Metatarsalgia: anatomy, pathology and management

Robert Cooke[1]
Christopher Manning[1]
Dhanushka Palihawadana[1]
Aamir I Zubairy[1]
Shah HM Khan[2]

Author details can be found at the end of this article

Correspondence to: Robert Cooke; robert.cooke@doctors.org.uk

Abstract

Metatarsalgia is a common presentation, particularly in middle-aged women. This review discusses the anatomical basis and classifies the different pathologies into primary, secondary and iatrogenic. The key elements to differentiate the pathologies within each classification which could cause a patient to suffer with metatarsalgia are outlined. Further investigations are briefly covered, but a linked article discusses the investigations of metatarsalgia in more detail. The article gives an overview of metatarsalgia to help clinicians to investigate and manage these symptoms.

Key words: Ankle metatarsalgia; Foot; Orthopaedics

Received: 11 June 2021; accepted following double-blind peer review: 18 June 2021

Introduction

Metatarsalgia is broadly defined as pain in the forefoot underneath the heads of one or more metatarsals (Besse, 2017). Metatarsalgia is a common reason for referral to a foot and ankle clinic, particularly in middle-aged women. The incidence of foot problems increases significantly with age, with a large proportion of these being forefoot problems (Chahal et al, 2020).

This review discusses the different pathologies which can cause metatarsalgia and covers the treatment options available, whether these be conservative or surgical.

Anatomy

When discussing metatarsalgia it is important to first understand the relevant anatomy and physiology of the forefoot within the gait cycle. The gait cycle is divided into the swing and stance phases. The swing phase accounts for 40% of the gait cycle, with the aim of providing sufficient foot clearance from the ground and preparing the foot for the heel strike at the start of the stance phase. Flexion of the hip and knee alongside dorsiflexion of the ankle enable the foot to clear the ground. The ankle dorsiflexion optimises the position of the foot for heel strike at the beginning of the stance phase. The power has to come from the tibialis anterior muscle for the ankle to dorsiflex efficiently, which also creates an inversion action. Subsequently, to counterbalance this, the extensors need to evert the foot to maintain a balanced foot position (Espinosa et al, 2010) (Figure 1).

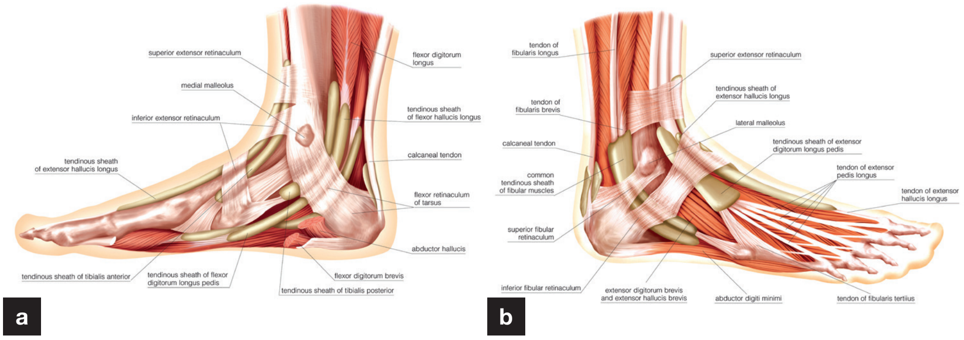

Figure 1. The muscles of the leg and foot: (a) medial view and (b) lateral view.

How to cite this article:
Cooke R, Manning C, Palihawadana D, Zubairy AI, Khan SHM. Metatarsalgia: anatomy, pathology and management. Br J Hosp Med. 2021. https://doi.org/10.12968/hmed.2021.0348

Table 1. Different types and causes of metatarsalgia		
Type	**Problem**	**Pathology**
Primary	Long second metatarsal	Increased point loading on second metatarsal head
	First ray deficiency (short first metatarsal or instability at first tarsometatarsal joint)	Increased pressure on lesser metatarsals
	Hyperextension of metatarsophalangeal joint	Abnormal loading on metatarsal heads
	Enlarged metatarsal head or condyle	Trauma, infection, arthritis, congenital malformation or hereditary factors
Secondary	Trauma	Malalignment of metatarsals
	Metatarsophalangeal joint instability	Acute trauma Plantar plate, medial and lateral collateral ligament tears
	Freiberg's disease	Avascular necrosis of the metatarsal head
	Morton's neuroma	Fibrosis of common digital nerves found in intermetatarsal spaces
	Systemic conditions	Rheumatoid and psoriatic arthritis Charcot–Marie–Tooth disease and other neurological conditions
Iatrogenic	Metatarsalgia post forefoot surgery	Hallux valgus surgery, lesser metatarsal osteotomies

