The year 2013 celebrated World Health Day marked by the theme hypertension. This fact exhibits not only the level of awareness among public but also rapidly rising cases of hypertension at a global level. Essential hypertension, as a risk factor has attracted many specialties of medical background due to broad range of clinical complications from cerebrovascular diseases to renal discomfort. Many markers selected on the basis of their physiological mechanisms were studied during the past several decades. Though most of them returned inconsistent results when replicated in various populations many of them did show a significant association with EH. Numerous common variants or rare variants remain to be discovered.

The present study is a case-control analysis including eleven markers such as rs2681492 and rs2681472 of plasma membrane calcium ATPase (ATP2B1) gene, rs11024074 of pleckstrin homology domain, family A, member 7 (PLEKHA7) gene, rs1052501 of unc-51-like kinase 4 (ULK4) gene, rs7200009, rs16960421, rs17177428 and rs11860907 of cadherin 13 (CDH13) gene, rs3184504 of lymphocyte-specific adapter protein coding gene (SH2B3), rs1474868 of mitofusin-2 (MFN2) gene and rs2286245 of solute carrier family 6, member 9 coding gene (SLC6A9).

- Among the markers studied, rs2681472 of ATP2B1 gene, rs7200009 and rs17177428 of CDH13 gene, rs3184504 of SH2B3 gene produced significant difference between the normotensive controls and hypertensive cases.
- The TT homozygous genotype and T allele of rs2681472 was found to be the risk genotype and allele. Gender based analysis also revealed that the T
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allele increases susceptibility of hypertension in females when compared to males. As predicted by the odds ratio, an individual with TT genotype or possessing T allele is 1.2 times more susceptible to essential hypertension, when compared to individuals with CC homozygous genotypes.

- The TT homozygous genotype of CDH13 gene (rs7200009) was found to be the risk genotype and allele in the population studied. This is evident from the recessive genetic model studied. TT homozygous individuals are 1.4 times at risk when compared to individuals with other genotypes. Similarly, the risk estimate remained the same even after adjusting for BMI. Further, the C allele was found to be the protective allele, which is evident from the additive model studied. TT genotype was also found to be an independent risk factor EH in females when compared to males in the population studied.

- Significant difference was observed in the case of rs17177428 marker between the two data sets studied. The GG homozygous genotype was found to be an independent risk factor for EH regardless of gender and BMI. Risk estimated from the GG genotype of dominant model is about 1.4 times when compared to GA or AA genotypes. In addition to the above results deviations from HWE were observed in three out of four markers on the CDH13 gene studied. Other SNP markers studied were in HWE. These deviations could be attributed to selection, drift, population admixture or forms of non-random mating.

- The CC homozygous genotype of rs3184504 marker was found to be the risk genotype after adjusting for BMI in females alone. The CC genotype risk as
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estimated from the dominant model in females after adjusting for BMI is 1.4 times in comparison to other two genotypes. The additive model estimates a risk of 1.3 times for C allele when compared to the T allele. Thus, the CC genotype and C allele were found to be the genetic risk factors associated with hypertension.

In conclusion, the present study shows the association of a few markers with essential hypertension in this population. Inconsistency in the results could be due to genetic and environmental heterogeneity among different ethnic groups. Since a wide variety of physiological systems exhibit pleiotrophic effects and interact in complex fashion to influence BP, the effect of the variants studied may be masked by the effect of unstudied variants. Thus, multiple variants need to be analyzed to decipher the molecular profile of this population. The knowledge acquired from the genetic association studies coupled with further functional studies will aid us to understand the underlying biological mechanisms of the observed associations and develop efficient preventive strategies or therapy for essential hypertension.