1. INTRODUCTION

Cardiovascular diseases (CVDs) especially, coronary artery disease (CAD) has become an epidemic worldwide and a major barrier to sustainable human development. According to “The 2017 Heart Disease and Stroke Statistics update” of the American Heart Association (AHA), it has been recently observed that around 16.5 million people who are above 20 years of age in United States of America (U.S.) suffer from CAD. Not only that, the prevalence increases in both the genders with a gradual increase in the age (Sanchis-Gomar, Perez-Quilis et al. 2016). The incidence in the developing countries like India is also alarming. In India, studies have reported a boost in CAD prevalence since past half century, from 1% to 9-10% in urban populations and <1% to 4-6% in the rural populations. There are a number of established key modifiable and non-modifiable factors like age, gender, genetics, smoking, dyslipidemia, hypertension, diabetes, obesity, high-fat diet, physical inactivity, drug abuse, alcohol consumption and mental stress attributing significant risk towards the disease.

An individual’s risk of harboring CAD is inflicted by the interplay between genetic and lifestyle factors established by the multifactorial nature of CAD. Genetic component in CAD is validated from the increased risk in the first degree relatives of the affected individuals, high lifetime risk in the offspring if parents are affected and high concordance in monozygotic than dizygotic twins. The first Genome Wide Association (GWA) studies for CAD were published in 2007 and since then, a number of genetic variants at various chromosomal loci specific to CAD in various populations have been identified (Scheffold, Kullmann et al. 2011).

Atherosclerosis and inflammation are the key processes in the development and progression of CAD and therefore, any changes occurring at the genetic level in both the processes hold a clinical significance particularly for developing preventive or therapeutic interventions for CAD. Analyzing the single nucleotide polymorphisms (SNPs) which are substantially associated with CAD in North Indian population will be useful to identify the promising SNP-CAD associations unique to the population. Moreover, CAD poses threat not only to an individual and his family but also to the community and the nation on the whole as the most productive years of one’s life is spent struggling with the disease.
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The drastic change in lifestyle and eating habits and the increased tendency to rely on machines and other forms of assistance has substantially decreased one’s physical effort and rendered the individuals highly susceptible to CAD. Comprehending the genetic foundation of CAD in the individuals is highly needed these days that will help in screening individuals at high risk and will also lay the groundwork for the coacervation of genetic data and routine clinical practice, which can one day spearhead the arena of “personalized medicine”.

Therefore, the present case-control study is designed to look into the genetic aspects of CAD by studying the SNPs in the genes involved in atherosclerosis and inflammation in the North Indian population. Additionally, an attempt has been made to study the gene-gene interactions as well as the gene-environment interactions to better understand the involvement of various genetic and environmental factors owing to the multifactorial nature of CAD.