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Hyperuricemia in Plantar Fasciitis- Attributable Cause or Associated Factor?

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Abstract:- Pain at the heel affects millions of people globally. The burden of this entity can be virtually visualised by the fact that around 7% of people over 65 years of age report heel pain and tenderness, the main differential diagnosis being plantar fasciitis, amongst others. Plantar fasciitis is one of the most common causes of pain around the heel and plantar aspect of the foot. 80% of patients with heel pain are suffering from plantar fasciitis. Plantar fasciitis has been linked to many mechanical abnormalities and biochemical derangements. The most common biochemical derangement in patients with plantar fasciitis is increased serum uric acid levels. There is increasing evidence that presence of asymptomatic hyperuricemia is a potential risk factor for development of plantar fasciitis. The mean serum uric acid levels in the patients with plantar fasciitis was 7.02 mg/dl, which is in close proximity to the upper limit of normal uric acid levels. Also, the same was true for males and females independently. Even though majority of patients with plantar fasciitis accounting to 52% had serum uric acid levels between 6-8 mg/dl, close to the upper limit of normal value, it cannot be categorised as hyperuricemia. By virtue of this study, we can infer that hyperuricemia per se, cannot be an attributable cause of plantar fasciitis, even though it is known to play an important role in pathogenesis of calcaneal plantar spur formation, which might lead to or augment the pre existing plantar fasciitis.

Keywords:- *Hyperuricemia, Plantar Fascitis, Gout, Heel Pain, Calcaneum, Associated Factor, Cause.*

I. INTRODUCTION

Pain at the heel affects millions of people globally. The burden of this entity can be virtually visualised by the fact that around 7% of people over 65 years of age report heel pain and tenderness, the main differential diagnosis being plantar fasciitis, amongst others like heel spurs, retrocalcaneal bursitis, Achilles tendinitis, tarsal tunnel syndrome, peripheral neuropathy, nerve entrapment syndromes and neuroma. Plantar fasciitis is one of the most common causes of pain around the heel and plantar aspect of the foot. 80% of patients with heel pain are suffering from plantar fasciitis. Patients often report pain at the anteromedial prominence of the calcaneum, which worsens with passive dorsiflexion of the toes. Typically, the pain is most intense when first standing after a period of rest, particularly in the morning. Although the pain generally decreases with initial walking, it usually doesn't completely subside and may even worsen with prolonged activities or exercise, especially on hard surfaces [1]. Plantar fasciitis has been linked to many mechanical abnormalities and biochemical derangements. The most common biochemical derangement in patients with plantar fasciitis is increased serum uric acid levels. There is increasing evidence that presence of asymptomatic hyperuricemia is a potential risk factor for development of plantar fasciitis. However, the association of hyperuricemia with plantar fasciitis is not clear. The aim of this study was to investigate the relationship between hyperuricemia and plantar fasciitis and to gain clarity with respect to whether hyperuricemia is just an associated finding in patients with plantar fasciitis or an attributable cause of plantar fasciitis.

II. MATERIALS AND METHODS

- Type of study : Retrospective study
- Study setting: Department of Orthopaedics, Justice KS Hegde Charitable Hospital, Deralakatte, Mangalore
- Study period: 1st July 2023 to 31st July 2024.
- Inclusion criteria : Patients between age group of 18-75 years who were diagnosed to have plantar fasciitis and were followed up in our department and had laboratory report of serum uric acid levels.
- Methods and Data collection: Patients with plantar fasciitis were identified from the Medical Records Department (MRD) of the hospital, by using the appropriate International Classification of Diseases (ICD)- code. All the demographic, laboratory and follow-up data were obtained from the patient files and OPEMR.

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The demographic data collected include:

- Age of patient
- Sex of patient,
- Body mass index (BMI)
- Serum uric acid level.

For this study, as per hospital Biochemistry laboratory curetted values,

- Normal serum uric acid levels for females : 2.50–6.20mg/dL.
- Normal serum uric acid levels for males : 3.50–8.50mg/dL.

Hyperuricemia can be defined as serum uric acid levels more than or equal to 6.0 mg/dl for females and more than or equal to 8.0 mg/dl for males.

BMI of the patient was also recorded and tabulated. BMI was grouped as normal, overweight and obese based on the values as shown below in Table (I).