The stance phase of the gait cycle aims to propel the body forward while maintaining stability. The stance phase has a three-rocker mechanism. The first rocker accounts for the initial 10% of the stance phase and involves the initial heel strike with the ground, with the tibia then rotating forward on the talus at the ankle joint. The entire foot making contact with the ground accounts for the next 20%, with the ankle providing the second rocker as the tibia rotates on the talus. The final stage of the stance phase is the toe off with the metatarsophalangeal joints in dorsiflexion. At this stage the gastrocnemius–soleus complex contracts causing the initial heel lift off, this results in only the forefoot being in contact with the ground at this point (Espinosa et al, 2008, 2010; Maceira and Monteagudo, 2019). In a normal foot, the metatarsal heads all remain the same distance from the ground with a reducing angle between the metatarsal heads and the ground from the first to fifth rays. The transverse inter-metatarsal ligament between the metatarsals enables the metatarsals to work synergistically with each other. Therefore disruption to this normal anatomy affects the distribution of weight across the whole forefoot unit, which will create increased pressure on the affected metatarsal head (Besse, 2017).

Patient assessment

When reviewing a patient with metatarsalgia, it is key that this is considered a symptom rather than a diagnosis. Many differing pathologies can cause a patient to present with metatarsalgia, and there can also be more than one cause within a single patient. This creates challenges in diagnosing the causative pathology of a patient's metatarsalgia. The cornerstone to being able to identify the cause of metatarsalgia is to revert to basic principles of good clinical history and examination. Pertinent questioning can help to guide the clinician to potential causes and underlying conditions which may be contributing to the patient's symptoms. This should include identifying if the patient's pain is different throughout the gait cycle as well as the exact site and character of pain. Clinical examination should be performed with the patient standing and in the supine position, as well as assessing the patient's gait. In the standing position deformities of the foot can be identified and this should include assessment of the mid and hindfoot as deformities of the mid and hindfoot could result in forefoot symptoms and metatarsalgia. Keratosis and previous scars can provide vital clues to aid the clinician (Besse, 2017; Lopez and Slullitel, 2019). Finally, it is important to perform a detailed neurological and vascular assessment.

When investigating metatarsalgia, initial dorsoplantar, oblique and lateral view radiographs of the foot are the starting point. These must be standing weight bearing views. Further imaging with ultrasound scans or magnetic resonance imaging can be beneficial, depending on the potential pathological cause (Espinosa et al, 2010). A further article detailing imaging of metatarsalgia is included in this issue (https://doi.org/10.12968/hmed.2021.0353).

The aetiology of metatarsalgia is commonly divided into three categories: primary, secondary and iatrogenic following forefoot surgery.

Primary metatarsalgia

Primary metatarsalgia is broadly described as anatomical abnormalities of the foot which result in increased load to the affected metatarsal (Espinosa et al, 2008). These abnormalities are not just confined to the forefoot, as abnormalities of the mid and hindfoot can also lead to increased loading of an affected metatarsal.

Patients who are presenting with symptoms of primary metatarsalgia may describe their pain being worse in the propulsive phase of gait as a result of abnormal load distribution or in the stance phase from a hyperextended metatarsophalangeal joint. The patient will likely have discrete tenderness over the affected metatarsal head. Repetitive point loading of this area during the gait cycle can cause the plantar soft tissue to become swollen which, over time, can lead to the skin thickening and developing plantar keratosis (Espinosa et al, 2008).

For the load to be adequately distributed during the gait cycle and prevent increased stress at a single point, both the relative length and the inclination of the metatarsals in relation to each other are important (Maceira and Monteagudo, 2019).

The most common abnormality causing primary metatarsalgia is a disproportionately long second metatarsal in comparison to the other metatarsals. This causes increased point loading on the second metatarsal head resulting in symptoms (Espinosa et al, 2008, 2010).

First ray deficiency can increase the pressure exerted on the lesser metatarsals. This can be caused by several pathologies occurring in the first ray, such as a congenitally short first ray or relative shortening caused by hallux valgus deformity. In addition, hypermobility at the first metatarsocuneiform joint can lead to transfer of load to the lesser metatarsals (Espinosa et al, 2010).

