

CONTINUING PROFESSIONAL UPDATE

Lesser Metatarsalgia: diagnosis, causes and management



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Introduction

Metatarsalgia is a common foot complaint and is a generalised term given to describe pain around the metatarsals, metatarsophalangeal joints (MTPJs), intermetatarsal spaces and the associated soft tissues. It is suggested that older patients are at a greater risk of developing metatarsalgia, with females being more often affected than males¹

Metatarsalgia is complex and can cause a diagnostic challenge due to a crossover of symptoms between pathologies, and the close and detailed anatomy; this leads to a risk of misdiagnosis and warrants a careful and thorough history-take and assessment.² Differential diagnoses for metatarsalgia include, but are not limited to: capsulitis, synovitis, 2nd MTPJ instability, plantar plate tear, neuroma, bursitis, intractable plantar keratoses, foreign bodies, infection, stress fracture, avascular necrosis/osteochondral defects, tumours, and inflammatory arthropathies.

This paper will discuss the diagnosis and conservative management of the commonly observed causes of central metatarsalgia.

Anatomy

The anatomy of the central forefoot is complicated and intricate, as visualised in Figure 1a and 1b.³ It is essential for clinicians to understand a normal joint, its function and the related anatomy. A lack of understanding of these factors will lead to difficulty identifying the important and injured structures, potentiating misdiagnosis and implementation of an incorrect treatment plan. The MTPJs have a fibrous capsule which attaches close to the articular surfaces of the metatarsal heads and phalangeal bases. The increased thickening at the sides and plantar aspect of the capsule forms the collateral ligaments and the plantar plate respectively, with the extensor expansion forming the roof of the joint capsule.⁴ The plantar plate is a broad trapezoidal fibrocartilaginous structure which traverses the plantar aspect of the MTPJs. It is thickest at the insertion point on the base of the proximal phalanx and medial and lateral to the midline, with a thinner portion centrally. This may be misinterpreted as a tear by inexperienced clinicians.⁵

The second MTPJ differs to the other lesser MTPJs in two ways. Firstly it has 2 dorsal interossei traversing the joint, whereas the others have both plantar and dorsal interossei. Secondly, the flexor digitorum longus tendon lies directly below the 2nd metatarsal head, unlike the slightly medial position it lies in at the other MTPJs.⁶

Knowledge of surface anatomy, as shown in Figure 2, is vital as it facilitates development of clinical skills. Educationalists believe surface anatomy should be considered an integral part of the core content of anatomy and clinical training.⁷

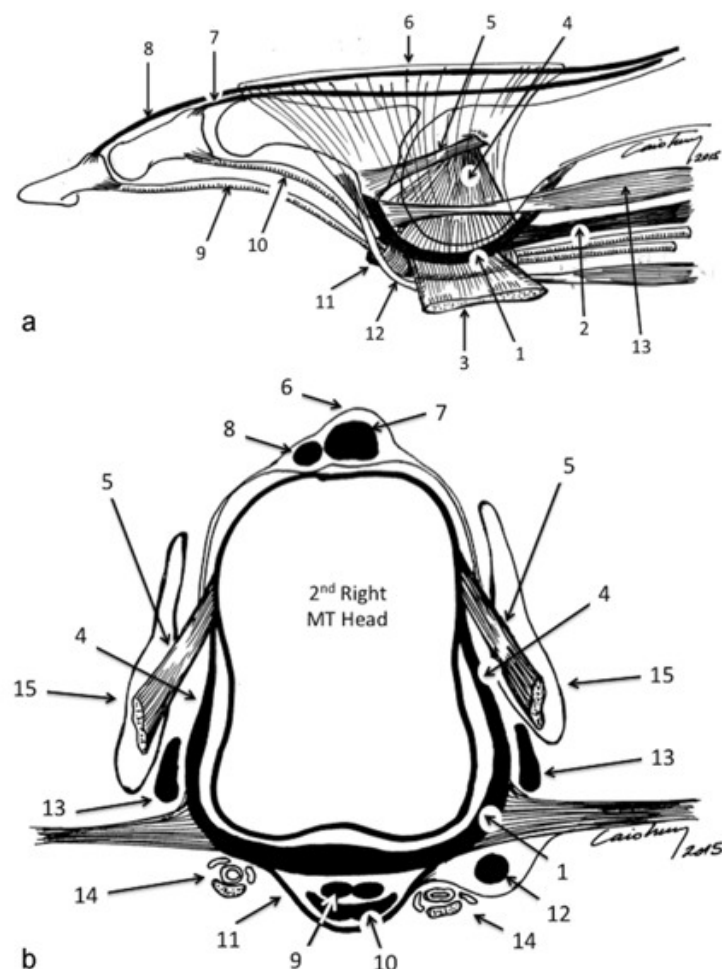


Figure 1 (above), Schematic illustration of the anatomy of the MTP joints of the lesser toes a) Sagittal view and b) Coronal view



Figure 2, The Surface anatomy of the central metatarsals

Aetiology

There are multiple proposed aetiologies for metatarsalgia including osseous, soft tissue, and systemic inflammatory conditions.

Espinosa et al. (2010)⁸ proposed a three-point classification of the causes of metatarsalgia:

1) Primary metatarsalgia relating to anatomical metatarsal abnormalities and their relationship to each other and the foot as a whole. For example, metatarsal parabola, pes cavus related excessive forefoot plantarflexion, and congenital deformities.

