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Original Research Article

Investigate the Levels of Hs Troponin I and Uric Acid in Individuals Diagnosed with Myocardial Infarction

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Abstract

Aim: Investigate the levels of Hs Troponin I and uric acid in individuals diagnosed with myocardial infarction.

Materials and Methods: This study was conducted in the Department of Biochemistry, Homi Bhabha Cancer Hospital (HBCH), Lahartara and Mahamana Pandit Madan Mohan Malaviya Cancer Centre (MPMMCC), Sundarbagiya, BHU Campus, Varanasi, Uttar Pradesh, India for one year. 200 patients who came to cardiac emergency. 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. 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 indicators in acute Myocardial Infarction.

Keywords: Hs Troponin I, Uric Acid, Myocardial Infarction.

Introduction

Myocardial infarction (MI), commonly known as a heart attack, is a critical medical condition characterized by the interruption of blood flow to a part of the heart, leading to the death of heart muscle cells. Early diagnosis and intervention are crucial to improve outcomes and reduce mortality. Among the various biomarkers used for diagnosing and assessing the severity of MI, high-sensitivity troponin I (Hs-Troponin I) and uric acid have gained significant attention. These biomarkers not only aid in the early detection of MI but also provide insights into the underlying pathophysiological processes and potential prognostic implications. [1-3] Hs-Troponin I is a highly sensitive and specific marker for myocardial injury. Troponins are regulatory proteins found in cardiac muscle, and their release into the bloodstream indicates damage to the heart muscle. High-sensitivity assays for troponin I allow for the detection of very low levels of this biomarker, enabling the identification of even minor cardiac injuries that might be missed by conventional assays. Elevated levels of Hs-Troponin I are a hallmark of MI and are used extensively in clinical practice to diagnose acute coronary syndromes (ACS) and stratify patients based on their risk of adverse cardiovascular events. Numerous studies have demonstrated the superiority of Hs-Troponin I over traditional markers in diagnosing MI, particularly in patients presenting with non-ST-segment elevation myocardial infarction (NSTEMI) . [4-6] In addition to Hs-Troponin I, uric acid has emerged as a potential biomarker in the context of cardiovascular diseases, including MI. Uric acid is the end product of purine metabolism and is known for its role in gout and kidney stones. However, elevated serum uric acid levels have also been associated with increased cardiovascular risk. Hyperuricemia can lead to endothelial dysfunction, inflammation, and oxidative stress, all of which are key contributors to atherosclerosis and subsequent cardiovascular events. Several studies have reported that high uric acid levels are linked with increased

incidence of MI and poor prognosis in patients with established cardiovascular disease . [7-10] The interplay between Hs-Troponin I and uric acid levels in patients with MI highlights the complex pathophysiology of the disease. While Hs-Troponin I serves as a direct indicator of myocardial injury, elevated uric acid levels may reflect underlying metabolic disturbances that exacerbate cardiovascular risk. For instance, oxidative stress and inflammation driven by hyperuricemia can potentiate myocardial damage, leading to higher troponin levels. Conversely, myocardial injury and the associated inflammatory response can result in elevated uric acid levels, creating a feedback loop that worsens patient outcomes . [11-13]

Materials and Methods

This study was conducted in the Department of Biochemistry, Homi Bhabha Cancer Hospital (HBCH), Lahartara and Mahamana Pandit Madan Mohan Malaviya Cancer Centre (MPMMCC), Sundarbagiya, BHU Campus, Varanasi, Uttar Pradesh, India for one year. 200 patients who came to cardiac emergency. 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. 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)

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

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

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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