongy parenchyma composed of 5-6 layered, thin-walled cells, traversed by a number of veins; rosette crystals of calcium oxalate present in a few cells; palisade ratio 3.0-4.5; stomatal index 13.0-14.5 on lower surface and 8.0-11.5 on upper surface.
Powder - Green; shows vessels, fibres, rosette crystals of calcium oxalate, fragments of spongy and palisade parenchyma.

C) Identity, Purity and Strength: 268
Foreign matter not more than 2 per cent, Appendix 2.2.2.
Total Ash not more than 10 per cent, Appendix 2.2.3.
Acid-insoluble ash not more than 1 per cent, Appendix 2.2.4.
Alcohol-soluble extractive not less than 13 per cent, Appendix 2.2.6.
Water-soluble extractive not less than 19 per cent, Appendix 2.2.7.

D) Chemical constituents: 269
About 100 chemical constituents mostly Triterpenoids of protolimonoids, Limonoids
few non Triterpenoids constituents are Azadirictin Azadiractoldeacetyl Azadiractol
and Sterols.

E) Part used: 268
Bark, leaf, flower, fruits, oil.

F) Properties and action: 268
1) Rasa: Tikta, Kshya,
2) Guna: Ruksha, Ushna, Laghu
3) Virya: Sheeta
4) Vipaka: Katu
5) Karma: 268
Grahi, Vatal, Pittashamak, Kaphashamak,Vranaropak, Vranashodhak, Putihar,
Daahaprashaman, Kandughna, Kushtahar, Rakttashodhak, Shothagna, Aampachk

G) Important formulations: 268
Kashishadi Taila, Jatyadi Ghruta, Arogyavardhini vati, PanchagunaTaila.

H) Therapeutic uses: 268
Jwara, Krumiroga, Kushtha, Netraroga, Prameha, Vrana, Amavat, Visharoga

I) Dose: 268
1-3 g. of the drug in powder form.
10-20 ml of the drug for decoction.
4. Classification:

A) Classification According to Samhita: ²⁷⁰

Table No.29: Classification of Nimba according to Samhita

<table>
<thead>
<tr>
<th>CharakaSamhita</th>
<th>SushrutaSamhita</th>
<th>AshtangaHridaya</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kandughana</td>
<td>Aragvadhadi, Pramehaghna</td>
<td>Vaman varga</td>
</tr>
<tr>
<td>Vamak</td>
<td>Guduchyadi, Dantashodhan</td>
<td>Guduchyadi</td>
</tr>
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<td>Langanarth</td>
<td>Lakshadi</td>
<td>Aragvadhadi</td>
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<td></td>
<td>UrdhwaBagh har</td>
<td>Tik Tak Varga</td>
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<td></td>
<td>Rakshoghana</td>
<td>PittaShamak Varga</td>
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<tr>
<td></td>
<td>Shodhan</td>
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</tr>
</tbody>
</table>

B) Classification According to Nighantu: ²⁶⁷

Table No.30: Classification of Nimba according to Nighantu

<table>
<thead>
<tr>
<th>Nighantu</th>
<th>Varga</th>
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<tbody>
<tr>
<td>AshtangNighantu</td>
<td>Guduchyadi/Aragwadadi Gana</td>
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<td>Dhanvantari Nighantu</td>
<td>Guduchyadi Varga</td>
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<tr>
<td>Shodhal Nighantu</td>
<td>Guduchyadi Varga</td>
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<tr>
<td>Madanpal Nighantu</td>
<td>Abhayadi Varga</td>
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<td>Raj Nighantu</td>
<td>Prabhadradi Varga</td>
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<td>Kaiydev Nighantu</td>
<td>Aushadhi Varga</td>
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<td>Bhavprakash Nighantu</td>
<td>Guduchyadi Varga</td>
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<tr>
<td>NidgantuAdarsh</td>
<td>Nimbadi Varga</td>
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C) Taxonomical Classification: ²⁷²

Table No.31: Taxonomical Classification of Nimba

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<th>Kingdom</th>
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<td>Rosids</td>
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<td>Meliaceae</td>
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<td>Genus</td>
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<tr>
<td>Species</td>
<td>A. Indica</td>
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</table>
D) Parayayi Nama of Nimba According to Nighantu

Table No. 32: Parayayi Nama of Nimba According to Nighantu

<table>
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<tr>
<th>Parayayi Nama</th>
<th>A.N.</th>
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</tbody>
</table>
J) Paryayinama Nirukati: 268

- **Arishta**: It eradicates many diseases.
- **Chardan**: It induces vomiting.
- **Hinguniryas**: Plant exudates secretion like Hingu.
- **Kakphala**: Fruits are eaten by crows.
- **Khrumighna**: It is anthelminthic.
- **Malak**: Useful for health in many ways.
- **Nimba**: Useful for health in many ways.
- **Niyamak**: Useful for health in many ways.
- **Paribhadra**: Useful for health in many ways.
- **Pichumanda**: Cures skin diseases.
- **Puyari**: It cures pus formation.
- **Shukapriya**: Parrots gather on this tree.
- **Sutiktak**: It is one of best better drug.
- **Vartwachya**: Bark is used as medicine
- **Sarvatobhadra**: It is good in all the ways.
- **Neta**: It is the first in medicine.

K) Vernacular names: 268

- **Sanskrit**: Arishtak, Pichumarda
- **Assamese**: Mahanim
- **Bengali**: Nim, Nimgach
- **English**: Margosa Tree
- **Gujrati**: Limba, Limbado, Limado, Kohumba
- **Hindi**: Nim, Nimba
- **Kannada**: Nimba, Bevu, Oilevevu, KahiBevu, Bevinama
- **Malayalam**: Veppu, Aryaveppu, Nimbam, Veppa
- **Marathi**: Balantanimba, Limba, Bakayan, Nim, Kadunimb
- **Oriya**: Nimba
- **Punjabi**: Nimba, Bakan, Nim
- **Tamil**: Vemmu, Veppu, Arulundi, Veppan
- **Telugu**: Vemu, Vep
L) Guna karma of Nimb:

Table No. 33: Gunapanchak of Nimb according to Nighantu:

<table>
<thead>
<tr>
<th>Properties</th>
<th>A.N.</th>
<th>D.N.</th>
<th>S.N.</th>
<th>M.N.</th>
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<tr>
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</table>

M) Dosha, karma and Rogaghanatta of Nimba:

Table No.34: Dosha, karma and Rogaghanata of Nimba according to Nighantu:

<table>
<thead>
<tr>
<th>Nighantu Karma</th>
<th>A.N.</th>
<th>D.N.</th>
<th>S.N.</th>
<th>M.N.</th>
<th>R.N.</th>
<th>K.N.</th>
<th>B.N.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dosha karma</td>
<td>Pittanuta Kaphaan uta</td>
<td>Pittanuta Kaphaan uta, Astrashodhan</td>
<td>Pittanuta Kaphaan uta</td>
<td>Vatkar Pittaghana Kaphakar</td>
<td>Pittanuta Kaphaan uta</td>
<td>Pittanuta Kaphaan uta Vatkar</td>
<td>Pittanuta Kaphaan uta Vatkar</td>
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<tr>
<td>Pramukha Karma</td>
<td>Vishodhan,</td>
<td>Apakwa vranapa chayet Vranapak washodhayet</td>
<td>Vranajee</td>
<td>Bhedan Kushtahar Vrana</td>
<td>Vrana</td>
<td>Shothaghn</td>
<td>Grahi Hrudya Krumihar Netrorogh har Agnikrut Hrudya</td>
</tr>
</tbody>
</table>
N) Shloka references of Various Nighantu for Paryayi Nama, Gunapanchak, Dosha Karma and Rogghanta of Nimb: 267

1. अष्टाङ्गनिष्ठ – ६. गुद्ध्यादिगणगुद्ध्यादिगण
guddhiyaparakarishadhanaakarkanchandana

2. कृष्णनिष्ठ – ३. गुद्ध्यादिगणगुद्ध्यादिगण
guddhiyaparakarishadhanaakarkanchandana

3. कृष्णनिष्ठ – ५. गुद्ध्यादिगणगुद्ध्यादिगण
guddhiyaparakarishadhanaakarkanchandana

4. कृष्णनिष्ठ – ६. गुद्ध्यादिगणगुद्ध्यादिगण
guddhiyaparakarishadhanaakarkanchandana
चक्षुष्णिनिम्बवपुष्पांचकृमिपितविषप्रणुत् ||८८३||
वातलंकुपाकंस्यातसर्वयोरोचकनाशनम् |
फलतिकरसापाककुपकंबेदनार्थु ||८८४||
अरूक्षमुण्यकुष्ठघनगुलमार्शृकृमिहनुत् |
निमबरसपकवम्पुरसतिक्षितविधधकशोणिपितिरोगे |
कपेशस्तन्त्रयनामस्येन्द्रकृमिकुष्ठविशोधन: ||८८५||
निमबबीजस्यमज्जाचकृमिकुष्ठविशोधन: |८८६|
3.धन्वन्तरिनिघण्डु - १. गुस्तच्यादिवरीगिमब
निमबिनियमनोनेतापिपुमन्दःसुतिकक:|
अरिष्ट:सर्वतोभःस्रम्दःपारिभद्रः||३१||
निमबस्तिकरसःशीतलघुःशेषासपितनुत्|
कुष्ठकण्ठःगुनान्त्वित्तिपाहारादिशीतलः ||३२||
अपकंवच्छेद्योजयाँगपुंयावंविशेषदेयेऽ ||३३||
4.धन्वन्तरिनिघण्डु - ६. सुर्णादिवरीगिमबतेत
नात्मयुण्यानिम्बजालंकृमिकुष्ठकापाहमः|
वातरकप्रशांन्तम्दमाश्रीलजापाहमः ||४३१||
5.भावहकाश-पूर्विकण्ड-मिश्रप्रकरण - ५. गुस्तच्यादिवरी-निमब
निमबःस्यालिपुचमर्दश्चिपुचमर्दशतिकक: |
अरिष्ट:पारिभद्रश्चहिर्गुलिया:सद्यापि ||८१||
निमबःशीतलघुःशीतलहुःपाककोऽपिन्यात्वनुत्|
अहःश्रमतःस्यवराराशिकृमिप्रणुत् ||८२||
ग्राणिपितककालिकुष्ठलासमेहनुत्|
निमबपत्रस्मूतलेज्याकृमिपितविषप्रणुत् ||८३||
वातलंकटुपाकचसवारोचककुष्ठनुत्

