

Evaluation of lesser metatarsophalangeal joint plantar plate tears with contrast-enhanced and fat-suppressed MRI

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Abstract

Objective To present findings of plantar plate (PP) lesions from MRI with administration of gadolinium and to differentiate PP lesions from others causes of metatarsalgia.

Materials and methods Two musculoskeletal radiologists reviewed 249 contrast-enhanced forefoot MRI scans from patients with metatarsalgia between June 2012 and June 2013. Evaluations focused on hyper-vascularized/fibrous tissue and other findings associated with PP tears.

Results Fifty-nine patients had PP tears, 59 % were female. Most of these patients, 48/59 (81.4 %), had a single metatarsophalangeal (MTP) PP lesion in one foot, although 7/59 patients had one lesion in each foot, 3/59 (5.1 %) had two in one foot, and 1/59 (1.7 %) had three lesions in one foot. The second MTP joint was the most common location for PP tears ($n=56$), followed by the third ($n=12$) and fourth ($n=3$) MTP joints. Lateral ($n=33$) and full thickness ($n=28$) PP lesions were the most frequent, and central ($n=3$) and lateral/central ($n=7$) tears were less prevalent. Fifty (70.5 %) PP lesions showed pericapsular fibrosis in pre-contrast sequences, and 21 (29.5 %) were visible only after administration of gadolinium. All PP lesions had collateral ligament involvement. Others findings included interosseous tendon lesions ($n=29$), interosseous tendon rupture ($n=29$), synovitis ($n=49$), flexor tenosynovitis ($n=28$), crossover toe ($n=2$), hammertoe ($n=1$), intermetatarsal space (IS) neuromas ($n=11$), and third IS neuromas ($n=12$).

Conclusion PP tears are a common cause of metatarsalgia, accounting for more than 20 % of cases in our sample. A substantial portion of the lesions (29.5 %) became visible only after the administration of gadolinium.

Keywords Plantar plate · Magnetic resonance imaging · Metatarsalgia · Metatarsophalangeal joint

Introduction

Metatarsalgia, or forefoot pain, is a common clinical problem that can lead to difficulty with ambulation and shoe wear. Causes of metatarsalgia include plantar plate (PP) injury, metatarsophalangeal (MTP) joint synovitis, stress fracture, osteonecrosis (Freiberg's infraction), arthritis, interdigital Morton's neuroma, intermetatarsal bursitis, and synovial cyst formation. Each of these conditions can be differentiated with forefoot magnetic resonance imaging (MRI) [1, 2].

Instability of the lesser MTP joint has been widely reported [3–10], and PP insufficiency may be a primary cause of this condition [8, 9, 11–15]. The PP is a fibrocartilaginous structure that lies deep in the metatarsal heads and plays a key role in providing stability to the MTP joints. The proximal origin of the PP attaches loosely to the metatarsal shaft. The distal end of the PP inserts firmly and directly into the plantar lip of the proximal phalanx, just distal to the articular surface. The PP also has attachments to the proper collateral ligament (PCL), accessory collateral ligament (ACL), plantar fascia, intermetatarsal ligaments, interosseous tendons, and extensor hood and sling, as well as the fibrous sheath of the flexor tendons (Fig. 1).

As the principal stabilizer of the MTP joint, the integrity of the PP is essential for securing the proximal phalanxes of the lesser toes. Attrition of the PP often results in metatarsalgia,

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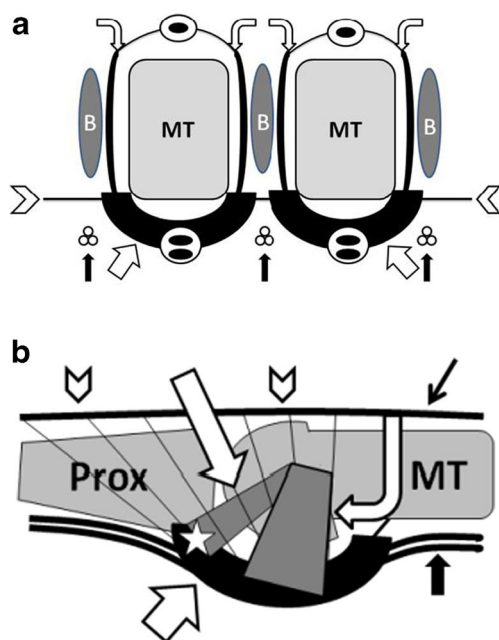


Fig. 1 a–b Schematic illustrations of the MTP PP and capsular supporting structures in the short (a) and long axis (b). *Thick arrows* (a) indicate the plantar plate, which cradles the flexor tendon sheath in a plantar midline groove. *Bent arrows* indicate the medial and lateral capsule, in continuity with the extensor hood, fanning out from the extensor tendon sheath. *B* indicates intermetatarsal bursae. *Small black arrows* indicate the plantar digital neurovascular (NV) bundles. *Chevrons* indicate the deep transverse intermetatarsal ligament (DTIL), which blends with the plantar plates. Note that the bursae lie above and the NV bundles lie below the DTIL. In the long axis (b), the *short thick white arrow* indicates the PP inserting into the base of the proximal phalanx (*Prox*). The most lateral insertion of the PP blends with the insertion of the phalangeal collateral ligament (PCL) (*long white arrow*); the *star* indicates a common site of tear at the lateral conjoint insertion of the PP and PCL. The accessory collateral ligament (*bent arrow*) is broader and more vertically oriented, inserting bilaterally onto the PP. The *thin black arrow* indicates the extensor digitorum longus tendon; *thin linear bands* (chevrons) spanning from the EDL tendon sheath to the PP represent the extensor hood. The *short black arrow* indicates the flexor digitorum tendons (the *B* lies below the longus tendon). Reproduced with permission of Umans et al. [16]

plantar swelling, hammertoe and clawtoe deformities, and lesser toe subluxation [1, 17–20]. Inadequate diagnosis and treatment of injuries to the PP can result in substantial disability, deformity, and dysfunction [18, 21].