2) Secondary metatarsalgia following trauma, such as: fracture leading to displacement and subsequent malposition of the metatarsal heads, secondary to 1st ray pathology including hallux rigidus or hallux valgus, inflammatory arthropathy, lesser MTPJ instability, intermetatarsal neuroma, Freiberg infraction, and ankle equinus.

3) Iatrogenic, such as following surgery to address digital deformity or 1st ray pathology, causing transfer of pressure to the lesser MTPJs.

It is suggested that 90% of metatarsalgia cases have an underlying biomechanical cause including 1st ray insufficiency or tight posterior muscle groups.⁹ Maceira & Montegudo (2019)¹⁰ instead described the theory of propulsive and non-propulsive metatarsalgia. Propulsive metatarsalgia occurs during the 3rd (forefoot) rocker where ground contact is limited to the forefoot only. At this stage the metatarsals are subjected to direct increases in pressure and dorsiflexory moments. This mechanical stress is more likely to lead to chronic plantar plate degeneration than the mechanical stress experienced in non-propulsive metatarsalgia. Trauma or direct hyperextension injury can also lead to acute plantar plate tears.¹¹

Non-propulsive metatarsalgia is the result of mechanical imbalances or impairments which are present at any other phase of the gait cycle. For example, during swing phase, the forefoot is not in ground contact, but can be exposed to deforming forces from excessive extensor recruitment which account for an equinus deformity. It is essential to address these underlying mechanics in the management of metatarsalgia.

Clinical assessment

Clinical examination and history taking is arguably the foundation of any clinical assessment and provides both the required clues to lead to a preferred differential diagnosis, which can then be supported or refuted by imaging modalities if required. Questioning should include the patient's personal and familial medical history and their medication usage. Due to the complexity of the forefoot anatomically and functionally, misdiagnosis is common. A well-structured

Onset	When did the pain start? How long have they had it? Was onset sudden or gradual? Was there any associated trauma or precipitating factors?
Location	Where is the pain? Is it at metatarsal head, metatarsophalangeal joint or intermetatarsal space? Is the pain plantar, dorsal, medial, or lateral? Is it localised, widespread, or radiating?
Duration	How long do the symptoms last when they occur? Are they constant or short-lived?
Character	How does the patient describe the pain? E.g. dull, sharp, throbbing, stabbing, electric shock, burning, pins and needles, walking on a pebble. Aggravating & Alleviating factors Are there any specific factors that increase in the pain? E.g. increased activities, certain footwear, particular exercises. Does anything they do relieve the symptoms? i.e. resting, removing footwear, analgesics.
Radiation	Is the pain localised or does it move? Is there any neuritic symptoms radiating into the toes?
Timing	Does the pain fluctuate depending on the time of day or activity?
Treatments	Have any previous treatments been trialled? What were they and were they effective?
Severity	How severe is the pain on a scale of 0-10? Does the pain interfere with daily activities or restrict the patient in any way?

Table 1, OLDCARTTS for Metatarsalgia

history take and clinical assessment are crucial.^{12,13}

When approaching the history of the presenting condition, the mnemonic OLDCARTTS (Table 1) can be useful to provide a systematic approach and ensure all appropriate details are recorded.¹⁴

Metatarsalgia is generally accepted to be pain located to the ball of the foot, of gradual onset and aggravated by activity or footwear changes¹⁵. However, symptoms can be vague making diagnosis difficult.

The clinical examination consists of 2 main components: visual inspection and palpation.

Visual inspection

The area of pain identified by the patient should be inspected thoroughly:

- Is there any visible swelling or colour change/ discolouration when compared to the surrounding area and the contralateral limb?
- This could indicate an infective, inflammatory, or traumatic diagnosis.
- Is there any deformity to the forefoot or hindfoot?
- Is there any callus or hyperkeratosis? Is it localised or diffuse?
- This can help determine the underlying mechanics contributing to increased pressures. Plantar keratoses which are located directly plantar to the metatarsal head are associated with the 2nd rocker issue relating to an abnormal metatarsal length or parabola for example, while more diffuse callus patterns are associated with abnormalities within the 3rd rocker.^{2,10}

- Are there signs of vascular compromise, such as atrophic changes?
- Vascular assessment should be undertaken as a standard part of foot assessment.
- A visual check should be undertaken for any open wounds, ulcerations, or foreign bodies.

Palpation

Clinical palpation to locate accurately the pain is essential as cross-over of symptoms can cause misdiagnosis, potentially due to the underlying anatomy as the interdigital nerve runs close to the joint capsule, any swelling of the capsule could cause nerve irritation. Similarly, if there is nerve inflammation this could potentially cause hypersensitivity to the joint innervation.¹⁶

Precise plantar palpation is required to determine the exact location of the patient's pain, taking care to differentiate between the metatarsal head, the MTP joint line and the intermetatarsal space. Dorsal palpation should differentiate between the MTP joint line, the metatarsal head, neck or midshaft, along with the intermetatarsal space. This can help to differentiate more accurately between lesser MTPJ pathology, interdigital neuroma, bursa and fracture.

