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Original Research Article

Hyperuricemia prevalence in Indian subjects with underlying comorbidities of hypertension and/or type 2 diabetes: a retrospective study from subjects attending hyperuricemia screening camps

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ABSTRACT

Background: To determine the prevalence of HU in Indian subjects attending the HU screening camps and in subjects with type 2 Diabetes Mellitus (T2DM), Hypertension (HTN), and T2DM+HTN.
Methods: This was a retrospective, non-interventional study where medical records of subjects attending HU screening camps across 592 locations in India, between June 2017 to May 2018, were analyzed.
Results: A total of 197097 subjects (T2DM: 19.69%; HTN: 14.08%; T2DM+HTN: 21.60%) attended the screening camps. Mean age of the study participants was 48.43±13.38 years (Male: 53.80%). A total of 48606(24.66%) subjects had HU. In the overall population, a higher proportion of subjects with T2DM + HTN (7.36%) had HU in comparison to subjects with T2DM (5.63%) and HTN (4.25%) alone. Similar results were reported when the data was evaluated only in HU subjects (T2DM+HTN: 29.85%; T2DM: 22.82%; HTN: 17.22%). Proportion of HU subjects increased with age, with the maximum prevalence evident in subjects aged >50 years (12.94%), followed by 30-50 years (10.65%) and <30 years (1.07%). Gender-wise, a slightly higher proportion of males (14.13%) were found to be hyperuricemic than females (10.53%). Higher proportion of subjects with disease (T2DM and/or HTN) duration of 2-5 years were found to be hyperuricemic in comparison with subjects with disease duration of >5 years or <2 years. Similar results were reported when the data was evaluated in the overall HU subjects and by indication.
Conclusions: Authors observed a high prevalence of HU among subjects attending HU camps and those with associated comorbidities. The prevalence of HU was higher in males and has an increasing trend with age. Furthermore, the prevalence of HU was observed to be higher in subjects with 2-5 years of duration of T2DM and/or HTN.

Keywords: Comorbidities, Hypertension, Hyperuricemia, Serum uric acid, Type 2 diabetes mellitus

INTRODUCTION

Uric Acid (UA) is a heterocyclic compound whose concentration in the body depends upon the balance between purine breakdown and rate of urate excretion.[1] Two-third (70%) of total UA produced daily is excreted unchanged through kidneys and the remaining one-third (30%) is broken down by intestinal flora and is excreted in the stools.[2,3] Hyperuricemia (HU) is characterized by elevated levels of Serum Uric Acid (SUA) due to deficiency of uricase enzyme or lower UA excretion resulting in the crystallization of UA into urate after exceeding the saturation level of 6.8 mg/dL at 370 C and pH 7.[4] Patients with SUA levels >7 mg/dL in men and >6 mg/dL in women were considered as hyperuricemic.[5] The prevalence of HU has steadily increased worldwide in the past 40 years with higher prevalence in Asian countries including Taiwan (10-52%), India (~25.8%), Japan (20-26%) and China (6-25%) in comparison to USA (21-22%), Brazil (13%) and Italy (9-12%).[6-10]

Hyperuricemia presents itself with symptoms like intense joint pain, redness, tenderness or swelling of joints (symptomatic HU) or without symptoms or signs of urate crystal deposition (asymptomatic HU). More than two-thirds of HU individuals are reported to have asymptomatic HU.[11] Symptomatic HU often presents as gout and nephrolithiasis due to precipitation of UA crystals in joints and tissues. Various published studies reported an association between increased SUA levels and high incidence and prevalence of gout. In a prospective 15-year study in 2046 healthy males, the annual incidence of gout was reported as 0.1% for SUA levels <7 mg/dL, 0.5% for levels 7.0-8.9 mg/dL, and 4.9% for levels >9.0 mg/dL.[12] Another study reported 5-year prevalence of gout as ~0.6% in patients with SUA <7 mg/dL and 30% in patients with SUA >10 mg/dL.[13]

In asymptomatic HU, the silent deposition of urate crystals may enhance the risk of Chronic Kidney Disease (CKD), Cardiovascular Disease (CVD), and insulin resistance syndrome.[14] Few studies have documented a higher prevalence of HU in patients with type 2 Diabetes Mellitus (T2DM) (25.35%), metabolic syndrome (47.1%), obesity (44.6%) and Hypertension (HTN) (37.33%) as compared to 14% prevalence in healthy normotensive individuals, suggesting HU to be a significant and independent risk factor for CVD, cerebrovascular diseases, HTN and T2DM.[15-17]

Hyperinsulinemia in T2DM decreases renal excretion, increases renal re-absorption and the production of UA.[18] The presence of HTN, CVD or CKD in patients with asymptomatic HU have an increased risk of urate deposition.[19] In another study, hypertensive patients with coexisting HU were at a greater risk of uncontrolled HTN, in spite of good compliance with the antihypertensive treatment.[20] Hence, patients with HTN, CVD or CKD should be screened for SUA levels to alleviate further development of urate deposition and prevent further disease-related morbidity and mortality.