Various modalities and techniques have been employed to study metatarsalgias. Although MRI is widely accepted as the preferred imaging modality for diagnosing PP tears [9, 16, 22–25], to our knowledge, there are no reports regarding the use of enhanced MRI for diagnosing PP tears. Terk et al. [26] used MRI with enhanced fat suppression to evaluate Morton’s neuroma. In this study, we characterized findings of PP lesions from fat-suppressed MRI both before and after administration of contrast material (gadolinium). We also developed the differential diagnosis between PP lesions and other causes of metatarsalgia with MRI.

Material and methods

Patients

This study was performed in accordance with the Declaration of Helsinki principles. Patients with metatarsalgia who underwent forefoot MRI with contrast enhancement participated in this retrospective study. We recruited 308 patients who had an MRI examination at one institution between June 2012 and June 2013. We excluded 99 patients from this study, with a history of previous MTP joint surgery, inflammatory arthropathy, peripheral neuropathy, diabetes, or peripheral vascular disease.

We enrolled 209 patients with pain in the lesser MTP joints who met the inclusion criteria. All patients had a double MRI examination, that is, a scan before and after intravenous administration of 0.1 mmol/kg intravenous contrast agent (GD-DOTA, Guerbert, France) (gadolinium). We found 71 PP lesions; distributed among 66 feet in 59 patients (some feet had more than one lesion). These patients contributed 249 forefoot MRIs to our analyses.

MRI

Imaging was performed in two closed 1.5-T MR scanners. One scanner was a GE HDTX with an 8-channel foot and ankle coil, and the other was a Siemens Symphony with a small flexible coil. We obtained T1-weighted and fat-suppressed proton density (PD) axial and coronal sequences, T2-weighted fat-suppressed PD coronal sequences, and T1-weighted fat-suppressed axial, coronal, and sagittal oblique planes after the administration of contrast (FOV 10–12 cm, 1–2 NEX, and 3 mm slice thickness). The sagittal oblique plane was imaged for each toe, oriented on coronal and axial planes.

Image processing and analysis

Two musculoskeletal radiologists who had more than 10 years of experience each in forefoot MRI analyzed the images. We used previously established MRI criteria for PP tear [22, 25, 27], interdigital neuroma [26, 28, 29], intermetatarsal bursitis [30–32], and fibrosis [16]. We also analyzed the findings after the use of contrast.

We considered PP degenerations as cases of PP morphologic alterations, specially thinning with increase signal intensity, and no contrast enhancement. Those cases were not included in our study (Fig. 2). In all cases of PP tears, we found lesions adjacent to the PP insertion into the base of the proximal phalanx. We classified PP lesions as medial, lateral, central, full thickness and combinations. We identified PP tears as bright signals on contrast enhanced fat-suppressed T1-weighted sequences, or as detachments of the PP at its insertion on the proximal phalangeal base. The position of a tear can be

