Concise report

Metatarsophalangeal joint pain in psoriatic arthritis: a cross-sectional study

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Abstract

Objective. The aim of this study was to identify independent predictors of pain at the MTP joints in patients with PsA.

Methods. Thirty-four consecutive patients with PsA (mean age 45.3 years, 65% female, mean disease duration 9.9 years) and 22 control participants (mean age 37.9 years, 64% female) underwent clinical and US examination to determine the presence of pain, swelling, synovitis, erosions, effusions and submetatarsal bursae at the MTP joints. Mean barefoot peak plantar pressures were determined at each MTP joint. Levels of pain, US-determined pathology and peak pressures were compared between groups. Binary logistic regression was used to identify demographic, clinical examination-derived, US-derived and plantar pressure predictors of pain at the MTP joints in the PsA group.

Results. The presence of pain, deformity, synovitis, erosions ($P < 0.001$) and submetatarsal bursae and peak plantar pressure at MTP 3 ($P < 0.05$) were significantly higher in the PsA group. MTP joint pain in PsA was independently predicted by high BMI, female gender and the presence of joint subluxation, synovitis and erosion.

Conclusion. These results suggest local inflammatory and structural factors, together with systemic factors (gender, BMI), are predominantly responsible for painful MTP joints in PsA, with no clear role for plantar pressure characteristics.

Key words: psoriatic arthritis, metatarsophalangeal joints, pain, ultrasound, plantar pressure.

Introduction

Forefoot structural damage and pain are common in patients with PsA [1–3] and has been shown to be associated with clinically important levels of impairment and disability [4]. While the magnitude of foot impairments and related disability in PsA is comparable to that reported in RA, to date, relatively few studies have integrated biomechanical and inflammatory measures to determine the underlying dominant mechanisms operating in PsA.

US-detected active disease (synovial hypertrophy, effusion) at the MTP joints is common in PsA and is detected with twice the frequency of clinical examination techniques, but is poorly correlated to pain [3]. There are likely some parallels between some of the structural pathology seen at the MTP joints in PsA and those in RA, namely synovial hypertrophy, joint subluxation and erosive changes. PsA, however, has a distinct bone morphology, not least of which is the propensity for new bone formation arising alongside erosion. It is recognized that the causes of pain at the MTP joint level are complex and may not be wholly related to inflammation [3] and that increased mechanical pressure over structurally damaged metatarsal heads may be important [5–6]. Indeed, in RA, MTP joint peak pressures are elevated and significant relationships between high forefoot pressures, structural damage, pain and foot-related disability have been demonstrated [6–8]. Therefore we hypothesize that the same mechanisms may be equally important in PsA—but this has not been investigated. Thus the aim...
of this study was to identify independent predictors of forefoot pain at the MTP joints in patients with PsA.

**Method**

Local ethical approval was obtained from the West of Scotland Local Research Ethics Committee (reference 09/S0704/14) and NHS Greater Glasgow and Clyde Research and Development Committee (reference GN09RH181) for this study, which was conducted according to requirements of the Declaration of Helsinki. All study participants gave written informed consent. Patients with a confirmed diagnosis of PsA, based on the CASification criteria for Psoriatic Arthritis (CASPAR) [9], were consecutively recruited from rheumatology outpatient clinics at the Glasgow Royal Infirmary and control participants from healthy (i.e. asymptomatic) university-based staff. In one data collection session, the feet of all participants were examined for MTP joint pain, tenderness and swelling, joint subluxation (i.e. dislocation or instability of the joint) and lesser toe bony deformity. Demographic data were collected, including disease duration, gender, age and BMI.

All participants underwent US examination using an Esaote Mylab 70 scanner (Esaote, Genoa, Italy) with multilinear 16–18 MHz probe. The sonographer was blinded to the clinical examination findings. All 10 MTP joints were scanned according to the protocol described by SzkuHale et al. [10]. Grey scale synovitis, erosions and effusions were defined according to the OMERACT definition [11] and scored dichotomously. Submetatarsal bursae were defined according to Hooper et al. [12]. Power Doppler (PD) signal was assessed with a pulse repetition frequency of 750 Hz, low wall filter, gain adjusted until the background signal was removed and minimal probe pressure. Barefoot mean peak plantar pressures were determined for each metatarsal head derived from five walking trials using an Emed X system (Novel GmbH, Munich, Germany) [13].

