The Indian Journal of Basic and Applied Research
Volume 1 |Issue 4| Feb 2016
Online & print@ www.ijbar.co.in
E- ISSN No: 2454-4639
International Innovative Journal Impact Factor (IIJIF)

ACE Gene Polymorphism in Cardiac Patients: An Enzymatic Experimental approach.

Dr. Hamendra Kumar Verma
Director, Lt. RKD women College Budwan Khaga Fatehpur India
vermahk@hotmail.com

Abstract:
Myocardial hypertension and infarction are multifactorial being influenced by both genetic and environmental factors. The angiotensin converting enzyme (ACE) gene has been recognized as a top candidate gene for cardio vascular research, the critical role of angiotensin converting enzyme gene in blood pressure regulation. Keeping in this view the expression of ACE deletion (D) and ACE insertion (I) alleles in the renin angiotensin system by means of polymerase chain reaction in the hypertensive patients, with coronary artery disorders (CAD).

Keywords: ACE gene, myocardial infraction, CAD, Hypertension.

Introduction:
Angiotensin converting enzyme (ACE) is a zinc-metallopeptidase which converts Angiotensin I to Angiotensin II (vasoconstrictor) and that degrades bradykinin (vasodilator), both for regulation of vascular tone and cardiac functions. ACE gene has been cloned earlier decades and identifies as somatic ACE resulting from gene duplication and expressed primarily in endothelial cells and germinal or testicular ACE. Myocardial hypertension and infarction are multifactorial being influenced by both genetic and environmental factors. The angiotensin converting enzyme (ACE) gene has been recognized as a top candidate gene for cardio vascular research, the critical role of angiotensin converting enzyme gene in blood pressure regulation. Between 1968 & 1976 the age adjusted mortality from CAD in United States progressively declined by 20%. Goldman & Cook in 1984 estimated that more than half of that decline was related to lifestyle changes, especially to decrease in serum cholesterol and cigarette smoking. Keeping in this view the expression of ACE deletion (D) and ACE insertion (I) alleles in the renin angiotensin system by means of polymerase chain reaction (PCR) in the hypertensive patients, with coronary artery disorders (CAD).

Methods:
Eighty patients diagnosed by Dr. Mahesh Yadava, Department of Medicine at Adarsh Hospital Khaga Fatehpur, were evaluated for cardiac disease. All these patients were included in present study. However the samples of matched control subjects were provided by Department of Genetics Govt. Medical College Allahabad. The controls were subjected to treadmill test to be sure that they were not suffering from any disease.

Results and Discussion:
Total 80 clinically diagnosed CAD patients were included in the present study and 50 normal healthy individuals were included in the control group. PCR amplification of genomic DNA produced fragments of 490 & 190 base pair yielding the genotypes II, ID & DD respectively. In the present study total of 30 chromosomes were found to be linked for I polymorphism and 70 chromosomes were found to be associated for D polymorphism in the normal group. Strong evidence exists for an association between the ACE gene insertion/deletion (I/D) found in intron 16 and plasma ACE activity, with increased levels found among persons with deletion allele (Rigat *et. al.*, Tiret *et. al.*, Zhu *et. al.*). Niu *et. al.* has shown a positive association between the DD genotype and its correlation with serum ACE activity in myocardial infraction patients. They reported a moderate impact of ACE polymorphism on the cardiovascular response to ACE inhibitor. However no definite consensus as to which allele confers a more pronounced effect is indicated. A strong association between these alleles and the level of serum ACE was detected. According to Rigat *et. al.* individuals homozygous for the D allele (genotype DD) displays serum ACE levels almost twice as high as individuals for the II alleles, meta-analysis confirmed that the ACE D allele could play a role in pre-disposition to acute brain ischemic events and to progressive deterioration of renal function in insulin dependent as well as in non-insulin dependent diabetes mellitus (Yoshida et. al.). The insertion & deletion (I/D) polymorphism of a 287- bp Alu elements in intron 16 of

E- ISSN No: 2454-4639

the ACE gene has attracted significant attention and has been extensively investigated in a spectrum of cardiovascular phenotypes because its correlation serum ACE activity.

References:

1. Kee F. *et. al.* (2000), Angiotensin II type I receptor and ACE polymorphism is a risk of myocardial infraction in men and women. *Europ. J Clin Invest*. 30, 1076-1082.
2. Niu, Marriam *et. al.* (1997), Genetic markers: Gene involved in human hypertension. *J Cardiovascular Risk*. 4, 341-345.
3. Rigat B. *et. al*. (1990), An insertion/deletion polymorphism in the angiotensin I converting enzyme gene accounting for half the variance of serum enzyme levels. *J Clin. Invest*. 86, 1343-1346.
4. Zhu X. *et al.* (2000), Localization of a small genomic region associated with elevated ACE. *Am. J. Hum. Gene*. 67, 1144-1153.