निम्बफलरसेतिकपाकेतुकुठुभेदनम्

स्निग्धलघूपाकृष्ठमुखुक्तमार्शःकृमिमेहनुत्।८४॥

6. भावप्रकाश-पूर्व खण्ड-मिश्रप्रकरण - ४. गुदचत्यादिवर्ग-पारिभ्रम

पारिभ्रमोनिमितस्वमेहनदरःपारिजातकः।

पारिभ्रमोनिमितस्वमेहनदःकृमिमेहनुत्।

तत्प्रतिरोगश्लंकणीयाचिविनाशनम्।८७॥

7. मदनपालनिघण्डु - १. अभयादिवर्ग-निम्ब

निम्बोनियमनोनेताशिरःस्यात्पारिभ्रमः।

सुतिकःसंरतोमदःपिचुमन्दःप्रभ्रमः।

कुठमदेवदत्तचिरसनिमितसूयकोः।१३७॥

निम्बःशीतोलघुहीकटपाकोनियागचकुकः।१३८॥

व्रणपितकःचछिकुकुमलासभेदनुत्।

निम्बप्रत्समतेर्ग्यकृमिपितविषप्रणुत्।१३९॥

तत्तथलभेदनस्निग्धहुणकुमहलस्तुः।

अपकवचयेनिम्बःपकवचपरिशोषयेत्।१४०॥

8. राजनिघण्ठु - ९. प्रभ्रमदि-प्रभ्रमः

अथनिगदितःप्रभ्रमःपिचुमम्दःपारिभ्रमकोनिमः।

काकफलःकीरेश्टोनेताशिरसवसतोमदः।१७॥

धमलोपिशीर्षपणापसतेःपीसारकःशीतः।

वरतिकोशिरसस्मालकस्थिहिंचुनियास।८॥

छदलपारिवधमलोचनामालःसातुदिशितः।९॥

प्रभ्रमः प्रभ्रमः शीतशृष्टिकः कफव्रणक्रिमिविशिष्टाश्च।
बलासभिद्रुविषपितदशस्त्रिद्वेष्टोहहदयविदाहशान्तिकृतयः॥९॥

9. राजनिघण्डु - १५. क्षीरादिवर्ग - निम्बतैल

निम्बतैलंतुल्यात्युण्ड्रिक्रियंकुषकानावहम्॥१७॥

10. शोढलिघण्डु

नामसंग्रह (प्रथमभाग) - १. गुदच्यादिवर्ग - निम्ब

निम्बोत्तियुसोनातापिचुमन्द्र - सुतिकांकः

अरिष्ट: सर्वतोभद्र: सुभाद्र: पारिवेदन: ||९८||

शुक्रप्रयोग्योणवृण्वल्लोहन्वत्वचः

छर्दनोहिष्कुर्मित्यास: पीतसारेरविप्रियवर्ग: ||९९||
III) Yashtimadhu (Glycyrrhiza glabra Linn)

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern Botany.

1. Vedic review of Yashtimadhu: 273

In Vedic literature Yashtimadhu is described as,

**Madudha:** According to KaushikSutra Jesthamadha, Shonakiya Atharvaved Samhita 6/101/3 Sayanbashya MadhuVruksha or Yashtimadhu. According to Mujumdar, Honey Plant. 274

**Klintak:** Gobhil Sutram1/10/10 275

**Jeshtimadhu:** VruschikdanshLepanarth, Streevashikaranpayog. KeshavPadhatti32/5, 35/21, 38/17 276

**Madhuk:** Atharvaparishista as a Samindha Dravy, for speech improvement Shonakiya Atharvved Samhita1/34/1 in marriage Manibandhanarth Shonakiya Atharvved Samhita 1/34 276

**Madhuyashtika:** Described in MoolavidhiPrakaran Praskar Ghruhya Sutra 1/21 277

**Mdhula:** Accordig to SayanBhashya itis describe as Vishanashini, Mashakjambhini. Sarpavisha Maitrayani Samhita 4/9/1 278

2. Samhitakalin Review: 279-281

I. Charak Samhita: 279

- As a constituent of Vamakdravya in Apamargatanduliya chapter. (Ch.Su.2/7)
- As a constituent of Vatahar Lepa in Argvadhdi chapter. (Ch.Su.3/21)
- As a Constituent of Sandhaniya, jeevaniya, KanthyaGana in Shadaveerechaniyashatashritiya chapter. (Ch.Su.4/9)
- As a constituent of Pratham Bramhya rasayan. (Ch.Chi.1/49)
- As a constituent of Jwaranashak kashaya in Jwarachikitsa. (Ch.Chi.3/19)
- As a constituent of Abhangya Taila in Kushtha chikitsa. (Ch.Chi.7/133)
- As a constituent of Jivantyadi Ghruta in Jwarachikitsa. (Ch.Chi.3/250)
- As a constituent of Haridradi Ghruta in Panduroghchikitsa. (Ch.Chi.16/53)
- As a constituent of Lepan Dravya in Visarpa chikitsa. (Ch.Chi.21/74)
- As a constituent of Lepan Dravya in Vranarp chikitsa. (Ch.Chi.21/74)
- As a constituent of Lepan Dravya for Shoolashaman in Vranachikitsa. (Ch.Chi.25/47)
- As a constituent of Lepan for Vranashodhan in Vranachikitsa. (Ch.Chi.25/48)
II. Sushrut Samhita: 280

- As a constituent of Lepan for Pittaj Arbud in. (Su.Chi.18/346)
- As a constituent of Lepa for Khardhaha in Kharapakvidhi Adhaya. (Su.Su.11/21)
- As a constituent of lepa for Rakattastravahar in Shonitvaniya Chapter. (Su.Su.14/36)
- As a constituent of Lepan for Asamyak Vrana in Karnavedhan Chikitsa. (Su.Su.16/6)
- As a Constituent of Kashay for Pittajanyashool in Mishrakadhaya. (Su.Su.37/4)
- As a constituent of jeevniya, Bruhaniya, Vrushya, kakolyadi Gana in Dravyasangrahaniya Chapter. (Su.Su.38/35)
- As a constituent of Sarivadi Gana for Pitta, Daha,Trushna Shaman, in Dravyasangrahaniya Chapter .(Su.Su.38/39)
- As a constituent of Anjanadi Gana for pitta and Dahashaman in Dravya Sangrahniya chapter (Su.Su.38/41)
- As a constituent of Lepa for Vranapandu karma in Vrana chapter. (Su.Chi.1/96)
- As a constituent of Lepa for Vranapratisaraniya Vrana chapter. (Su.Chi.1/99)
- As a constituent of Kwath for Kaphaj vataraka chapter. (Su.Chi.5/10)
- As a constituent of Siddhaghruta for dusita vrana in Vidharadhi Chapter. (Su.Chi.16/17)
- As a constituent of Hita dravya in VishaChikitsa. (Su.Chi.8/131, 32)

III. AshtangHriday: 281

- As a constituent of JeevaniyaGana in Shodhanadi gana chapter.(A.Hr.Su.15/8)
- As a constituent of Nyogrodhadi Gana in Shodhanadigan chapter (A.Hr.Su.15/41)
- As a constituent of VranaLepa in Shastrakarmavidhi chapter,(A.Hr.Su.29/55)
- As a constituent of VranaLepa in Ksharagnikarma chapter.(A.Hr.Su.30/34)
- As a constituent of Siddhaghruta in Rajyakshma chikitsa chapter. (A.Hr.Chi.5/16)
As a constituent of Kashya in VidradhiVrudhi chikitsa chapter.
(A.Hr.Chi.13/11)

As a constituent of Lepa for daha in Vatarakta chikitsa chapter.
(A.Hr.Chi.22/28)

As a constituent of Siddha taila for Ropan in Sadyovrana chapter.
(A.Hr.Ut.26/55)

As a constituent of Kashya in Shotharoga chikitsa chapter.
(A.Hr.Chi.17 31/)

As a constituent of Vrana lepa in Vrana chikitsa chapter.
(A.Hr.Chi.1/96)

IV. Nighantu Kaal: 282

It is mentioned in Sarivadi/Vachadi Varga of Ashtanga Nighantu.

It is mentioned in Guduchyadi Varga of Dhanvantari Nighantu.

It is mentioned in Guduchyadi Varga of Shodhal Nighantu.

It is mentioned in AushadhiVarga of Kaiyadev Nighantu.

It is mentioned in Abhayadi Varga of Madanpal Nighantu.

It is mentioned in Pippalyadi Varga of Raj Nighantu.

It is mentioned in Mishrakprakaran Haritkyadi Varga of Bhavprakash Nighantu.

It is mentioned in Palashadi Varga of Nighantu Adarsha.

3. Morphology of Yashtimadhu: 283

Yashtimadhu consists of dried, unpeeled, stolon and root of Glycyrrhizaglabra Linn, (Fam. Leguminosae), a tall perennial herb, upto 2 m high found cultivated in Europe, Persia, and Afghanistan and too little extent in some parts of India.

1. Description: 283

a) Macroscopic:
Stolon consists of yellowish brown or dark brown outer layer, externally longitudinally wrinkled, with occasional small buds and encircling scale leaves, smoothed transversely, cut surface shows a cambium ring about one-third of radius from outer surface and a small central pith, root similar without a pith, fracture, coarsely fibrous in bark and splintery in wood, Odour, faint and characteristic taste is sweetish.

b) Microscopic:
Stolon- transverse section of stolon shows cork of 10-20 or more layers of tabular cells, outer layers with reddish-brown amorphous contents, inner 3 or 4 rows having thicker,colourless walls,secondary cortex usually of 1-3 layers of radially arranged
parenchymatous cells containing isolated prisms of calcium oxalate, secondary phloem abroad band, cells of inner part cellulosic and outer lignified, radially arranged groups of about 10-50 fibres, surrounded by a sheath of parenchyma cells, each usually containing prism of calcium oxalate about 10-3 μ long, cambium form tissue of 3 or more layers of cells, secondary xylem distinctly radiate with medullary rays, 3-5 cells wide, vessels 168 about 80-200μ in diameter with thick, yellow, pitted, reticulately thick end walls, groups of lignified fibres with crystal sheaths similar to those of phloem, xylem parenchyma of two kinds, those between the vessels having thick pitted walls with out inter-cellular spaces, the remaining with thin walls, pith of parenchymatous cells in longitudinal rows, with inter-cellular spaces. Root-transverse section of root shows structure closely resembling that of stolon except that no medulla is present, xylem tetra , usually four principal medullary rays at right angles to each other unpeeled, drug cork shows phelloderm and sometimes without secondary phloem all arenchymatous tissues containing abundant, simple, oval or rounded starch grains, 2-20μ in length.

