Painful Calcaneal Spur Often Misdiagnosed for Lumbar Radicular Pain

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Abstract---Background: Persistence of painful calcaneal spur (PCS) and loss of long-term effect may be related to unrecognized low back pain (LBP), dysfunction and/or lumbosacral radiculopathy (LSR), but prevalence of LBP in PCS patients has not been established. Purpose: to determine the prevalence of LBP among individuals with and without PCS. Methods: A cross-sectional study of individuals with (n = 26) and without (n=27) PCS. X-ray used to determine calcaneal spur. MRI and X-ray (as available), and clinical tests used to determine LBP/pathology/LSR. Results: A greater percentage of individuals with PCS had LBP (88.5% vs. 33% in controls), lumbar pathology (58 vs. 19), and LSR (54 vs. 15). Conclusion: Individuals with painful calcaneal spur had a greater prevalence of LBP/lumbar pathology/LSR. Treatment to address impairments related to lumbar spine may be necessary to enhance the treatment of PCS.

Keywords---calcaneal spurs, lumbosacral radiculopathy, misdiagnosed, painful calcaneal spur (PCS), plantar fasciitis.

Introduction

Heel pain (HP) affects 10–44% of patients from the general population, and 80% of cases caused by plantar fasciitis (PF) (1,2). Calcaneal spurs found in 45% of patients with (3) and in 10–60% without PF or HP (3-5), and not found in 46% of patients with painful heel (4), at age 40–60, aggravated while taking the initial few

How to Cite:
steps after long period of inactivity (6). Several factors can affect PF/HP as long standing (7-8), Neuromuscular deficit (9), High body mass index (10), Fascial thickening (11), foot pronation, high arch and tight Achilles tendon (12,13), Plantar nerve entrapment (2), Heel pad atrophy (14). Causal relation of spur to pain is contradicted by several studies and findings. Near half of patients had unilateral plantar heel pain, yet both feet had spurs or are exposed to same risk factors (4,15). Osteophytes as spurs are common above age of 50 years, not because of disease but coming with age (16). Fascial thickening precedes the formation of calcaneal spurs (9). Wearing et al. (17-19) stated that there is extremely little proof that any one of numerous recognized risk elements are in fact the reason for HP/PF, yet instead, these variables act to modulate the pain levels once it exists. pain and spurs might establish from an usual underlying pathologic condition (2). These studies doubt that spur or fascial thickening are primary cause of HP and other source of pain may be masked (i.e., spine), as lumbosacral radiculopathy (LSR) that may lead to neuro-muscular impairments in lower limb that lead to overloading soft tissues of foot leading to fascial thickening and the spur.

Studies about PF/HP excluded neurological disorders, definite osteoarthritis of lumbo-sacral spine (20,21), peripheral neuropathy (21). But, they did not present how they could identify that. Other claimed that an elderly patient, who had foot pain in dorsal and plantar surface with burning sensation for 6 months, had normal lumbar and ankle motion, strength, sensation and reflexes (22). Heel pad atrophy, common with PF/HP, associated with spondyloarthropathy (23). Clinical guidelines, based on international classification of function and diseases, diagnose PF with only moderate evidence, when patient had; plantar medial heel pain: most obvious with first steps following a duration of inactivity but additionally worse after extended weight bearing, HP precipitated by a recent rise in weight-bearing activity, Pain with palpation of the plantar fascia proximal insertion, Positive windlass test, Negative tarsal tunnel tests, Limited passive and active ankle joint dorsiflexion range of motion (ROM), Abnormal foot posture index score, High BMI in nonathletic individuals (24) with no strict exclusion of spine. This leaves a gap that spine may be affected the foot indistinctly or indirectly.

PF/HP treated with local stretching and strengthening (2,13,24,25), NSAIDs, orthoses (2), tape (24), iontophoresis (25, 26), and laser (25,28), in addition to platelet-rich plasma (PRP) (29), corticosteroid injections (CI), and extracorporeal shock wave therapy (ECSWT) (21,27, 29-34), and local surgeries (21, 35, 36). Guidelines not recommend electrical stimulation, Ultrasound, or weight loss (24). No agreement has been gotten about the top treatment way of PF with PCS and the results of the treatment procedures. Several treatments have been inconsistent and short term (29). ECSWT altered the morphology of musculoskeletal conditions, potentially reflecting variations in underlying pathophysiological processes, irrespective of parameters of SWT dosage (33). Findings of these studies may suggest presence of another factor (as spine). Pain that patients report as originating from the heel may likewise be associated with issues of the of the lumbar spine (37). HP may be pain of neural origin or others (38). Ankle pain was associated with LBP. more study is required to make clear the system of this association, which will give beneficial details for avoidance and also therapy of LBP (39). Less and asymmetry of maximal voluntary contraction at
plantar flexors maybe associated with advancement of the LBP throughout long-term standing in the back-healthy people. additional research is required to explore plantar flexors relation to development of LBP during prolonged standing (40). Self-myofascial release had an immediate clinical benefit on the flexibility of the hamstrings and lumbar spine (41). Handgrip strength was adversely associated with functional status in Sagittal imbalance and symptomatic lumbar spinal stenosis (42). A newly, highly valid and reliable, developed system score for PHP had weighted spur only 2 points but walking 4 points, VAS 5 points (43). These studies may suggest that proximal source may be the cause of PF/HP not the spur, but more research is needed to support.

