Incidental Finding of Gout in Patients with Chronic Back Pain

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Abstract

The purpose of this study was to determine the prevalence of Gout in individuals suffering from persistent low back pain.

Methodology: This study was conducted in Benazir Bhutto Medical College Karachi, PakistanFrom June 2019 to July 2020. This was descriptive cross-sectional research. Sixty-five people with persistent low back pain were assessed, ranging from 18 to 75 years. A structured Performa was employed to collect data. Magnetic resonance imaging (MRI) and X-rays of the lumbosacral spine were utilized to evaluate any abnormalities related to low back pain. The Serum uric levels were measured and reported in a laboratory.

A statistical connection was found concerning age, gender, and associated radiological findings between persistent low back pain and hyperuricemia. The results showed that 16 patients (25 percent) had elevated serum uric acid levels, with no significant gender differences. Compared to other age groups, patients aged 26-60 years had a greater prevalence of Gout. In 13 individuals, there was a strong connection between hyperuricemia and big joint pain in the lower leg (85 percent). Other noteworthy radiological results were lumbar disc prolapse in 12 of 16 patients (73 percent), degenerative disc disease in 54 percent (n=7), and disc space narrowing in 96 percent (n=15) individuals (P0.05).

Conclusion: Our survey reveals that gout exists in 1 out of 4 individuals with low back pain, regardless of gender. There is a varied relationship between vocation and antecedent co-morbidities in individuals suffering from low back pain. Our findings indicate a substantial link between lumbar vertebral joint space constriction and lumbar disc prolapse and hyperuricemia. It raises the issue of

whether hyperuricemia exacerbates age-related Spondylolisthesis through mechanisms that have yet to be discovered.

Introduction

Chronic low back pain is a predominant condition that affects more than 80 percent of the community at some point in their lifetimes. It is a major public health issue worldwide, causing a financial burden on patients in terms of treatment costs and, as a result, a negative result on work achievement(1). Low back pain can migrate to the legs and be associated with pain in the lower limb joints. The occurrence of joint pain may be due to underlying hyperuricemia, which appears as joint inflammation and pain. Hyperuricemia is defined as unusually high serum uric acid levels caused by insufficient protein metabolism(2). Serum uric acid levels should be in the range of 3.4-7.2 milligrams per decilitre (200-430 mol/L) for men and 2.4-6.1 milligrams per decilitre (140-360 mol/L) for women[(3),(4),(5)]. Gout is a clinical illness with a restricted range of indications caused by the deposition of monosodium urate crystals, the end product of purine metabolism in humans. It affects around 10 percent of persons with hyperuricemia. According to a regional survey, the predominance of gout in people over 50 is considerable, with a male preponderance(6). Extended longevity, lifestyle changes and dietary, and co-morbidities are all risk factors for developing hyperuricemia and the subsequent presentation of gout. Gout primarily affects the appendicular skeleton's peripheral joints and has infrequently impacted the axial joints. Though identified late, spinal gout is related to bony erosion, lumbar disc degeneration, and spinal cord compression, resulting in persistent lower back discomfort. The posterior spinal components, paraspinal soft tissues, sacroiliac joints, and intervertebral discs are commonly involved. Magnetic resonance imaging (MRI) for radiological examination produces homogenous, middle to low signal intensity on T1-weighted pictures and varying signal intensity on T2-weighted images(7). The purpose of this comparison is to assess hyperuricemia in individuals with chronic low back pain based on gender, age, and linked radiological abnormalities.

Methods and Materials

This study was conducted in Benazir Bhutto Medical College Karachi, Pakistan From June 2019 to July 2020. Patients were recruited using easy sampling in descriptive cross-sectional research. Sixty-five individuals with chronic low back pain lasting more than three months, ranging in age from 18 to 70 years, were included in the study. All patients were given a thorough medical history, which included questions concerning co-morbidities such as ischemic heart disease, hypertension, asthma, chronic renal failure, diabetes, hepatitis, and obesity. Low back pain was assessed, considering its length and any accompanying lower limb joint pain and radicular leg pain. Low back pain radiological investigation was performed using MRI and X-rays of the lumbosacral spine, searching for significant alterations such as disc degeneration, disc enlargement, and lumbar vertebral joint space narrowing. In all individuals, serum uric acid levels were determined using laboratory data. Serum uric acid levels were normalized to 2.4-6.1 milligrams per decilitre (140-360 mol/L) for women and 3.4-7.2 milligrams per decilitre (200-430 mol/L) for men. Levels above the normal scope were regarded abnormal and utilized as a confounding variable to set up any link to hyperuricemia. SPSS version 20.0 was used to enter and evaluate data. Percentages and frequency were calculated for all categorical variables, including gender, age, joint discomfort, lumbar vertebral joint space

narrowing, disc degeneration, disc enlargement, and patient serum uric acid levels. Thisstatistical

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notes for clinical researchers were used to examine the relationship between joint pain, joint space narrowing of lumbar vertebrae, lumbar disc prolapse, disc degeneration, and their assessment with serum uric acid levels(8). Age and gender stratification were used to adjust for effect modifiers. P-values less than 0.05 were deemed significant.