Considering the growing incidence and high mortality rates of HTN and DM in the developing and the developed countries and a positive association between high SUA levels and impaired renal function, insulin resistance and high cardiovascular and other disease-related complications, more emphasis should be put on the strategy of early screening of SUA levels in these patients. This, in turn, would help in early detection, prevention, and management of T2DM and HTN, given that the prevalence of HU has increased worldwide.

There is a dearth of large-scale data (in terms of gender, age, and duration of disease) on the prevalence of HU in subjects with T2DM and/or HTN in the Indian population. Hence, this multicentric retrospective study was undertaken to determine the prevalence of HU in subjects with T2DM and/or HTN attending the HU screening camps conducted by Abbott Healthcare Pvt Ltd across India. Further, this study also determined the association between HU and age, gender and duration of disease in subjects with T2DM and/or HTN.

METHODS

Study design

This was a multicentric, retrospective, non-interventional study in which data were collected from the medical records of subjects who attended 1,50,000 screening camps. These camps were held at 592 locations from June 2017 to May 2018 across India. The camps were conducted at consulting physicians' clinics and a trained phlebotomist performed the UA detection test. All the subject records for which UA level was performed and results were available were included in the study while the subject records with incomplete information were excluded from the study. The study design has been elaborated elsewhere.[10] Since this was a retrospective data collection study, informed consent was not required. Patient confidentiality was maintained during data entry and analysis process.

Study variables

The primary outcome of the study was to determine the proportion of HU subjects by indication (T2DM, HTN, and T2DM + HTN). The secondary study outcomes were to determine the demographic characteristics (age, gender, and geographical location), clinical profile and mean UA level (mg/dL). The other study outcome was to determine the relationship between HU and different age categories (≤30 years, 31-50 years and ≥50 years), gender (men and women), underlying condition (T2DM, HTN, and T2DM + HTN) and duration of disease (≤2 years, 2-5 years and >5 years).

Statistical analysis

No formal sample size calculation was done as this was a retrospective, non-interventional study. All the subject records collected during the study period were analyzed. The statistical analysis was done using Statistical Analysis System® version 9.3 software. Descriptive statistics were used. No missing data imputation was carried out. To see the association between different indications, Pearson's chi-squared test at 5% level of significance was used.

RESULTS

Subject population

A total of 197097 subjects (T2DM: 38799 [19.69%]; HTN: 27742 [14.08%]; HTN + T2DM: 42585 [21.60%]; other underlying diseases: 87971 [44.63%]) attended the HU screening camps during the study period. The data of all the subjects were analyzed in the study. More than 30% of the subjects (31.6%) were from the southern region of India (Figure 1). The mean age of the overall population was

48.43±13.38 years. The majority (51.04%) of the subjects were in the age group of 30-50 years. The proportion of males was higher than females (53.80% versus 46.20%). The mean age was comparable across subjects with T2DM, HTN, and T2DM+HTN (Table 1).

Table 1: Baseline characteristics.

Characteristics		Data
Age (Years), Mean±SD		48.43±13.38
Gender, n (%)	Female	91068(46.20%)
	Male	106029(53.80%)
Age (years), n (%)	≤30	14321(7.26%)
	30-50	100589(51.04%)
	≥50	82187(41.70%)
Comorbidities, n (%)	T2DM	38799(19.69%)
	HTN	27742(14.08%)
	T2DM+HTN	42585(21.60%)
	Others	87971(44.63%)
Subjects with comorbidities and hu, n (%)	T2DM	11,091(5.63%)
	HTN	8,372(4.25%)
	T2DM+HTN	14,507(7.36%)
	Others	14,636(7.43%)
Age (years) of subjects with comorbidities, mean±sd	T2DM	50.16±12.51
	HTN	51.67±12.57
	T2DM+HTN	52.64±12.75
	Others	44.6±13.26

Prevalence of hyperuricemia

Out of 197097 subjects, a higher proportion of subjects with T2DM + HTN had HU in comparison to subjects with T2DM and HTN alone (14507(7.36%) versus 11091(5.63%) and 8372(4.25%), respectively). The proportion of HU subjects in the overall population increased with age; the maximum prevalence was evident in subjects aged >50 years (25500(12.94%)), followed by age groups of 30-50 years (20992(10.65%)) and <30 years (2114(1.07%)). Gender-wise, a higher proportion of males were hyperuricemic than females (27856(14.13%) versus 20750(10.53%)).

