Discussion


**DISCUSSION**

Many metabolic and hormonal changes occur in the body during pregnancy. These are the physiologic changes in response to pregnant state. Changes in the maternal carbohydrate metabolism is one of them, the overall effects of these alterations in maternal carbohydrate metabolism in normal nondiabetic woman are reduced fasting blood sugar and aminoacid levels, but increased postprandial blood sugars, free fatty acids, ketone, triglycerides and insulin secretions in response to glucose (Phelps et al). Though the insulin secretion is increased, but simultaneously body develops resistance to insulin. Women who are not able to increase the pancreatic insulin secretion sufficiently, to overcome pregnancy induced insulin resistance, develop gestational glucose intolerance.

Maternal hyperglycemia leads to various neonatal and maternal complications. Present study highlights various neonatal complications in infants born to mothers with abnormal gestational glucose tolerance.

Overall incidence of gestational diabetes is reported as approximately 2 percent (O' Sullivan et al). Deodari et al in their study reported the incidence of gestational diabetes as 55/11,920, while Tallarigo and associates in 1987 reported the incidence of impaired gestational glucose tolerance (GIGT) as 16%. In our study we found the incidenc
of GDM as 14%, and that of IABG and GIGT as 12% each. Incidence of GDM was found to be higher in our study, as compared with earlier studies, while incidence of GIGT was nearly similar to the observations by Tallarigo.

The major finding of this prospective study of infants born to mothers with abnormal gestational glucose tolerance was that macrosomia was the most frequent complication. It was found in 44.4% infants of GDM, 24.3% infants mothers with IABG and 16.6% infants of mothers with GIGT. In earlier studies Deodari and Coworkers reported 34.5% incidence of macrosomia in IGDM. E Stenninger has reported 27% incidence of macrosomia in insulin treated GDM. While Tallarigo has reported the macrosomia in 27.5% of infants born to mothers with GIGT. Our observations possess a quite similarity with previous studies. Postulated mechanism of macrosomia is that, maternal hyperglycemia results in fetal hyperglycemia and excessive stimulation of fetal pancreas to produce insulin. Insulin facilitates the transport of nutrients like glucose, aminoacids and free fatty acids into cells leading to increase in number and size of cells (Pederson et al). Arginine or Leucine in the presence of glucose can markedly enhance the fetal insulin release. With effective control of metabolic derangements in mothers, it is possible to prevent macrosomia in their babies.
Another important finding of our study was congenital malformations. We found that 2 infants (14.4%) out of 14 IGDM had major congenital malformations and one infant (8.3%) out of 12 infants of mothers with IABG had congenital malformation. Thus total 3 infants of mothers with abnormal GTT had congenital malformation, one had anencephaly, one had tracheoesophageal fistula and one had multiple small anomalies. None of the woman with normal GTT gave birth to congenitally malformed baby. This is probably because the incidence of congenital malformations in general population is very low, that of anencephaly is 1/1000 live births and tracheoesophageal fistula is 1/3000-4500 live births. Thus from the above observations it seems that incidence of congenital malformations is higher in infants born to mothers with abnormal GTT as compared with infants of mothers with normal GTT. Difference was found statistically significant (P<0.05) also. A.Y. Ranade, A.K. Deodari and Tallarigo has reported a somewhat lesser incidence of congenital malformations in infants of mothers with abnormal GTT, while Joslin's clinic has reported the incidence of congenital malformations as 9% major and 5% minor in IGDM. Our observations are near to the observations by Joslin's clinic. The cause of congenital malformations is probably related to alteration in metabolic milieu in early pregnancy,
kenone bodies in combination with glucose are responsible for teratogenic effect (Joslin's diabetes clinic). For this reason good control of diabetes is essential in earliest possible weeks of pregnancy.

Increased incidence of hyperbilirubinemia is another frequent complication of diabetic pregnancies. The cause of it is presumed to be related to functional prematurity of hepatic enzymes (Osler & Coworkers). Hyperbilirubinemia was present in our study in 14.4% infants of GDM, 16.6% infants of mothers having IABG and 16.6% infants of mothers having GIGT, while jaundice was present in 7.7% infants of mothers with normal GTT. Pederson and Coworkers noted the hyperbilirubinemia in 38% infants of GDM. Similar observations was done by Essex and Coworkers. Moshe Hod et al has reported the prevalence of hyperbilirubinemia as 8.2 - 16.1%, this observation is nearly similar to our observation.

