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RESEARCH ARTICLE

ORIGINAL RESEARCH ARTICLE: STATUS OF GGT, URIC ACID AND SERUM ELECTROLYTES IN MYOCARDIAL INFARCTION

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ABSTRACT

Background: Cardiovascular diseases are the first leading cause of death for adult men and women in developed countries. Surprisingly in developing countries CVD also become the first leading causes responsible for one third of all deaths.
Aims and Objectives: To evaluate the level of Seum GGT,S.uric acid and S.electrolyte in myocardial infarction patients.
Material and Methodology: 100 patients of MI and 50 age matched healthy controls are involved in this study. Estimation of GGT, Uric acid, S.electrolyte was done from all participant. Results obtained are analysed statatically by calculating p-value using online student t-test calculator. P-value less than 0.01 was consider as a significant.
Result:S.GGT(IU/L),S.uricacid(mg/dl),S.sodium(mmol/l),S.pottasium(mmol/l),S.sodium(mmol/l),S.pottasium(mmol/l),S.chloride(mmol/l) in case group is 28.12±14.72, 7.11±1.08, 135.45±2.16, 3.43±0.35, 105.97±3.6 respectively and in control group is 11.09±3.8, 4.2±0.79, 142±2.76, 4.5±0.37, 103.62±2.01 respectively
Conclusion: In conclusion our study indicated a direct association between serum GGT and uric acid in MI. Although GGT is often measured as a marker of liver health, this study provides evidence that it may also be useful in the identification of patients at elevated risk of MI.

Key Words: GGT, Uric Acid, Electrolyte, MI

INTRODUCTION

Myocardial infarction is also known as heart attack. It is a condition of heart muscles death when one or more coronary arteries which supply oxygen-rich blood to the heart muscle become suddenly blocked (Lee and Goldman, 1986). Blockage results from plaques made of fats and cholesterol. The accumulation of this plaque is known as coronary artery disease (Harem, 2010). The accumulation of plaque is a process and also can produce chest pain symptom known as angina pectoris. A myocardial infarction occurs when a plaque rupture suddenly and it causes a rapid accumulation of clotting factors at the rupture site which leads a sudden obstruction of blood flow in the coronary artery. In India incidence of CVD is greater in urban population than in rural population (Gupta et al., 2008). In 2004, CVD was the leading cause of death in India, leading to 1.46 million deaths (14% out of a total of 10.3 million deaths) 130.7 deaths per 100,000. According to projection the number of deaths due to cardiovascular diseases worldwide were 7.2 millions (12.2% out of a total of 58.8 millions deaths), 134.0 deaths per 100,000 (Huffman et al., 2004). In India, CVD is projected to be the largest cause of death and disability by 2020, (WHO, 2002) with 2.6 milliom Indians predicted to die due to coronary heart disease, which constitutes 54.1% of all CVD deaths. Nearly half of these deaths are likely to occur among young and middle aged individuals (30-69 years) (Pareek et al., 2014). This has the potential to adversely affect India's economy with 52% of CVD deaths occurring in those below the age of 70 years compared to 23% of countries in established market economics.

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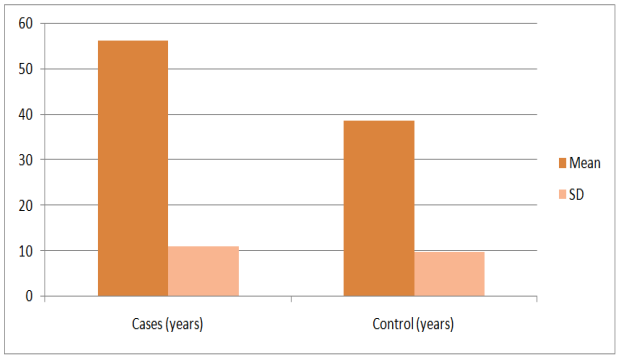
Although uric acid can act as an antioxidant, excess serum accumulation is often associated with cardiovascular disease (Solmon et al., 1981). The present study has been undertaken to evaluate status of GGT uric acid and electrolytes in MI patients, to verify the role of GGT, uric acid and electrolyt.