Lumbosacral radicular pain is characterized by aggravating pain of a shooting, sharp, or burning sensation, with or without paresthesia in one or several lumbar or sacral dermatomes; it might or might not be come with other radicular irritation symptoms or disability. The most usual cause is intervertebral disc herniation, wherein the leaving nerve root is placed under stress by the displaced disc material. An additional significant source of lumbosacral radicular pain is stenosis of lumbosacral canal. It is the most common neuropathic pain with annual prevalence of 9.9% to 25%. Conservative management includes physical therapy, exercise and medication (44-46). For a picked population, less than 15%, a surgical treatment leads to a more rapid minimizing of the acute radicular problems in contrast with a conservative method, however the results after one to two years are equal (47-49).

**Methods**

This study aimed to determine and compare the prevalence of LBP among individuals with and without PCS.

**Design**

A cross-sectional study was done.

**Setting**

Study was conducted at Kasr AlAiny hospital, Cairo, Egypt.

**Participants**

Individuals with (n = 26) and without (n = 27) painful calcaneal spur (PCS) with age from 20-70 years were included.

- Inclusion criteria; Cases should have; Heel pain, Calcaneal spur on X-ray, and Both sexes
- Exclusion criteria; Trauma, Fractures, or Neurological diseases

**Procedures**

- Prevalence of LBP among individuals with and without PCS was calculated, as percentage of cases with LBP or LSR in each group.
- Cases with calcaneal spur were identified based on presence of inferior calcaneal osteophyte on Plain lateral X-ray.
- Heel pain and LBP determined by asking participants, Do you have heel pain? Do you have LBP?
- If they had calcaneal spur and heel pain, entered PCS group. If they had no heel pain, entered control.

Then presence of LSR determined by reporting dermatomal pain and/or paraesthesia and/or weak dorsiflexors and/or reduced knee (extensor) and ankle (plantar) reflexes, during SLR test, sensory testing, reflex testing. Lasègue test: A straight leg-raising test with the patient supine is deemed positive if radicular pain (in one leg, combined with one or more positive neurological signs that indicate a nerve root irritation or neurological loss of function (50), is elicited between 10° and 60° and is indicative of compression at L4/L5, often lumbar herniated disc (44,51) with sensitivity 0.91 and specificity of 0.26 (52, 53).

MRI and X-rays of the cases, as available, were seen to determine confirm presence of lumbar pathology.

**Statistics**

Demographics reported as range in continuous and count and percent in nominal variables. Prevalence of LBP among individuals with and without PCS reported as no (%), and compared with Z-test. Odds ratio (OR) was calculated. P<0.5 was set.

**Results**

Demographic data of the individuals existed in table (1).

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Cases</th>
<th>Controls</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>49 (10)</td>
<td>48 (19)</td>
<td>0.4</td>
</tr>
<tr>
<td>Duration of pain (ms)</td>
<td>1.5-36</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Male percentage</td>
<td>5/26</td>
<td>17/27</td>
<td>0.001</td>
</tr>
<tr>
<td>Side of spur</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>6</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>5</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Bilateral</td>
<td>15</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

A higher percent of individuals with PCS had LBP 23 (88.5%) versus 8 (33%) of controls, odds ratio = 2.7, $P <0.001$ and LSR (54% versus 15% of controls, odds ratio =3.6, $P=0.001$), symptomatic lumbar pathology on imaging (57.7% versus 18.5% of controls, odds ratio =1.9, $P=0.002$), see table (2).
Table 2
Prevalence of LBP/LSR in PCS

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>PCS (n=26) No. (%)</th>
<th>Control (n=27) No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBP</td>
<td>23 (88.5)</td>
<td>9 (33)</td>
</tr>
<tr>
<td>Lumbar pathology</td>
<td>15 (57.7)</td>
<td>5 (18.5)</td>
</tr>
<tr>
<td>SLR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Left</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Bilateral</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>14 (54)</td>
<td>4 (15)</td>
</tr>
</tbody>
</table>

In individuals with PHP and LSR, agreement is present between side of LSR and heel pain in 13 cases (93%) (being the same in 6 cases + unilateral in LSR and bilateral in heel pain in 6 (assuming that LSR caused abnormal loading on other foot and hence PCS) + bilateral in LSR and unilateral in HP in 1 (assuming that LSR was more to one side, hence PCS on one side), and no agreement in 1 case (7%) (right on one and left on other). If assuming that unilateral on one and bilateral on other is disagreement, agreement was 43% (data not shown).