In the presence of an interdigital neuroma, a palpable 'Mulder's' click may be felt on palpation of the webspace while applying forefoot compression; if this recreates the symptoms, it is suggestive of a neuroma, although this is a non-specific clinical test. Clinical assessment with the "thumb index- finger squeeze test" is the most sensitive screening test

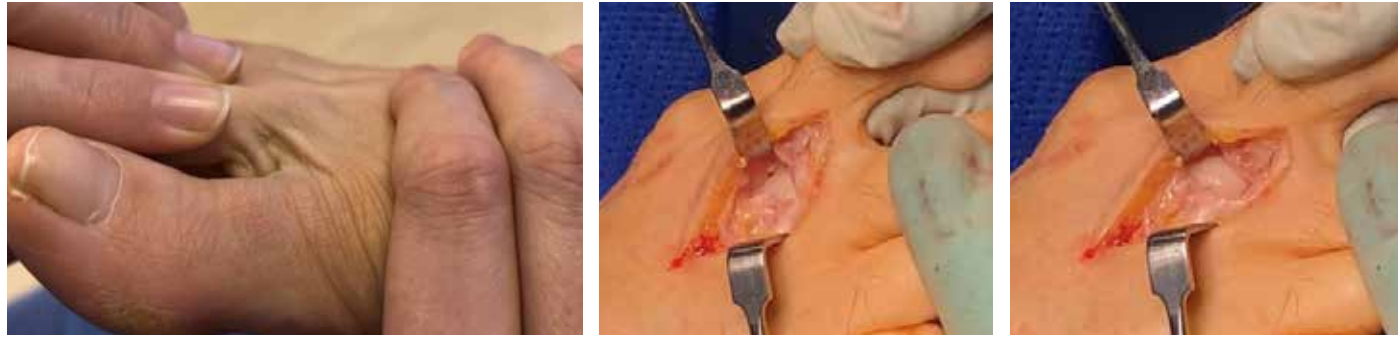


Figure 3, The Lachman's test, a) clinical example of a positive Lachman's test, b) intra-operative photograph of the 2nd MTPJ and c) intra-operative photograph of a positive Lachman's test

and was comparable to ultrasound (US) in diagnosing neuroma.¹⁷

Weightbearing and non-weightbearing assessment should be completed and the range and quality of motion at the lesser MTPJs should be assessed. Pain is often reported at the end range of motion. Stiffness and crepitus of the MTPJ may be noted and is suggestive of an arthrosis pathology. A dorsal drawer test, also referred to as the vertical stress or Lachman test, (Figure 3a, b & c) may be used to assess stability of the MTPJ. While holding the foot with one hand and the base of the toe firmly in the other, the toe is dorsally translated. An increased degree of displacement which recreates the reported symptoms is suggestive of synovitis or MTPJ instability.^{15, 18}

Any associated digital deformity should be assessed for its flexible or fixed nature and the patient questioned on changes to toe position. Metatarsalgia may precede digital deformity,¹⁵ particularly in the case of plantar plate tears. Splaying of the digits is considered more in keeping with capsular issues including instability as opposed to neuroma. However, severe hyperextension at the MTPJ may cause soft tissue attenuation and predispose to a rupture of the plantar plate. Similarly, claw toe deformity with hyperextension at the MTPJ can encourage distal displacement of the plantar fat pad, reducing resilience to the increased plantar pressures at the metatarsal heads.

Range of motion of the 1st MTPJ, midfoot, ankle and rearfoot should also be noted. Incompetence of the 1st ray due to hallux valgus, hallux rigidus, a short 1st metatarsal, or previous surgery can result in overloading of the lesser MTPJs¹⁹ and subsequent development of metatarsalgia. Restricted ankle joint range of motion due to arthrosis, Achilles contracture, or soft tissue equinus is also associated with an increase in forefoot pressures and a propensity to the development of metatarsalgia.^{9,15,18} The Silfverskiöld test (Figure 4a & 4b) should be performed to assess for the presence of gastrocnemius-soleal tightness/soft tissue ankle equinus.

To perform this test:

- The subtalar joint should be in neutral and the knee in full extension.
- The foot is then dorsiflexed to 90° and an attempt is made to dorsiflex further.
- Equinus is considered present when there is less than 10° further dorsiflexion available.
- This is then repeated with the knee flexed, if additional dorsiflexion is achieved a gastrocnemius equinus is present.²⁰

It is suggested that 90% of all cases of metatarsalgia can be explained by biomechanical causes⁹. Assessment of footwear, wear patterns, and gait should be observed, in addition to palpation, range of motion, non-weightbearing and weightbearing assessment of deformity.

Consideration must be given to any red flag features reported which could indicate the presence of a sinister pathology. These may include reports of pain at night, fever, unexplained weight loss, and widespread neurological changes and should prompt urgent investigation and onward referral as required. Similarly, infection should be investigated and discounted in the presence of active inflammation. Systemic pathology including anaemia, vitamin B12 or D deficiency, alcoholic neuropathy, diabetes, thyroid disorders, inflammatory disorders and radicular causation must also be considered. This is especially important when pain is global, atypical, the clinical assessment is largely normal, or reported pain is above anticipated levels.

Diagnostic imaging

After considering the clinical signs and symptoms along with thorough clinical assessment, diagnostic imaging may be required to aid diagnostic accuracy of the specific cause of central metatarsalgia.