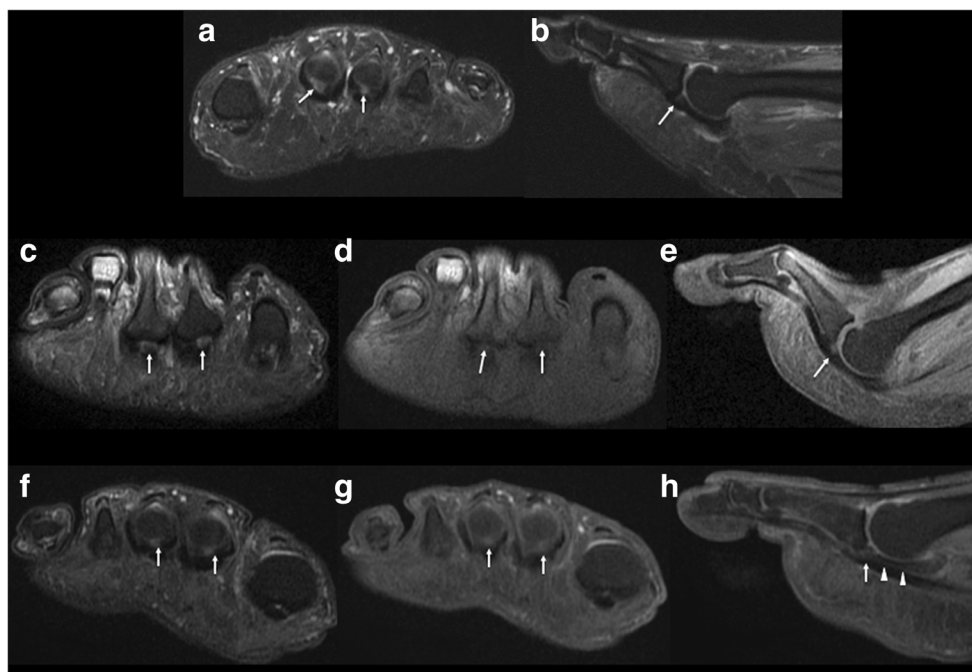


Fig. 2 Coronal short (a) and sagittal axis (b) proton-density-weighted fat-suppressed images obtained in a 49-year-old female with forefoot pain and intact PP. *White arrows* indicate increased signal intensity near the distal attachment (central area), that should not be confused as PP tear, and can represent PP recess or hyaline cartilage undercutting the PP phalangeal insertion. Coronal short axis (c) proton-density-weighted fat-suppressed image and coronal short (d) sagittal axis (e) contrast enhanced fat-suppressed T1-weighted images, obtained in a 50-year-old female with metatarsalgia and dorsi-flexion MTP joint. *White arrows* also

indicate increased signal intensity near the distal attachment without contrast enhancement, excluding PP tear. Coronal short axis (f) proton-density-weighted fat-suppressed image, coronal short (g) and sagittal axis (h) contrast enhanced fat-suppressed T1-weighted images obtained in a 62-year-old female with 1-year second MTP pain. *White arrows* indicates increased signal intensity near the distal attachment without contrast enhancement excluding PP tear, and sagittal image shows central PP thinning that may represent PP degeneration

seen well in the coronal plane with a hyperintense signal in fat-suppressed PD images [2, 16, 22, 25] and enhancement with bright signals on contrast-enhanced T1-weighted fat-suppressed images. Retraction of the tear is best assessed in the sagittal plane [25]. Transverse complete distal tears usually occur along the phalangeal base, with dislocation of the PP into proximal phalanx. We also recorded the affected MTP joint and the tear's location and type (Fig. 3).

We used the coronal axis images to improve assessment of hyper-vascularized/fibrous tissue in and around PP lesions, ligaments, interosseous tendons, and capsule. We identified hyper-vascularized/fibrous tissue in soft tissue from intermediate signals on T1- and T2-weighted images and somewhat strong fat-suppressed PD signals. We also identified hyper-vascularized/fibrous tissue in soft tissue that appeared similar to blood vessels in contrast-enhanced fat-suppressed T1-weighted images [33], was marked by thickening, and abutted the capsule, PP, collateral ligaments, or interosseous tendons eccentrically within the IS (Fig. 4) [16].

The PCL and ACL are indistinguishable from the capsule in MRI [16]. Therefore, we considered only components of the ligaments and ignored capsule ruptures. We interpreted coronal fat-suppressed PD and contrast-enhanced T1-weighted fat-suppressed images of the ACL junction with the lateral

margin of the PP as ACL injuries or tears when they were marked by discontinuity and hyperintense signals. We found evidence of discontinuity and injury to the capsule and PCL in axial fat-suppressed PD and contrast-enhanced T1-weighted fat-suppressed images of the most lateral insertion of the PP into the base of the proximal phalanx, with or without phalanx deviation (Fig. 5).

Hyperintense signals at the insertion of the interosseous tendon into the phalangeal base reflect lesions on the interosseous tendon in fat-suppressed PD and contrast-enhanced T1-weighted fat-suppressed images. These lesions were generally associated with hyper-vascularized/fibrous tissue, capsulitis, or ligamentous lesions (Fig. 5).

We identified flexor tenosynovitis with hyperintense signals and increased fluid around the tendons in PD/T2 sequences. Intensified signals inside the tendon sheath in contrast-enhanced T1-weighted fat-suppressed images were also due to synovitis and were always associated with a PP tear (Fig. 4).

In contrast-enhanced T1-weighted fat-suppressed images, hyperintense signals in the synovial part of the MTP joint (similar to blood vessels) represented synovitis. T2-weighted turbo spin-echo and fat-suppressed PD-weighted images do not differentiate between synovitis and joint fluid [33].

