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

Himanshu Patel¹*, Dhiren Shah²

¹Department of Nephrology, Consultant, Zydus Hospitals, Thaltej, Gujarat, India
²Department of Nephrology, Consultant, Jaslok Hospital, Mumbai, Maharashtra, India

Received: 19 December 2020
Accepted: 16 January 2020

*Correspondence:
Dr. Himanshu Patel,
E-mail: himanshu667@icloud.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

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 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 7.² 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. 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. 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.

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. 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.

Hyperinsulinemia in T2DM decreases renal excretion, increases renal re-absorption and the production of UA. The presence of HTN, CVD or CKD in patients with asymptomatic HU have an increased risk of urate deposition. 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. 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. 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%)).

### 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 urea levels |
|----------------------------------|---------------------------------------------|
|                                 | T2DM (N=38799)                             |
|                                 | HTN (N=27742)                              |
|                                 | T2DM+HTN (N=42585)                         |
| Age (years)                     |                                             |
| <30                              | 232(0.60%)                                 |
| 30-50                            | 4696(12.10%)                               |
| >50                              | 6163(15.88%)                               |
| Gender                           |                                             |
| Female                           | 4998(12.88%)                               |
| Male                             | 6093(15.70%)                               |
| Duration (years) of disease      |                                             |
| <2                               | 1524(3.93%)                                |
| 2-5                              | 1817(4.68%)                                |
| >5                               | 1156(2.98%)                                |
| Unknown                          | 6594(17.0%)                                |

Figure 1: Region wise distribution of subjects.

Figure 2: Hyperuricemic Subjects as per Age Groups and Gender (n=48606).
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 | p value |
|-----------------|------------------------|---------|
| Age (years)     |                        |         |
| <30             | 232(2.09%)             |         |
| 30-50           | 4696(42.34%)           |         |
| >50             | 6163(55.57%)           |         |
| Gender          |                        |         |
| Females         | 4998(45.06%)           | p<0.0001|
| Males           | 6093(54.94%)           |         |
| Duration (years) of disease |         |         |
| <2              | 1524(13.74%)           |         |
| 2-5             | 1817(16.38%)           |         |
| >5              | 1156(10.43%)           |         |
| Unknown         | 6594(59.45%)           |         |

### 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 | p value |
|-----------------|------------------------|---------|
| Age (years)     |                        |         |
| <30             | 2114(4.35%)            |         |
| 30-50           | 20992(43.19%)          |         |
| >50             | 25500(52.46%)          |         |
| Gender          |                        |         |
| Females         | 20750(42.69%)          |         |
| Males           | 27856(57.31%)          |         |

### 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, hypertiglyceridemia, 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.29 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.23 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.20,15,27-29 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. 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. 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). 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. 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. 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. 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). 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. 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.

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. In a meta-analysis, UA lowering was reported to be associated with significant reduction in serum creatinine concentration and an increase in estimated eGFR, 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%). 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.

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.

ACKNOWLEDGEMENTS

GCE Solutions provided writing assistance and Dr Shalini Nair (Abbott) provided additional review and editorial support for the manuscript.

