Hyperuricemia: an Emerging Health Problem of the Society Invites Considerations

Ashique Ali Arain¹, Mohammad Ali², Aftab Ahmed Shaikh³ and Muhammad Hamid Ali⁴

¹Department of Pharmacology, Assistant Professor Isra University Hyderabad, Pakistan
²Department of Orthopedics, Assistant Professor Liaquat University of Medical and Health Sciences Jamshoro, Pakistan
³Department of Pharmacology, Associate Professor Al-Tibri Medical College Karachi, Pakistan
⁴Department of Neurosurgery, Assistant Professor Liaquat University of Medical and Health Sciences Jamshoro, Pakistan

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*Corresponding author: Ashique Ali Arain, Department of Pharmacology, Assistant Professor Isra University Hyderabad, Sindh, Pakistan; Tel no: 03333389250; Email: ashiquepcmd77@yahoo.com

Abstract

Background: Joint pain is the most common complaint from the patient presenting to orthopedic clinics. Elevated uric acid levels in plasma result in accumulation of the same in the joint spaces causing inflammation. Gouty arthritis has dual management, pharmacological and dietary management.

Study Design: Observational study.
Study setting: Consultant Orthopedic OPD of Liaquat University of medical and health sciences.
Study Duration: 3 months from November 2016-feb 2017.
Sample Size: 100 Patients.
Sampling technique: probability sampling.
Methodology: Serum uric acid levels were measured through device method with the permission of study patients.
Statistical analysis: Results were compiled using SPSS 20 Data analysis was performed using t-test.

Results: Study population male/female ratio was 1:1. Mean of the serum uric acid was found to be 4.91 mg/dl while the range of the same was 1.0 mg/dl. The minimum value for uric acid was 2.0 mg/dl and the maximum 12.0 mg/dl. 10% of the population showed elevated uric acid levels >7.5 mg/dl while 15% of the patients were having a borderline values of 6.5 mg/dl and 22% of the study population was found to have their uric acid levels above the optimal level (6mg/dl). There was a significant difference between the mean of male and female patient p-0.002.

Conclusion: It is concluded that 47% of the study population was suffering from the uric acid levels above the optimal levels and were higher in females as compared to males. Patient education is needed especially regarding the non pharmacological management.

Keywords: Uric Acid; Arthralgia; Xanthane Oxidase.

Introduction

Hyperuricemia puts the patients on developing gouty arthritis as well as renal uric acid stones. The mechanism behind is the deposition of uric acid crystals that invite inflammatory response through leucocyte infiltration leading to release of inflammatory mediators, leukotriene and hydrolytic enzymes. The cause behind elevation of uric acid is either its overproduction or diminished excretion by the kidneys. Renal tubular reabsorption occurs for 90% of filtered urate through the transporters URAT1 and GLUT9 [1]. Uric acid is synthesized from the metabolism of purine derivatives of DNA and RNA that get converted first into xanthine and then to uric acid from the hypoxanthine by the action of enzyme xanthine oxidase. High protein diet like meat, pulses, beans, chocolates are the main risk factors for developing hyperuricemia. Drugs also contribute in the etiology of hyperuricemia theses include aspirin, loop and thiazide diuretics, antituberculous drugs like ethambutol and pyrazinamide.
Symptoms produced by elevated uric acid levels are pain and swelling of the Big toe, painful joint especially the knee joint. Many patients even remain symptom free despite of high uric acid levels patients with gout alone or combined with renal stones should be distinguished prior to treatment. Elevated uric acid is a risk factor for diabetes, HTN, HHD and metabolic syndrome [2]. A US study showed that 8.3 million Americans have their uric acid levels raised and gave a prevalence rate of gout in adult population of 3.9%, with 5.9% and 2.0% in male and female respectively [3]. There are many treatment options but compliance rate is 18-26% that is much low [4]. Uric acid levels should be kept below 6 mg/dl anoptimal level required for therapeutic goal as suggested by the European Guidelines for chronic hyperuricemia [5]. Therapy for this purpose consists of

1) Non-pharmacological, which includes encouraging a diet with low purine contents while discouraging with high purine contents.

2) The pharmacological, which includes colchicine, NSAIDS, uricosuric agents, xanthine oxidase inhibitors, piglactone, glucocorticoids and interleukin-1 inhibitors [6].