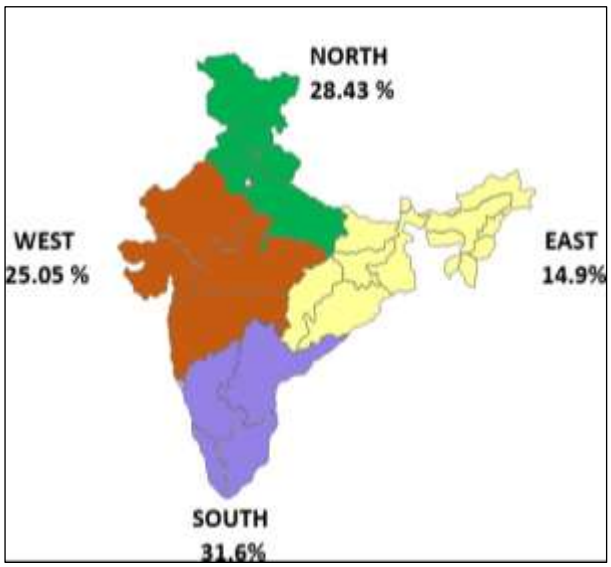

Figure 1: Region wise distribution of subjects.

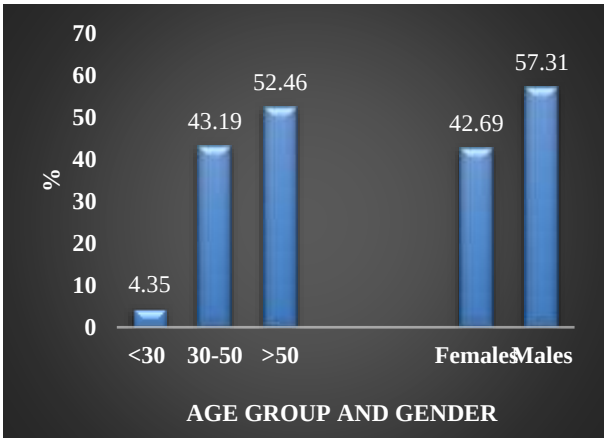

Figure 2: Hyperuricemic Subjects as per Age Groups and Gender (n=48606).

Table 2: Proportion of hyperuricemic subjects by age, gender and duration of disease in subjects with type 2 diabetes, hypertension and type 2 diabetes + hypertension.

Characteristics		Number (%) of subjects with high uric acid levels		
		T2DM (N=38799)	HTN (N=27742)	T2DM+HTN (N=42585)
Age (years)	<30	232(0.60%)	219(0.79%)	262(0.62%)
	30-50	4696(12.10%)	3478(12.54%)	5344(12.55%)
	>50	6163(15.88%)	4675(16.85%)	8901(20.90%)
Gender	Female	4998(12.88%)	3573(12.88%)	6422(15.08%)
	Male	6093(15.70%)	4799(17.30%)	8085(18.99%)
Duration (years) of disease	< 2	1524(3.93%)	1606(5.79%)	351(0.82%)
	2-5	1817(4.68%)	3342(12.05%)	2794(6.56%)
	>5	1156(2.98%)	2400(8.65%)	2716(6.38%)
Unknown		6594(17.0%)	1024(3.69%)	8646(20.30%)

The similar results were obtained across different indications in the overall population (Table 2) and even when the data was evaluated in HU subjects as overall (n=48606; 24.66%) (Table 3, Figure 2) and by indications (T2DM: 11091(22.82%); HTN: 8372(17.22%); T2DM+HTN: 14507(29.85%); (Table 4). Subjects with disease duration of 2-5 years were more hyperuricemic in comparison to subjects with disease duration of >5 years or <2 years among subjects with T2DM (n=38799), HTN (n=27742), and T2DM+HTN (n=42585) (Table 2). The similar results were reported in HU subjects with T2DM, HTN, T2DM+HTN (Table 4). In the HU subjects (n=48606), a statistically significant association was reported between HU and age and gender across different indications (p<0.0001). There was also a statistically significant association between subjects with high UA levels and duration of HTN and T2DM+HTN (p<0.0001) (Table 4).

Table 3: Characteristics of hyperuricemic subjects (n=48606).