2 Identity, purity and strength:  
Total Ash not more than 10 per cent, Appendix 2.2.3.
Acid-insoluble ash not more than 2.5 per cent, Appendix 2.2.4.
Alcohol-soluble extractive not less than 10 per cent, Appendix 2.2.6.
Water-soluble extractive not less than 20 per cent, Appendix 2.2.7.

3. Constituents:  
Glycyrrhizin, prenylatedbiaurone, licoagrone, 7-acetoxy-2methyl-isoflavone, asrtagalin, liquiritigenin, Glycyrrhizin, glycyrrhizic acid, glycyrrhetinic acid, asparagine, Sugars, resin and starch.

4. Properties and action:  
1) Rasa: Madhura
2) Guna: Guru, Snigdha
3) Virya: Sheet
4) Vipaka: Madhur
5) Karma: Baly, Dahaprashamanan, Keshya, Shothhar, Medhya, Trushnanigrahani, Vatanuloman, Mrudurechan, Shonitsthapan, Kapanissaran, Kanthya, Mutral, Shukravardhan, Varnya, Jeevaniya, Rasayan, Chakshushya, Sandhaniya
5. **Important formulations:**

   Eladigulika, Yashtimadhutailaa, Kalyanleha.

6. **Therapeutic uses:**


7. **Dose:** 2-4 gm of the drug in powder form.

8. **Classification According to Samhita:**

   **Table No.35: Classification of Yashtimadhu according to Samhita**

<table>
<thead>
<tr>
<th>Charaka Samhita</th>
<th>Sushruta Samhita</th>
<th>Ashtanga Hridaya</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jeevaniya</td>
<td>Haridradi Gana</td>
<td>Ambashtadi Varga</td>
</tr>
<tr>
<td>Sandhaniya</td>
<td>Bruhatyadigana</td>
<td>Niruhan Varga</td>
</tr>
<tr>
<td>Varnya</td>
<td>KakolyadiGana</td>
<td>PittasanshmanGana</td>
</tr>
<tr>
<td>Kanthya</td>
<td>Sarivadi Gana</td>
<td>JeevaniyaGana</td>
</tr>
<tr>
<td>Kandughna</td>
<td>AnjanadiGana</td>
<td>SarivadiGana</td>
</tr>
<tr>
<td>Snehopag</td>
<td>Ambashthdai Gana</td>
<td>Patoladi Varga</td>
</tr>
<tr>
<td>Vamanopag</td>
<td>Shodhan Gana</td>
<td>Haridradi Varga</td>
</tr>
<tr>
<td>Asthapanopag</td>
<td>Sthanyajanan Varga</td>
<td>Nyogrodhadi Varga</td>
</tr>
<tr>
<td>Angamarda Prashaman</td>
<td>Garbha</td>
<td>Madhur Skanda</td>
</tr>
<tr>
<td>Shoonitsthapangan</td>
<td>Keshyaranjan Varga</td>
<td>VamakGana</td>
</tr>
<tr>
<td>Garbhasthapan</td>
<td>Medhya Rasayan</td>
<td>Niruhaupyoji Varga</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RaktasthambhakVarga</td>
</tr>
</tbody>
</table>
9. Classification According to Nighantu: 282

Table No.36: Classification of Yashtimadhu according to Nighantu

<table>
<thead>
<tr>
<th>Nighantu</th>
<th>Varga</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ashtang Nighantu</td>
<td>Sarivadigana, Vachadigana</td>
</tr>
<tr>
<td>Dhanvantari Nighantu</td>
<td>Guduchyadivarga</td>
</tr>
<tr>
<td>Shodhal Nighantu</td>
<td>Guduchyadivarga</td>
</tr>
<tr>
<td>Madanpal Nighantu</td>
<td>Abhayadivarga</td>
</tr>
<tr>
<td>Raj Nighantu</td>
<td>Pipalyadivarga</td>
</tr>
<tr>
<td>Kaiydev Nighantu</td>
<td>Aushadivarga</td>
</tr>
<tr>
<td>Bhavprakash Nighantu</td>
<td>Hritakyadi Varga</td>
</tr>
<tr>
<td>Nighantu Adarsha</td>
<td>Palashadi Varga</td>
</tr>
</tbody>
</table>

10. Taxonomical Classification: 286

Table No.37: Taxonomical Classification of Yashtimadhu

<table>
<thead>
<tr>
<th>Kingdom</th>
<th>Plantae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unranked</td>
<td>Angiosperms</td>
</tr>
<tr>
<td>Unranked</td>
<td>Edicots</td>
</tr>
<tr>
<td>Unranked</td>
<td>Rosids</td>
</tr>
<tr>
<td>Order</td>
<td>Fabales</td>
</tr>
<tr>
<td>Family</td>
<td>Leguminaceae / Faboideae</td>
</tr>
<tr>
<td>Genus</td>
<td>Glycyrrhiza</td>
</tr>
<tr>
<td>Species</td>
<td>Glabra</td>
</tr>
</tbody>
</table>
11. Vernacular Names:  
- **Assamese**: Jesthimadhu, Yeshtmadhu  
- **Bengali**: Yashtimadhu  
- **English**: Liquorice root  
- **Gujrati**: Jethimadha, Jethimard, Jethimadh  
- **Hindi**: Mulethi, Mulathi, Muleti, Jethimadhu, Jethimadh  
- **Kannada**: Jestamadu, Madhuka, Jyeshtamadhu, Atimadhura  
- **Kashmiri**: Multhi  
- **Malayalam**: Irattimadhuram  
- **Marathi**: Jesthamadh  
- **Oriya**: Jatimadhu, Jastimadhu  
- **Punjabi**: Jethimadh, Mulathi  
- **Tamil**: Athimadhuram  
- **Telugu**: Atimadhuramu  
- **Urdu**: Mulethi, Asl-us-sus

12. Parayayi Nama of Yashtimadhu according to Nighantu:  

Table No. 38: Parayayi Nama of Yashtimadhu according to Nighantu

<table>
<thead>
<tr>
<th>Nighantu →</th>
<th>A.N.</th>
<th>D.N.</th>
<th>S.N.</th>
<th>M.N.</th>
<th>R.N.</th>
<th>K.N.</th>
<th>B.N</th>
<th>N.A.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ParyayiNama ↓</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yashtimadhu k</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Yashti</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Mdhuk</td>
<td>+</td>
<td>Madhuyastya</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Klintak</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Mdhuparni</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Madhu-valli</td>
</tr>
</tbody>
</table>
13. Paryayi Nama:²⁸⁷

निरुक्ति

1. Madhuk: Sweet as like honey.

2. Yashtimadhu: It is available in sweet wooden form


14. Gunakarmatmaka Vivechana:²⁸²

Rasapanchaka i.e. Guna, Rasa, Virya, and Karma.; with these characteristics, the drug can work. These are described in all Nighantus.

Table No. 39: Gunapanchak of Yashhtimadhu:²⁸²

<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Guna</td>
<td>Sheet</td>
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<td>+</td>
<td>+</td>
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<td>+</td>
<td>+</td>
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<tr>
<td></td>
<td>Guru</td>
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<td>+</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Snigdha</td>
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<td></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Rasa</td>
<td>Madhur</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
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<td>Tiktta</td>
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<td></td>
<td></td>
<td>+</td>
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<td></td>
<td>+</td>
</tr>
<tr>
<td>Virya</td>
<td>Sheet</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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</tr>
<tr>
<td>Vipaka</td>
<td>Madhur</td>
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<td>+</td>
</tr>
</tbody>
</table>
Table No.40: Dosha, karma and Rog-ghnata of Yashtimadhu according to Nighantu:

15. Shloka references of Various Nighantu for Paryayi nam, Gunapanchak, Dosha Karma and Rogghanta of Yashtimadhu:

1.अष्ठाङ्गनिघंडु  –

1) 2.सारियादिग्राण

सारियोशीरकाश्मर्यमधूकशिशिरद्वयम्

यष्टीपुष्पकंहलिताहपियासर्वसंख्याज्ञरान्॥२३॥
सारिवादिगणवक्ष्येपुराप्रोकतातुसारिवा

वीरण्यमव(भय)लामञजकोशीरसमृणालकम्|२४||

वीरवीरणमूलंचबहुमूलंरणोऽहया

सारिवादिगण

यष्टीमधुकंपञ्च्यामाामधुकंपञ्च्यामा|पशुकोमृदुफलोरोषजोधुवनंसः|

२४||

1. अष्टाङ्गोऽकांड - १९. वचापदिगण

वचाजलदेवामातृतोवषामयाः

हरिद्रायष्ट्यायकलशीकुट्टोमः||४४||

वचाहरिद्रायणामातीसारनाशनाः

मेदःककायथष्ट्यायकमुस्तातुजलदाहः||

गाडणीकुकुविन्दाचायदेवामाभ्रमस्कमः||५५||

२. धन्वन्तरिलिङ्गाय - १. गृहयादिवर्ग

मधुयषी

मधुयषीचयषीचयषीमधुमुस्वा

यष्टीकंमधुकंपञ्च्यामाामधुकंपञ्च्यामा|५५||

मधुयषीस्वातुरसाशीस्तपितविनाशिनी

वृष्णयशक्षयहरविचच्छदिविनाशिनी||५६||

३. शोभ्रलिङ्गाय

(प्रथमभाग) - १. गृहवदिवर्गमधुयषी

मधुयष्ट्यायष्ट्यायष्ट्यायमधुमुस्वा

मधुकंपञ्च्यालितकासाचकलीतलंचमधूपृलिका||६५||
मधुपणरसासौयाविरसाशोषनाशिला

t| 4. मदनपालनिघण्डु - १. अभयादिवर्गमधुयशी
मधुयशीक्लीतनकंथीमधुमधूलिका
यष्टयागमधुकंथिमधुकंजलजाम्
मधुयशीगुरुःशीताबल्यात्तुचिदिपितजिनत
5. राजनिघण्डु - ६. पिपपल्यादिवर्गयशीमधु
यशीमधुमधुयशीमधुवल्लिमधुसः
मधुकंमधुकंमधुयशीमधुसः
मधुरंयशीमधुकं
चक्षुष्यितहदु चय्यशोष्यत् यन्नाण्यपहम्
6. कैयदेवनिघण्डु - १. ओषधिवर्गयशीमधु
यशीमधुकंमधुकंमधुयशीमधु
यशीमधुकंमधुकंमधु
यशीकमपरांभोजामधुपणराशोषनार्रणालिसत्
यशीहिमागुरुःस्वादु चक्षुष्यावल्लणकृत्
सुस्त्रिष्यशुक्लाकेश्यायमधुपणराशोषनालिसत्

ब्रणशोफविषच्छदित्तु यन्ताग्नालिसतापहः
१. ओषधिवर्गयशीमधु
यशीमधुकंमधुकंमधु
यशीमधुकंमधु
मधुयशीमधुपणराशोषनालिसत्
मधुकंमधुपणराशोषनालिसत्
सुस्त्रिष्यशुक्लाकेश्यायमधुपणराशोषनार्रणालिसत्
 सच्चातासतुचिदियात्श्रोफव्रणालिसत्
7. भावप्रकाश-अपूर्वेक्षण-सिद्धप्रकरण - २. हरितक्ष्यादिवर्गं यष्टीमधु
यष्टीमधुतथायष्टीमधुकणिकलितस्तथा
अन्यत्रकणिकलितस्तुभेवतोयमधूलिखिता॥१२८॥
यष्टीहिमाणुःस्वादीवचक्षुप्यावलवर्णकृत्
सुर्दिगापधुकेशयास्वयंपितातिलासतित्
ग्राणशोथविष्ट्युदितःपणागलास्तिक्ष्यायपि॥१२७॥
IV. Til (Sesamum indicum)
The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern Botany.

