

Original Research Article

Atypical manifestation of hyperuricemia in the Indian population: a clinical and biochemical assessment and treatment guidelines

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ABSTRACT

Background: Podagra, the clinical presentation of gout presents no problem in diagnosis but hyperuricemia leading to some unusual presentation with musculoskeletal disorders and is often missed. This study had been conducted to analyze the disturbances of uric acid metabolism and subsequent role of uric acid in the pathogenesis of various disorder including cardiovascular, renal and various musculoskeletal disorders and to determine the disease related upper level of SUA in Indian population in both vegetarians and non-vegetarians.

Methods: Serum uric acid (by nephelometric method) was assessed in 1084 individuals during January 2018-December 2022 at a single center at Max Super Specialty Hospital, Patparganj, New Delhi with complaint of pain and swelling in joints and various other musculoskeletal problems. Out of these 48.80% were vegetarians and 51.20 were nonvegetarians.

Results: Looking at the symptomatology, only 18.50% presented with podagra as their chief complaint followed by 15.40% with generalized myalgia, 14.39% cases of plantar fasciitis and rest including tennis elbow, knee and ankle synovitis, interscapular fibroscititis, bursitis anserine and various other musculoskeletal problems. In 76% cases with uric acid level more than 7mg/dl were having hypertriglyceridemia. Mild to moderate hypertension had been noted in 53% cases of those having SUA level more than 7 mg/dl.

Conclusions: In present study, it concluded that musculoskeletal symptoms related uric acid level should be lower around 5.5 mg/dl. SUA levels in vegetarians and non-vegetarians did not differ significantly. The high haemoglobin percent in most of the individuals gives an idea that hyperuricemia is related to good nutritional status.

Keywords: Uricemia, Podagra, Bursitis, Fibroscititis, Hypertriglyceridemia, Unusual manifestations

INTRODUCTION

Uric acid a weak organic acid (PKA 5.75) at pH 7.40 and temp 37 degree, is produced from xanthine as the end product of purine metabolism with the help of xanthine oxidase in human beings and about 98% of uric acid is ionized to monosodium urate.

Except the human beings most mammals have enzyme uricase, that catalyses the degradation of insoluble uric acid to readily excretable compound allantoin. The SUA level depends on purine content of diet, rate of

biosynthesis, degradation, renal excretion and salvage pathway. In the male individuals' important associates of uricemia are obesity, hypertension, hyperlipidaemia, diabetes and ischemic heart disease.

Ruilope et al after their Study found that the hyperuricaemia commonly seen in clinical practice, neither represents a specific disease nor it is an indication of therapy.¹ Hyperuricaemia is normally present in 2% adults in USA, 17% adults in France, 7% of adults in Spain (Noguera et al) and 0.2-2% adult population in India with increasing age.²⁻⁵ The defect in excretion is the most common cause of elevated serum uric acid.⁶

Ultimate excretion is the balance between secretion and resorption. Muscelli et al in their study stated that resorption is linked to Na⁺/H⁺ exchange mechanism and insulin increases both sodium and uric acid resorption.⁷ Seegmiller et al after their isotopic studies, provided the evidence of heterogeneity of the mechanism of uricemia.⁸ Campion et al had seen the association of increased gout and nephrolithiasis at levels >7 mg/dl.⁹ Measurable increase occurs in the level of uric acid in males after puberty and after menopause in females. Nicholas et al had seen low urate level in premenopausal females due to estrogen.¹⁰

It is still debatable how uric acid is associated to the clusters of diseases, Few studies suggested that increased serum uric acid with LDL-lipoproteins might be the Part of insulin resistance syndrome.^{11,12} The insulin resistance and hyperlipidemia probably decreases urate excretion independent of obesity and decreases Creatinine clearance by enhancing tubular resorption of sodium (Rathmann et al, Ficchini et al, Cappucio et al, Lindau et al, Krentz et al and Mohan et al).¹³⁻¹⁸ Yu et al reported that uricemia is common in insulin resistant cases and may be a factor that links hyperlipidemia, hypertension, and hyperuricemia.¹⁹

Cannon et al, Levya et al, Messerli et al and Borghi et al had shown that the hypertensive patients with coronary heart disease and renal dysfunction are likely to have hyperuricemia.²⁰⁻²³ Malignant hypertension is associated with hyperuricemia but not as an independent predictor (Iossa et al, Wang et al and Selby et al).²⁴⁻²⁶ These findings led to the basis for epidemiological studies in population in Europe and USA regarding association of uric acid as risk factor for coronary heart disease (Moriarty et al, Klien et al, Bengtsson et al and Lehto et al).²⁷⁻³⁰ Levine et al in a prospective study of 2400 workers in Chicago industrial heart institute had concluded serum uric acid is associated with increased morbidity and mortality in CAD.³¹ Freedom et al in their study found that baseline serum uric acid is associated with increased coronary heart diseases in females.³² Another reported study had observed that SUA level is below 7mg/dl in this part of world.³³ The present study was aimed to know the association of serum uric acid level, its manifestations both classical podagra and various other musculoskeletal symptoms, role of vegetarian and non-vegetarian diets and nutritional status of individual and to determine what should be the treatable range of serum uric acid in Indian population.