MATERIALS AND METHODS

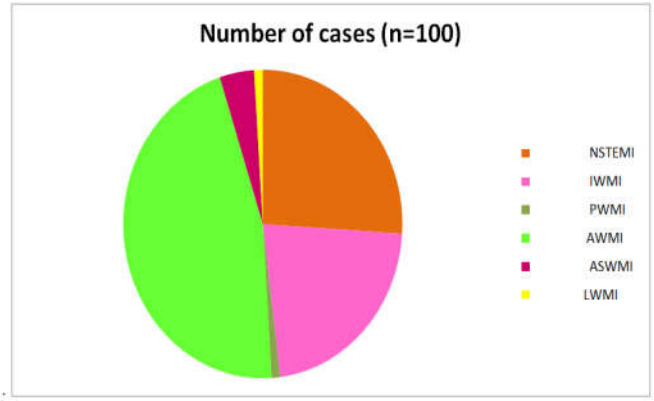
The present case control study was conducted on 100 patients of Myocardial infarction who were admitted in the Department of Cardiology (CCU) of Geetanjali medical college and hospital after obtaining permission of ethical committee. 50 healthy subjects were selected from same instution. Group A (study group)- study group were consisted of 100 patients had MI of different age group (n=100).Group B (control group) – study group were consisted of 50 volunteers had same age & sex matched healthy control (n=50).

Exclusive criteria: Cofounding factors which could interfere in the biochemical analysis of study subjects and alter the results are active inflammatory diseases, nutritional deficiencies patients undergoing treatment for any liver disorder, kidney failure, thyroid disorder, malignancy and autoimmune disease other than diabetes, alcoholics, smokers.

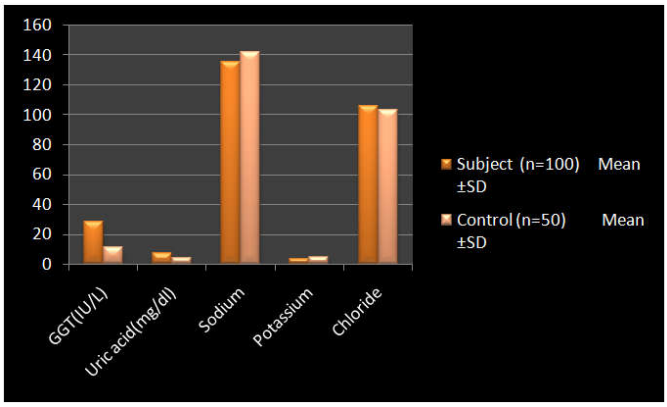
After obtaining informed consent 10 ml blood was collected from all participants. The collected samples were incubated at 37^{o}C for 15 minutes in the incubator and then centrifuged for 10 minutes at approximately 3000 rpm and serum. The serum separated from the samples will be analyzed for following biochemical parameters- GGT, Uric acid, and Electrolytes.



Graph 1. Showing age wise distribution between study group and control group



Graph 2. Showing the type of MI according to ECG pattern among study group



Graph 3. Graphical presentation of the mean concentration of various biochemical parameter in both case and control group

RESULTS

The present case control study was conducted on 100 patients of Myocardial infarction who were admitted in the Department of Cardiology (CCU). Out of 100 patients there were 67 males and 33 females.

Table 1. Showing male and female distribution between study group and control group

Sex	Cases (n=100)	Control (n=50)
Male	67%	55%
Female	33%	45%

Table 2. Showing age distribution between study group and control group

Age (years)	Cases (years)	Control (years)
Mean	56.41	38.64
SD	10.98	9.87

Table 3. Smoking status among study group and control group

Smoking status	Cases% (n=100)	Control% (n=50)
Smoker	30%	5%
Non smoker	70%	95%

Table 4. Showing alcohol drinking status among study group and control group

Alcohol drinking	Cases% (n=100)	control (%) (n=50)
Alcoholic	24%	4%
Non alcoholic	76%	96%

Table 5 .Showing type of MI according to ECG pattern among study group

ECG indings	Number of cases (n=100)
NSTEMI	26
IWMI	22
PWMI	1
AWMI	46
ASWMI	4
LWMI	1
TOTAL	100

Table 6. The mean concentration of various biochemical parameter in both case and control group

Parameter	Subject (n=100) Mean ±SD	Control (n=50) Mean ±SD	P- value
GGT(IU/L)	28.12±14.72	11.09±3.8	0.0001
Uric acid(mg/dl)	7.11±1.08	4.2±0.79	0.0001
Sodium	135.45±2.16	142±2.76	0.0001
Potassium	3.43±0.35	4.5±0.37	0.0001
Chloride	105.97±3.6	103.62±2.01	0.0001

The mean age of patients were 56.4±10.98, (n=100) whereas in controls, it was 38.64±9.87 (n=50).