Mann–Whitney U and chi-square tests were used to compare the PsA and healthy control groups. To identify predictors of MTP joint pain, a binary logistic regression analysis was performed. The dependent variable was the presence or absence of pain in the joint upon clinical examination. Age, gender, other features found on clinical examination (swelling, deformation, subluxation) or US examination (synovitis, erosion, effusion, PD, bursae) and plantar pressure were included in the initial model as potential predictors. Through a process of backward stepwise elimination, the model was reduced to include only those predictors with a P-value below the removal threshold value (P-out) of 0.10. This regression analysis was performed at the individual joint level, i.e. K = 340 joints from N = 34 participants.

**Results**

Thirty-four PsA patients and 22 control participants were recruited. A total of 340 and 220 MTP joints were examined in the PsA and control groups, respectively. The demographics, clinical and US features and peak plantar foot pressures are presented in Table 1. In the PsA group, 129 joints were found to be painful, compared with no joints in the control group. Although subluxation and deformity of the joints were found in the control group, these were more prevalent in the PsA group. Effusion was the most prevalent US feature in both groups (46% PsA, 41% control). The prevalence of synovitis and erosion were both 14% in the PsA group, while these were absent in the control group. For these features, additional

### Table 1 Demographics and clinical, US and biomechanical features

<table>
<thead>
<tr>
<th></th>
<th>PsA (n=34)</th>
<th>Control (n=22)</th>
<th>Test result</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (s.d.) (range), years</td>
<td>45.3 (13.0) (21–71)</td>
<td>37.9 (10.4) (22–60)</td>
<td>t = 2.24, df = 54</td>
<td>0.03</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>22 (64.7)</td>
<td>14 (63.6)</td>
<td>χ² = 0.01, df = 1</td>
<td>0.94</td>
</tr>
<tr>
<td>BMI, mean (s.d.) (range)</td>
<td>25.1 (3.7) (18.8–32.9)</td>
<td>24.4 (2.7) (20.2–28.8)</td>
<td>t = 0.81, df = 54</td>
<td>0.42</td>
</tr>
<tr>
<td>Clinically painful MTP joints, n (%)</td>
<td>129 (32)</td>
<td>0 (0)</td>
<td>χ² = 109.2, df = 1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Clinically swollen MTP joints, n (%)</td>
<td>10 (3)</td>
<td>0 (0)</td>
<td>Fisher’s exact test</td>
<td>0.08</td>
</tr>
<tr>
<td>Clinically subluxed MTP joints, n (%)</td>
<td>173 (51)</td>
<td>40 (18)</td>
<td>χ² = 60.6, df = 1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Clinically deformed MTP joints, n (%)</td>
<td>233 (69)</td>
<td>53 (24)</td>
<td>χ² = 105.6, df = 1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>US synovitis MTP joints, n (%)</td>
<td>47 (14)</td>
<td>0 (0)</td>
<td>χ² = 33.2, df = 1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>US erosion MTP joints, n (%)</td>
<td>46 (14)</td>
<td>0 (0)</td>
<td>χ² = 32.4, df = 1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>US effusion MTP joints, n (%)</td>
<td>158 (46)</td>
<td>91 (41)</td>
<td>χ² = 1.41, df = 1</td>
<td>0.24</td>
</tr>
<tr>
<td>US PD-positive MTP joints, n (%)</td>
<td>6 (2)</td>
<td>0 (0)</td>
<td>Fisher’s exact test</td>
<td>0.086</td>
</tr>
<tr>
<td>US MTP joint bursa, n (%)</td>
<td>92 (27)</td>
<td>42 (19)</td>
<td>χ² = 4.66, df = 1</td>
<td>0.03</td>
</tr>
<tr>
<td>PP MTP joint 1, median (IQR)</td>
<td>263.1 (186.3–488)</td>
<td>318.3 (266.7–397.5)</td>
<td>U = 1253, z = -1.265</td>
<td>0.206</td>
</tr>
<tr>
<td>PP MTP joint 2, median (IQR)</td>
<td>420.5 (313.3–667.3)</td>
<td>364 (291–467.5)</td>
<td>U = 1139, z = -1.955</td>
<td>0.051</td>
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<tr>
<td>PP MTP joint 3, median (IQR)</td>
<td>396.5 (317.5–392)</td>
<td>330 (267–392)</td>
<td>U = 979.5, z = -2.921</td>
<td>0.003</td>
</tr>
<tr>
<td>PP MTP joint 4, median (IQR)</td>
<td>255 (202–299.5)</td>
<td>235 (198–301.7)</td>
<td>U = 1302, z = -0.969</td>
<td>0.333</td>
</tr>
<tr>
<td>PP MTP joint 5, median (IQR)</td>
<td>228.5 (130.4–315.8)</td>
<td>163 (118–341.7)</td>
<td>U = 1248, z = -1.295</td>
<td>0.195</td>
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</table>