1. Vedic review: 288

Jaetil: Mostly it is wild variety of Tila. The Yavagu of this Tila is mentioned. 289

Tila is mentioned in Atharvaveda at 8-8-3, 5-3-10, 4-7-3, 2-8-3, 6-140-2. 290

Til is not mentioned in Rugveda, but mentioned in other Samhita with Masha, and Shishir Dhanya. As Gramya and Aranya types. Tail is extracted from it. Tilapishti is used as a fuel, as a dietary item, Keshya. 291

2. Samhitakalin review: 292-94

I. Charak Samhita: 292

- As a constituent of Haradradi Lepa dravya in Aaragvadhadi chapter. (Ch.Su.3/14)
- As a constituent of Sthavarsneha yoni adrvya in Snehaadhyya chapter. (Ch.Su.13/10)
- As a Rakttadushti hetu dravya in Vidhishonit chapter. (Ch.Su.24/6)
- As a Shukravardhak dravya in Annapanvidhi chapter. (Ch.Su.27/270)
- As a Prameha hetu dravya in Prameha nidan chapter. (Ch.Ni.4/5)
- As a constituent of lepa in Raktaj Arsha chikitsa chapter. (Ch.Chi.14/220)
- As a constituent of Upnahdravya in Dwivraniya chapter. (Ch.Chi.25/51)
- As a constituent of VatakVrana Lepa dravya in Dwivraniya chapter. (Ch.Chi.7/74)
- As a constituent of Daha in Dwivraniya chapter. (Ch.Chi.7/78)
- As a constituent of Lepaniya dravya in Vatavyadhi chapter. (Ch.Chi. 28/114)

II. Sushrut Samhita: 293

- As a constituent of Vranaropakdravya in Dwivraniya chapter. (Su.Chi.1/65)
- As a constituent of Vranashodhakdravya in Dwivraniya chapter. (Su.Chi.1/69)
- As a constituent of Kalka in PittajVrana in Dwivraniya chapter. (Su.Chi.2/93)
- As a constituent of Kalka in Kaphaj Vrana in Dwivraniya chapter. (Su.Chi.2/94)
- As a constituent of Siddha Tail in Bhagnna chikitsa chapter. (Su.Chi.3/55)
As a constituent of Sarvakushtanashak lepa Dravya in Kushtarog chikitsa chapter. (Su.Chi.9/10)

As a constituent of Lepa in Vidradhi chikitsa chapter. (Su.Chi.16/13)

As a constituent of Taila in VranaShodhan in Granthi, Apachi chikitsa chapter. (Su.Chi.18/7)

III. AshtangaHriday: 294

The Guna of Tila is mentioned in Annaswaroopvidnyaniya chapter. (A.Hr.Su.6/23)

As a constituent of Madyamkshar Dravya in Ksharagnikarma chapter. (A.Hr.Su.30/12)

As a constituent of Vataj vidradhi lepa Dravya in Vidradhi-Vrudhi chikitsa chapter. (A.Hr.Chi.13/4)

As a constituent of Pittaj vidradhi lepa Dravya in VidradhiVrudhi chikitsa chapter. (A.Hr.Chi.13/5)

As a Constituent of Kaphaj vidradhi lepa Dravya in Vidradhi Vrudhi chapter. (A.Hr.Chi.13/6)

As a Constituent of lepa Dravya for shool in Vataraktta chikitsa chapter (A.Hr.Chi.22/30)

As a constituent of lepa Dravya for Upanah in Vranachikitsa chapter. (A.Hr.Ut.25/35)

As a constituent of lepa Dravya for Vranashodhan in Vranachikitsa chapter. (A.Hr.Chi.25/47)

As a constituent of lepa Dravya for Shodhan ropan in Vranachikitsa chapter. (A.Hr.Chi.25/54)

A) Nighantu Kaal: 295

It is mentioned in SuvarnadiVarga of Dhanvantari Nighantu.

It is mentioned in Taila Varga of Shodhal Nighantu.

It is mentioned in Paniyadi and Dhanyak Varga of Madanpal Nighantu.

It is mentioned in Kariradi, Ksheeradi, Shalyadi Varga of Raj Nighantu.

It is mentioned in Dhanyavarga, TailaVarga of Bhavprajkash Nighantu.

It is mentioned in TilaVarga of Nighantu Adarsha.
3. Morphology of Til

Description: 296

a) Macroscopic:
Seed white, brown, grey or black, flattened, ovate in shape, smooth or reticulate, 2.5 to 3 mm long and 1.5 mm broad, one side slightly concave with faint marginal line and an equally faint central line; taste- pleasant and oily.

b) Microscopic:
Testa of seed shows single layered palisade-like, thin-walled, yellowish coloured cells, and the rest of the testa composed of collapsed cells; endosperm 3 layered, rarely 2 layered, consisting of cellulosic polygonal cells of parenchyma containing fixed oils and small aleurone grains; cotyledons two, externally covered with thin cuticle; single layered epidermal cell, followed by a single row of palisade-like cells; rest of the tissues consist of polygonal, parenchyma cells containing fixed oil and aleurone grains.

Powder - Blackish coloured; shows palisade-like cells in surface view, parenchyma cells, aleurone grains and oil globules.

c) Identity, purity and strength: 296
Foreign matter not more than 2 per cent, Appendix 2.2.2.
Total Ash not more than 9 per cent, Appendix 2.2.3.
Acid-insoluble ash not more than 1.5 per cent, Appendix 2.2.4.
Alcohol-soluble extractive not less than 20 per cent, Appendix 2.2.6.
Water-soluble extractive not less than 4 per cent, Appendix 2.2.7.
Fixed Oil not less than 35 per cent, Appendix 2.2.8

T.L.C.-T.L.C. of alcoholic extract on Silica gel 'G' plate using Toluene: Ethylacetate (9 : 1) shows under UV (366 nm) three fluorescent zones at Rf. 0.57, 0.64 (both light blue) and 0.72 (blue). On exposure to Iodine vapour five spots appear at Rf. 0.08, 0.57, 0.64, 0.72 and 0.94 (all yellow). On spraying with Vanillin-Sulphuric acid reagent and heating the plate for ten minutes at 110° C seven spots appear at Rf. 0.08, 0.57, 0.64, 0.72 (all violet), 0.76, 0.84 (both light violet) and 0.94 (violet).

D) Constituents: 297
Natural lipid, glycolipids, phospholipid, arginine, cysteine, histidine, isoleucine, isoleucine, leucine, lysine, methionine, phenyalanine, threonine, tryptophan, tyrosine, valine, α- and β-globulin, ascorbic acid, pyridoxine, riboflavin, sesamol, thiamine, galactose, glucose, lycine.
E. Properties and action: 296
1. Rasa: Madhura, Katu, Tikta, Kashya
2. Guna: Vyavayi, Guru, Snigdha, Sukshma
3. Virya: Ushna
4. Vipaka: Madhura

F. Important formulations: 296
Narasimha Churna, Jatiphaladya Chura, Haridradi Lepa, Tiladi Upanaha, Tiladi Yoga, Priyaladi Yoga, Mustadi Upanaha, Sunthyadi Churna, PathyadiGulika, Hingvadi Yoga, Bhallatakadi Modaka.

G. Therapeutic uses: 296

H. Dose: 296 Powder 5-10 gm/day.

5. Classification:

A) Classification According to Samhita: 298

Table No.41: Classification of Tila according to Samhita

<table>
<thead>
<tr>
<th>CharakaSamhita</th>
<th>SushrutaSamhita</th>
<th>AshtangaHridaya</th>
</tr>
</thead>
<tbody>
<tr>
<td>SthavarSneha</td>
<td>Pachak gana</td>
<td>Snehavarga</td>
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<td>Agryasangraha</td>
<td>Shodhak Gana</td>
<td>GundushDravya</td>
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<td>Vatavijay Sneha</td>
<td>Dantashodhan Dravya</td>
<td>DhupanDravya</td>
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<td>Kesharajan Dravya</td>
<td>DravPadarth</td>
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<td>Vajikaranarth</td>
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</table>
B) Classification according to Nighantu: 295

Table No.42: Classification of Tila according to Nighantu

<table>
<thead>
<tr>
<th>Nighantu</th>
<th>Varga</th>
</tr>
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<tbody>
<tr>
<td>Dhanvantari Nighantu</td>
<td>Suvarnadi Varga</td>
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<tr>
<td>Shodhal Nighantu</td>
<td>Taila Varga</td>
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<tr>
<td>Madanpal Nighantu</td>
<td>Paniyadi, DhanyaVarga</td>
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<tr>
<td>Raj Nighantu</td>
<td>Karveeradi, Ksheeradi, ShlyadiVarga</td>
</tr>
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<td>Kaiydev Nighantu</td>
<td>Drava, Dhanya, TailaVarga</td>
</tr>
<tr>
<td>Bhavprakash Nighantu</td>
<td>Drava, TailaVarga</td>
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<tr>
<td>Nighantu Adarsha</td>
<td>TilaVarga</td>
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C) Taxonomical Classification: 299

Table No.43.: Taxonomical Classification of Tila

<table>
<thead>
<tr>
<th>Kingdom</th>
<th>Plantae</th>
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<td>Genus</td>
<td>Sesamum</td>
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<tr>
<td>Species</td>
<td>Indicum</td>
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</table>
6. Vernacular names:

- **Sanskrit**: Tila
- **Assamese**: Simmasim
- **Bengali**: Tilagachh
- **English**: Sesame, Gingelly-oil Seeds
- **Gujrati**: Tall
- **Hindi**: Tila, Teel, Tili
- **Kannada**: Accheellu, Ellu
- **Malayalam**: Ellu
- **Marathi**: Tila
- **Oriya**: Til
- **Punjabi**: Til
- **Tamil**: Ellu
- **Telugu**: Nuvvulu
- **Urdu**: Kunjad

Table No.44: Parayayi Nama of Tila according to Nighantu:

<table>
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<tr>
<th>Nighantu→ ParayayiNama ↓</th>
<th>A.N.</th>
<th>D.N.</th>
<th>S.N.</th>
<th>M.N.</th>
<th>R.N.</th>
<th>K.N.</th>
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<td>Taruni</td>
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</table>
7. लिखितः

Til: It is Snigdha. It gives Snigdha to all over body.

