

Hyperuricemia in Patients with Chronic Low Back Pain: Experience from a Single Institutional Neurosurgical OPD

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Abstract

Objective: To evaluate the frequency of hyperuricemia in patients with chronic low back pain.

Methodology: A descriptive cross sectional study was conducted over a period of 4 months from August 2015 to November 2015 at the Neurosurgery Clinic, Liaquat National Hospital and Medical College, Karachi. A total of 104 patients were evaluated between the ages of 18 to 75 years with chronic low back pain. Data was collected by means of a structured Performa. X-rays and Magnetic Resonance Imaging (MRI) of lumbo-sacral spine were used to assess any findings in relation to low back pain. Serum uric levels were laboratory tested and recorded. A statistical association was established between hyperuricemia and chronic low back pain with respect to age, gender and related radiological findings.

Results: Twenty six patients (25%) had elevated serum uric acid levels with no significant difference in genders. Patients in the age group of 26-60 years showed a higher frequency of hyperuricemia as compared to other age groups. There was a significant association of hyperuricemia with large joint pain of the lower limb as seen in 22 patients (85%). Other significant radiological findings were lumbar disc prolapse found in 19 out of 26 patients (73%), degenerative disc disease in 54% (n=14) and disc space narrowing in 96% (n=25) patients (P<0.05).

Conclusion: Our study suggests existence of hyperuricemia in 1 out of every 4 patients with low back pain, irrespective of the patient's gender, with higher . There is a variable association of occupation and pre-existing co-morbidities of the patients with low back pain. Our study predicts a strong association of lumbar disc

prolapse and joint space narrowing of the lumbar vertebrae with hyperuricemia. It raises a question that perhaps hyperuricemia augments the age related spondylolesthesis by mechanisms not understood so far.

Keywords: Lowbackpain; Hyperuricemia; Serumuricacid; Spinalgout

Introduction

Chronic low back pain is a common symptom experienced by more than 80% of the population at some time during their lives [1]. It presents a significant health problem worldwide leading to economic burden of the patients in terms of treatment cost and subsequent impact on job performance. Low back pain may radiate towards the legs and can be associated with joint pain of the lower limbs. The presence of joint pain can be a result of underlying hyperuricemia, which manifests as inflammation of joints leading to joint pain. Hyperuricemia is abnormally high serum levels of uric acid which occurs due to inadequate protein metabolism. The normal range of serum uric acid is 3.4-7.2 mg/dL (200-430 µmol/L) for men and 2.4-6.1 mg/dL (140-360 µmol/L) for women [2,3]. About 10% of people with hyperuricemia develop gout, which is monosodium urate crystal deposition disorder in and around large joints. According to a local study, the prevalence of gout is high in patients over 50 years of age with a male predominance [4,5]. Risk factors for the development of hyperuricemia and consequent presentation of gout are related to increasing longevity, dietary and lifestyle changes, and presence of co morbidities [6,7]. Gout typically affects the peripheral joints of the appendicular skeleton, and has been reported to rarely involve the axial joints. Spinal gout, though diagnosed late, is associated with spinal cord compression, osseous erosion and lumbar disc degeneration leading to chronic lower back pain [8]. It usually involves the

posterior spinal elements, para spinal soft tissues, sacroiliac joints, and even the intervertebral disc [9,10]. The use of Magnetic Resonance Imaging (MRI) for radiological analysis yields homogeneous, intermediate to low signal on T1-weighted images and variable signal intensity on T2-weighted images [11]. This study aims to evaluate hyperuricemia in patients presenting with chronic low back pain with respect to age gender and correlated radiological findings.

Material and Methods

This study was conducted over a period of 4 months from August 2015 to November 2015. A descriptive cross sectional study was performed, and patients were recruited through convenient sampling. A total of 104 patients, with low back pain of more than 3 months duration aged 18 to 70 years were included. A detailed history was taken from all patients to inquire about co morbidities like hypertension, diabetes, ischemic heart disease, tuberculosis, chronic renal failure, asthma, hepatitis and obesity. Low back pain was evaluated, keeping in view its duration and any associated radicular leg pain and joint pain of the lower limb. Radiological analysis of low back pain was done through X-rays and MRI of lumbosacral spine, looking for significant changes such as disc enlargement, disc degeneration and joint space narrowing of the lumbar vertebrae. Analysis of serum uric acid was done through laboratory results in all the patients. The normal range of serum uric acid was taken as 3.4-7.2 mg/dL (200-430 μ mol/L) for men and 2.4-6.1 mg/dL (140-360 μ mol/L) for women. Levels above the normal range were considered elevated and used as the confounding variable to establish any association with hyperuricemia.

Data was entered and analyzed using SPSS version 20.0. Frequency and percentages were evaluated for all the categorical variables like age, gender, joint pain, disc enlargement, disc degeneration, joint space narrowing of lumbar vertebrae and the patient serum uric acid levels. Chi-square test and Fischer's exact test were applied to check association between joint pain, lumbar disc prolapse, disc degeneration, joint space narrowing of lumbar vertebrae and their respective evaluation with serum uric acid levels. Effect modifiers were controlled through stratification of age and gender. P-value < 0.05 was considered as significant.