Xanthine oxidase Inhibitors are in most common use for gouty arthritis they are also supposed to reduce the cardiovascular complicationsof hyperuricemia [7]. 3,4-dihydroxy-5-nitrobenzaldehyde (DHNB), is a derivate of natural protocatechuic aldehyde that inhibits XO in a time-dependent manner similarly to allopurinol. It interacts with the molybdenum center of XO and is able to directly scavenge free radicals [8].

NSAIDs produce their effects through the inhibition of the synthesis of prostaglandin by COX-2 that is the most important step in the initiation of inflammation of gouty arthritis [9]. Sevelamer is a nonabsorbable phosphate binder that increases gastrointestinal elimination of uric acid while BCX4208 is an inhibitor of purine nucleotide phosphorylase and pegloticase reduces theurate levels through enhanced metabolism [10]. Current study was conducted to analyze the male/female distribution of hyperuricemia and to evaluate the percentage of patient presenting with joint pain in which the cause was elevated uric acid and those with non-uric acid etiology.

Methodology

100 patients presenting to the orthopedic OPD with joint pain were selected through non probability purposive sampling. All age groups were included with 1:1 male/female ratio. Mean of the study population male/female ratio was 1:1. Mean of the study population male was 10.19mg/dl while the range of the same was 10mg/dl. The minimum value for uric acid was 2.0mg/dl and the maximum 12.2mg/dl. 10% of the population showed elevated uric acid levels >7.5 mg/dl while 15% of the patients were having a border line values of 6.5mg/dl and 22% of the study population was found to have their uric acid levels above the optimal level (6mg/dl). There was a significant difference between the mean of male and female patient p-value 0.002 (Figure 1) (Tables 1 & 2).

Results

Study population male/female ratio was 1:1. Mean of the serum uric acid was found to be 4.91mg/dl while the range of the same was 10mg/dl. The minimum value for uric acid was 2.0mg/dl and the maximum 12.2mg/dl. 10% of the population showed elevated uric acid levels >7.5 mg/dl while 15% of the patients were having a border line values of 6.5mg/dl and 22% of the study population was found to have their uric acid levels above the optimal level (6mg/dl). There was a significant difference between the mean of male and female patient p-value 0.002 (Figure 1) (Tables 1 & 2).

Discussion

Yanyan Zhuet al (2011) were inconsistent to the results of the current study regarding gender involvement with a mean of uric acid 6.14 mg/dl and 4.87 mg/dl in men and women respectively [3]. While the current study shows higher levels in female 5.1mg/dl as compared to male 4.6mg/dl. Jamshed et al. (2016) studied uric acid levelsin coronary artery diseases patients and reported the mean uric acid in male patients as 7.2mg/dl while it was 5.9mg/dl in female patients [11]. That was in contrast to our findings the reason may be diet or vascular events. Study by Dasti MA et al. (2015) were in accordance to our results who declared a population mean of 13.74mg/dl while the mean in male and female groups was 11.74mg/dl and 14.43mg/dl respectively [12]. However his work was based on a hypertensive population while we studied patients of arthralgia only the vascular events

Table 1: Mean and range of uric acid levels in study population.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Male</th>
<th>Female</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Uric Acid</td>
<td>4.6mg/dl</td>
<td>5.1mg/dl</td>
<td>4.91mg/dl</td>
</tr>
<tr>
<td>Range</td>
<td>12.2-10mg/dl</td>
<td>10.6-8.3mg/dl</td>
<td>10mg/dl</td>
</tr>
</tbody>
</table>

Table 2: Population requiring management for uric acid.

<table>
<thead>
<tr>
<th>Uric Acid Levels</th>
<th>Male</th>
<th>Female</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;7.5mg/dl</td>
<td>4(8%)</td>
<td>6(12%)</td>
<td>10(10%)</td>
</tr>
<tr>
<td>&gt;6.5mg/dl</td>
<td>5(10%)</td>
<td>10(20%)</td>
<td>15(15%)</td>
</tr>
<tr>
<td>&gt;6.0mg/dl</td>
<td>8(16%)</td>
<td>14(28%)</td>
<td>22(22%)</td>
</tr>
</tbody>
</table>
may have some impact on the uric acid levels. The current study was on small scale so we recommend large scale studies in general population as well as a population suffering from joint pain. Public education or awareness campaigns regarding the diet modifications and non-pharmacological management for controlling the uric acid need to be conducted national wide.

Conclusion

We conclude that 47% of the patients with joint pain were found to be their serum uric acid levels higher than the optimal levels and females were more affected in comparison to males. While 53% patients were having their uric acid in control and in optimal range.

References