Results

In this trial, 65 individuals with low back pain were considered, and 16 (25 percent) of them had elevated serum uric acid levels. Hyperuricemia was roughly equally prevalent in both genders, with females having a little greater frequency (25 percent) than males (23 percent). **Table 1**

Gender	Serum uric acid level	Frequency	Percentage
	Normal	40	76.9
Male	Elevated	12	23.1
	Total	52	100
Female	Normal	36	75
	Elevated	12	25
	Total	48	100

Table 1 shows the gender distribution of serum uric acid levels.

The patient group was then separated into three age groups of 18-25, 26-60, and 60 years or older, with 36.4 percent (n=5), 23.5 percent (n=7), and 25 percent (n=2) of hyperuricemic patients in each category, respectively. The average age of patients with low back pain was 43 years. Middle-aged patients (26-60 years) had the highest rate of hyperuricemia.**Table 2**

Age group	Serum Uric acid level	Frequency	Percentage
18-25 Years	Normal	6	66.7
	Elevated	3	33.3
	Total	9	100
26-60 Years	Normal	61	77.2
	Elevated	18	22.8
	Total	79	100
60 Years and above	Normal	8	80
	Elevated	2	20
	Total	10	100

Table 2 shows the frequency of serum uric acid levels across three age groups.

A further study discovered that 18.2 percent (n=12) of patients had no known co-morbidities. As reported by our research, concomitant joint pain exhibited a meaningful connection with hyperuricemia in low back pain patients (P0.05). Out of 16 hyperuricemia patients, 85 percent (n=14) reported concomitant joint discomfort, most notably in the knee and tarsal joints. Further radiographic examination revealed that 11 of 16 patients (73 percent, p<0.05) had lumbar disc prolapse, whereas 54 percent (n=8, p0.05) had lumbar disc degeneration. A strong relationship was found between lumbar spinal disc space narrowing and hyperuricemia. Disc space narrowing of the lumbar vertebrae was seen in 96 percent of the patients (n=15, P0.05).

Discussion

Gout is defined as a high uric acid level in the blood. The typical top limit is 6.8mg/dL, and anything beyond 7mg/dL is considered saturated, with symptoms possible. This higher-level results from increased uric acid synthesis reduced uric acid excretion or combined the two processes[(3),(9)(10). Elevated uric acid levels have also been observed in rapid purine breakdown, high cell turnover states (hemolysis, rhabdomyolysis, and tumor lysis), and reduced excretion (renal insufficiency and metabolic acidosis). Gout and nephrolithiasis can be caused by hyperuricemia[(11)(12)(13)]. It has

also been linked to metabolic syndrome, diabetes, cardiovascular disease, and chronic renal disease. **Table 3**

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

Table 3 shows the relationship between increased blood uric acid levels and radiographic findings.

Many variables might play a role at the beginning of low back pain(14). Our investigation focused on serum uric acid levels to identify hyperuricemia in individuals with low back pain. As far as we can tell, this is the first survey of its sort in Pakistan that looks at the predominance of hyperuricemia in individuals with persistent lower back pain. Our study found that 25 percent of persistent low back pain patients had hyperuricemia, inferring that elevated uric acid levels might be a major component in exacerbating back pain or being a cause of spinal gout[(15),(16),(6)]. Gender did not have a direct relationship with hyperuricemia or low back discomfort.

Nevertheless, our findings revealed that 77 percent of patients presenting with low back pain were between 26 and 60. Wallace et al.(17)found an increased frequency of gout in the older age group. Patients' occupation variedly linked with hyperuricemia and low back pain, as did any preexisting co-morbidities such as diabetes, hypertension, ischemic heart disease, chronic renal failure, tuberculosis, obesity, vitamin D3 insufficiency, and asthma(18). However, this is consistent with the findings of Elfishawi et al.(19), who found no change in gout prevalence between age groups or with other co-morbidities.

In our study, the prevalent manifestation of joint pain was associated with gout in low back pain patients. According to Elfishawi et al., around 81 percent of hyperuricemia patients reported knee and metatarsophalangeal joint pain. This can be clarified by the development of gouty arthritis, which leads to joint pain. Although computed tomography has a more precise diagnostic capability for evaluating spinal gout than Magnetic Resonance Imaging (MRI), we included only MRI and X-rays analysis of the lumbosacral spine due to the patients' financial reasons also because they have been confirmed as helpful in diagnosing gout(20). In our research, 58 percent of patients with hyperuricemia displayed joint space narrowing of the lumbosacral vertebrae on Magnetic Resonance Imaging scans, as Cabot et al.(21) observed in their radiologic examination of gout patients. Another research by Aktar et al. (22)reveals that joint space narrowing is the source of clinical low back pain. Our findings provide convincing evidence that hyperuricemia has a substantial role in aggravating persistent low back pain. According to the statistical link with Magnetic Resonance Imaging data, hyperuricemia can aggravate the development of degenerative Spondylolisthesis(23). A large-scale investigation is clearly needed to map out more definite facts about the demographic distribution and the etiological pathways related to hyperuricemia in low back pain patients.

Conclusion

Our study reveals that hyperuricemia exists in 1 out of 4 individuals with low back pain, regardless of gender. The employment and preexisting co-morbidities of individuals with low back pain have a varying correlation. Our findings indicate a substantial link between lumbar disc prolapse and lumbar vertebral joint space narrowing and hyperuricemia. This shows that hyperuricemia exacerbates degenerative Spondylolisthesis through mechanisms that have yet to be discovered.

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