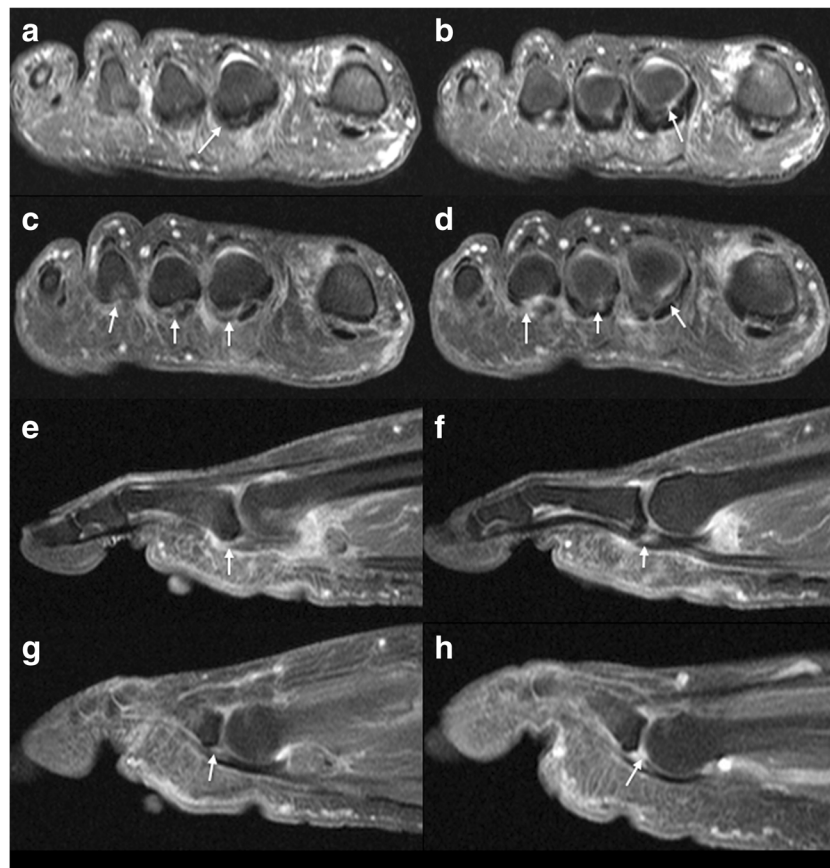


Fig. 3 Coronal (a–d) and sagittal (e–h) axis of the right foot of a 58-year-old woman with metatarsalgia (a–b are fat-suppressed PD and c–h are contrast enhanced fat-suppressed T1-weighted images). Image a shows the tear at the distal lateral insertion of the second MTP PP (white arrow). Image b shows a midline PP phalangeal side bright signal (white arrows), that can represent tears of PP, recess, or hyaline cartilage undercutting the

PP phalangeal insertion. c–d Images show contrast enhancement that reveals PP tears extending from the lateral side to the midline insertion onto the base of the second, third, and fourth toe phalanxes (white arrows). e–f Images confirm these findings in the sagittal plane (white arrows) for the second toe (e–f), third toe (g) and fourth toe (h)

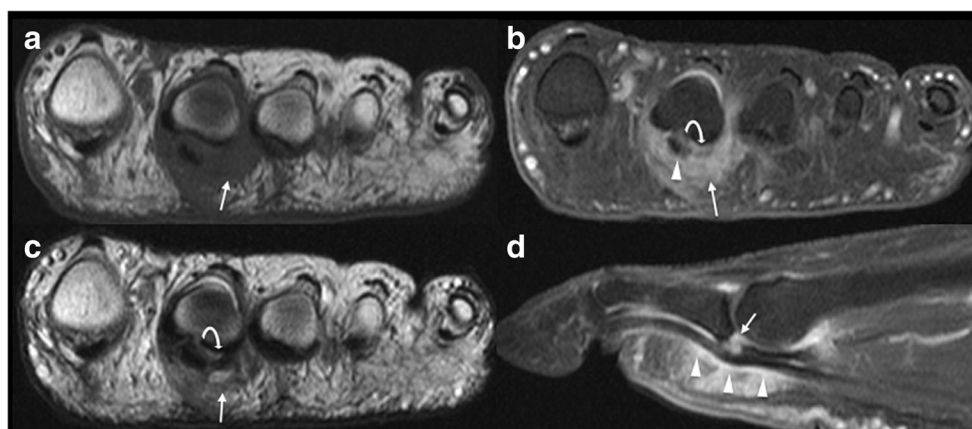


Fig. 4 Coronal (a–c) and sagittal (d) axis of the left foot of the same patient in Fig. 1. a T1-weighted (b and d) contrast-enhanced fat-suppressed T1-weighted and c T2-weighted images. b–c The curved arrows demonstrate PP and ACL tears, respectively. White arrows (a and c) show intermediate T1 and T2 signals of a soft tissue mass thickening and abutting the capsule, PP, ACL, flexors tendons, and

second IS. These features may represent pericapsular hyper-vascularized/fibrous tissue because they appear similar to blood vessels in fat-suppressed T1-weighted images in c. Confirmation of a PP tear in the sagittal plane (white arrow) (d). In contrast-enhanced b and d, tenosynovitis is also present around the flexor tendons (arrowheads)