METHODS

This single group observational study was conducted on 1084 individuals. Inclusion criteria were any person who had complaints of joint pain, tennis elbow, retrocalcaneal bursitis, planter fasciitis, bursitis anserina, metatarsalgia, intercostals myalgia, generalized muscle pain and various other musculoskeletal disorders. Those with known other reason of joint pain like rheumatoid arthritis, seronegative

poly arthralgia, psoriatic arthritis and reactive arthralgia were excluded. Serum uric acid (by nephelometric method) was assessed during January 2018-December 2022 at a single center at Max Super specialty Hospital, Patparganj, New Delhi with complaint of pain and swelling in joints and various other musculoskeletal problems.

There were 882 male and 202 females with average age of 31.66 + 8.62 years ranging from 15-78 years including 48.80% vegetarians and 51.20% nonvegetarians. Each individual had been investigated for Hb; TLC, DLC, ESR, S-uric acid, S-urea, S-creatinine, blood sugar, and lipid profile especially triglycerides. Blood Pressure was recorded regularly. The general physical examination including records of height and weight was kept systematically. The uric acid was measured by nephelometric method. The symptomatic cases were followed for the period of 6 months to 4 years. In the cases with uric acid level 4.5-5.5mg/dl, after the dietary restriction the clinical condition were more or less same as before and less than 15% cases showed improvement. Cases with uric acid level about 5.5 mg/dl had been treated with dietary restriction and NSAIDS showed moderate relief in 30-40% cases. Addition of allopurinol in same group improved the clinical response in 85% cases. The individuals with uric acid level >7.0 mg/dl had been treated with febuxostat/colchicine (during acute attacks) and NSAIDS along with dietary restriction showed drastic improvement.

The hypertensive hyperuricemia had been treated with febuxostat/ allopurinol/ colchicine along with dietary restriction, responded very well and the doses of antihypertensive medication was reduced.

RESULTS

The average age was 31.66±8.62 years, ranging from 15-78 years (Table 1).

Table 1: Age distribution.

Age (in years)	SUA (<5%)	SUA (>5%)	Total
<20	34	52	86
21-40	118	452	570
41-60	112	210	322
61-80	20	102	122
Total	284	800	1084
Mean±SD	30.26±8.33	33.06±9.91	31.66±8.62

*P>0.10, r=0.027 (insignificant)

The average weight was 59.9 kg (range: 36-93.5 kg). The average height was 163.1 cm (range: 136-184.5 cm). Fasting blood sugar was above normal in 84 cases (average 128 mg%). Rheumatoid factor was positive in 56 cases (11-20 IU). S-urea and creatinine were normal in all cases.76% of cases had uric acid above 7 mg/dl and mild hypertriglyceridemia. Labile hypertension to mild

hypertension was found in 53% of cases with SUA >7 mg/dl.

Table 2: Distribution of cases according to symptoms.

Main symptom	N	Percentage (%)	Average SUA (mg/dl)
Podagra	201	18.50	7.6
Generalized myalgias	166	15.31	5.4
Plantar fasciitis	126	11.65	5.2
Tennis elbow	120	11.06	5.5
Retrocalcaneal bursitis	111	10.24	5.4
Pain in joints with synovitis	72	6.64	5.9
Interscapular fibrofasciitis	56	5.16	7.7
Flexor tendon synovitis	55	5.07	5.2
De Quervain's disease	52	4.79	5.3
Bursitis anserine	39	3.59	5.8
Golfer's elbow	23	2.12	5.1
Subacromial bursitis	22	2.12	5.2
Intercostal myalgia	18	1.66	5.5
Metatarsalgia	18	1.66	5.3
Achilles tendonitis	18	1.66	5.4
Bicipital tendonitis	12	1.10	5.7
Trochanteric bursitis	9	0.83	5.5
Leg pain with oedema	8	0.73	5.4
Total	1084	100	5.7

Summary

The 85% responded well to allopurinol treatment, 68.95% had Hb >12 gm%, 80% of cases with SUA >5 mg/dl also had Hb >12 gm%, p>0.05 (significant) ESR was below 20 mm in 81.00% of cases, but those with SUA >9 mg/dl had ESR between 25-40 mm in the 1st hour.

This signifies that individuals with increased SUA had increased destruction of soft tissues and bones, leading to elevated ESR (Tables 3 and 4).