Results

In this study, a total of 104 patients with low back pain were evaluated and out of which 26 (25%) showed increased levels of serum uric acid. Hyperuricemia had nearly equal occurrence in both genders, showing a slightly higher frequency in females 26% as compared to males 24% (Table 1).

Table 1: Distribution of serum uric acid levels with respect to gender.

Gender	Serum uric acid level	Frequency	Percentage
	Normal	41	75.9

Male	Elevated	13	24.1
	Total	54	100
	Normal	37	74
Female	Elevated	13	26
	Total	50	100

The patient population was further divided into three age groups of 18-25, 26-60 and 60 years or above, the number of hyperuricemic patients in each group was 36.4%(n=4), 23.5% (n=19) and 25%(n=3) respectively. The mean age of patients presenting with low back pain was 43 years. The highest frequency of hyperuricemia was seen in middle aged patients (26-60 years) (Table 2).

Table 2: Distribution of serum uric acid levels in the 3 age groups.

Age group	Serum Uric acid level	Frequency	Percentage
18-25 years	Normal	7	63.6
	Elevated	4	36.4
	Total	11	100
26-60 years	Normal	62	76.5
	Elevated	19	23.5
	Total	81	100
60 years and above	Normal	9	75
	Elevated	3	25
	Total	12	100

Further analysis revealed that 18.2% (n=19) patients did not have any known co-morbidities. According to our study concomitant joint pain had a significant association with hyperuricemia in low back pain patients (P<0.05). Out of 26 patients with hyperuricemia, 85% (n=22) had associated joint pain, particularly in the knee and tarsal joints. On further radiological assessment, lumbar disc prolapse was present in 19 out of 26 patients (73%, p<0.05) whereas, lumbar disc degeneration occurred in 54% patients (n=14, p<0.05). A significant association was observed between disc space narrowing of lumbar vertebra and hyperuricemia. Disc space narrowing of the lumbar vertebrae was found to exist in 96% of the patients (n=25, P<0.05).

Discussion

Hyperuricemia is raised level of uric acid in the blood that may occur as a consequence of defective protein metabolism. The normal range of serum uric acid is 3.4-7.2 mg/dL (200-430 μ mol/L) for men and 2.4-6.1 mg/dL (140-360 μ mol/L) for women [2,12,13]. Hyperuricemia has been found to correlate with co-morbidities such as cardiovascular diseases, genetic factors, dietary factors, alcohol consumption, metabolic

syndrome (including hypertension and obesity), diuretic use and renal disease [6,7,13,14]. Hyperuricemia manifesting as gout usually presents as pain in the large joints of the appendicular skeleton and inflammation of the articular surfaces (Table 3).

Table 3: Association of elevated serum uric acid levels with radiographic findings.

	Frequency	P Value
Joint pain	22/26	<0.05
Lumber disc prolapse	19/26	<0.05
Disc space narrowing	25/26	<0.05
Disc Degeneration	14/26	0.579

Many factors can contribute to the onset of low back pain. In our study we laid emphasis on the corresponding serum uric acid levels to access hyperuricemia in low back pain patients. To the best of our knowledge this is the first study of its kind to assess the prevalence of hyperuricemia in patients with chronic lower back pain in Pakistan. Our study showed a frequency of 25% hyperuricemia in chronic low back pain patients, suggesting that increased uric acid levels can be of prime importance in factors responsible for aggravating back pain or a cause of spinal gout. Gender showed no direct correlation with hyperuricemia and low back pain. However our results showed that 77% of the patients presenting with low back pain were between the ages of 26-60 years. Weaver et al. [15] also reported a elevated prevalence of gout in the higher age group. Occupation of patients showed a variable relationship with both low back pain and hyperuricemia accompanied by any pre-existing co morbidities like hypertension, diabetes, ischemic heart disease, TB, chronic renal failure, asthma, vitamin D3 deficiency and obesity. However this correlates with the study of Rukmini et al. [16] who did not find any difference in prevalence of gout between different age groups or with other comorbidities.

The common presentation of joint pain had a significant association with hyperuricemic low back pain patients in our study. About 81% of the patients with hyperuricemia presented with pain in the knee and metatarsophalangeal joints, as concluded by Rukmini et al. [16]. This can be explained by the presentation of gouty arthritis that leads to development of joint pain [17].