Funding: The study was funded by Abbott Healthcare India Private Limited
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Singh V, Gomez V, Swamy S. Approach to a Case of Hyperuricemia. Ind J Aerospace Med. 2010;54(1):40-6.
2. Johnson RJ, Rideout BA. Uric acid and diet-insights into the epidemic of cardiovascular disease. New Eng J Med. 2004 Mar 11;350(11):1071-3.
3. Eggebeen AT. Gout: an update. Am Fam Phys 2007;76(6):801-8.
4. Martillo MA, Nazzal L, Crittenden DB. The crystallization of monosodium urate. Curr Rheumatol Rep. 2014 Feb 1;16(2):400.
5. Chizynki K, Rozycka M. Hyperuricemia. Pol Nefrol. 2004;19(113):693-6.
6. Edwards NL. The role of hyperuricemia in vascular disorders. Curr Opin Rheumatol. 2009 Mar 1;21(2):132-7.
7. Smith E, March L. global Prevalence of Hyperuricemia: A Systematic Review of Population-based Epidemiological Studies: abstract Number: 2236. Arthr Rheumatol. 2015 Oct;67:2690-2.
8. Wang J, Chen RP, Lei L, Song QQ, Zhang RY, Li YB, et al. Prevalence and determinants of hyperuricemia in type 2 diabetes mellitus patients with central obesity in Guangdong Province in China. Asia Paci J Clin Nutr. 2013;22(4):590.
9. Lin CS, Lee WL, Hung YJ, Lee DY, Chen KF, Chi WC, et al. Prevalence of hyperuricemia and its association with antihypertensive treatment in hypertensive patients in Taiwan. Inter J Cardiol. 2012 Apr 5;156(1):41-6.
10. Billia G, Dargad R, Mehta A. Prevalence of hyperuricemia in Indian subjects attending hyperuricemia screening programs: a retrospective study. J Assoc Phys Ind. 2018 Apr;66:43-6.
11. Luk AJ, Simkin PA. Epidemiology of hyperuricemia and gout. Am J Manag Care. 2005 Nov 1;11(15 Suppl):S435-42.
12. Campion EW, Glynm RJ, Delabry LO. Asymptomatic hyperuricemia. Risks and consequences in the Normative Aging Study. Am J Med. 1987 Mar 1;82(3):421-6.
13. Agudelo CA, Wise CM. Crystal-associated arthritis. Clin Geriatr Med. 1998 Aug 1;14(3):495-514.
14. Becker MA, Mount DB. Asymptomatic hyperuricemia. 2019. Available at https://www.uptodate.com/contents/asymptomatic-hyperuricemia. Accessed 7 January 2020.
15. Mundhe SA, Mhasde DR. The study of prevalence of hyperuricemia and metabolic syndrome in type 2 diabetes mellitus. Int J Adv Med. 2016 Apr;3:241-9.
16. Remedios C, Shah M, Blasker AG, Lakdawala M. Hyperuricemia: a reality in the Indian obese. Obes Surg. 2012 Jun 1;22(6):945-8.
17. Shrivastav C, Kaur M, Suhalka ML, Sharma S, Basu A. Hyperuricemia-A Potential Indicator to Diagnose the Risk of Essential Hypertension. J Clin Diag Res: JCDR. 2016 Mar;10(3):CC01.
18. Modan M, Halkin H, Karasik A, Lusky A. Elevated serum uric acid-a facet of hyperinsulinaemia. Diabetol. 1987 Sep 1;30(9):713-8.
19. Kerr G, Dowell S, Wells A, Haddad R, DeMarco P, Joseph J, et al. FRIO236 Associations between comorbidity and urate deposition in subjects with asymptomatic hyperuricemia: a pilot study Annal Rheum Dis. 2018;77:659.
20. Cho J, Kim C, Kang DR, Park JB. Hyperuricemia and uncontrolled hypertension in treated hypertensive patients: K-MetS Study. Medicine. 2016 Jul;95(28).
21. Grayson PC, Kim SY, LaValley M, Choi HK. Hyperuricemia and incident hypertension: a systematic review and meta-analysis. Arthr Care Res. 2011 Jan;63(1):102-10.
22. Li L, Yang C, Zhao Y, Zeng X, Liu F, Fu P. Is hyperuricemia an independent risk factor for new-onset chronic kidney disease?: A systematic review and meta-analysis based on observational cohort studies. BMC Nephrol. 2014 Dec 1;15(1):122.
23. Lv Q, Meng XF, He FF, Chen S, Su H, Xiong J, et al. High serum uric acid and increased risk of type 2 diabetes: a systemic review and meta-analysis of prospective cohort studies. PloS One. 2013;8(2).
24. Viazzi F, Bonino B, Ratto E, Desideri G, Pontremoli R. Hyperuricemia, diabetes and hypertension. G Ital Nefrol. 2015;32(62).
25. Kuwabara M. Hyperuricemia, cardiovascular disease, and hypertension. Pulse. 2015;3(3-4):242-52.
26. Kuwabara M, Niwa K, Nishi Y, Mizuno A, Asano T, Masuda K, et al. Relationship between serum uric acid levels and hypertension among Japanese individuals not treated for hyperuricemia and hypertension. Hyper Res. 2014 Aug;37(8):785-9.