**Discussion**

The current study found high prevalence of LBP/LSR in patients with PCS (88.5%). This finding come in line with McClinton et al. (54) who found a higher percentage of individuals with PHP had LBP (74% versus 37% of controls, odds ratio = 5.2, P = 0.009), but the present study found a higher percent. As well, Yabe et al. (39) found 13% of LBP in players with LL pain and OR of LBP of 3.8 in ankle pain. LBP/dysfunction/LSR may lead to neuromuscular deficit creating strength deficits in the intrinsic or extrinsic muscles of the foot can make overload and asymptomatic thickening of an otherwise healthy plantar aponeurosis (9). Isolated manual therapy reduced pain and function in PF (55). There was no significant benefit of physical therapy treatment along with common podiatry management in the main result of foot and ankle ability measures (FAAM) change at 6 months (56). This finding may support our results that other factor rather than foot may be present (spine).

Current study finding support treatment incorporated clinical guidelines and recent evidence that combine manual therapy, patient education, stretching, resistance training, and neurodynamic interventions for PF treatment (54). Persons with PHP symptoms more than 7 months need further attention and added examination of efficient methods to enhance treatment reaction (57). Based on finding of our study, this consideration and these strategies may be needed to be directed to spine. Treatment directed to lumbosacral nerve roots and paraspinals in patient diagnosed by the podiatrist as bilateral “plantar fasciitis, was effective (58), supporting our result that HP may be from spine and treatment should be directed to spine.
Measurement of plantar fascia is not useful as a diagnostic or prognostic method for functional recovery from PF (34). No correlation between clinical outcome and radiologic changes, as no significant radiologic change after ESWT therapy, however pain shortly decreases (30). These suggest there is other source of pain rather than plantar fascia or spur. Most patients with radicular pain (~60%) reported allodynia, on the leg, often extending into the foot. The level of allodynia associated with the level of background pain. The existence of allodynia recommends that the peripheral nerve creators of background leg and back pain have also made central sensitization. The distal (foot) site of the allodynia in patients who have it shows that the nociceptive drive that keeps the central sensitization arises paraspinal (ectopically) in injured ventral ramus afferents; this is not an example of somatic referred pain (59). So, HP may arise centrally not locally.

Batesha and Abd Elrazik Gad Elhak (60) found relation between trunk control and foot plantar pressure and that trunk control training improved plantar pressure distribution in LSR patients. This agrees with our finding that lumbar spine may be the primary cause of HP not spur or fascial thickening. Mohamed Badr found that up to 16% of patients with no evidence of LSR (using MRI, NCV criteria) had LSR symptoms (atrophy, reduced jerks) (unpublished data). This supports our view that even LBP without LSR may cause symptoms in LL especially foot that may cause HP/PF

**Conclusion**

Individuals with painful calcaneal spur had a greater prevalence of LBP. Treatment to address impairments linked to LBP may be necessary to enhance the treatment of PCS.

**References**

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29. Meriç Uğurlar, Mesut Mehmet Sönmez, Özge Yapıcı Uğurlar, Levent Adıyake, Hakki Yıldırım, Osman Tuğrul Eren. Effectiveness of Four Different Treatment Modalities in the Treatment of Chronic Plantar Fasciitis During a 36-Month Follow-Up Period: A Randomized Controlled Trial, J foot ankle surg 2018;m 57 (5): 913-918 https://doi.org/10.1053/j.jfas.2018.03.017 Get rights and content


31. Meng-Chen Yin, Jie Ye, Min Yao, Xue-Jun Cui, Ye Xia, Qi-Xing Shen...Wen Mo. Is Extracorporeal Shock Wave Therapy Clinical Efficacy for Relief of Chronic, Recalcitrant Plantar Fasciitis? A Systematic Review and Meta-Analysis of Randomized Placebo or Active-Treatment Controlled Trials. Archives of Physical Medicine and Rehabilitation 2014;95:1585-93


44. Koen Van Boxem; Jianguo Cheng; Jacob Patijn; Maarten van Kleef; Arno Lataster; Nagy Mekhail, Jan Van Zundert. 11. Lumbosacral Radicular Pain; EVIDENCE-BASED MEDICINE: Evidence-Based Interventional Pain Medicine according to Clinical Diagnoses. Pain Practice; 10 (4), 2010 339–358.
47. Atlas SJ, Keller RB, Wu YA, Deyo RA, Singer DE. Long-term outcomes of surgical and nonsurgical management of lumbar spinal stenosis: 8–10 year results