	Characteristics	Number (%) of subjects
Age (years)	<30	2114(4.35%)
	30-50	20992(43.19%)
	>50	25500(52.46%)
Gender	Females	20750(42.69%)
	Males	27856(57.31%)

Table 4: Proportion of hyperuricemic subjects by age, gender and duration of disease in subjects with type 2 diabetes, hypertension and type 2 diabetes + hypertension and hyperuricemia.

Characteristics		Number (%) of subjects with high uric acid levels			p value
		T2DM (N=11091)	HTN (N=8372)	T2DM+HTN (N=14507)	
Age (years)	<30	232(2.09%)	219(2.62%)	262(1.8%)	p<0.0001
	30-50	4696(42.34%)	3478(41.54%)	5344(36.84%)	
	>50	6163(55.57%)	4675(55.84%)	8901(61.36%)	
Gender	Females	4998(45.06%)	3573(42.68%)	6422(44.27%)	p<0.0001
	Males	6093(54.94%)	4799(57.32%)	8085(55.73%)	
Duration (years) of disease	< 2	1524(13.74%)	1606(19.18%)	351(2.42%)	p<0.0001
	2-5	1817(16.38%)	3342(39.92%)	2794(19.26%)	
	>5	1156(10.43%)	2400(28.67%)	2716(18.72%)	
Unknown		6594(59.45%)	1024(12.23%)	8646(59.60%)	

DISCUSSION

Hyperuricemia is a highly prevalent disorder which is increasing gradually not only in the advanced countries but in the developing countries as well. The elevated SUA levels are caused by underexcretion (due to renal dysfunction) or overproduction (by the liver) of UA. Serum UA has been identified as a potential biomarker for predicting the development of HTN, DM and CKD.[21-23] At the tissue level, chronic exposure to increased UA promotes vascular changes leading to renal ischemia and stimulation of renin-angiotensin system and development of insulin resistance, hypertriglyceridemia, and hepatic steatosis through pro-oxidative mechanisms.24 Therefore, early screening of UA levels is advisable to prevent and manage complications of elevated levels of SUA, especially in subjects with CVD, CKD and metabolic syndrome, including T2DM and HTN. There is limited information available regarding the burden of HU in the Indian subcontinent, hence the current retrospective pan India study with large sample size (n=197097) was conducted to determine the prevalence of HU in subjects examined in HU screening camps and to analyze the possible association of HU with age, gender, disease duration and comorbidities (T2DM, HTN, and T2DM+HTN).

In the study, 31.6% of the enrolled subjects were from the southern region of India followed by northern (28.43%), western (25.05%) and eastern (14.9%) regions, encompassing subject enrolment from all four zones of the country. The prevalence of HU among subjects was 24.66%. The results were in concordance with previously published study where ~25.8% of the Indian subjects were reported to have HU.[10] Several other studies also demonstrated similar HU prevalence across different countries in the Asian continent but the prevalence of HU in this study was much higher in comparison to countries outside the Asia.[7-9] This could be due to multiple reasons: 1) enrolment of high-risk patients and not the healthy general population in the study, 2) intake of purine-rich foods/poor diet/high-fructose containing drinks/alcohol/high-fat dairy products, 3) sedentary lifestyle, or 4) excessive use of diuretics and cyclosporine.[25] Of all HU subjects (n=48606), females were less hyperuricemic than males (42.69% versus 57.31%) possibly due to high estrogen levels in premenopausal females which promotes SUA excretion by inhibition of renal urate reabsorption via organic ion transporter.[26] Similar results were reported across various literature.[10,15,27-29] Further, battery of published literature supports an increase in HU prevalence with advancing age, which may be due to inheritance of acquiring age-related diseases (such as metabolic diseases, cardiovascular or renal-related diseases),

adverse effects of medications (due to its rampant usage), endogenous synthesis of purines or lower excretion of UA with age progression.[10,30-32] In this study as well, the proportion of HU subjects in the overall population increased with age; maximum subjects were evident in the age category >50 years (12.94%), followed by age categories of 30-50 years (10.65%) and <30 years (1.07%). Similar results (gender- and age-wise) were obtained when the data were evaluated by comorbidities (T2DM, HTN, and T2DM+HTN) and across only HU subjects.