27. Tuomilehto J, Zimmet P, Wolf E, Taylor R, Ram P, King H. Plasma uric acid level and its association with diabetes mellitus and some biologic parameters in a biracial population of Fiji. Am J Epidemiol. 1988 Feb 1;127(2):321-36.
28. Conen D, Wietlisbach V, Bovet P, Shamlaye C, Riesen W, Paccoud F, et al. Prevalence of hyperuricemia and relation of serum uric acid with cardiovascular risk factors in a developing country. BMC Pub Health. 2004 Dec 1;4(1):9.
29. Ogbera AO, Azemao AO, Hyperuricaemia and the metabolic syndrome in type 2 DM. Diabetol Metab Syndr. 2010 Dec 1;2(1):24.
30. Mikkelsen WM, Dodge HJ, Valkenburg H, Himes S. The distribution of serum uric acid values in a population unselected as to gout or hyperuricemia: Tecumseh, Michigan 1959-1960. Am J Med. 1965 Aug 1;39(2):242-51.
31. Culeton BF, Larson MG, Kannel WB, Levy D. Serum uric acid and risk for cardiovascular disease and death: the Framingham Heart Study. Ann Int Med. 1999 Jul 6;131(1):7-13.
32. Povoroznyuk VV, Dubetska GS. Hyperuricemia and age. Gerontol. 2012;13(3):149-53.
33. Kuwabara M, Niwa K, Nishi Y, Niinuma H, Nishihara S, Anzai H, et al. The positive relationship between uric acid and hypertension in Japanese people not taking antihypertensive drugs. J Hyper. 2011;29:e32-e33.
34. Agarwal V, Hans N, Messerli FH: Effect of allopurinol on blood pressure: a systematic review and metaanalysis. J Clin Hyper. 2013;15:435-42.
35. Anthonia O, Alfred O. Hyperuricemia and metabolic syndrome in type 2 DM. Diabetol Metab Syndr. 2010;2:24.
36. Kinsey D, Smithwick R, Walther R, Whitelaw G, SISE H. Incidence of hyperuricemia in 400 hypertensive patients. Circulation. 1961;24(4):972.
37. Kolbel F, Gregorova I, Souka J. Serum uric acid in hypertensives. Lancet. 1965;1:519.
38. Kahn HA, Medalie JH, Neufeld HN, Riss E, Goldbourt U. The incidence of hypertension and associated factors: the Israel ischemic heart disease study. Am Heart J. 1972 Aug 1;84(2):171-82.
39. Shrivastav C, Kaur M, Suhalka ML, Sharma S, Basu A. Hyperuricaemia-A Potential Indicator to Diagnose the Risk of Essential Hypertension. J Clin Diag Res: JCDR. 2016 Mar;10(3):CC01.
40. Levy GD, Rashid N, Niu F, Cheetham TC. Effect of urate-lowering therapies on renal disease progression in patients with hyperuricemia. J Rheumatol. 2014 May 1;41(5):955-62.
41. Ali N, Perveen R, Rahman S, Mahmood S, Rahman S, Islam S, et al. Prevalence of hyperuricemia and the relationship between serum uric acid and obesity: a study on Bangladeshi adults. PLoS One. 2018;13(11):e0206850.
42. You L, Liu A, Wuyun G, Wu H, Wang P. Prevalence of hyperuricemia and the relationship between serum uric acid and metabolic syndrome in the Asian Mongolian area. J Athero Throm. 2014 Apr 24;21(4):355-65.
43. Grassi D, Ferri L, Desideri G, Di Giosia P, Cheli P, Del Pinto R, et al. Chronic hyperuricemia, uric acid deposit and cardiovascular risk. Curr Pharma Design. 2013 Apr 1;19(13):2432-8.
44. Perlstein TS, Gumieniak O, Williams GH, Sparrow D, Vokonas PS, Gaziano M, et al. Uric acid and the development of hypertension. Hypertension. 2006;48:1031-6.
45. Quinones Galvan A, Natali A, Baldi SI, Frascerra SI, Sanna GI, et al. Effect of insulin on uric acid excretion in humans. Am J Physiol-Endocrinol Metab. 1995 Jan 1;268(1):E1-5.
46. Messerli FH, Frohlich ED, Dreslinski GR, Suarez DH, Aristimuno GG. Serum uric acid in essential hypertension: an indicator of renal vascular involvement. Ann Intern Med 1980;93(6):817-21.
47. Woyesa SB, Hirigo AT, Wube TB. Hyperuricemia and metabolic syndrome in type 2 diabetes mellitus patients at Hawassa university comprehensive specialized hospital, South West Ethiopia. BMC Endo Dis. 2017 Dec 1;17(1):76.

Cite this article as: Patel H, Shah D. Hyperuricemia prevalence in Indian subjects with underlying comorbidities of hypertension and/or type 2 diabetes: a retrospective study from subjects attending hyperuricemia screening camps. Int J Res Med Sci 2020;8:794-800.