The SUA was ≤5 mg/dl in 24.20% of cases, whereas it was >5 mg/dl in 75.80% of cases.

Mean±SD for SUA in this study: 5.7%±2.68, p>0.05 (significant), out of the total, 14.22% of cases had SUA

>7 mg/dl, while 63.04% of cases were between 5-8 mg/dl. SUA>11 mg/dl was seen in only 1.93% of cases (Table 5).

Table 3: Distribution of cases by SUA and Hb%.

Hb (in gm%)	SUA (<5 mg%)	SUA (>5 mg%)	Total
8-10	10	4	14
10-12	162	168	330
12-14	112	484	596
14-17	0	124	124
Total	284	800	1084
Mean±SD	12.06±1.72	13.16±1.29	12.8±1.53

Table 4: Distribution of cases by SUA and ESR.

ESR (in mm)	SUA (<5%)	SUA (>5%)	Total
<10	24	122	146
10-20	216	496	712
>20	44	182	226
Total	284	800	1084
Mean±SD	14.16±12.50	17.40±13.20	16.55±12.84

Table 5: Distribution of cases by SUA.

Serum uric acid (mg/dl)	N	Percentage (%)
<5	262	24.20
5-8	686	63.04
8-11	116	10.83
11-13	16	1.50
>13	4	0.43
Total	1084	100

In the present study, only 66 cases were over 60 years old, and only 46 of them had SUA >5 mg/dl. 322 cases were aged 40-60 years, with 210 having SUA >5 mg/dl. 570 cases were aged 20-40 years, with 452 having SUA >5 mg/dl. This shows that age had no significant correlation with SUA level.

DISCUSSION

In this series of 1084 cases, the prevalence of SUA >5 mg% was 65.80%:and out of these vegetarians were 48.80% and non-vegetarians were 51.20%. As the proportions were nearly equal, diet was not found to have a significant correlation with uric acid level.

The cases of plantar fasciitis, tennis elbow, retrocalcaneal bursitis, joint pain with synovitis, interscapular fibrofasciitis, bursitis anserine, and others who had SUA levels >5.0 mg/dl without any other causative factor proved clinically and biochemically, responded well to febuxostat/allopurinol in 85% of the cases.

The cases with SUA >7 mg% were 14.22% in our series. Another study reported that nearly 5% of India's randomly chosen population had SUA >7 mg. This study shows a definite increase in SUA levels in the Indian population.

The association of hyperlipidaemia with SUA and coronary heart disease has been explained by the pathological process that SUA increases the production of free radicals (Becker et al and Levya et al).^{21,34} The increase in peroxidative free radicals facilitates peroxidation of LDL in the arterial wall, possibly playing a role in atherosclerosis (Hoeschen, Borghi et al and Kuriyama et al).³⁵⁻³⁷ The pathological role of SUA in producing arthritis by precipitation of synovial crystals and deposition in synovial fluid is well documented. The possible explanation for all these pathogeneses is that acidity favours the precipitation of uric acid.³⁸ Even after saturation of plasma at 6.8 mg/dl, SUA does not precipitate until ~80.00 mg/dl because of solubilizing agents in plasma keep it dissolved. As joints and bursae are under constant strain, with relatively poor blood supply, uric acid can precipitate locally due to hypoxia and lack of solubilizing agents. Different types of connective tissue may have their own predisposition for precipitation and pathogenesis.³⁹

In this study, dietary restriction for an average of three weeks reduced SUA by only 1.0-1.5 mg/dl in individuals. This suggests that imbalance in renal excretion and secretion is more important than diet in causing hyperuricemia. The 85% cases with SUA >7% were associated with hyperlipidaemia. These findings stress the need to evaluate other factors influencing SUA metabolism in humans. Pollution, pesticide use, and chemicals in food lead to breakdown of nucleic acids, increasing purine production and interfering with purine metabolism.

Humans cannot convert purine into soluble allantoin, unlike some animals. This means diet must be balanced regarding fertilizer/pesticide exposure and activity levels. Sedentary life with long sitting hours increases the risk.

Limitations

The limitations of present study are its single group observational study as it was done at a prestigious private institute with high turnover of patients and creating a group and their regular involvement is not possible. Teaching institutes with research facility should conduct more study on such important discussions. These results need further evaluation in randomly chosen populations in unbiased manner.

CONCLUSION

In conclusion, in India, disease related uric acid level should be lower around 5.5 mg/dl. SUA levels in vegetarians and non-vegetarians did not differ

significantly. The high haemoglobin percent in most of the individuals gives an idea that hyperuricemia is related to good nutritional status. Cases with SUA >9 mg/dl often had higher ESR, indicating more musculoskeletal disturbance and destruction.

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