Table 1 Grouping Based on BMI of Patients

NORMAL =	18.5 – 24.9
OVERWEIGHT =	25 – 29.9
OBESE =	>/= 30

III. RESULTS

Total number of patients diagnosed to have plantar fasciitis as per the data retrieved from the medical records department of the hospital on entering respective ICD code were forty seven. Out of these forty seven patients, sixteen patients did not have records of laboratory report of serum uric acid levels and hence were excluded.

Total of thirty one patients having plantar fasciitis with laboratory report of serum uric acid levels were included in this study, as illustrated in figure (I).

The mean serum uric acid levels in the thirty one patients with plantar fasciitis included in this single centre retrospective study was 7.02 mg/dl.

The mean serum uric acid levels in males was 7.8 mg/dl and in females was 6.2 mg/dl, as shown in table (II).

The mean serum uric acid levels in patients who had normal BMI was 6.2 mg/dl, in patients who were overweight was 7.9 mg/dl and in obese patients was 8.8 mg/dl respectively as shown in table (III).

Patients were grouped into 3 groups according to their age as given in table (IV) and the mean uric acid levels in patients aged more than or equal to seventy years was 8.6 mg/dl, in patients aged between fifty years to sixty nine years was 6.7 mg/dl and in patients aged between thirty years to forty nine years was 6.3 mg/dl.

Serum uric acid levels of all the thirty one patients was tabulated and grouped as shown in table (V) and expressed as frequency and percentages.

Four (12.9 %) of the patients with plantar fasciitis had uric acid levels less than 4 mg/dl, five (16.1 %) of them had uric acid levels between 4 mg/dl-6 mg/dl, sixteen (51.6 %) of them had uric acid levels between 6 mg/dl-8 mg/dl, six (19.3 %) of them had uric acid levels more than 8 mg/dl.

Table 2 Gender wise mean serum uric acid levels

SEX OF THE PATIENT	MEAN SERUM URIC ACID LEVELS (mg/dl)
MALE	7.8
FEMALE	6.2

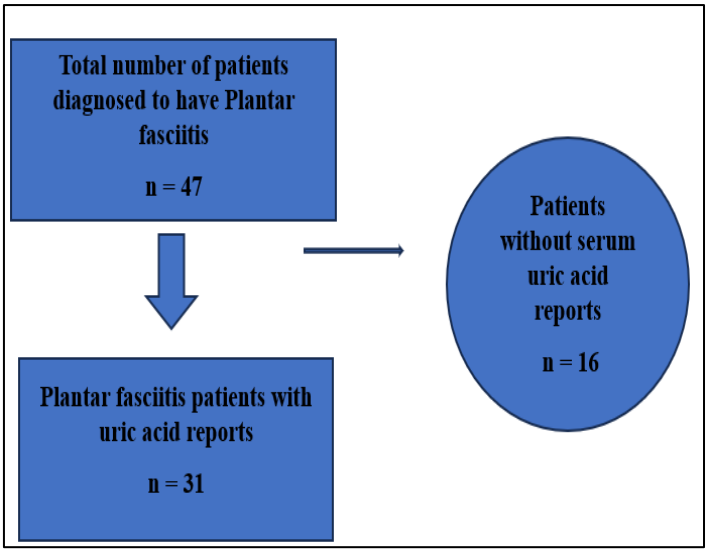

Fig 1 Showing Algorithm of Number of Patients Included in the Study after Diagnosis of Plantar Fasciitis with Exclusion of those without Uric Acid Report.

Table 3 Mean Serum Uric Acid Levels based on Patient's BMI Grouping.

BMI OF THE PATIENT (Kg/M2)	MEAN SERUM URIC ACID LEVELS (mg/dl)
NORMAL (18.5 – 24.9)	6.2
OVERWEIGHT (25 – 29.9)	7.9
OBESE (>/= 30)	8.8

Table 4 Mean Serum Uric Acid Levels based on Different Age Grouping of Patients.

AGE OF PATIENT (YEARS)	MEAN SERUM URIC ACID LEVELS (mg/dl)
>/ = 70	8.6
50-69	6.7
30-49	6.3

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Table 5 Distribution of Serum Uric Acid Level in Patients Expressed as Frequency and Percentage.