8. Gunakarmatmaka Vivechana:
Rasapanchaka i.e. Guna, Rasa, Vipaka, Virya, and Karma; with these properties the drug can work. These are described in all Nighantus.

Table No.45: Gunapanchak of Tila

<table>
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<tr>
<th>Properties</th>
<th>A.N.</th>
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Table No.46: Dosha, karma, and Rogghanata of Til: 295

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<th>Gunakarma</th>
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<th>D.N.</th>
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8. Shloka references of Various Nighantu for Paryayi nam, Gunapanchak, Dosha, Karma and Rogghanta of Til:

1. धनंजयनिनिघंडु - ६. मुन्यांदिवंगतिः
    तिलस्तुलखोदायंस्यात्मपरिवर्तिःपितृदत्तपुरी;पापधनःपुत्रान्यञ्चजित्वतस्तुवणेनन्द्रः॥१९५॥
    तिलोकेकुंदकोषपूरस्तुवरोगुः;
    विपाकेकुंकु:स्वादः:स्त्रिःस्थलाण:कपफितनुतः॥१९६॥
    वल्यःकेल्हिजरसपाश्चस्वच्छ:स्तन्योग्यविनानिः;
    दल्योऽपलमूद्रकुद्वामावायिनोऽग्रिमातिप्रद॥१९७॥
    तिलतेल (तेलविशेष)

स्नानाभ्यांस्माणाहेषुतिलतेलतेलविशेषवते|
तदद्वितिष्कवानेषुक्षकारापिपुरुषाः॥१२०॥
अन्तवानविधिवातिप्रयोज्यंताततशैवते|
छिन्नमिल्लयुतलयपस्यनस्यक्षतापतिते॥१२१॥
भगनेपुतितविद्वारगिनिजङ्गस्विनिशितारिते|
भाषामिल्लिनेन्हम्:गात्यालादिविनानिः॥१२२॥
तैलयोगपरस्कारात्सर्वोगायोप्यामः॥१२३॥

3. शोढलिनिघंडु: गुणसह: (द्वितीयभाग) - १४. तेलपर
    तिलतेलकंपायानुरस्वलयचयोपितक्रुतः
    दीपपरकावित्विदाथस्वादु:स्त्रिःकु:त्वक्ष्य्वायिच॥७६४॥
    त्वंन्युश्चकु:त्वक्ष्य्वायिच्:सूक्ष्मोऽयानकाक्षः
    कृष्णांबृहणायांस्तुलाकर्त्ता:नायच॥७६५॥
    कृष्णिन्द्रनवंदविष्णूविन्यंस्कारात्सर्वोगायोप्यामः
छिन्नभिन्नचुतीतप्यतमयितोतलाङ्कर्मणि||५६॥
भिन्नस्पुष्टितब्राह्मणदर्शिष्टदारिते||
तथोपहततिमेषुगृहग्यालादिभिषेते||५७॥
सेकामयुक्तायागास्पुष्टिततैलविशिष्यते||
तददस्तितपुणेनभेक्षणाक्षेपणपूर्णे||५८॥
अनुपालनविदार्शीचाप्रयोज्यंवातशान्ते||
त्वचयेश्वरंदययुक्तादेवकालिन्द्रियतपरणम्||५९॥
तैलप्रयोगादुरानन्विधानिजातश्रमा::
४.मदनपालिनघु-८. पानीयादियण-तैल
tैलमुणुण्णुरुस्वैयुबलवरणकर्णसरम्||
वृष्णिकाशिविशिष्टोदयपालसपायो::||१६॥
सोपणकाययुरसंतित्कश्रेष्ठानिलापत्तम्||
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श्रेष्ठमलंकुमुष्कृतवरणग्राहमणयोधनम्||
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मनुष्टपुच्छन्नमनंसमयाविषादियु श्रेष्ठत्वपतिपद्धति
क्षत्रेणघिनदर्पंत्तपंपाणामहाभंगादिभि.सदा::||१९॥
घृतमवतायुपंचशौन्तंघिपालसरम्
tैलविन्नंकेत्याग्निभैुद्धयादियणपूर्णम्||२०॥
मदनपालिनघु-१०.धान्यवर्ण-तिल
tिलपुष्पस्तैलफलस्तितिलफियोप:सिता::
जातिवशनजातःस्यादादृकतूत्वरीमता::||२१॥
तिलःकषायो मधुरस्तिककःकटुकोरसे
तिलोग्राही मुःःस्वादःःस्निग्धोःसककपितलः||४८||
बल्यःकेशोहिमस्पर्शस्त्वच्योग्रणाहितःपरमः
बन्योःप्रभुःकृतःतुनाशनोःगर्मतिप्रदः||४९||
कृण्णःश्रेष्ठमस्तेवुःकलोधयमःसितः
अन्येहीनतराःप्रोकास्ततजैरकादयस्तिलाः||५०||
5. राजनिघण्डु-१०. करवीरादिवर्ग-तिलक
तिलकोविशेषःस्थानमुखमण्डनकिण्डुःपुणः
स्थिरपुणःविनरीहोदधर्थुरोरेशकिण्डुमुखजीवी ||४२||
तरुणीकटाकःकोमोवासःसुदर्शनाः||
भालविभूषणाःजोविजेष्यःपञ्चदशनामा||४३||
तिलकोमधुःस्निग्धोयातपितककाकः
बलपुट्ठकोइयोलपुःरमदोज्विधनः||४४||
तिलकत्वकपशायोणःपुःस्त्वधनीदस्तोभिनुतः
क्रमशोफ्रणाःहलितकर्त्तविनाशनी ||४५||
राजनिघण्डु - १५. क्षीरादिवर्ग-तिलमल
तिलमलरङ्गकरोतिकशेषमधुररतिकपशायमुषणातिक्रमः
बलकृतङ्गोवातजनमस्तुखुम्ब्रकुण्डूस्तिरभ्रंस्कारतिदायि||१०६||
राजनिघण्डु - १६. शाल्यादिवर्ग-तिल
तिलस्तुकोमधात्वायस्तविधिःपितुतपरणः
पापपःपूर्तशाल्याचजित्स्तुकोलोऽद्रवः||१४७||
स्निग्धोयवान्नासमिवृद्धिजननस्तुन्त्वयातिलध्निमृुः
सोष्णःपितकरोःस्निग्धमृतकरणःकेशोःतिपठोग्रणः
सड़ग्राहीमधुरःकषायसहितस्तिकोविपाककेतुः
कृष्णःपत्थवतःसितोऽलंपुयुणःक्षीणास्तथान्येति।||११२||
6. कैयदेवनिघण्डु - ४. द्रवयग्नि तेलवर्ग
तैलंस्तेनहोतमस्तेन्हमुख्यंचितसर्वभवम्
अभ्यजनंचयपदलंखंसारंचितकेतीम् ||२९६||
एरण्डकलसमूहमेत्रेणकमितिस्मृतम्
उमातेलंखांमस्यात्तकुर्तेलंसारंपुलम् ||२९७||
कसुमुद्बैतंकोऽनुमृशेषऽरुपकरमुष्यकिम्
तुर्वीरत्योतवरस्योतिन्द्रेतेलंसरिम्बजम् ||२९८||
कैयदेवनिघण्डु - ४. द्रवयग्नि- तेलसामान्यगुण
tैलंयोनित्वत्रिततेलवर्गुरुः
कषायानुसंतितंकपुरुसरस्सपाकतः ||२९६||
विकाशविशदसुखमुण्डसंस्पर्श्वीयोः
मेदोविकेशनकेशंतर्पणरक्तपितृक् ||३००||
विहस्तकेवलंकान्तफुकुंचदीपनम्
ब्रजजन्तुप्रमहचन्डवयाथिकफुकुंचच||३०१||
मेदामांसविद्यश्वरणमादिवशुककुकूल्
बद्रमृपुरीपञ्चगर्भमाशयविशेषधनम् ||३०२||
योनिकरणेषिरेशुलशमंतुभारकरम्
त्वंधोजितचक्षुषप्रभाग्नाशयुभिषोधनम् ||३०३||
श्रीकृष्णपुरीपञ्चवनातिस्वलितन्तुप्रवर्त्येत्
रूपादिकृत्यधवनसोतःसड्कोचतोयदि
रसीदसम्यकवहनकारण्यकृत्यदकायवर्धयत् ||३०४||
तेषुप्रविषंयसतःसौम्यस्तिंग्धत्वमादैवः
३०५
तैलंकृशानांतेनबृंहणम्
३०६
शनैःहकुञ्जतेतैलंतेनेा×थौषायनाशनम्
३०७
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३०८
शनैःहकुञ्जतेतैलंतेनेा×थौषायनाशनम्
३०९
सेकाभयाेगावाहेषुपाननावनवुंते
३१०
संकारेचाûनपानानांहयोïयिमदमेवǑह
३११
कैयदेविनघÖटु
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धाûयवगि-3. धान्यवर्ग-तिल

tilastailfal: पूर्व: स्नेहपुरफोलपर:

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til: कषायोमधुरस्तकः कुचकोरसः

