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Study of Metabolic Syndrome in Correlation With Hyperuricemia

KEYWORDS: Hypertension; Insulin resistance; Metabolic syndrome; Obesity; Uric acid

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ABSTRACT ***BACKGROUND:*** *Insulin decreases uric acid excretion. So there must be some link between hyperuricemia and insulin resistance. But the prevalence of the metabolic syndrome by recent definitions among individuals with hyperuricemia remains unclear. Our objective was to determine the prevalence of the metabolic syndrome according to serum uric acid levels in a medical college hospital.*

METHODS: *By using data from 850 participants who aged 20 years and more who attended OPD during the period of January 2014 to May 2015, the prevalence of the metabolic syndrome at different serum uric acid levels was determined. Metabolic syndrome was defined as per National Cholesterol Education Programme Adult Treatment Panel (NCEP/ATP) III criteria.*

RESULTS: *The prevalences of the metabolic syndrome according to the NCEP/ATP III criteria were 16% for uric acid levels less than 6 mg/dL, 25% for uric acid levels from 6 to 6.9 mg/dL, 42% for uric acid levels from 7 to 7.9 mg/dL, 52% for uric acid levels from 8 to 8.9 mg/dL, 60% for uric acid levels from 9 to 9.9 mg/dL, and 66% for uric acid levels of 10 mg/dL or greater. The prevalence of individual metabolic abnormalities of the metabolic syndrome were also calculated*

CONCLUSIONS: *With increasing levels of serum uric acid the prevalence of the metabolic syndrome increased substantially. Hence metabolic syndrome must be considered as a frequent comorbidity of hyperuricemia and should be treated to prevent serious complications.*

INTRODUCTION

Metabolic syndrome, previously named syndrome X, describes a group of risk factors occurring in the same individual and a common denominator of insulin resistance[1]. Risk for atherosclerotic cardiovascular disease and type 2 diabetes,[2,3] as well as mortality from cardiovascular disease[4,5] are increased in metabolic syndrome patients because of the multiple interrelated conditions.

A number of studies reported significant associations between serum uric acid levels and individual components of the metabolic syndrome,[4,5] but the scope of prevalence of the metabolic syndrome among individuals with hyperuricemia is unknown. Renal clearance of uric acid is inversely related to the degree of insulin resistance. Thus, the decreased renal excretion of uric acid among patients with the metabolic syndrome may explain the increased frequency of hyperuricemia. On the basis of these data, hyperuricemia has been suggested as a simple marker of the metabolic syndrome.[6] However, potential utility of the hyperuricemia as a marker remains unclear.

To study the hyperuricemia as a useful marker, we determined the prevalence of the metabolic syndrome among individuals with different serum uric acid levels.

METHODS

This study is conducted between January 2014 and May 2015, sample of patients attending OPD, was selected randomly. In the current study, we analyzed data for 850 individuals who were aged at least 20 years who attended the medical examination, had fasted at least 8 hours before the blood collection, and had complete information to allow definition of the metabolic syndrome and measurement of serum uric acid levels.

We used National Cholesterol Education Program Adult Treatment Panel (NCEP/ATP) III criteria to define the metabolic syndrome. According to the NCEP/ATP III criteria, participants with 3 or more of the following criteria were defined as having the metabolic syndrome: abdominal obesity (waist circumference 102 cm in men and 88 cm in women); hypertriglyceridemia (150 mg/dL [1.70 mmol/L]); low high density lipoprotein (HDL) cholesterol (40 mg/dL [1.04 mmol/L] in men and 50 mg/dL [1.30 mmol/L] in women); high blood pressure (130/85 mm Hg); and high fasting glucose (100 mg/dL [5.6 mmol/L]). We counted participants who reported currently using antihypertensive or antidiabetic medication (insulin or oral agents) as participants with high blood pressure or diabetes, respectively.

RESULTS

Out of 850 individuals who participated in our study males were 62% and females were 38%. The mean age of the individuals in our study is 39 yrs.

The prevalence of the metabolic syndrome (%)among the total study population was calculated according to 6 categories of serum uric acid levels: less than 6 mg/dL, 6 to 6.9 mg/dL, 7 to 7.9 mg/dL, 8 to 8.9 mg/dL, 9 to 9.9 mg/dL, and 10 mg/dL or more.

There was graded increase in the prevalence of the metabolic syndrome according to NCEP/ATP III criteria among individuals with hyperuricemia, from 16% in people with serum uric acid level < 6 mg/dl upto 66% among individuals with serum uric acid level of 10 mg/dl or greater as shown in table 1.

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Table 1: prevalence of metabolic syndrome according to serum uric acid levels

	Serum uric acid levels					
	<6 mg/dl	6-6.9mg/dl	7-7.9mg/dl	8-8.9mg/dl	9-9.9mg/dl	>10mg/dl
No. of patients	107	193	226	154	125	35
Prevalence No.of pts	17	48	95	80	75	23
Prevalence Percentage	16%	25%	42%	52%	60%	66%

Similarly, the prevalence of individual metabolic abnormalities increased with increasing levels of serum uric acid, except for a slight decrease in the prevalence of hypertriglyceridemia in the highest category of serum uric acid level> 10 mg%. There is strong positive correlation between hypertension and fasting blood glucose with serum uric acid levels as shown in table 2.