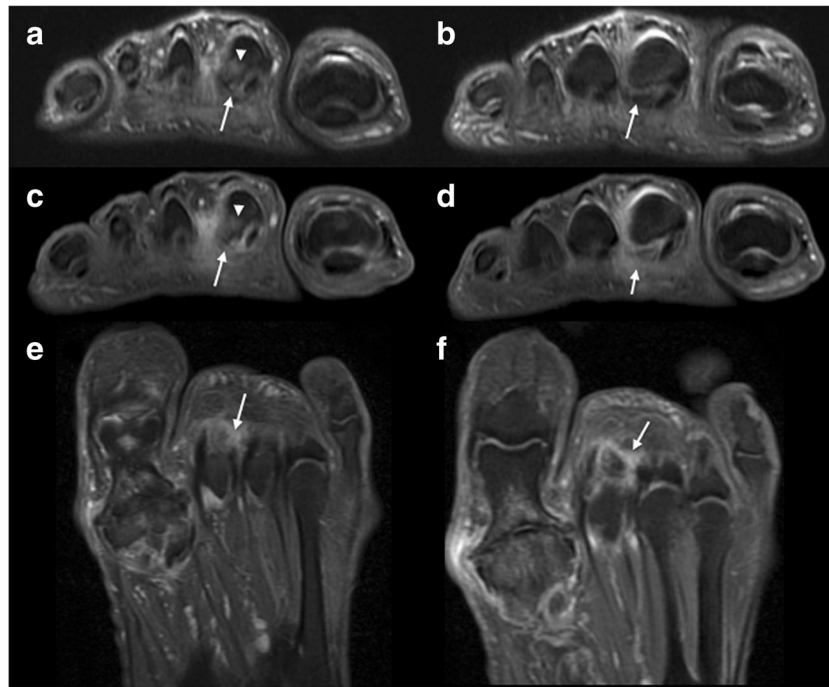


Fig. 5 Coronal (a–d) and axial (e–f) axis of a 73-year-old woman with left forefoot pain and hallux arthrosis. Images a and b are fat-suppressed PD images, and c–f are contrast-enhanced fat-suppressed T1-weighted images. Images (a, c, e and f) show discontinuity of the PCL with the most lateral insertion of the PP into the base of the proximal phalanx, in association with hyper-vascularized/fibrous tissue, and a lesion on an

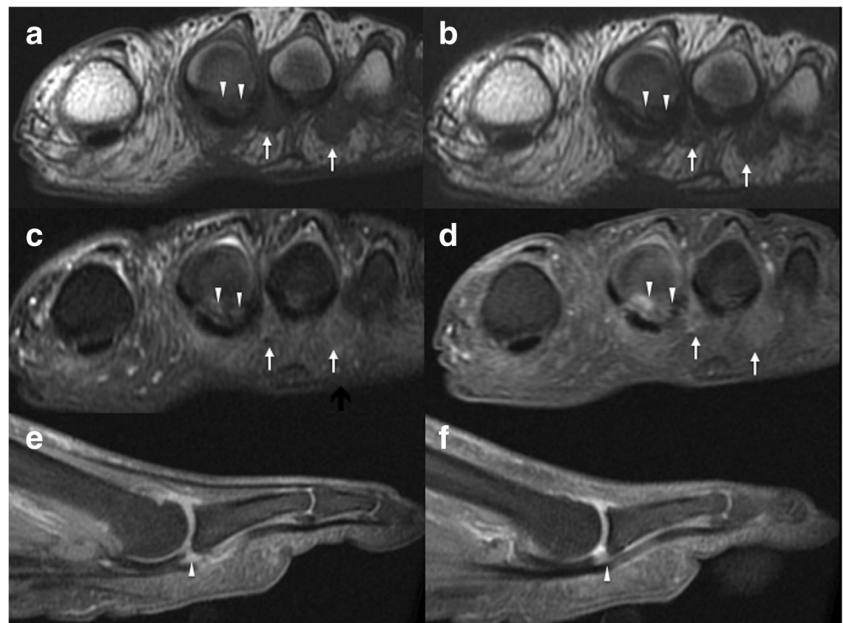
interosseous tendon (*white arrows*). Osteitis can be detected as a hyperintense signal at the base of the phalanx in fat-suppressed PD-weighted (a) and contrast-enhanced fat-suppressed T1-weighted (c) images. Images show an ACL lesion in association with inflammatory tissue and a PP lesion (b and d)

Cortical irregularities at the phalangeal enthesis on fat-suppressed PD and contrast-enhanced T1-weighted fat-suppressed images reflected phalangeal bone irregularities on the lateral tubercle of the base of the phalanx. These irregularities sometimes were associated with marrow edema, as revealed by very strong signals in axial fat-suppressed PD

images. We considered osteitis when bone marrow had bright signal after contrast enhancement (Fig. 5).

The axial and sagittal images provided the best means for assessing toe deformities. A hammertoe deformity implies a flexion deformity of the PIP joint, with dorsiflexion of the MTP joint and either a neutral position or hyperextension at

Fig. 6 Coronal (a–d) and sagittal (e–f) axis of the right foot of a 53-year-old woman with forefoot pain. T1-weighted (a) and T2-weighted (b) images show a teardrop-shaped lesion (*white arrow*) in the second and third intermetatarsal spaces typical for Morton's neuromas. These neuromas appear as mild lesions in fat-suppressed PD-weighted (c) and contrast-enhanced fat-suppressed T1-weighted (d) images. This patient also had a PP tear extending from the lateral side (e) to the midline (f) (*arrowheads*)



the DIP joint. A clawtoe deformity typically involves more than one toe and is similar to a hammertoe deformity, with additional flexion deformity of the DIP joint [34]. A medial crossover toe is characterized by medial inclination and deviation of the second toe, which initially results in widening of the second interdigital [18]. Over time, a progressive sagittal plane deformity results in hyperextension at the second MTP joint, and the second toe overlaps the great toe.

We also noted other abnormalities. Hallux valgus and long second metatarsals are commonly associated with PP tears [35]. We diagnosed nodular tissue masses as interdigital neuromas if they had moderately intense signals in T1- and T2-weighted images, with or without contrast enhancement, and were centered in the IS between the plantar and deep transverse intermetatarsal ligaments [26, 29]. We also followed the neurovascular bundle in the coronal images to confirm that the fibrous tissue mass was involved (Fig. 6). Fluid collection with a bright signal centered in the IS represented intermetatarsal bursitis in coronal fat-suppressed PD and T2-weighted images, with or without contrast enhancement (Fig. 7).