Hyperextension of the metatarsophalangeal joints can lead to abnormal loading underneath the metatarsal heads. This can occur as a result of hindfoot abnormalities such as pes cavus, gastrocnemius–soleus complex tightness or contracture as well as any hindfoot problem which causes increased pressure on the forefoot (Besse, 2017). Gastrocnemius–soleus complex contracture or tightness increases the involvement of the extensors of the lesser toes which results in metatarsophalangeal joint disorders with the plantar fat pad moving distally, exposing the metatarsal heads to increased load (Besse, 2017; Lopez and Slullitel, 2019).

An enlarged metatarsal head or condyle is a less frequent cause of pressure below the metatarsal head. This can occur as a result of trauma, infection, arthritis, tumour, congenital malformation or hereditary factors (Espinosa et al, 2008, 2010; Besse, 2017).

Secondary metatarsalgia

Secondary metatarsalgia is when metatarsal loading is increased by conditions having either a direct or indirect action on the metatarsal. It can be caused by local conditions which have an effect on the metatarsals or systemic conditions which cause overloading of the foot (Espinosa et al, 2008; Chahal et al, 2020).

Trauma

Trauma to the metatarsals can result in malalignment. If the malalignment causes depression of the metatarsal head this can cause direct pressure symptoms. However, if the malalignment causes the metatarsal head to become elevated this could transfer pressure to neighbouring metatarsals (Espinosa et al, 2010).

Table 2. Clinical staging system for metatarsophalangeal instability

Stage	Alignment			Clinical examination			
	Degree of malalignment	Deformity	Other features	Joint pain	Swelling	Toe purchase	Drawer test
0	None	None	Prodromal phase with pain	Yes	Yes	Diminished	Negative
1	Mild	Medial deviation	Webspace widening	Yes	Yes	Diminished	<50% subluxation
2	Moderate	Medial, lateral or dorsal deformity	Hyperextension	Yes	Yes	None	>50% subluxation
3	Severe	Dorsal deformity	Possible toe overlap and hammer toe	Yes	Yes	None	Dislocatable toe
4	Severe	Dislocation	Fixed hammer toe	Yes	Yes	None	Dislocated toe

From Doty and Coughlin (2014)

Metatarsophalangeal joint instability

Metatarsophalangeal joint instability is seen when the base of the proximal phalanx dorsally subluxes or dislocates from the metatarsal head (Maas et al, 2016). This can occur in an acute trauma setting but is not seen commonly. More frequently, there are chronic attritional changes to the stabilising soft tissue structures of the metatarsophalangeal joint which ultimately cause these structures to fail (Doty and Coughlin, 2014). The plantar plate and the collateral ligaments fail as these structures initially undergo elongation, then attenuation followed by rupture (Maas et al, 2016). Synovitis from conditions such as rheumatoid arthritis and other connective tissue disorders can contribute to this process. Other causative factors include wearing high-heeled shoes or shoes with narrow toe boxes (Doty and Coughlin, 2014). The theory regarding high-heeled shoes causing metatarsophalangeal instability is believed to be linked to the repetitive strain caused by hyperextension forces on the metatarsophalangeal joints (Nery et al, 2012). The patient may describe the pain as 'walking on marbles' and have particular discomfort in the toe off phase of the gait cycle. Walking barefoot may cause symptoms to become exacerbated, but the pain may subside with rest (Doty and Coughlin, 2014). The degree of metatarsophalangeal instability can be assessed using the 'drawer test' which involves holding the metatarsophalangeal joint in a neutral position and then assessing the amount of movement of the proximal phalanx in a dorsal and plantar direction (Doty and Coughlin, 2014; Nery et al, 2015). The outcome of the drawer test plus the amount of deformity and malalignment of the toe can be scored to stage the degree of the metatarsophalangeal instability demonstrated in Table 2 (Doty and Coughlin, 2014).

Weight-bearing radiographs can help to identify plantar plate injuries, particularly with a dislocated metatarsophalangeal joint. If there is an element of doubt in the diagnosis then magnetic resonance imaging or an ultrasound scan can be performed to confirm a suspected tear of the plantar plate or collateral ligaments (Hsu et al, 2018).