X-rays (XRs) are first line imaging to exclude stress fractures. Fractures may be missed if XR imaging is conducted early after the injury occurred as it can take up to 14 days for bony callus to become evident radiographically.²¹ XRs can also be used to appreciate the metatarsal parabola, MTPJ dislocation, stage Freiberg's Disease and for surgical planning.



Figure 4, The Silfverskiöld test, a) Limited range of dorsiflexion with the knee extended and b) Increased range of dorsiflexion with the knee flexed

Ultrasound (US) is relatively cheap, can be useful in visualising neuromal bursal complexes as round to ovoid, well-defined, and with hypoechoic lesions in the interspaces that can have mixed echotexture.²² Neuromas are non-compressible and Mulder's sign may be visualised.²³ A systematic review conducted by Xu et al. (2015)²⁴ found US to be 90% sensitive and 88% specific in identifying Morton's neuroma, with MRI having 96% sensitivity but only 68% specificity. US is however noted to be very operator dependent. The dynamic nature of US can be of assistance when assessing for a Mulder's click or when assessing MTP joints for instability.

Magnetic Resonance Imaging (MRI) +/- contrast or arthrography are available to assess the interspaces for lesions and lesser MTP joints for pathology. Regarding the use of imaging in diagnosing plantar plate tears, there are conflicting opinions. Klein et al. (2012)²⁵, after prospectively comparing preoperative US findings with observed intraoperative findings for 50 patients undergoing surgical correction of plantar plate tears, found that MRI and US are appropriate soft-tissue imaging modalities when the anatomy is understood by the technician and radiologist. They established that both modalities can detect plantar plate pathology, but that MRI is more specific at localising tears therefore, although US is less costly and quicker, it should not replace MRI in all cases.

In 2013, Klein et al. further stated that ultrasound is highly dependent on the technician with the best sonographic images being captured by a technician who is highly skilled. However, a retrospective review by Dinoá et al. (2016)²⁶ of forefoot MRIs found that 30% of the plate defects only became visible after enhancement with gadolinium. Carlson et al. (2013)²⁷, in a retrospective case series of only 8 patients found US displayed comparable sensitivity in identifying plantar plate tears compared with MRI, with considerably less financial cost. Feuerstein et al. (2014)²⁸ found the

sensitivity and accuracy of US was best when dynamic assessment of the plantar plate was employed and stated that caution should be used when relying solely on static images to diagnose subtle injuries. In a systematic review, Maas et al. (2016)²⁹ stress the importance of plantar plate anatomy and integrity for MTP joint stability, and the review highlights the lack of primary data regarding plantar plate anatomy and MTP joint stability. A meta-analysis including seven studies involving 246 plantar plate tears showed MRI provided more diagnostic accuracy than US for the detection of plantar plate tears, with higher sensitivity and specificity showing MRI had superior diagnostic accuracy than US.³⁰

Arthrography injects contrast media into the joint under fluoroscopic guidance with subsequent MRI to evaluate the integrity of the MTP joint capsule and plantar plate to clearly delineate capsular tears and associated abnormalities.³¹ Its invasive nature and cost means it is not frequently utilised.

Diagnostic imaging is no substitute for a thorough clinical examination. Table 2 illustrates a diagnostic imaging matrix for central metatarsalgia. Once the appropriate imaging modality has been chosen it is imperative that the clinical history and assessment information are added to the referral to enable the reporting clinician to access as much information as possible to make an accurate diagnosis.

Treatment options

Treatment is determined by the diagnosis and underlying cause identified. For treatment to be successful, addressing any biomechanical or contributory factors is paramount regardless of the pathology being treated. First line treatment is conservative, although the authors recognise that there is a paucity of high-quality evidence (from randomised controlled trials) to support the various conservative treatment options.

Simple analgesics, non-steroidal anti-inflammatory

	X-Rays	Ultrasound	MRI	Arthrography
Stress fracture	✓			
Keratosis	✓			
Capsulitis		✓		
Neuroma bursal complex		✓	✓	
Synovitis/inflammatory arthritis	✓	✓		
Freiberg's Disease	✓			
Lesser MTP joint instability	✓	✓	✓	✓
MTP dislocation	✓			

Table 2, Diagnostic Imaging Matrix for Central Metatarsalgia

drugs, rest from aggravating activities, footwear advice and adaptations, callus management, physical therapy, and lifestyle advice such as weight loss and smoking cessation are applicable to many causes of metatarsalgia and therefore are first line treatment modalities, with additional treatments being condition specific.^{15,18,32,33}

In the presence of soft tissue ankle equinus, a calf stretching programme has been suggested to be beneficial in the management of metatarsalgia symptoms.⁹ This may include active calf stretching exercises or incorporating the use of postural exercises using a calf-stretching board. Orthotic therapy designed to address the underlying contributory foot mechanics and redistributing load from high-pressure areas has been shown to be effective in reducing metatarsalgia.¹²

Lesser MTPJ instability and plantar plate tears

Capsulitis and synovitis can be considered part of a continuum of lesser MTPJ instability which if untreated can, through joint capsule distension, lead to instability, plantar plate tear, and lesser toe deformity, ultimately ending with dislocation at the MTPJ. It is worth noting that acute plantar plate tears can also occur with hyperextension trauma.