Results

We detected 71 PP lesions in 59 patients. Most (35, or 59 %) of these patients were female. They ranged in age from 24 to 81 years, with an average of 46 years. Seven patients had one lesion in each foot, three patients had two lesions in the same foot, and one patient had three lesions in the same foot. We found PP tears in the left feet of 38 patients and in the right feet of 33 patients. Fifty-six lesions involved the second MTP joint, 12 involved the third MTP joint, and three involved the fourth MTP joint.

Of the 71 PP lesions, 33 were lateral only, 3 were central only, 7 were lateral and central, and 28 were full thickness. Twenty of the 28 full thickness tears had proximal displacement. Fifty PP lesions showed pericapsular hyper-vascularized/fibrous tissue, but 21 of these were visible only after administration of gadolinium. All PP lesions had collateral ligament involvement. Thirty-nine had PCL and ACL lesions, 23 had ACL lesions (1 medial and 22 lateral), 32 had PCL lesions, and 19 had PCL ruptures. In addition, 29 PP lesions had interosseous tendon lesions, 29 had interosseous tendon ruptures, 49 had synovitis, and 28 had flexor tenosynovitis. Four PP lesions had toe deformities, including two crossover toes, one hammertoe associated with dorsal and lateral luxation, and one second valgus toe deformity.

Furthermore, we found some osseous alterations on the proximal phalanx, in correlation with the ACL, PCL, and interosseous tendon lesions and ruptures. These alterations included 13 irregularities on the lateral tubercle of the base of the phalanx, which were associated with osseous edema or osteitis

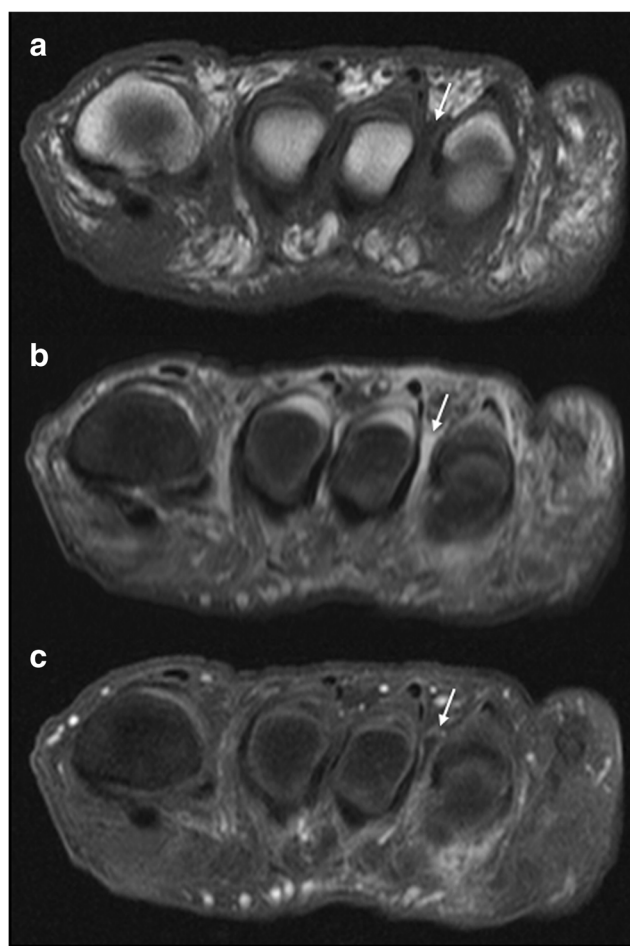


Fig. 7 Coronal axis of the right foot of a 48-year-old woman with forefoot pain. **a** The image is T1-weighted, **b** the image is fat-suppressed PD, and **c** the image is contrast-enhanced fat-suppressed T1-weighted. *White arrows* indicate evidence of third intermetatarsal bursitis with a hypointense signal (**a**), a hyperintense signal (**b**), and peripheral enhancement of the bursal wall (**c**)

in seven cases. We also found 19 hallux valgus and 37 long second metatarsals, which are predisposing factors to PP lesions.

The 59 feet with PP lesions had other lesions related to metatarsalgias, including 23 Morton's neuromas (11 in the second IS, 12 in the third IS), and five callosities on the affected PP. Our other findings in these feet included hallux arthrosis ($n=12$), turf toe ($n=2$), adventitia bursa ($n=1$), sesamoiditis medial ($n=1$), hallux MTP synovitis ($n=1$), submetatarsal synovial cysts or ganglions ($n=10$), and intermetatarsal bursitis ($n=6$).

Discussion

Metatarsalgias a common clinical complaint, affecting not only the metatarsals, but also the MTP joint and intermetatarsal and submetatarsal soft tissues [36, 37]. Instability of the lesser MTP joints is a common cause of forefoot pain and deformity.

Detailed anatomical dissection has suggest that the PP is the major stabilizing structure of the MTP joint, due to its central location and multiple strong attachments; following its disruption, MTP joint instability thus develops [6, 10, 17, 21]. Previous reports [14, 37, 38] have indicated that frequent use of high-heeled or ill-fitting footwear is a major predisposing factor for lesser MTP joint instability [7, 9, 10]. A clinical diagnosis of instability can be made based on physical examination [2, 23, 39, 40]. The degree of pain may be disproportionate with other physical examination findings, and this pain may be difficult to differentiate from neuroma pain, particularly when edema in the joint has caused effusion into the second interspace [16, 19, 37]. In some instances, patients have been treated by one or several other medical professionals without resolution of symptoms, and begin to question the validity of their foot pain.