विपाकेकटुकः स्वादु: सुस्निग्धोबलकृत्युः

हकुञ्जतेनेा×थौषायनाशनमः

दल्योस्तथायस्तिंग्धत्वमादैवः

तिलेशुषुकलः कृषणः प्रधानोमध्यमः सितः

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अन्योजनादयोऽजेयामृतातरस्तिताः||८३||
7.भावप्रकाश-पृंवेङ्कण-मिश्रप्रकरण - ९. धान्यवर्ग-तिल
tिल:कृष्णःसितोऽकःसवृंवाूपलितःस्मृतः
tिलोरसेकुस्तितकोमितारस्तुवरोगुः
विपक्कुकःस्वादुःसितस्वरःकपिलस्नुनुतः५३||
बल्यःकेश्योहिमस्वर्णस्तवच्छ्यःसवन्योऽणहितः
दल्ल्योऽल्पमुक्तद्याहीवातन्त्रोऽक्षिणमतिप्रदः||५४||
कृष्णःश्रेष्ठमस्तुपुरुषकलोधयमःसितः
अन्येर्र्र्नतःप्रोकास्तजसेरकादयस्तिताः||५५||
भावप्रकाश-पृंवेङ्कण-मिश्रप्रकरण - २०. तैलवर्ग-तैल
tिलादिस्तिःस्तवस्तुस्ततेनस्तैलरोगुः
ततुवातहस्तस्विशेषायितस्मभवः ||७||
भावप्रकाश-पृंवेङ्कण-मिश्रप्रकरण - २०. तैलवर्ग-तिलतैल
tिलतैलस्पृंवमृतवस्तुलरोगुः
वृष्ण्विकारिषिविशद्मपुराणस्थायोः||२||
सुष्ऩंकशायानुरस्तिकंवातक्कःफवः
वीर्योन्नाहिमस्वरूपुःहृणस्त्रिपितकृतः३||
लेखनस्वच्छविशिरंगमंवशस्यविशेषाधनम्
दीपलच्छुनिभंद्मितच्छमच्छवाधिग्रामेहनुतः४||
श्रृवंशोलिशिरःशूलकाशरलपुताकरम्
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छेन्न्योन्तुस्तोतिप्राणमितक्षतपितकियते
भगवस्फुटितविद्वाणिदधविषिठदारिते||६||
तथा|भिन्दनिर्मुखगमण्डलगत्यालाहादिविक्षते
|वस्तौपानेन्नसंस्कारत्वे|करणक्षिप्पूरणे||
|सेवकायिङ्गावाहेषुपुतिलतैलप्रशस्त्यते||
|स्त्रादिदृप:पयन:सोत:सहकोचयेददा||
|रसोस्मयगवहल्कारयुक्ताकालाध्यवधयन्||
|तेषुपवेशस्वसोक्तस्त्वस्मिन्धत्तमादः||
|तैलक्षमसंस्तुकुशानान्तेनबृंहणम्||
|व्यवायिषयसुखमतीक्षणोऽसर्वेषांदसःक्षयम्||
|शानेप्रकुसतेतेलसूचनेशमसेरितम्||
|दु:तपूर्णवधनातितिःक्षतितंप्रवत्येत्||
|ग्राहकंसारकर्षापितेनतैलमुदीरितम्||
|भावप्रकाशपूर्वनृङ्गप्रक्रमण:- २०. तैलवर्ग-धृत, तैलपरिभाषा
|घृतमवदात्यस्रंपकविहीनप्रशा|जयते
|तैलंपवमपवमचिदरस्थायिगुणाधिकम्||
V. Madhu/ Honey (Apisindica Mellifera)

The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern science.

1. Vedic review: 300-303
   - As a ahariya Dravya Atharvaveda. 300,303
   - As a Pranij Dravya Atharvaveda 9-1-1 to 24 301,303
   - As a Madhu, Saragh, Sapptamadhur Padarth Superior in category. Atharva Veda: 9-1-22, 23, 19, 17, 16, 4, 3, 2, 1 300,303

2. Samhita kalin review: 304-6

   I. Charaksamhita: 304
   - It is used for Mamsa and Shonitvardhan in Kshatakasheena chapter. (Ch Chi 11/30)
   - The Properties of Madhu are mentioned in Annapaanavidhi chapter (Ch Su 27/243-246)
   - It is used for the healing of open wound (Ch Chi 25/65)
   - It is constituents of Nimbadi kalka for shodhana and ropana of Vrana. (Ch Chi 25/83-85)
   - It is mentioned in Twakvishuddhikar yoga in Vranachikitsa (Ch Chi 26/114)

II. Sushrut Samhita: 305
   - The Properties of Madhu are mentioned in Dravadravyavidhi chapter. (Su Su 45/132)
   - It is use during operative process of Baddhagudodar and Chidrodar. (Su chi 15/17)

III. Ashtanga Hriday: 306
   - It is mentioned in Twakvishuddhikar yoga in Vranachikitsa. (A.U.25/58)
   - It is mentioned in kalka dravya for Vranalepa in Vranachikitsa. (A.U.25/55)
   - The Properties of madhu are mentioned in Dravdravyavidnyaneeyam chapter (AH .Su 5/52-53)
   - As a constitueion of Gandusha dravya for ropana in Mukharoga. (AH Su 22/7)

IV. Nighantu Kaal: 307
   - It is mentioned in Suvarnadi Varga of Dhanvantari Nighantu.
   - It is mentioned in Namasanghrah, Parishishtya of Shodhal Nighantu.
   - It is mentioned in Aushadhi Varga of Kaiyadev Nighantu.
It is mentioned in Ikshukadi Varga of Madanpal Nighantu.

It is mentioned in Paniyadi Varga of Raj Nighantu.

It is mentioned in Madhu Varga of Bhavprakash Nighantu.

5. Constituents: Honey has approximately 40% fructose, 30% glucose, 5% sucrose and 20% water. It also contains several amino acids, antioxidants, vitamins, minerals, glucose oxidase, which produces hydrogen peroxide, and gluconic acid, which gives the honey an acidic pH of 3.2-4.5.

6. Properties and action:

- **Rasa**: Madhur, Kashya
- **Vipaka**: Madhur
- **Virya**: Sheet
- **Guna**: Rukshya, Laghu, Grahi
- **Karma**: Lekhan, Vrushya, Pittavatghna, Stanya, Chakshushya, Mukharoghar, Varnya, Vranaropak, Krumighna, Atisaraghna, Shwasaghna, Kasahar, Kanthya.


8. Therapeutic uses: Mukharog, Netrarog, Shwas, Kas, Kshya, Atisar, Vrana, Kushta, Varnya, Bastidravya, Strotoshodhan.

9. Dose: 5-7 gm/day orally

10. Classification:

A. Classification According to Samhita:

Table No.47: Classification of Madhu according to Samhita:

<table>
<thead>
<tr>
<th>Charaka Samhita</th>
<th>Sushruta Samhita</th>
<th>Ashtanga Hridaya</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vamanopag Dravya</td>
<td>Lehaniya Gana</td>
<td>NiruhanVarga</td>
</tr>
<tr>
<td>Aahariya Dravya</td>
<td>Kumar Rasayan</td>
<td>Madhurskanda</td>
</tr>
<tr>
<td>Agryasangraha</td>
<td>Annapansangraha, Dravdravya</td>
<td>Dravapadarth</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mehaghanavarga</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gandush Dravya</td>
</tr>
</tbody>
</table>
B. Classification According to Nighantu: 

Table No. 48: Classification of Madhu according to Nighantu

<table>
<thead>
<tr>
<th>Nighantu</th>
<th>Varga</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dhanvanri Nighantu</td>
<td>Suvarnadi</td>
</tr>
<tr>
<td>Shodhal Nighantu</td>
<td>Parishishtya, Namasangraha</td>
</tr>
<tr>
<td>Madanpal Nighantu</td>
<td>Ikshukadi</td>
</tr>
<tr>
<td>Raj Nighantu</td>
<td>Paniyadi</td>
</tr>
<tr>
<td>Kaiyadev Nighantu</td>
<td>Aushadhi</td>
</tr>
<tr>
<td>Bhavprakash Nighantu</td>
<td>Madhu</td>
</tr>
</tbody>
</table>

C. Taxonomical Classification:

Table No. 49: Taxonomical Classification of Madhu

<table>
<thead>
<tr>
<th>Kingdom</th>
<th>Amimalia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phylum</td>
<td>Arthropoda</td>
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<tr>
<td>Class</td>
<td>Insecta</td>
</tr>
<tr>
<td>Order</td>
<td>Hymenoptera</td>
</tr>
<tr>
<td>Family</td>
<td>Apidae</td>
</tr>
<tr>
<td>Genus</td>
<td>Apis</td>
</tr>
<tr>
<td>Species</td>
<td>Mellifera</td>
</tr>
</tbody>
</table>

11. Vernacular names:

- **Sanskrit**: Madhu
- **Bengali**: Madhu
- **English**: Honey
- **Gujrati**: Madh
- **Hindi**: Shahad
- **Kannada**: Jenutupa
- **Malayalam**: Taen
- **Marathi**: Madha
- **Tamil**: Taene
- **Telugu**: Taene
- **Urdu**: Shahad
12. Parayayi Nama of Madhu according to Nighantu:

Table No.50: Parayayi Nama of Madhu according to Nighantu:

<table>
<thead>
<tr>
<th>Nighantu →</th>
<th>A.N.</th>
<th>D.N.</th>
<th>S.N.</th>
<th>M.N.</th>
<th>R.N.</th>
<th>K.N.</th>
<th>B.N.</th>
<th>N.A.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ParyayiNama ↓</td>
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<tr>
<td>Madhu</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Pushparasa</td>
<td>+</td>
<td>+</td>
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<td>+</td>
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<td>+</td>
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<tr>
<td>Pushpaasav</td>
<td>+</td>
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<td>Makarand</td>
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</tr>
</tbody>
</table>

13. Madhu Nirukti:

- Madhu (honey) is derived from the verb ‘dham’ (to blow) reversed.
- Madhu (honey) means Soma is derived from (the root) mad (to exhilarate) and is compared with soma (because the analogy of exhilaration). This other meaning of Madhu (wine) is derived from the same root also.

14. GunakarmatmakVivechana:

Rasapanchaka i.e. Guna, Rasa, Vipaka, Virya, and Karma. with these properties the drug can work. These are described in all Nighantus.

