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Original Research Article

A Hospital Based Study to Assess the Association of HS Troponin I & Uric Acid in Patients of Myocardial Infarction

Abhilasha Abhilasha[1], Purushottam Kumar[2], Jully Kumari[3], Uday Narayan Singh[4]

[1]Tutor, Department of Biochemistry, Nalanda Medical College and Hospital, Patna, Bihar, India

[2]Tutor, Department of Biochemistry, Nalanda Medical College and Hospital, Patna, Bihar, India

[3]PG Student, Department of Biochemistry, Nalanda Medical College and Hospital, Patna, Bihar, India

[4]Professor, Department of Biochemistry, Nalanda Medical College and Hospital, Patna, Bihar, India

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Corresponding Author: Dr. Purushottam Kumar
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Abstract

Aim: The aim of the present study was to assess the Correlation of Hs Troponin I & Uric Acid in patients of Myocardial Infarction.

Methods: 200 patients who came to cardiac emergency in the Department of Medicine, Nalanda medical College and Hospital, Patna, Bihar, India. Serum samples were taken for Hs Troponin I and Uric Acid for patients of Myocardial Infarction and run on VITROS 5600/7600 which is based on dry chemistry.

Results: Among the 200 patients of more than 40 years of age 120 were males & 80 were females. For both males & females age mean & SD was 60.6±11.72 and 58.6±12.70. For Hs Trop I males were 22.78±46.84 & females 15.75±54.56. For uric acid for males were 6.534±3.750 & for females 6.316±1.860. For Hs Trop I males were 22.78±46.84 & females 15.75±54.56. For uric acid for males were 6.534±3.750 & for females 6.316±1.860. Therefore Hs Trop I & uric acid were both significant when compared with age P value was 0.0040.Whereas when compared with sex that is male and female to both Hs Trop I and uric acid then Hs Trop I was more significant with P value 0.0001.

Conclusion: In acute MI, patients with hyperuricemia had higher mortality. Serum uric acid levels correlated with Killip classification in acute MI. Serum uric acid can be used as a marker of short term mortality in patients. Hyperuricemia is an indicator of poor prognosis in acute MI. Uric acid is an economical biomarker that is readily, quickly and reliably obtainable, it can be one of the predictable prognostic indicator in acute Myocardial Infarction.

Keywords: Hs troponin I, Serum uric acid, Myocardial Infarction

Introduction

Chest pain is a common complaint for patients presenting to the emergency department (ED). Worldwide, more than 8 million patients presented to ED for chest pain in 2019. [1] While chest pain accounts for approximately 10% of emergency department visits, only 10 to 20% of chest pain is diagnosed as cardiac chest pain [2], with a majority being attributed to a multitude of other diagnoses. Differentiating chest pain that originates from cardiac ischaemia, such as acute coronary syndrome (ACS), remains a diagnostic dilemma, as patients may present atypically. Conditions such as pulmonary embolism, aortic dissection, or pneumothorax may mimic ACS and present with symptoms such as chest pain and shortness of breath. [3] At time of presentation, approximately 80% of patients with chest pain do not have a clear diagnosis of ACS [4], with a further need to be monitored and worked up while in the emergency department or emergency observation units. While clinical evaluation of patients and repeated electrocardiograms (ECG) are essential, these alone are not enough to reliably rule out ACS, and have to be used in conjunction with other investigations. [5]

The use of cardiac biomarkers, especially cardiac troponin, remains a cornerstone in the diagnosis of myocardial infarction. [6] The troponin complex consists of three subunits (troponin I, T, and C), and plays a vital role in regulating cardiac excitation and contraction. [7] In the setting of myocardial infarction, both troponin I and T are released from the damaged myocardium. [8] Conventional troponin assays are limited by their low sensitivities at the time of patient presentation, due to delay in the increase in circulating levels of troponin, requiring

repeated testing 6–9 h after presentation [9] and are deemed as unlikely to be cost-effective. [10] With the introduction of high-sensitivity troponin, new protocols were generated to help in the risk stratification of patients presenting to the emergency department with chest pain or symptoms suggestive of cardiac ischaemia for myocardial infarction as quickly as within one to two hours. [11,12]

The aim of the present study was to assess the Correlation of Hs Troponin I & Uric Acid in patients of Myocardial Infarction.

Materials and Methods

200 patients who came to cardiac emergency in the Department of Medicine, Nalanda medical College and Hospital, Patna, Bihar, India for one year. Serum samples were taken for Hs Troponin I and Uric Acid for patients of Myocardial Infarction and run on VITROS 5600/7600 which is based on dry chemistry.