Table 2: prevalence of metabolic syndrome abnormalities according to serum uric acid levels

Uric acid levels	<6 mg/dl	6-6.9mg/dl	7-7.9mg/dl	8-8.9mg/dl	9-9.9mg/dl	>10mg/dl
Abdominal obesity	17%	19%	26%	33%	38%	40%
Hypertriglyceridemia	16%	25%	31%	37%	45%	42%
Low HDL cholesterol	20%	25%	35%	42%	46%	60%
High blood pressure	30%	31%	35%	45%	55%	62%
Fasting glucose>100 mg/dl	25%	31%	39%	43%	64%	80%

DISCUSSION

In this study we found that there was a graded increase in the prevalence of metabolic syndrome among people with increasing levels of hyperuricemia, upto 66% among individuals with highest serum uric acid levels > 10 mg/dl. The prevalence was approximately four times that among adults with the lowest serum uric acid levels(< 6mg/dl). The increasing prevalence of individual metabolic abnormalities with increasing levels of hyperuricemia was apparent.

There are several important implications of our results. The prevalence estimates determined in the current study provide the probabilities of concomitant presence of the metabolic syndrome among individuals with differing degrees of hyperuricemia. The presence of hyperuricemia, particularly at higher levels, should trigger a high level of clinical suspicion and investigation for a potential coexistence of the metabolic syndrome. If present, the syndrome needs to be recognized as a potentially more life threatening factor than hyperuricemia, given the serious associated complications. The cornerstones of treatment for the syndrome are managing body weight and ensuring appropriate levels of physical activity. Recent studies demonstrated that lifestyle interventions or medications may delay or prevent the transition from impaired glucose tolerance to type 2 diabetes mellitus and provide relevant treatment paradigms for patients with the metabolic syndrome.

Long-term dietary recommendations for the majority of individuals with hyperuricemia or gout should take this frequent comorbidity into account. For example, conventional dietary recommendations for hyperuricemia or gout have focused on restriction of purine intake, although low-purine diets are often high in carbohydrate and saturated fat. These macronutrients are associated with an increased risk of the insulin resistance syndrome and associated major consequences. Furthermore, these macronutrients tend to lead to higher serum insulin levels, which are known to reduce renal excretion of urate, thus potentially further increasing the serum uric acid level. Given the frequent association between hyperuricemia and the metabolic syndrome, it is imperative to develop appropriate dietary and other lifestyle guidelines taking into account improving hyperuricemia and overall long-term health effects. In addition, the growing epidemics of obesity and the metabolic syndrome present a substantial challenge in the prevention and management of gout with hyperuricemia[5] Because these conditions would likely also share important parts of public health and clinical management approaches, future studies may need to focus on developing the overall optimal strategies for improving these concurrent conditions.

It is uncertain whether elevated levels of uric acid are the result or the cause of metabolic syndrome. The syndrome has been attributed to insulin resistance. Indeed, several studies have shown that hyperinsulinemia (the consequence of insulin resistance) is inversely related to 24 hour urinary uric acid clearance[7]. One mechanism linking hyperinsulinemia with hyperuricemia is a decreased renal excretion of uric acid. Moreover, insulin enhances renal tubular sodium absorption, which reduces renal excretion of uric acid. On the other hand, animal models have shown that uric acid has a role in the development of Metabolic Syndrome and that decreasing uric acid levels can prevent or reverse features of Metabolic Syndrome[8]. Two mechanisms have been suggested to explain how hyperuricemia[9] might induce metabolic syndrome. The first mechanism is related to the fact that hyperuricemia has been shown to induce endothelial dysfunction , which leads to a decreased release of nitric oxide from those cells[10] . Features of syndrome were shown to develop in mice lacking endothelial nitric oxide synthesis[11]. The second mechanism concerns the inflammatory and oxidative changes induced by uric acid in adipocytes[12], essential for inducing Metabolic Syndrome in obese mice. A recent review suggests some possible explanations for the association between the traditional components of this syndrome and elevated uric acid levels. Although hyperuricemia in obese patients is mainly the result of insulin resistance, it may also be due to elevated levels of leptin .Hypertension leads to vascular disease and increased vascular resistance, resulting in decreased renal blood flow, which in turn stimulates urate absorption. Increased triglyceride levels may be associated with decreased uric acid excretion. Apolipoprotein E polymorphism affecting TG levels may also affect uric acid levels. And lastly, elevated serum glucose levels, hypertension and obesity have all been associated with chronic kidney disease, which again leads to hyperuricemia.

CONCLUSION

The prevalence of the metabolic syndrome increases with increasing serum uric acid levels. This should rise clinical suspicion for the concomitant presence of the metabolic syndrome. Metabolic syndrome should be recognised as a

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frequent comorbidity of hyperuricemia and treat it to prevent serious complications.

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