Table No.51: Gunapanchak of Madhu

<table>
<thead>
<tr>
<th>Properties</th>
<th>A.N</th>
<th>D.N</th>
<th>S.N</th>
<th>M.N</th>
<th>R.N</th>
<th>K.N</th>
<th>B.N</th>
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<tbody>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
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<td>Vishad</td>
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<td>Gunaka rma</td>
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<td>S.N.</td>
<td>M.N.</td>
<td>R.N.</td>
<td>K.N.</td>
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<td>Pitt-aughna</td>
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<td>Pitta-ghna Vata-ghna</td>
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<td>Stanyadoshhara</td>
<td>Vrushya</td>
<td>Shoshhara</td>
<td>Sho-shvinashi</td>
<td>Balya</td>
<td>Chaksus-hya</td>
<td>Chakshu, Bala, Varna-kruta, Shukral</td>
<td>Chakshu, Bala, Varna-kruta, Shukral</td>
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</tbody>
</table>
6. Shloka references of Various Nighantu for Paryayi nam, Gunapanchak, Dosha, Karma and Rogghanta of Madhu:

6. कैयदेवलिखितमधु - १. ओषधिप्रयगमधु

पुष्पासवः पुष्पसमुखिकाविद्वसारंगम

मालिकंपणीतिकंकॉलंध्रभणमधुजातयः।॥१७३॥

मालिकंसदल्वकाशपन्तिकंधुत्वरणकम्

क्षौंत्रतुकलिंवियात्थ्रभ्रमंशिशिवणकम्॥१७४॥

2. सामान्यमधुगुण

मधुस्वादु हिंमसंक्षणायानुसंगतधु

दीपलंवाहिकंसुधविद्वस्यवणविलेपनम्॥१७५॥

सौकुमारयकरंसुधविद्वससोतोविश्वधनम्

सूक्ष्ममधुकरंसुधविद्वसवरणम्॥१७६॥

विशंदरोवनखंसंप्रसादनजनजयेऽि

मेदंपितकमधुस्वाहिह्मेदमेधमिशवान्॥१७७॥

दीपंकायतिसरसस्त्रुणादहविषकृमीन्

कुष्ठाशरकपितद्वन्योगवाहिचवातलम्॥१७८॥

वातेकंद्वोगेशिवसुधुश्चयते॥१७९॥

2.धन्वतरिनिघण्डु - ६. सुधवादिवर्ग मधु

मधुसुंदमलुमधाकरंमालिकंकृमुसाववम्

पुष्पासवसारंधुतच्चपुष्पसशस्मृतम्॥२२७॥

मालिकंभभ्नञ्चारंपणीतिकंधृतकंतथा

आध्यमौदलकंदादलमित्यहमधुजातयः॥२२८॥

मालिकंपल्लवणस्यालंहारंत्रजलिंभवेऽि

पौतिकंधृतवर्णतुथेतभ्रमंसुधुच्यते॥२२९॥
आपीतवण्छानाक्रयंपित्साेइगंचाच्यंनामकम्
औद्यलिंस्यसहशंदालचपाललस्मृतम्॥२३०॥
7. सामान्यमधुगुण
कषायानुरसंधीतलमधुरम्
दीपनतेलखनत्यंग्रणरोपणमुषमम्॥२३१॥
सन्धानलघुच्छुष्यंसक्ष्यंहंत्रिनिर्देशनुत्
छदितिकविषशाकासकाशोपातिसरिजित्॥२३२॥
रक्षितहरुंग्नातिर्कतरूपोढहत्परम्॥२३३॥
8. धन्वन्तरिंघण्डु - ६. सुवणादिर्ग्राममधुम् (विशिष्टमधुगुण)
पौरिष्यत्सवादुरुप्तवादुभागरुपसस्द्धितम्
भामरुपकुस्तेजायमत्यत्तमधुरपुर्णत्॥२३३॥
धन्वन्तरिंघण्डु - ६. सुवणादिर्ग्रामक्षौंद्रमधु
क्षौंद्रविशेषात्सेयेशीतलमधुलेखनम्॥२३४॥
धन्वन्तरिंघण्डु - ६. सुवणादिर्ग्राममाक्षिममधु
तस्मालघुरुपक्षोङ्गाक्षिकप्रक्षरस्स्मृतम्॥२३४॥
उष्णविशृथुंतन्त्रविषालयंत्यालघु
उष्णातरण्रणायात्त्तिन्तितथाविषम्॥२३५॥
तत्सौकुमार्याच्चत्यावस्याेइयनींथपीव्यासमहवाच्च
उष्णविशृथुंतन्त्रविषालयस्तुतथासस्त्तरिक्षेणजलेवादिप्य॥२३६॥
4. मदनपालिंघण्डु - १. इक्षुकादिर्ग्राममधु
मधुपुप्पासव-पुप्पासोमाक्षिकमीरितम्
माक्षिकपातिकशीष्याम्रमधुपंद्वस्सरात्॥२३॥
माक्षिकलत्सुकाशपातिक्षसंहितमम्
क्षौंद्रकपिलवर्णस्याद्भामरस्त्वकोमक्षणम् ॥२४॥
मधुशीतंलघुवादु स्त्रंगाहिवितेखनम्
चक्रुप्यंदीपनस्यव्रणशोधनरोपणम् ॥२५॥
वण्यंमेधाकरुप्यंविशदरयेरनजयत्
कुष्ठशःकासपितासृकफमेहक्तमक्रिमीन् ॥२६॥
मदनुष्णावमिथासहिककातीसारहदहान्
दाहक्षतश्यास्तुयोगवान्लयवालम् ॥२७॥
मातिकंपटुषुशेखरनामयहरलघु
पीतिकंपुसरुष्णांपितदाहस्यातकृत् ॥२८॥
क्षौंद्रमातिकंपंयंविशेषान्मेहनाशनम्
भामरस्त्वकितहंमूजाड़यकरंगुरुः ॥२९॥
नविंस्यमथ्यभिध्यन्तिरितस्यश्वेष्वमहंरसरम्
5. राजरथयुतः १४. पाणीयादिवर्गः मधु
मधुश्रीद्वरंचमाश्रीकमातिकंकुसमासवम्
पुष्पास्यपितचप्यंपुप्परसाइयम् ॥११४॥
मातिकंभामरस्त्वातीर्थिताक्षात्रक्रत्वात्
आध्यायमौदालकेकालमित्यश्रीमधुजातयः ॥११५॥
नानाध्यायमधुरससाहारः कपिलवनमस्काः
या:स्थूलाभ्यक्षितपनस्यमधुमातिकमुच्चते ॥११६॥
येश्विनरघाङनगळोभा: पुष्पास्यपरायणः
भगरैजनितैैस्तुभामरमधुभणयते ॥११७॥
पिडंगनामितिका: सूक्षमा: शुद्धः श्रीतिहितिविश्रुतः
तामरस्तपदितं तु त्त्वापुस्रुपकृतयते ॥११८॥
अन्नजामशिकाःपिन्दुःपुत्रिकातितिकौतिताः
साधारण्यामधुहितततुल्यामधुशरकर्||१३३||
ऊषण्:सहोषणकालेवास्यमुणामथापिवाः
आममधुमुन्याणवाविषयवात्तादधायकम्||१३४||
कीटकादियुतममलद्धितत्यतचप्पर्युङ्गितकंमधुस्वत||
करण्कोटरगतन्त्रमेचक्कंतचगेहजनितजचदीपकृत||१३५||
दण्डेनिहित्:ययु:पातमापास्तदशंतांस्तव:निधंमधुरसायन:योऽगयोऽगम्||१३६||
हिन्दकागुदाङ्कुरश्रोफकफःृणादिदोषांभवतिदोषदमण्यथाचेत्
3.शोधलनिघण्टु-नामसङ्ग्रह (प्रथमभाग) - ९०. परिशिष्टमधु
क्षौद्रमधुमाशिक्षकमाक्षराकसारंवततततत||
पुष्पासव:पुष्पसोमकरन्दरसस्तथा||६||
गुणसङ्ग्रह (द्वितीयभाग) - १५. मधुवर्ग
मधुवर्ग
मधुस्वरहितमेध्यांतेलीखनदीपनम्||
चक्रुष्यांछिन्दिन्तृ:श्रेष्ठविषपिस्पितनुतु||७९५||
कुष्टमहकृमिकछिदिदशसात्मसारिजिततः
ब्रणैंधनसन्धानरोपणावात्तकोपनम्||७९६||
मधुरकटकंपाकेततुल्यामधुशरकर्||
VI. Ghruta
The drug review includes review from Veda, Ayurved Samhita, Nighantu and modern science.

1. Vedic review: \(^{314}\)
As a Aahariya Dravya.

2. Samhitakalin review: \(^{315-17}\)

I. Charak Samhita: \(^{315}\)
- It is mentioned in vranashodhan lepa in Dwivrameeya chapter (Ch Chi 25/83-85)
- It is used for Mamsa and shonitvardhan in Kshatakshena chapter (Ch Chi 11/30)
- It is mentioned for Ghritapaan in vaatpittaj jwara (Ch Chi 3/164)
- The properties of Ghrita are mentioned in Annapaanvidhi chapter (Ch Su 27/231-233)
- Indication of ghritapaan in various condition are mentioned in snehaadhyay (Ch Su 13/41-43)
- Sanskar Anuvartan quality of Ghrita is mentioned snehaadhyay (Ch Su 13/13)
- Properties of Ghrita as Dahashaman is mentioned in snehaadhyay (Ch Su 13/41-14)

II. Sushrut Samhita: \(^{316}\)
- The Properties of Ghrita are mentioned in Dravdravyavidnyaneeyam chapter (Su Su 45/96)
- The Properties of Cow Ghrita are mentioned in Dravdravyavidnyaneeyam chapter (Su Su 45/97)
- It is mentioned for snehapaan in different types of sdyovrana (Su Chi 2/23-25)
- It is mentioned for lepa, Parishek for dahashaman in sadyovrana (Su Chi 2/27)
- It is mentioned in Shashti Upakrama of Vranachikitsa (Su Chi 1/8)
- It is constituents of Shodhan ghrita for Pittaj dhah in deep wound (Su Chi 1/56)
- It is mentioned in nirwapan lepa chikitsa in shashtiupakrama of vrana (Su Chi 1/49)
- It is mentioned for the nirwapan of sadyovranachikitsa (Su chi 1/130)
- It is mentioned for Ropan karma in all types of Vrana (Su chi 1/79)
It is used during operative process of Baddhagudodar and Chidrodar (Su chi 15/17)

- It is mentioned in Twakvishuddhikar yoga in Vranachikitsa (A.U25/58)
- It is mentioned in kalkadravya for Vranalepan in Vranachikitsa(A.U25/55)
- It is constituents of Yashtimadhu Ghrita for Vedanasthapana in sadyovranachikitsa (AH U 26/6)
- It is constituents of Pittashamaklepa in sdyovranachikitsa (AH u 26/8)
- The Properties of Ghrita are mentioned in Dravdyavidnanyaneeyam chapter (AH Su 5/37-39)
- The Properties of Puran Ghrita are mentioned in Dravdyavidnyaneeyam chapter (AH Su 5/40)
- It is mentioned for ghrita sevana in Bheshaj ksheena sharir. (AH Su 5/30)

**III. NighantuKaal: 318**

- It is mentioned in ViprrakirnaPrakaran of AshtangaNighantu.
- It is mentioned in SuvarnadiVarga of DhanvantariNighantu.
- It is mentioned in Gunasagharah GhrutaVarga of Shodhal Nighantu.
- It is mentioned in PaniyadiVarga of MadanpalNighantu.
- It is mentioned in Drava ghrutaVarga of Kaiyadev Nighantu.
- It is mentioned in Mishraprakaran GhrutaVarga of Bhavprakash Nighantu.