Another important cause of neonatal morbidity and mortality in infants born to mothers with abnormal GTT is respiratory distress syndrome. Epstein and Coworkers in their study concluded that maternal hyperglycemia leads to fetal hyperglycemia resulting in fetal hyperinsulinemia and this results in reduction in ability of fetal lungs to synthesize, store and release lecithin, the principal component of surface active material in lungs.
A.Y. Ranade and Associates in their study reported 7% infants with RDS in GDM, similar observations was done by Robert and Associates. Our observations was nearly similar to earlier observations, as we found RDS in 14.4% infants of GDM, 8.3% infants of mothers with IABG and 8.3% infants of mothers with GIGT, while only 3.1% infants of mothers with normal GTT had RDS. Thus the frequency of RDS seems higher with abnormal GTT as compared with normal GTT.

Prematurity was reported in 5% IGDM by A.Y. Ranade et al, while Deodari et al had reported prematurity in 20% infants of GDM. But, we did not found any case of prematurity in GDM, while 16.6% infants of IABG and 16.6% infants of GIGT were premature. Various factors like maternal hydramnios, macrosomia, placental insufficiency are incorporated in the etiology of prematurity.

A common problem in infants born to mothers with abnormal GTT is reported as early postnatal hypoglycemia, Secondary to excessive insulin secretion after division of umbilical cord and termination of placental transfer of glucose. Hypoglycemia occurs most frequently 2 hours after birth (E. Stenninger et al). In our study hypoglycemia was found in 7.2% IGDM as compared with 1.5% infants of mothers with normal GTT. So the frequency of hypoglycemia appears higher in IGDM. Near similar observations was done by A.Y Ranade et al and A.K. Deodari. studies indicate that risk of
neonatal hypoglycemia increased with increasing maternal
blood glucose at delivery (Kuhl et al). Thus good control
of maternal hyperglycemia at term can reduce the risk of
neonatal hypoglycemia.

Since very little data are available regarding neonatal
complications in IABG and GIGT, we could not compare our
findings with other studies except macrosomia, which was
studied by Tallarigo in GIGT.

Though RDS, hypoglycemia, hyperbilirubinemia were
found with higher frequencies in infants of mothers with
abnormal GTT, the number of cases was small and no statistical
correlation was found. We believe, it will be necessary to
study a large number of cases to evaluate a possibility of
a relation between abnormal gestational glucose tolerance
and various neonatal complications.

Prevalance of polycythemia was reported 3.8 - 13.8% in
IGDM by Moshe Hod et al, similar observations was done by
A.Y. Ranade and Coworkers and A.K. Deodari. Prevalance of
polycythemia (Hematocrit) in normal neonatal population is 1-2
percent. But in our study, we did not found any case of
polycythemia in either study or control group. Though appar-
etly mean Hb level seemed higher (15.15 gm) infants born to
mothers with abnormal GTT as compared with infants born to
normal GTT (Mean Hb 11.15 mg/dl). But the difference was
not statistically significant.
Following advances in the management of gestational diabetes and fetal monitoring, perinatal mortality has responsibly decreased. A study conducted by Gabbe et al in 1977 showed that perinatal mortality was nearly 19/1000 in IGDM. Deodari et al reported perinatal mortality as 3.5% in IGDM. In our study perinatal mortality was 21.2% in IGDM. No mortality was found in other two categories of abnormal gestational glucose tolerance, while in normal mothers perinatal mortality was 6.2%.

Commonest cause of perinatal mortality in our study appeared to be major congenital malformations as two infants out of 3 had major congenital malformations in the form of anencephaly and tracheooesophageal fistula. This observation was similar to observations by Pederson, who showed that 40% of perinatal mortality in IGDM is due to major congenital malformations. One infant died of respiratory distress syndrome, which is another major cause of mortality in infants of diabetic mothers (Driscoll et al).

The goals of care in the management of pregnant diabetic are to achieve adequate metabolic control throughout pregnancy,
as it is known to be associated with improve perinatal outcome (Karesson et al).

Since fetal hyperinsulinemia is implicated in most of the adverse neonatal outcome, management of abnormal gestational glucose tolerance is aimed at the prevention of fetal hyperinsulinemia.

American College of Obstetrician & Gynaecologists and American diabetes association suggest that fasting plasma glucose should be maintained below 105 mg/dl and 2 hour post prandial values below 120 mg/dl for gestational diabetic pregnancies. Gestational diabetics can be managed by dietary modifications in most of cases, only 10-15 percent of gestational diabetics require insulin therapy (Ranade et al)

In our study most of the antenatal mothers came at term and delivered within one or two days, so no effective treatment could be given to these mothers. Only 9 women with abnormal glucose tolerance approached before term, and they were first treated on dietary regime, but dietary therapy failed to achieve required glycemic control in four women and these women were treated with insulin therapy.

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