The 2nd MTPJ is most affected: 40% of metatarsalgia cases have had plantar plate tears reported, with 90% occurring at the 2nd MTPJ.³⁴ This is postulated to be due to its anatomical prominence as the longest metatarsal.^{3,15,35}

Capsulitis is inflammation of the joint capsule and surrounding ligaments, whereas synovitis is inflammation of the synovial membrane (the joint lining). Inflammatory arthropathies can present as synovitis, including rheumatoid arthritis (which has a predilection to metatarsophalangeal joint

destruction).^{36,37} Blood tests may be of benefit in these patients including C-reactive protein, erythrocyte sedimentation rate, anti-cyclic citrullinated peptide antibody, and rheumatoid factor.^{15,38}

2nd MTPJ instability/capsulitis can be diagnostically challenging with a risk of being misdiagnosed as an interdigital neuroma.^{3,39,40} Patients typically report pain with or without swelling of the affected joint. There may be localised erythema affecting the dorsum of the joint. Pain is often characterised as a sensation of walking on a pebble, which is predictably aggravated by increased activity, hard-surfaces, or thin-soled or high-heeled footwear and relieved

by rest.^{18,35,39} Neuroma however, is more likely to cause neuritic symptoms which are more vague and more unpredictable.

KEY POINT: Capsulitis is inflammation of the joint capsule and surrounding ligaments, whereas synovitis is inflammation of the synovial membrane (the joint lining).

Any precipitating trauma or recent change in toe position should be identified. A positive dorsal drawer test (described previously) is highly suggestive of MTPJ instability with 80.6% sensitivity and 99.8% specificity.⁴¹ There may or may not be a digital deformity present, with loss of toe purchase or development of a crossover 2nd toe deformity, which may be more obvious on weightbearing assessment.¹⁵ In early stages there may be no accompanying toe deformity, however as the peri-articular soft tissues continue to attenuate, progressive deformity typically develops; it is usually this progression of deformity which prompts individuals to seek treatment.^{35,42} Pain is often recreated at the plantar crease or base of the affected toe and aggravated by range of motion testing. It is important to note that the presence of clinical instability is not specific to plantar plate tears and may also be present in the presence of ligamentous laxity and chronic soft tissue attenuation.

Coughlin et al. (2011)⁴³ provided a classification system for pre-dislocation syndrome as shown in Table 3. While other systems are available, Coughlin's classification incorporates assessment of digital alignment and physical examination.

Conservative management involves off-loading the affecting joint with cushioning pads, rocker-soled footwear, and bespoke orthotics^{15,18,33,35,40,44,45}, in addition to the standard treatments previously discussed of activity modification and simple analgesics.

Grade	Alignment	Physical Examination
0	MTP joint alignment; prodromal phase with pain but no deformity	MTP joint pain, thickening or swelling of MTP joint, reduced toe purchase, negative drawer
1	Mild malalignment at MTP joint; widening of web space, medial deviation	MTP joint pain, swelling of MTP joint, loss of toe purchase, mild positive drawer (<50% subluxable)
2	Moderate malalignment; medial, lateral, dorsal, or dorsomedial deformity, hyperextension of toe	MTP joint pain, reduced swelling, no toe purchase, moderate positive drawer (>50% subluxable)
3	Severe malalignment; dorsal or dorsomedial deformity, second toe can overlap the hallux; may have flexible hammertoe	Joint and toe pain, little swelling, no toe purchase, very positive drawer (dislocatable MTP joint), flexible hammertoe
4	Dorsomedial or dorsal dislocation; severe deformity with dislocation, fixed hammertoe	Joint and toe pain, little or no swelling, no toe purchase, dislocated MTP joint, fixed hammertoe

Figure 5 (below), Darco toe alignment splint
a) dorsal view with 2nd toe in plantarflexed and lateral position and b) plantar view of splint



Table 3 (above), Classification system for predislocation syndrome (Coughlin et al., 2011)

Loop strapping of the toe is recommended with the proximal phalanx held in 10 degrees plantarflexion with the aim of providing sagittal plane stability, reducing pain and promoting fibrosis of the plantar plate degeneration or tear.^{40,46} As the plantar plate is primarily composed of Type 1 collagen, some authors argue taping is ineffective in severe tears where dislocation is present, as it has a lower innate ability to spontaneously heal.⁴⁷

Loop strapping may be achieved by taping, by a Darco® toe splint (as pictured in Figure 5a & 5b) or a Budin splint. Care must be taken when applying the strapping to ensure a tourniquet effect of the digit does not occur due to swelling.