PP: plantar pressure; IQR: interquartile range; t: Student’s independent groups t-test result; df: degrees of freedom; χ²: chi-square test for independence; U: Mann–Whitney U test result with Monte Carlo exact significance; z: z score.
Comparing these findings with the present results, associated with some measures of pain and impaired function synovial pathology has been shown to be variably associated and osteophytosis in OA [17]. Moreover, in hand OA, reduced joint function correlated with joint space narrowing and osteophytosis in OA [17]. Of combining measures of inflammation and function are painful. Studies in OA have previously shown the value of plantar pressure and for plantar pressure predicting which joints can be replicated, with limited evidence for elevated plantar pressure has been shown to be elevated at the MTP joints, and this has been associated with pain and disability [6–8]. In this PsA study, these results could not be replicated, with limited evidence for elevated plantar pressure and for plantar pressure predicting which joints are painful. Studies in OA have previously shown the value of combining measures of inflammation and function [17–18]. In contrast to the findings reported here, US features of synovitis did not correlate with MTP pain, but reduced joint function correlated with joint space narrowing and osteophytosis in OA [17]. Moreover, in hand OA, synovial pathology has been shown to be variably associated with some measures of pain and impaired function [18]. Comparing these findings with the present results, which are dominated by inflammatory factors, indicates that models of joint pain may be disease and joint specific. A different balance between inflammatory and mechanical factors may exist even in superficially similar arthritic disorders.

There are several potential limitations to this study. First, the sample size is relatively small. Second, no causal inferences can be made in this cross-sectional data set. Finally, the assessment of joint pain was done in a qualitative (absent/present) manner during clinical examination and did not take into account pain severity or frequency, nor the subjective nature of pain reporting.

In summary, this work supports an explanatory model of peripheral joint pain in PsA that includes local inflammatory and structural factors as well as systemic characteristics (gender, BMI).

### Discussion

This study has identified that MTP joints that are subluxed and have US-proven synovitis or erosion in female PsA patients with higher BMI are most likely to be painful. No clear role for plantar pressure was found in explaining PsA-related MTP joint pain.

Anatomical site-specific models of disease burden and pain have been studied in other arthritic conditions. In RA, plantar pressure has been shown to be elevated at the MTP joints, and this has been associated with pain and disability [6–8]. In this PsA study, these results could not be replicated, with limited evidence for elevated plantar pressure and for plantar pressure predicting which joints are painful. Studies in OA have previously shown the value of combining measures of inflammation and function [17–18]. In contrast to the findings reported here, US features of synovitis did not correlate with MTP pain, but reduced joint function correlated with joint space narrowing and osteophytosis in OA [17]. Moreover, in hand OA, synovial pathology has been shown to be variably associated with some measures of pain and impaired function [18]. Comparing these findings with the present results, which are dominated by inflammatory factors, indicates that models of joint pain may be disease and joint specific. A different balance between inflammatory and mechanical factors may exist even in superficially similar arthritic disorders.

### Funding

Grant support was received from Arthritis Research UK (grant references 17832 and 18381) to D.T. and R.B.

### Disclosure statement

The authors have declared no conflicts of interest.

### References