The inclusion of patients in this study was based only on the consideration of metatarsalgia history. Most other studies have included patients with metatarsalgia and clinically detected pathologic PP features, including some degree of instability [2, 9, 11, 16, 22–25, 41], and some have also included patients who have shown inadequate response to conservative treatment [9, 24, 41]. We found pathologic PP features in 66 of 249 forefoot MRI examinations. In addition, we detected 21 PP lesions only after gadolinium administration. We believe that the use of MRI with gadolinium enables the detection of PP lesions prior to the manifestation of typical pathologic clinical findings such as some degree of MTP joint deformity and/or instability.

In 1994, Yao et al. [22] found that conventional arthrography of the MTP joint was a practical, but invasive, way to confirm the diagnosis of PP rupture. In the same study, they found that MRI was a superior and noninvasive method because it could detect PP rupture and other abnormalities such as degeneration, synovitis, and Morton's neuroma. As they had no histologic basis for signal heterogeneities in the PP, the authors presumed that advanced plate degeneration would be difficult to distinguish from frank rupture [23]. They described a case of PP thinning with increased signal intensity in a 61-year-old woman, which they classified as age-related degeneration. In PP rupture, they reported that signal changes in the PP were typically more intense and widespread, extending beyond the immediate area of the plate attachment on the proximal phalangeal base [23]. We found no histologic study in the literature describing the differentiation of PP degeneration from tearing [10, 42]. In our work, we considered only PP tears. The only cadaveric study describing PP attenuation is by Deland and Sung [35]; they described this condition in only one specimen, with no histologic reporting, and we do not know what this attenuation actually represents. We believe that PP degeneration may be represented by PP thinning, but with no contrast enhancement.

PP injury is typically seen as a focal, intrasubstance increase in the signal of the plate on a T2-weighted image [2,

22, 23, 36]. In some instances, discrete tears are visible. On MRI, the area on the distal aspect of the PP immediately adjacent to the base of the proximal phalanx in the tear zone (central position) is poorly defined, which may make the identification of tears difficult. Therefore, one should take care when evaluating the integrity of the PP using MRI. Mohana-Borges [32] evaluated MTP joints in 12 cadaveric feet with MR arthrography and MR bursography. In that study, 47 % of joints examined had recesses on the phalangeal side of the PP, observable in the midsagittal plane. These recesses should not be confused with lesions. In addition, hyaline cartilage underlying the PP on reformatted sagittal images may be mistaken for frank PP tearing [2]. Umans et al. [16] considered >2.5-mm elongation of the normal bright T2 signal zone at the midline insertion of the PP to indicate a tear. In this study, we demonstrated that PP tears are more conspicuous on contrast-enhanced, fat-suppressed T1-weighted images in the same sagittal plane. Because contrast agents enhance the signal intensity of tears selectively, without enhancing the brightness of the PP recess or artifacts, they can be used to facilitate the differentiation of tears from the PP recess on the phalangeal side.

Gregg et al. [42] described the sonographic appearance of lesser metatarsal PPs in cadavers and correlated these findings with MRI and histologic findings. Sonography and MRI were used to examine six soft-embalmed cadaveric feet. The authors obtained proton density (PD) fast-spin echo sequences and T2-weighted fat-suppressed fast-spin echo sequences in the coronal and axial planes. The MRI findings of PP tearing were plate discontinuity and an area of increased signal intensity at the attachment of the PP to the base of the proximal phalanx. Sonography was used to examine one young fresh cadaver before histologic assessment. Eight of the 24 lesser metatarsal PPs were removed for histologic examination. Histologically, PP tearing appeared as dense fibrocartilage disruption proximal to its insertion on the proximal phalanx, with abrupt borders, and replacement cellular structures and vessels. The average volume densities of these tissues, calculated in soft-embalmed cadavers, were—fat cells, 6 %; hydroptic tubules (watery fluid), 10 %; blood vessels, 18 %; loose cellular tissue (excluding fat cells), 26 %; dense irregular tissue, 23 %; and dense regular tissue, 18 %. Histologic analysis of the central fibers of the fresh cadaver PPs was performed, and normal histologic volume densities were calculated. Regular dense connective tissue had a volume density of 64 %, blood vessels had a density of 7 %, irregular dense connective tissue had a density of 9 %, and loose cellular tissue (excluding fat cells) had a volume density of 20 %. We believe that the reduced quantity of regular dense connective tissue and major increase (more than doubling) in the average number of blood vessels in the abnormal PP may explain our finding of contrast enhancement at the site of the PP tear. Further studies are needed to confirm this hypothesis.

Umans et al. [16] sought to establish the differential diagnosis between second MTP PP pathology with concomitant second IS masses and interdigital neuroma using MRI. They considered pericapsular fibrosis in cases of intermediate T1 and T2 signal intensity, indicating soft-tissue thickening that abutted the capsule and PP eccentrically within the IS, typically with an adjacent degenerative or torn PP and/or capsule. All but one case of second MTP PP tearing in their series was accompanied by a second IS lesion, which they considered to be pericapsular fibrosis with no adjacent IS neuroma. They argued that second MTP PP tears and adjacent pericapsular second IS reactive fibrous masses fall along a spectrum of the same condition, rather than constituting two different concomitant lesions. Their study was limited by its retrospective design, lack of histologic correlation, and presumptive diagnosis of pericapsular fibrosis based on the eccentric pattern of reactive soft-tissue thickening along the contour of the torn MTP capsule and PP, as well as intermediate T1/T2 signal suggesting fibrous tissue.