It is recognised that steroid injection therapy may be beneficial in the management of lesser MTPJ capsulitis and pre-dislocation syndrome, however it is a contentious topic. Coughlin et al. (2011)⁴³ suggest that selective use of corticosteroid injections may be diagnostic and therapeutic, but could mask symptoms and allow further capsular and plantar plate degeneration, resulting in joint subluxation and dislocation. Other authors have argued that while conservative measures can temporarily relieve pain, they will not permanently correct the deformity so may be of limited value. Where steroid injection therapy is considered appropriate, the patient must be fully informed of the potential risks including the worsening of deformity and soft tissue rupture. There is no consensus within the literature regarding the most appropriate steroid choice although agreement is present that the smallest effective dose should be used. Kilmartin, (2017)⁴⁸, suggests 0.5ml of 4mg/ml betamethasone. Stabilising the toe by taping is considered critical for 6 weeks following the injection.⁴⁹

Surgical treatment of 2nd MTPJ instability and plantar plate tears is indicated when symptoms continue or when conservative treatments have been exhausted



Figure 6, US imaging showing a) transverse and b) longitudinal views of Morton's neuroma

or in severe deformity. As many patients often do not present in early-disease stages, the role of conservative treatment may be limited¹¹, and most of the conservative management literature covers injuries of Coughlin grade 0-2. Surgery aims to restore normal mechanical alignment and function of the affected MTPJ and correct any associated digital deformity.³ This may include proximal interphalangeal joint arthroplasty or arthrodesis, flexor tendon transfers, plantar plate repair, and lesser metatarsal osteotomies and possible 1st ray correction (i.e. hallux valgus correction) in the presence of 1st ray insufficiency.^{3,15,45}

Hammer toe deformity can occur due to instability at the lesser MTPJs, however toe deformity may also occur without instability, but retrograde pressure can cause metatarsalgia. Management of toe deformity includes cushioning covers, footwear advice (wide/deep toebox) and corn/callus management.^{15,44} When these options fail to improve symptoms then surgery may be indicated including tenotomies, proximal interphalangeal joint arthroplasty or arthrodesis, and lesser metatarsal osteotomies. In cases of severe toe deformity conservative measures may be of limited benefit. However, these should be exhausted prior to surgical intervention due to the risks associated with surgery.^{2,15}

Intractable plantar keratoses

Intractable plantar keratoses whether discrete or diffuse can cause significant pain and disability.⁵⁰ It is imperative to assess these fully to determine the underlying cause and precipitating factors including deformity, biomechanical factors, fat pad atrophy and lifestyle factors such as smoking. Ensuring correct diagnosis is also imperative to successful treatment outcomes. In a study by Lopez & Kilmartin (2016)⁵⁰, which assessed outcomes of full thickness excision of intractable plantar keratoses, 51% of the excised lesions were identified by histopathology as verrucae. This highlights the potential for misdiagnosis and the risk of unnecessary intervention.

Discrete lesions located beneath the metatarsal head may be associated with underlying bony abnormalities

such as a condylar prominence, whereas diffuse lesions are more typically associated with a long metatarsal, a retracted digit causing retrograde pressure, transfer lesions from 1st ray insufficiency, previous surgery or fracture resulting in transfer lesions, or previous fracture with subsequent healing in a plantarflexed position.¹⁸

First-line treatment involves debridement of the lesion, however this typically only provides short-term relief if the underlying factors are not addressed. Additional non-surgical treatment modalities include cushioning pads and orthoses to redistribute pressure.

While considering callosities, it is also important to determine the patient's smoking status. Smoking has been indicated as a significant risk factor for the development of plantar hyperkeratotic lesions. This is postulated to be attributable to the effect of nicotine on skin receptors, which essentially upregulates keratinocyte adhesion and migration to the epidermis, increasing production.⁵¹ In Lopez & Kilmartin's study (2016)⁵⁰ 76% of their cohort were cigarette smokers, and 60% of those developed a recurrence following surgical excision of the lesion. This highlights the importance of a holistic approach and patient education in treatment planning and outcomes.

Key Point: . Smoking has been indicated as a significant risk factor for the development of plantar hyperkeratotic lesions.

Electrosurgery is an alternative treatment modality, but requires specialist equipment, and carries the risk of scarring and high recurrence rates.⁵² When non-surgical treatments fail, lesion excision may be considered with primary or secondary intention healing⁵³ or where there is an underlying anatomical abnormality, lesser metatarsal osteotomy may be considered with the aim of restoring normal mechanical alignment. However, this procedure also possesses inherent risks including transfer lesions to adjacent metatarsals, joint stiffness, fixation irritation, and floating toes.^{18,50}

Interdigital neuroma

Interdigital neuroma, known as Morton's neuroma in the third webspace, is a non-neoplastic lesion characterised



Figure 7, Freiberg's Disease with secondary arthrosis

by perineural fibrosis, vascular proliferation, endoneural oedema, and axonal degeneration of the interdigital nerve.⁵⁴ The pathophysiology is not fully understood but repetitive microtrauma and compression of the nerve which lies plantar to the transverse intermetatarsal ligament are accepted causes.³³ Symptoms include paraesthesia and pain which is worse with weight-bearing or wearing narrow footwear and relieved by removing shoes and massaging the foot.

Diagnosis can be made clinically¹⁷ but US is frequently used to support clinical diagnosis, with MRI being utilised in uncertain cases or to exclude adjacent pathology. Figures 6a & 6b illustrate the US appearances of a neuroma.

