

52nd CARDIOCON 2023: ABSTRACT**CYTOCHROME P450 2C19*2 GENETIC POLYMORPHISM IN PATIENTS WITH ACUTE CORONARY SYNDROME****Nazeef Ullah¹**¹Hayatabad Medical Complex, Peshawar, Pakistan

Objectives: The study aimed to investigate the association between CYP2C19*2 loss of function polymorphism and acute coronary syndrome (ACS) patients receiving Clopidogrel. It compared genotype frequencies of heterozygous GA, homozygous GG, and homozygous AA between ACS patients and healthy controls. The study assessed the statistical significance of the association in the Pakistani cohort. It contributed to understanding the role of genetic polymorphisms in response to Clopidogrel therapy in ACS patients and provided insights into the need for pharmacogenomics testing for patients with CAD, particularly in the Asian population, who are poor metabolizers and require long-term Clopidogrel therapy.

Methodology: COHORT Study.

Results: The study found no statistically significant association between the CYP2C19*2 loss of function polymorphism and acute coronary syndrome (ACS) in the Pakistani cohort. The frequency of the CYP2C19*2 heterozygous GA genotype was 38.3% in ACS patients and 29.5% in control subjects, while the homozygous GG genotype was 58.7% in patients and 59.0% in controls. The homozygous AA genotype was 3.4% in patients and 11.4% in controls.

The genotype frequency analysis showed that the wild type GG genotype was lower in patients (58.7%) compared to controls (59.0%), while the mutant AA genotype was also lower in patients (3.4%) compared to controls (11.4%). The heterozygous GA genotype was higher in patients (38.3%) compared to controls (29.5%).

The population studied was in Hardy Weinberg equilibrium, with a frequency of mutant allele A at 0.26 and wild type allele G at 0.73.

Conclusion: The study concludes that the CYP2C19*2 loss of function polymorphism is not associated with acute coronary syndrome (ACS) in the Pakistani cohort. The distribution of the CYP2C19*2 genetic polymorphism was not statistically significant between ACS patients and healthy controls.

The genotype frequency analysis showed no significant differences in the distribution of the heterozygous GA, homozygous GG, and homozygous AA genotypes between the patient and control groups.

The population studied was in Hardy Weinberg equilibrium, with a frequency of mutant allele A at 0.26 and wild type allele G at 0.73.

Keywords: Genetic polymorphism, CYP2C19, acute coronary syndrome, restriction fragment length polymorphism

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