Similar to Umans et al. [16], we found an eccentric pattern of reactive soft-tissue thickening along the contour of the torn MTP capsule and PP, and an intermediate T1/T2 signal suggesting fibrous tissue; however, we used gadolinium contrast, which enhanced these tissues and associated lesions and growths [33]. Gregg et al. [42] did not describe a similar fibrous tissue pattern, perhaps because they used one slide representing the central fibers of the PP for each soft-embalmed specimen. Further studies are needed to evaluate this presumed fibrous tissue pattern found in association with PP injury. In our study, not all neuromas were enhanced after gadolinium administration; hence, we believe that contrast can be used to help differentiate pericapsular fibrosis, which is enhanced by contrast, from neuroma, which may not be enhanced.

Coughlin et al. [19] reported a 20 % incidence of concomitant second Morton's neuroma in a study of 121 patients with adjacent PP tears. All cases of neuroma in their series were confirmed by independent pathologic evaluation. However, whether they could differentiate concentric perineural fibrosis with classic third Morton's neuroma from eccentric digital nerve encasement by pericapsular fibrosis in reaction to a PP and/or lateral capsular tear in their surgical specimens is not clear. Correct distinction of PP tearing from neuroma is of great clinical importance, as metatarsalgia is commonly treated with intra-articular MTP and/or IS steroid injection [43]. These injections can result in soft-tissue atrophy and may precipitate tearing of the capsule and/or PP [18, 21].

Coughlin and Nery [41] graded 100 MTP joints in 68 patients according to their anatomical grading system for PP tears to determine the appropriate surgical procedures. They suggested that surgical repair of early-stage PP tears has better outcomes with regard to restoration of function and anatomic alignment [21, 41]. Early and accurate diagnosis of PP tearing is thus clinically important. In addition, anatomic grading of

this condition is important for accurate treatment. The anatomical grades of PP tears were: grade 0, PP or capsular attenuation, and/or capsular discoloration; grade 1, transverse distal tear adjacent to insertion into the proximal phalanx (<50 %); grade 2, transverse distal tear (\geq 50 %); grade 3, transverse and/or longitudinal extensive tear (may involve collateral ligaments); grade 4, extensive tear with button hole (dislocation). The authors did not describe PP attenuation (grade 0) in their cadaveric study [10]; they described PP attenuation and/or discoloration based on arthroscopic observations, without histologic confirmation. Based on the histologic appearance of the abnormal PP described by Gregg et al. [42], we believe that PP lesions detected only after gadolinium enhancement in our study may represent intrasubstance partial PP tears or plantar partial PP tears, and that they fall within the anatomic grade of 0, described arthroscopically [9, 41]. Further studies are needed to clarify these issues. The anatomical grading system for MTP PP tears proposed by Coughlin and Nery [7, 9, 41] did not describe fibrous tissue, described by Umans [16].

Treatment of PP tearing can be conservative or surgical [13, 18, 44, 45]. In early-stage PP lesions with no or mild toe deformity, sagittal plane instability can be treated with MTP taping, to maintain alignment during scarring or healing [21]. Taping, however, is unsuccessful in cases of complete capsular disruption or moderate to severe subluxation [8, 9, 18, 28], and long-term taping has been reported to cause skin ulceration and chronic edema [18]. Other conservative treatments include metatarsal pads, nonsteroidal anti-inflammatory drugs, orthotics, and intra-articular corticosteroid injections; as mentioned previously, the latter should be used with caution due to the risk of PP injury. In light of the frequent failure of conservative treatment in cases of lesser MTP joint subluxation or dislocation, numerous surgical procedures have been advocated to correct this deformity [12, 17, 18, 43, 46–49]. Nery et al. [41] demonstrated good to excellent outcomes for their proposed treatment protocol for grades 0 and 2 PP lesions, good results for grades 1 and 3 tears, and moderate results for grade 4 tears. Therefore, they concluded that success rates are lower for more extensive tears than for lower-grade tears.

Our study was limited by the absence of histologic correlation with our diagnoses. We did not have histological samples because patients receiving surgical treatment underwent Weil osteotomy via dorsal arthrotomy, without exploration of the PP tear, pericapsular fibrous tissue, or IS. Despite this limitation, MRI is widely accepted to be the preferred imaging modality for the diagnosis of PP tears [2, 9, 16, 50].

Conclusion

PP tearing is a common cause of metatarsalgia, found in more than 20 % of patients in our sample. The use of contrast facilitates radiologists' evaluation of PP lesions. Areas of

enhanced contrast increase the precision of lesion type diagnosis, relative to the use of PD-weighted fat-suppressed images, which sometimes register bright signals in the absence of true lesions. Therefore, we believe that the use of contrast-enhanced and fat-suppressed MRI enables the diagnosis of PP tearing or attenuation in the early stages, before the development of instability, which improves treatment and outcome.

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