There are a variety of treatment options for interdigital neuroma with the simple measures including analgesics, metatarsal pads and wide fitting footwear. In a systematic review of non-surgical treatment for Morton's neuroma Thomson et al. (2020)⁵⁵ identified 486 studies, 22 proceeded to full-text review. Nine non-operative treatment modalities were identified: corticosteroid injection, alcohol injection, extra-corporeal shockwave therapy (ESWT), radiofrequency ablation (RFA), cryoablation, capsaicin injection, Botulinum toxin, orthosis and YAG laser therapy. Corticosteroid injection showed a statistically significant reduction in mean pain scores over all the studies, with 50% success at 12 months. Alcohol showed

promising short-term pain-relieving results only. Orthotics, capsaicin injections, cryoablation, Botulinum toxin, RFA and ESWT did show statistically significant improvements but with limitations to their application. A meta-analysis, carried out by Matthews et al. (2019)⁵⁶, found corticosteroid injections and manipulation/mobilisation had the strongest evidence for pain reduction, however high-quality evidence for a gold standard intervention was not found.

Operative management is indicated for patients with persistent symptoms who fail non-operative management. Surgical decompression with release of the transverse intermetatarsal ligament can be done via minimally invasive⁵⁷ or endoscopic⁵⁸ approaches, but due to low levels of evidence the mainstay of surgical treatment remains neurectomy. Pace et al. (2009)⁵⁹, reported an 82% patient satisfaction rate in 82 feet (78 patients) but a 71% rate of footwear restriction at 4.6 years post-operatively. In 2011 Lee et al.⁶⁰ published their 10-year retrospective case series of 19 feet following Morton's neurectomy with a 61% satisfaction rate with the main complication of plantar interspace numbness.

Freiberg's Disease

Osteonecrosis of the lesser metatarsal head is a rare but well-recognised condition which has been referred to by various names⁶¹⁻⁶⁵ since it was first described in 1906 by Alfred Freiberg. Multiple aetiologies have been proposed including traumatic, mechanical, vascular, or systemic causes, which is why the condition has multiple names. Freiberg's Disease most commonly affects the second metatarsal head (68%), with involvement of the third and fourth metatarsals occurring less frequently, 27% and 3% respectively.⁶⁵ Unusually for osteochondroses, there is a higher prevalence in females with a ratio of 5:1.⁶⁶ It is considered a juvenile pathology but there are reported cases in the later life, and it can be asymptomatic until secondary arthrosis causes symptoms, as illustrated radiographically in Figure 7.

Key Point: Freiberg's Disease most commonly affects the second metatarsal head

The Smillie staging system (Figure 8) published in 1967 describes 5 stages of disease based on plain film radiographs to determine severity of the pathological process.⁶⁷ Plain radiographs are the most common modality for initial diagnosis but are known to be insufficiently sensitive to detect early-stage disease whereas Torriani et al. (2008)⁶⁸ found MRI to be beneficial for all stages. Advanced cross-sectional imaging (e.g. MRI) is of benefit for treatment planning as an appreciation of the entire joint profile can be gained. There is no optimal protocol for imaging to diagnose or plan treatment.