DISCUSSION

In the present study there were 57 patients from urban area and there were 43 patients from rural population. Similar type of pattern was observed by Gupta, et al. 1994. These results are collaborating with these workers who also reported similar observations (Ruttmann et al., 2005) suggested a positive association between serum GGT and the risk of heart failure, especially in those aged<60 years. A similar trend was also observed in two other studies on the association of GGT with CVD (Lee et al., 2003) found that distribution of increased GGT values in the female and male patient groups did not differ significantly (Flear and Hilton, 1979) found that the prognostic role of serum GGT in older subjects was not as strong as in younger subjects. Gandiah et al., 2013, concluded that Elevated SUA levels have been associated with an increased risk for cardio-vascular disease. The potential mechanisms by which SUA may directly cause cardiovascular risk include enhanced platelet aggregation and inflammatory activation of the endothelium.

In few studies, the association of SUA with cardiovascular disease was uncertain after multivariate adjustment as in the Framingham Heart study (1985) and the ARIC study (1996), but in the other association remained certain and significant. Two of the previous studies have reported that a high concentration of uric acid is a strong marker of an unfavorable prognosis of moderate to severe heart failure and cardiovascular disease. Large cohort studies have shown that uric acid is an important independent risk factor for association between cardiovascular mortality.

The role of uric acid in coronary heart disease is less clear. Some studies reported an association between uric acid and coronary heart disease but others only found an association in women, and in yet others, the associations disappeared after adjustment after adjustment for confounders. Because elevated serum uric acid is correlated with several risk factors including renal dysfunction, hypertension insulin resistance, hyperhomocystinemia and hyper-lipidemia,it is debated whether SUA is an independent cardiovascular risk factor. The results of epidemiologic studies have been contradictory, although most conclude that serum uric acid is a risk factor for CAD. Electrolyte imbalance are fairly common in the acute phase of myocardial infarction patients. When measured within 48 hours of admission (Fauci and Braunwald, 2012).

The sodium potassium levels were found to be significantly reduced in our study when compared to control group. Hypokalemia was evident in a large number of patients in MI, hypokalemia has been found to be associated with an increased risk of ventricular tachycardia and ventricular fibrillation. Hypokalemia prolongs ventricular repolarisation, often with prominent U waves (Jeldsen, 2010). The incidence of ventricular fibrillation has been found to be five – fold higher in patients with a low serum potassium[11]. Skeletal muscle acts as a reservoir pool for potassium, maintaining potassium in vital organs such as the heart and brain. This hypokalemia is mostly due to the stress induced catecholamine response (predominantly epinephrine from the adrenal medulla) that function as hormones, in such patients causing increased K^+ uptake into cells.

The beta 2- adrenoceptor agonists stimulate Na^+ /K^+ pump mediated potassium uptake in skeletal muscles of experimental animals and humans [11]. A study done by Goyal et al found the mortality in MI patients to be the least in patients with potassium levels of (3.5 – 4.5 mmol/l), thus showing a relation between the hospital mortality mad mean post admission levels. Hyperkalemia too has been shown to be associated with reduced ventricular excitability and other causes of cardiac arrest, like sinus arrest and complete block. Similar studies done by Goldberg et al observed that hyponatremia on admission or observed that hyponatremia on admission or during the first 72 hours of hospitalization in STEMI was independently associated with increased 30- day mortality risk and more post-discharge re- admission for heart failure and death in long – term follow-up.

It was similarly found that hyponatremia was more often associated with increased morbidity and mortality in MI patients. The risk of in hospital mortality is found to increase with the severity of hyponatremia.

Conclusion

In conclusion our study indicated a direct association between serum GGT and uric acid in MI. Although GGT is often measured as a marker of liver health, this study provides evidence that it may also be useful in the identification of patients at elevated risk of MI.

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