Subjects with metabolic diseases such as HTN, T2DM, dyslipidemia, CKD, and obesity presents with high SUA levels.[26,33,34] In a recent study, Mundhe and Mhasde reported a significantly higher prevalence of HU in subjects with metabolic syndrome against those without metabolic syndrome (47.1% vs. 7.3%; $p<0.05$).[15] In this study, the prevalence of HU was lower in subjects with T2DM (5.63%) and HTN (4.25%) in comparison to earlier reports wherein 25% of T2DM subjects35 and 26%-56% HTN subjects had reported HU.[36-39] The lower HU prevalence among our T2DM and HTN subjects as compared to other studies could be possibly attributed to the higher diagnostic cut off for SUA level or variability in the diagnosis procedure. In this study, subjects with SUA levels >7 mg/dL were considered as hyperuricemic, while some trial defined HUA as UA greater than 7 mg/dL in males and greater than 6 mg/dL in females.[40-42] Moreover, authors also speculate that the majority of subjects who visited camps were not of severe disease category, hence the prevalence of HU could have been underestimated. In addition, participants who were taking certain antihypertensive therapies such as thiazides and other diuretics were not identified in this study. It is noteworthy that these antihypertensive medications reduce UA excretion leading to enhanced SUA levels.[43] In a study, it was reported that the prevalence of HTN increases by 1.2 fold with an increase in SUA levels by 1 mg/dL, after adjusting age, BMI, dyslipidemia, diabetes, smoking, and estimated glomerular filtration rate (eGFR).[26] Another prospective study involving more than 2000 patients demonstrated that high SUA level predicts the development of future HTN independent of age, alcohol use or renal function.[44] Hence, it is imperative to screen the SUA level amongst T2DM, HTN and HTN+T2DM cases, in particular with uncontrolled nature of the disease as it may increase the risk of CKD-, CVD- and other disease-related complications due to reduced excretion of urates.[45]

In T2DM+HTN cases, the prevalence of HU was higher in comparison to T2DM and HTN cases (7.36% versus 5.63% and 4.25%, respectively) suggesting an exacerbating effect of both diseases in UA retention. HTN increases renal vasoconstriction and T2DM increases hyperinsulinemia, which further increases renal re-absorption and UA production.[18,46] In a meta-analysis, UA lowering was reported to be associated with significant reduction in serum creatinine concentration and an increase in estimated eGFR8, suggesting the possibility that early treatment of HU may prevent the development of HTN. Apart from managing HU, individualized diet management through health education measures is of utmost importance.

Duration of T2DM and/or HTN plays an important role in increasing the SUA levels. In this study, HU prevalence predominated in subjects with 2-5 and >5 years of duration rather than those with <2 years of disease. This may be possibly due to the reason that with the progression of the disease, SUA levels rises. However, the same trend was not observed in T2DM cases as subjects with >5 years of duration had the least prevalence of HU. Similar observation was reported elsewhere where subjects with less or equal to 10 years of diabetes duration were 3-times more hyperuricemic than subjects with longer (>10 years) duration of diabetes (26.4% vs. 7.3%).[47] Nevertheless, it is unclear in this study whether the observed findings could be generalized because the duration of disease was not known in more than half (59.45%) of T2DM subjects. This result was contrary to authors previous observation wherein an increasing trend was recorded between HU positive cases and duration of T2DM and HTN.[10]

Though this study is the first of its kind where a larger subset of population was assessed for SUA, however, the interpretation of the present results is confronted by some limitations. Firstly, the data analyses were restricted to the retrospectively collected data from different healthcare clinics, which limited the viability of these findings. Also, the sample size was not calculated statistically. Secondly, authors did not collect data on serum insulin levels, which is an index for insulin resistance and would have been an important parameter for meaningful interpretation of this study results. Thirdly, there was no healthy comparator group, which restricted ability to compare the SUA levels between different comorbidities and healthy population. In addition, retrospective analyses limited ability to explore the association between the SUA levels and different stages of HTN, other comorbidities. Furthermore, this study involved patients who are at high risk of HU and is not a true representation of the Indian population. Nevertheless, this cross-sectional multicentric study has provided baseline data on the prevalence of HU in the Indian population and different comorbidities. This data can be useful in clinical practice in improving the management of HU and in preventing the complications associated with escalated SUA levels.

CONCLUSION

The overall prevalence of HU among subjects attending hyperuricemia camps in Indian population was 24.66%. The HU burden was also higher in subjects with T2DM and HTN (7.36%) in comparison to subjects with T2DM (5.63%) and HTN (4.25%). The prevalence of HU was higher in males and showed an increasing trend with age. Furthermore, a higher HU prevalence was observed

across all comorbidities, with a disease duration of 2-5 years. Hence, a regular screening of HU is of utmost importance, particularly when a patient is at high risk of HU, had uncontrolled T2DM, HTN, and T2DM+HTN, CKD or CVD. Further well-designed prospective and randomized case-controlled studies are warranted to evaluate the prevalence of HU in patients with comorbid diseases.

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