RANGE OF SERUM URIC ACID LEVEL (mg/dl)	FREQUENCY (PERCENTAGE)
< 4 mg/dl	4 (12.9 %)
4 – 6 mg/dl	5 (16.1 %)
6 - 8 mg/dl	16 (51.6 %)
> 8 mg/dl	6 (19.3 %)

IV. DISCUSSION

In this study, we aimed to explore the relationship between hyperuricemia and plantar fasciitis. Previous researches have shown that imaging studies reveal monosodium urate crystal deposits in 25-40% of patients with both plantar fasciitis and hyperuricemia [13]. These crystals typically accumulate in joints and periarticular structures, particularly in the first metatarsophalangeal joint, midfoot, and knee [14].

Ames et al. suggested that hyperuricemia could damage microvascular structures and impair tissue oxygenation, potentially leading to Achilles tendinopathy in individuals with gout [15].

However, there is a lack of studies specifically investigating whether hyperuricemia alone can cause plantar fasciitis or if the two conditions simply coexist. This study seeks to address this gap by examining the relationship between hyperuricemia and plantar fasciitis.

Research by Comberg and Schach found a significant association between elevated uric acid levels and musculoskeletal joint pain, with urate levels impacting non-specific joint pain in areas such as the lumbar spine, cervical spine, shoulder, and knee [16]. Stewart et al. observed that patients with asymptomatic hyperuricemia also reported foot and leg pain, even in the absence of gout symptoms [17].

Other studies have linked hyperuricemia to knee and foot pain, even without clinical gout, with elevated uric acid levels being more common in individuals experiencing joint pain compared to those without pain, and predominantly affecting those over 40 years of age. Similarly, Andersson and Leden's prospective study of 124 women found a notable association between serum uric acid levels and chronic non-gouty musculoskeletal pain [18].

Studies have well-established that hyperuricemia contributes to the development of gout, its potential role in causing plantar fasciitis remains unexplored. The degree of hyperuricemia is a known predictor for gout, but its influence on plantar fasciitis have not been studied. Nossent et al. found that despite a high prevalence of hyperuricemia (10.7% of the population), uric acid levels were not independently predictive of unexplained musculoskeletal pain. However, Hsu et al. reported that in renal patients, hyperuricemia could independently predict musculoskeletal symptoms [19].

Given the absence of studies directly linking hyperuricemia with plantar fasciitis, this research aims to provide a foundation for future investigations and help establish an understanding of their relationship.

This study has several limitations: Firstly, it involved a retrospective design with patients already diagnosed with plantar fasciitis, which introduces selection bias. Secondly, the research was conducted at a single hospital, which may lead to hospital-specific biases. Lastly, while many patients had lateral foot radiographs, ultrasonographic imaging of the plantar fascia was not performed, which may have affected the accuracy of diagnosing plantar fasciitis and ruling out other causes of heel or plantar pain.

V. CONCLUSION

The study aimed to fill the lacunae in research regarding the relationship between hyperuricemia and plantar fasciitis. By exploring this potential connection, it aimed to lay the groundwork for future investigations and enhance our understanding of whether hyperuricemia could influence the development of plantar fasciitis.

The mean serum uric acid levels in the patients with plantar fasciitis was 7.02 mg/dl, which is in close proximity to the upper limit of normal uric acid levels. Also, the same was true for males and females independently. Even though majority of patients with plantar fasciitis accounting to 52% had serum uric acid levels between 6-8 mg/dl, close to the upper limit of normal value, it cannot be categorised as hyperuricemia.

By virtue of this study, we can infer that hyperuricemia per se, cannot be an attributable cause of plantar fasciitis, even though it is known to play an important role in pathogenesis of calcaneal plantar spur formation, which might lead to or augment the pre existing plantar fasciitis. It is also imperative to note that routine screening of plantar fasciitis patients for hyperuricemia might not be feasible as it is just an associated finding and might not influence or alter the course of the treatment. The initiation of uricosuric medications in patients of plantar fasciitis with hyperuricemia having a beneficial role in resolution of plantar fasciitis is the question yet to be answered and more research is required to bring out clarity regarding the same.

This study concludes that hyperuricemia is a co-incidental or associated finding in plantar fasciitis and cannot be designated as an attributable cause for plantar fasciitis.

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