Results

Table 1: Distribution of Hs Tropi and uric acid in age and observed

Parameters	Male	Female	P Value
	Hs Tropi	Uric acid	
	Mean±SD	Mean±SD	
Age	60.6±11.72	58.6±12.70	1
Observed	21.58±52.58	6.374±3.180	0.0040

Among the 200 patients of more than 40 years of age 120 were males & 80 were females. For both males & females age mean & SD was 60.6±11.72 and 58.6±12.70.

Table 2: Distribution of Hs Tropi and uric acid in male and female

Parameters	Male	Female	P Value
	Mean±SD	Mean±SD	
HS Tropi	22.78±46.84	15.75±54.56	0.0001
Uric acid	6.534±3.750	6.316±1.860	0.634

For Hs Trop I males were 22.78±46.84 & females 15.75±54.56. For uric acid for males were 6.534±3.750 & for females 6.316±1.860.
Therefore Hs Trop I & uric acid were both significant when compared with age P value was 0.0040.Whereas when compared with sex that is male and female to both Hs Trop I and uric acid then Hs Trop I was more significant with P value 0.0001. (Table 1 and Table 2)

Discussion

The Global burden of disease Study reported that in 1990 there were 5.2 million deaths from cardiovascular diseases in economically developed countries and 9.1 million deaths from the same causes in developing countries. [13] The prevalence of CAD in India increased from 1% in 1960 to 9.7% in 1995 in urban populations & in rural population it is most doubled in past decade. [14] There has been growing interest in the link between uric acid levels, xanthine oxidoreductase and cardiovascular disease. Previous studies have reported that a high concentration of uric acid is a strong marker of an unfavourable prognosis of moderate to severe heart failure and cardiovascular disease. [15,16] Uric acid levels may be elevated in heart failure and provide important prognostic information. [17]

Among the 200 patients of more than 40 years of age 120 were males & 80 were females. For both males & females age mean & SD was 60.6±11.72 and 58.6±12.70. For Hs Trop I males were 22.78±46.84 & females 15.75±54.56. For uric acid for males were 6.534±3.750 & for females 6.316±1.860. For Hs Trop I males were 22.78±46.84 & females 15.75±54.56. For uric acid for males were 6.534±3.750 & for females 6.316±1.860. Therefore Hs Trop I & uric acid were both significant when compared with age P value was 0.0040.Whereas when compared with sex that is male and female to both Hs Trop I and uric acid then Hs Trop I was more significant with P value 0.0001. Serum uric acid and MI has been debated with conflicting results in previous studies. The AMORIS study [18] and the Rotterdham study [19] have been demonstrated a significant association between Serum uric acid & MI. In contrast, the Tromso study [20] and the NHANES (National Health and Nutrition Examination Survey) III study [21] have failed to establish an independent association between Serum uric acid and MI. First, Serum uric acid is a product of xanthine oxidoreductase, which is known to be one of the most important sources of reactive oxygen species, High Serum uric acid is therefore associated with increased vascular endothelial function, vascular smooth muscle cell proliferation and oxidative stress thereby increasing the risk of MI and all-cause mortality. [22,23] Second, high Serum uric acid exerts a plethora of deleterious effects in cells and thus may be directly involved in the pathophysiological characteristics of MI and all-cause mortality. [24] Third, high Serum uric acid is correlated with almost all known

cardiovascular risk factors, such as metabolic syndrome [25] and chronic kidney disease thus, a higher level of Serum uric acid may be seen as correlation of cardiovascular risk or an epiphenomenon of coexisting cardiometabolic risk factor.

While the usage of high-sensitivity troponin may identify a group of patients with troponin elevation without myocardial infarction, elevated levels of high-sensitivity troponin among patients who present to ED with chest pain is associated with higher rates of mortality and MACE, regardless of index visit diagnosis. [26,27] High-sensitivity troponin should still be used over conventional troponin, as this allows identification of patients who are at high risk and should be followed up more closely, and may be considered of prognostic value for future events even in patients with detectable levels below the 99th percentile or with stable low-level elevations. [28]

Conclusion

In acute MI, patients with hyperuricemia had higher mortality. Serum uric acid levels correlated with Killip classification in acute MI. Serum uric acid can be used as a marker of short term mortality in patients. Hyperuricemia is an indicator of poor prognosis in acute MI. Uric acid is an economical biomarker that is readily, quickly and reliably obtainable, it can be one of the predictable prognostic indicator in acute Myocardial Infarction.

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