Treatment options depend on the presentation. In

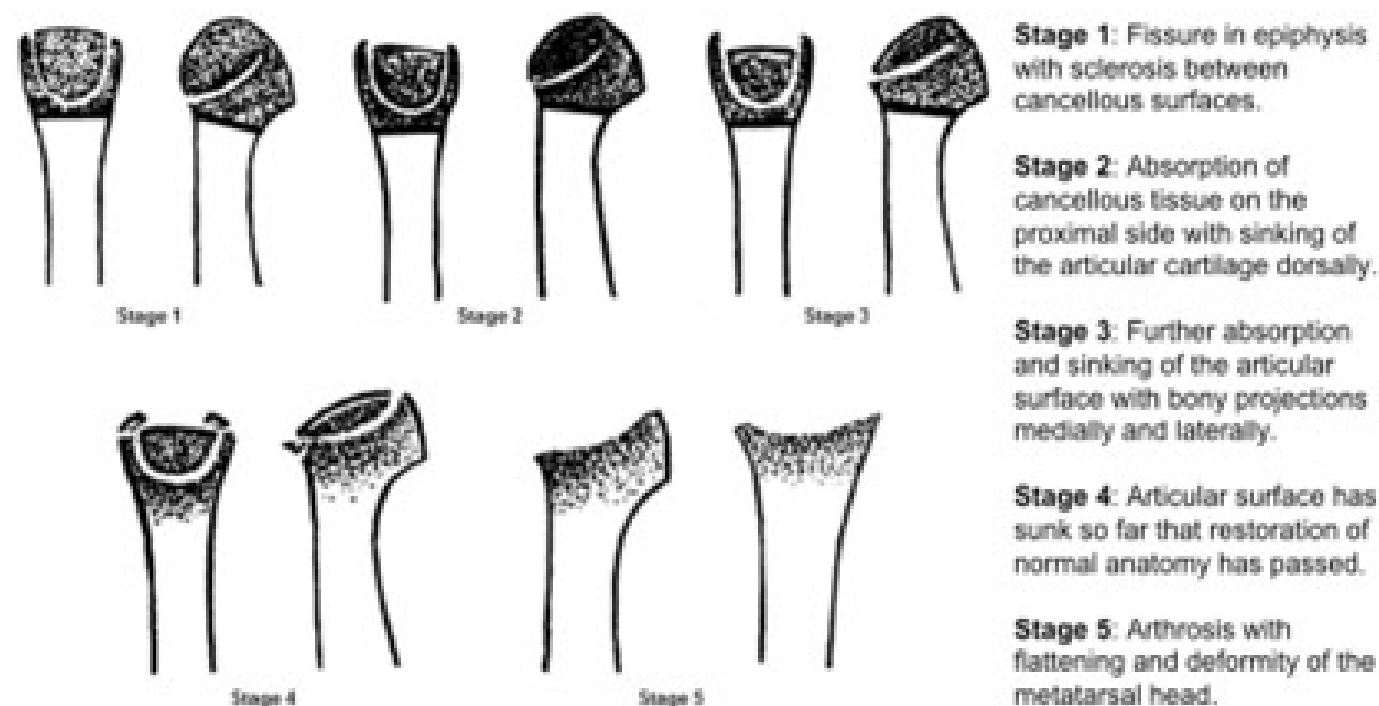


Figure 8, The Smillie staging system for Freiberg's Disease

paediatric cases treatment is aimed at offloading, preventing collapse and further damage to the metatarsal head. This may be achieved with activity modification, strapping, orthoses, stiff soled footwear and immobilisation in casts/air boots. In the adult population, treatment is like that of any small foot joint with arthrosis including offloading, intra-articular steroid injections, and hyaluronic visco-supplementation injections. These can be useful modalities in the treatment pathway although evidence specific to Freiberg's is weak. When conservative measures fail, surgical management is indicated for progressive pain, joint destruction and joint deformity.

Surgical management of Freiberg's can be divided into joint sparing and joint sacrificing procedures (Table 4). Case studies and reviews have suggested the use of core decompression^{69,70}, synthetic cartilage graft⁷¹, osteochondral autograft transfer⁷² and lesser MTP joint Cartiva synthetic resurfacing.⁷³ A systematic review by Alhadhoud et al. (2021)⁷⁴ identified procedural sub-groups and the evidence to support their use. The procedures reviewed: rotational metatarsal osteotomy, interposition arthroplasty, debridement, microfracture,

auto/allograft, hemiarthroplasty, resection arthroplasty, were all deemed to have weak grade 'C' evidence to support their use. Metatarsal shortening Weil osteotomy had insufficient evidence to support its use in the treatment of Freiberg's.

Various surgical approaches have been described throughout the literature in both joint sparing and joint sacrificing groups but there is a lack of high-quality evidence which highlights the need for further work to be undertaken to justify their use.

Stress fracture

Central metatarsal stress fractures are a common foot injury and result from cyclic loading forces that exceed the strength of the bone. Stress injuries occur in normal bone subjected to excessive force (fatigue injury) or weakened bone subjected to normal physiologic force (insufficiency injury).³³

The second metatarsal is most commonly affected⁷⁵ possibly due to its length and morticed position in the midfoot. Associated foot deformities e.g. hallux valgus or a cavo-varus foot type, may predispose to these injuries and should be evaluated.⁵ Symptoms of a stress fracture can include pain, swelling and an inability to

JOINT SPARING	JOINT SACRIFICING
Arthrotomy (removal of loose bodies)	MTP joint synthetic resurfacing (Cartiva)
Core decompression (Drilling/Microfracture)	Implant arthroplasty
Synthetic cartilage graft	Interposition arthroplasty
Osteochondral Autograft Transfer	Metatarsal head excision
Metatarsal osteotomy (dorsal closing wedge)	Basal phalangectomy

Table 4, Surgical management of Freiberg's Disease



Figure 9, Healed 3rd metatarsal fracture on AP X-ray bear weight. Diagnosis is made by plain radiographs with MRI being useful for occult fractures or to identify bone marrow oedema.³³ Metabolic evaluation should be considered for fragility fractures to determine if osteopenia or osteoporosis is a contributing factor requiring treatment.²¹

Key Point: Stress injuries occur in normal bone subjected to excessive force (fatigue injury) or weakened bone subjected to normal physiologic force (insufficiency injury).

Treatment for central metatarsal stress fractures is usually conservative offloading for six weeks to allow the fracture site to heal with minimal displacement. This is achieved by wearing a stiff soled post-operative shoe, walking boot or offloading cast/air boot. Figure 9 shows a healed metatarsal fracture. If displacement has occurred, as seen in Figure 10, there is a risk of malunion which may lead to transfer metatarsalgia or plantar keratosis.¹⁸ If significant displacement is identified in the acute phase, treatment with closed reduction and percutaneous fixation, or open reduction and internal fixation may be required to avoid the risk of transfer metatarsalgia and non-union. If diagnosis is delayed, then surgery with metatarsal osteotomy may be required to restore alignment.²¹



Figure 10, Dorsally displaced 3rd metatarsal fracture on oblique X-ray

Conclusion

Central metatarsalgia is complex and requires in-depth clinical assessment and a thorough understanding of anatomy and normal mechanics. It is essential to consider any diagnostic tests within the context of the clinical signs, symptoms and individual history to ensure correct diagnosis and appropriate treatment planning.

KEY POINTS
Anatomy is key – must know surface anatomy and what's normal
A thorough history and clinical examination is fundamental
Treat the patient not the imaging
A clear diagnosis is required to inform the treatment plan
Management of patient expectations is essential – how long will it take to resolve, what should be expected from treatment?
Consider alternative diagnoses when symptoms are atypical and pain is heightened above anticipated levels
Consider a holistic approach such as smoking cessation and weight management

Table 5, Key points

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