**3. Constituents: 319**

Gross Composition of Ghee. Cow ghee Buffalo Ghee Fat 99.0—99.5% 99.0—99.5%
Saturated fat 46% cis-monoene 29% trans-monoene 7% Diene 13% Polyene 5%
¹riglycerides (triacylglycerols) SSS 42% 49% SSU 42% 39% SUU 14% 11% UUU
2% 1% Diglycerides (diacylglycerols) 4% Monoglycerides (monoacylglycerols) 1%
°nsaponifiable matter Cholesterol 300 mg Lanosterol 9 kg 100 g~1 Lutein 4 kg 100
g~1 Squalene 60 kg 100 g~1 Vitamin A 9 kg 100 g~1 Vitamin E 28 kg 100 g~1
Ubiquinones 6 kg 100 g~1 S"saturated, U"unsaturated. Table adopted from the works
reviewed by Achaya (1997).

**4. Properties and action: 318**

1) **Rasa** : Madhur
2) **Guna** : Snigdha,Sheet,Guru
3) **Virya** : Sheeet
4) **Vipaka** : Madhur
5) **Karma:** Balya, Rasayan, Vayasthapan, Jeevaniya,Bruhaniya, Indriyauttam, Vatpittaghana, Kantiojakar, Vranaropak.

6. **Important formulations:**
   Chavanprash, Kalyanakghruta, TiktaKghruta, Panchatikakghrutagugul.

7. **Therapeutic uses:**
   Jirnajwara, Yakshma, Rakttapitta, Unmmad, Apasmar, Netraroga, Vajeekaran, Shwas, Kas, Mutrakruchha.

8. **Classification According to Samhita:**

   ![Table No.53: Classification of Ghruta according to Samhita](image)

<table>
<thead>
<tr>
<th>Charaka Samhita</th>
<th>Sushruta Samhita</th>
<th>Ashtanga Hridaya</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bruhan dravya</td>
<td>Rakshoghna Gan</td>
<td>DravapadarthVarga</td>
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<tr>
<td>Snehan dravya</td>
<td>Bruhaniya Gan</td>
<td></td>
</tr>
<tr>
<td>Agryasangharah</td>
<td>Sarvapathyavarga</td>
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</tr>
<tr>
<td>Pittashamangan</td>
<td>KumarrasayanVarga</td>
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</tr>
<tr>
<td></td>
<td>Agryasanghrahah</td>
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<tr>
<td></td>
<td>VajeekaranVarga</td>
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</tr>
</tbody>
</table>

9. **Classification According to Nighantu:**

   ![Table No.54: Classification of Ghruta according to Nighantu](image)

<table>
<thead>
<tr>
<th>Nighantu</th>
<th>Varga</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ashtang Nighantu</td>
<td>Viprakirna Prakaran</td>
</tr>
<tr>
<td>Dhanvantari Nighantu</td>
<td>Suvarnadi Varga</td>
</tr>
<tr>
<td>Shodhal Nighantu</td>
<td>Gunasangraha Ghrut Varga</td>
</tr>
<tr>
<td>Madanpal Nighantu</td>
<td>Paniyadi Varga</td>
</tr>
<tr>
<td>Raj Nighantu</td>
<td>Ksheera Varga</td>
</tr>
<tr>
<td>Kaiydev Nighantu</td>
<td>Dravavarga Ghruta Varga</td>
</tr>
<tr>
<td>Bhavprakash Nighantu</td>
<td>Ghruta Varga</td>
</tr>
<tr>
<td>Nighantu Adarsha</td>
<td></td>
</tr>
</tbody>
</table>

10. **Vernacular names:**

    - **Sanskrit:** Ghrut
    - **Bengali:** Ghee
    - **English:** Clearified Butter
    - **Gujrati:** Ghee
11. Parayayi Nama of Ghruta according to Nighantu: 318

<table>
<thead>
<tr>
<th>Nighantu → ParayayiNama↓</th>
<th>A.N.</th>
<th>D.N.</th>
<th>S.N.</th>
<th>M.N.</th>
<th>R.N.</th>
<th>K.N.</th>
<th>B.N</th>
<th>N.A.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ghrut</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Ajjya</td>
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<td>Havi</td>
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<td>Sarpi</td>
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<tr>
<td>Navneetam</td>
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<tr>
<td>Ghrutalya</td>
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<tr>
<td>Jeevaneeya</td>
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<tr>
<td>Pavitra</td>
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<td>Amruta</td>
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<td>Snehottam</td>
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<td>Tejasam</td>
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<td>Abhidhara</td>
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</tbody>
</table>
12. Gunakarmatmaka Vivechana:318
Rasapanchaka i.e. Guna, Rasa, Vipaka, Virya, and Karma. with these properties the
drug can work. These are described in all Nighantus.

Table No.56: Gunapanchak of Ghruta

<table>
<thead>
<tr>
<th>Properties</th>
<th>A.N.</th>
<th>D.N.</th>
<th>S.N.</th>
<th>M.N.</th>
<th>R.N.</th>
<th>K.N.</th>
<th>B.N.</th>
<th>N.A.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guna</td>
<td>Snigdh</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Guru</td>
<td>+</td>
<td>+</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Sheet</td>
<td>+</td>
<td>+</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Rasa</td>
<td>Madhur</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>Virya</td>
<td>Sheet</td>
<td>+</td>
<td>+</td>
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<td>+</td>
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</table>

13. Dosha, karma, Rogghnata of Ghruta

Table No.57: Dosha, karma, Rogghnata of Ghruta:318

<table>
<thead>
<tr>
<th>Nighantu</th>
<th>A.N.</th>
<th>D.N.</th>
<th>S.N.</th>
<th>M.N.</th>
<th>R.N.</th>
<th>K.N.</th>
<th>B.N.</th>
</tr>
</thead>
</table>
| Dosha karma   | Vattapittagha
ana, | Vattapittaghana, | Vattapitta ghana, | Vattapittaghana, | vatpitthar         | vatpitthar         |vatpitthar, ka
p-hakar |
| Pra
mu
ka | Vrushya, Jeevaneeya, Vayastapan ,Balya, Rasayan,Dhee,Dhruti, Medha, Smruti | Snehan Brhun, Vrushya, Rasyan, Dhee, Dhruti, Smruti ,Medha | Vrushya, Vayastapan , Rasayan, Medha, | Snehan Brhun, Vrushya, Rasyan, Dhee, Dhruti, Smruti ,Medha | Snehan, Brhun, Vrushya, Rasayan, Dhee, Dhruti, Smruti ,Medha | Snehan, Brhun, Vrushya, Rasayan, Dhee, Dhruti, Smruti ,Medha |
| Karma        | Kshatseen aVisha, Chakshurog Vrana, | Udavarta, unamadaJwaraShool a ,Anaha, Vrana, rana, | Vrana, Chakshus
- ya,Rakta pitta,Vra
na,vatara
ga,Khata
ksheen,A
gnimand ya, Ajirna, udavarta, Ykshma. | Udavarta, unamadaJwara,Shoola ,Anaha, Vrana, | Udavarta, unamadaJwara,Shoola ,Anaha, Vrana, |
Karma and Rogghanta:318

1.अष्टाणगसिकाः - २५-विविभ्वक्यप्रकरण
घृतमाज्यःहि:सर्पः-नवनीतङ्गुलावलयः||३२||

2.धन्वन्तरिनिघटु - ६-सुवर्णादिवर्गघृत
घृतमाज्यःहि:सर्पः-पवित्रनवनीतजम्
अमृतांक्षिप्तार्थाधीनलीलायंप्रकीर्तितम्||४०||
सहस्रीयविधिवदृतकमसहस्रकृत:४१||
शस्त्रविद्वायप्रभालयः-शुकचक्रकाम्
बालवृद्धप्रजाकालित्सौकुमार्यस्थिरायिनाम्||४२||
क्षतकीणपरीरस्थातात्तिकितपितामहम्
विपाकेम्पुरुषकीर्तलयातपितिविवाहम्||४२||
चक्रुष्यांवल्यमन्यगच्छत्यसर्पिलुणोतरम्||४३||

3.शोढलिनिघटु-गुणसिर (द्वितीयाग) - १३-घृतवर्ग
घृतंहिंदीस्त्रृत्यगनिवलयः-शुकचक्रकाम्
बालवृद्धप्रजाकालित्सौकुमार्यस्थिरायिनाम्||५४||
क्षतकीणपरीरस्थातात्तिकितपितामहम्
वातपितिभोजनायायशालक्ष्मीवराहम्||५४||
स्लेहानामुनमंशीतवस्त्रापांरसम्
सहस्रविधिविकारचक्रसहस्त्रकृत:||५५||
पुराणान्तिरिहर्षीश्चर-करणाशियानिजान्
मदापस्मारमृत्युंग्यांशोधनरोपणम्||५५||
उग्रगणांपुराणस्याध्येदिनस्थत्वंधृत:||

149
लाक्षणिकंशीतंत्रविद्यंश्रवापहम्||७५२||
अपृथकारोपश्चाद्यंतरंश्चविशेषतः||
पूर्वकांथाधिकान्तक्यांशुरण्तदमृतोपमम्||७५३||
तदस्तिष्टतमण्डोःपिरुक्षस्तीक्षणस्तनुषां||
गृथयंततुष्कुशुष्क्यातिपितकपपहम्||७५४||
विपाकंभृतशीतलयमण्डगुणोपमम्
आज्ञूंचंदीपनीयंचक्षुष्क्यंवलयंचनम्||७५५||
कासेशास्नेरा्मणापप्पकक्षतलच्चु||
औषधकं:पृतपकृमेशोफोदरपपहम्||७५६||
दीपपणकायातच्चंचकुशुगुलमोदरपपहम्
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पूर्वेखण्ड-मिश्रप्रकरण - १८. घृतवर्ग

घृतमाज्यंधवि: सर्पि: कथयत्तेतदुणाधि

घूंतरसायलंस्वादु चक्षुप्यंवह्वङ्गिदिपनम्

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अल्पाभिध्यन्तिन्द्रकांत्योजस्तेजोलावणयुवद्विकृत्

स्वरस्वृतिकरंसेधमायुग्यंबलकृष् ॥२॥

उदायत्वरोहमादस्मुनाह्यणाह्यरेव्

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बल्यपविन्तमायुग्यंसुमुहंगल्यंनस्यनम्

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