Although the diagnostic capability of computed tomography for assessing spinal gout is precise than Magnetic Resonance Imaging (MRI), we included only X-rays and MRI analysis of the lumbo-sacral spine due to financial constraints of the patients and also because they have been reported as useful in diagnosing gout [18,19]. In our study, 58% patients having hyperuricemia had joint space narrowing of the lumbosacral vertebrae on MRI scans, as was seen by Bloch et al. [3] in his radiologic evaluation of gout patients. Another study by Michael et al. [20] suggests that joint space narrowing causes clinical low back pain. Our results show a clear evidence that there is a significant role of hyperuricemia in aggravating

chronic low back pain. The statistical correlation with MRI findings suggests that hyperuricemia can accentuate the process of degenerative spondylolesthesis. There is a definitive need for a large scale study to map out more concrete evidence regarding the demographical distribution as well as etiological mechanisms associated with hyperuricemia in low back pain patients.

The limitations of our study were a small sample size, and the fact that this study was carried out in a single hospital setting. Although the study had been conducted in a tertiary hospital specialty clinic, fortunately for us, the patients in our clinic come from all over Pakistan representing a broad spectrum of socio-economic groups of the country's population.

Conclusion

Our study suggests existence of hyperuricemia in 1 out of every 4 patients with low back pain, irrespective of the patient's gender. There is a variable association of occupation and pre-existing co-morbidities of the patients with low back pain. Our study predicts a strong association of lumbar disc prolapse and joint space narrowing of the lumbar vertebrae with hyperuricemia. This suggests that hyperuricemia augments degenerative Spondylolisthesis by mechanisms not understood so far.

Author Contribution

All the authors contributed equally to the following:

The conception and design analysis and interpretation of the data. The critical revision for important intellectual content, critical appraisal of findings with the literature search and actual write up of manuscript.

References

1. Freburger JK (2009) The rising prevalence of chronic low back pain. *Arch Intern Med* 169: 251-258.
2. Boss GR, Seegmiller JE (1979) Hyperuricemia and gout: Classification, complications and management. *N Engl J Med* 300: 1459-1468.
3. Bloch C, Hermann G, Yu TF (1980) A radiologic reevaluation of gout: a study of 2,000 patients. *AJR Am J Roentgenol* 134: 781-787.
4. Akram M (2011) Prevalence of gout in Gadap town, Karachi Community, Pakistan. *African Journal of Biotechnology* 10: 7893-7895.
5. Smith EU, Díaz-Torné C, Perez-Ruiz F, March L M (2010) Epidemiology of gout: an update. *Best Pract Res Clin Rheumatol* 24: 811-827.
6. Choi HK, Mount DB, Reginato AM (2005) Pathogenesis of gout. *American College of Physicians; American Physiological Society. Ann Intern Med* 143: 499-516.
7. Arromdee E, Michet CJ, Crowson CS, O'Fallon W M, Gabriel S (2002) Epidemiology of gout: is the incidence rising? *J Rheumatol* 29: 2403-2406.

8. Igbiniedion BO, Akhigbe A(2011) Correlations of Radiographic Findings in Patients with Low Back Pain. *Niger Med J* 52: 28-34.
9. Popovich T, Carpenter JS, Rai AT, Carson LV, Williams HJ, et al. (2006) Spinal cord compression by tophaceous gout with fluorodeoxyglucose-positron-emission tomographic/MRfusion imaging. *AJNR Am J Neuroradiol* 27: 1201-1203.
10. Kelly J, Lim C, Kamel M, Keohane C, O'Sullivan M (2005) Topacheous gout as a rare cause of spinal stenosis in the lumbar region. Case report. *J Neurosurg Spine* 2: 215-217.
11. Hsu CY, Shih TT, Huang KM, Chen PQ, Sheu JJ, et al. (2002) Tophaceous gout of the spine: MR imaging features. *Clin Radiol* 57: 919-925.
12. Aarsand AK, Sandberg S (2013) How to achieve harmonisation of laboratory testing -The complete picture. *Clin Chim Acta* doi: 10.1016/j.cca.2013.12.005. [Epub ahead of print].
13. Roddy E, Doherty M (2010) Epidemiology of gout. *Arthritis Res Ther* 12: 223.
14. Keenan RT, Pillinger MH (2009) Hyperuricemia, gout, and cardiovascular disease--an important "muddle". *Bull NYU Hosp Jt Dis* 67: 285-290.
15. Weaver AL (2008) Epidemiology of gout. *Cleve Clin J Med* 75 Suppl 5:S9-12.
16. Konatalapalli RM (2009) Gout in the axial skeleton. *J Rheumatol* 36: 609-613.
17. Neugebauer V, Han JS, Adwanikar H, Fu Y, Ji G (2007) Techniques for assessing knee joint pain in arthritis. *Mol Pain* 28: 3-8.
18. McQueen FM, Doyle A, Dalbeth N (2011) Imaging in gout--what can we learn from MRI, CT, DECT and US? *Arthritis Res Ther* 13: 246.
19. Dalbeth N, McQueen FM (2009) Use of imaging to evaluate gout and other crystal deposition disorders. *Curr Opin Rheumatol* 21: 124-131.
20. Hall MC, Selin G (1960) Spinal involvement in gout, a case report with autopsy. *JBJS Case Connector* 42: 341-343.