Freiberg's disease

Freiberg's disease describes avascular necrosis of the metatarsal head (Figure 2), most commonly seen in the second metatarsal (Cerrato, 2011; Trnka and Lara, 2019). This avascular necrosis alters the metatarsophalangeal joint, causing flattening and collapse of the metatarsal head and subsequently causing arthritis of the joint (Cerrato, 2011). The aetiology is believed to be multifactorial, with the main theories being trauma and vascular insufficiency. Trauma is thought to be caused by repetitive overload of the metatarsal head resulting in trabecular stress injury. Repetitive trauma could be postulated as a causative reason for this to occur in athletes. The second and third metatarsals are the longest and therefore the most likely to develop metatarsal overload. Insufficiency of the arterial supply to the metatarsal head is thought to be the result of anatomical variants. In these cases, the metatarsal heads are supplied by collaterals from the first and third metatarsals rather than

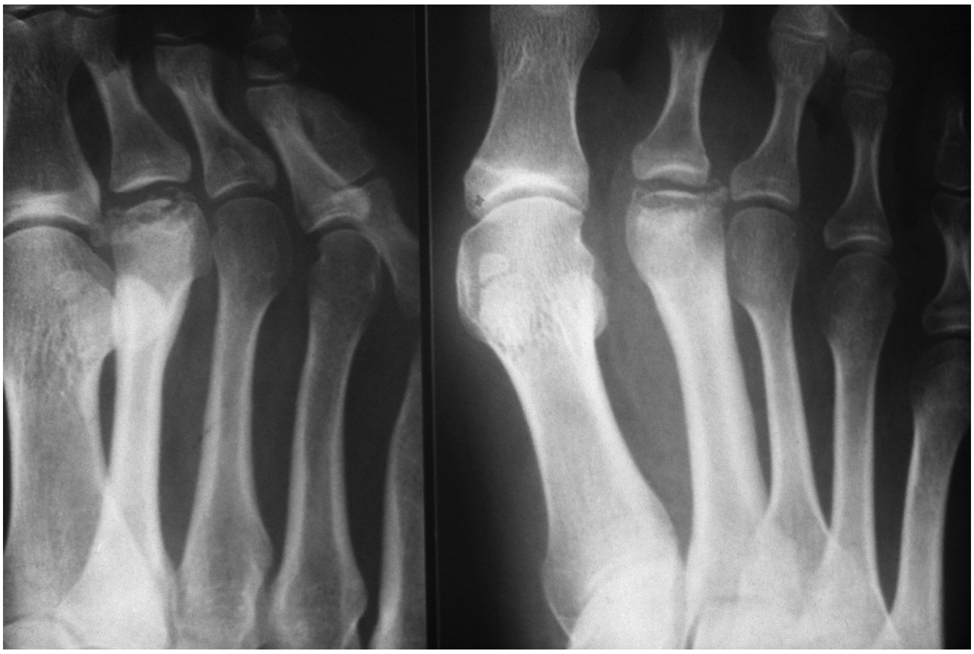

Figure 2. X-ray showing Freiberg's disease.

the dorsal and plantar metatarsal arteries. These variants are thought to be more susceptible to occlusion in conjunction with increased stress through the metatarsal head.

Several systemic conditions are potential risk factors for developing Freiberg's disease, including diabetes mellitus, systemic lupus erythematosus and hypercoagulability (Cerrato, 2011; Trnka and Lara, 2019; Wax and Leland, 2019). It has been postulated that repetitive dorsiflexion at the metatarsophalangeal joint from wearing high-heeled shoes could be a contributing factor. Evidence of Freiberg's disease may not be seen on plain weight-bearing radiographs in the early phase of the disease and is appreciated best on magnetic resonance imaging sequences (Wax and Leland, 2019).

Morton's neuroma

Morton's neuroma is fibrosis of the common digital nerves found in the intermetatarsal spaces (Kay and Bennett, 2003; Briggs, 2006; Valisena et al, 2018; Bhatia and Thomson, 2020) (Figure 3). There are several aetiological theories which include chronic traction damage, intermetatarsal bursitis, the deep transverse intermetatarsal ligament causing compression of the nerve, and ischaemia of the vasa nervorum (Valisena et al, 2018). There is evidence of neural oedema, axonal demyelination and fibrinoid degeneration histologically (Kay and Bennett, 2003; Bhatia and Thomson, 2020).

Clinically the patient will often describe a burning pain or paraesthesia in the forefoot. The pain is often worsened by walking, particularly barefoot or in high-heeled shoes. Some patients may describe night pain but usually the pain is resolved with removal of offending footwear and rest (Kay and Bennett, 2003; Briggs, 2006; Bhatia and Thomson, 2020). A Mulder's click test can be performed which involves the foot being dorsiflexed and then squeezing the metatarsal heads together: an audible click can be heard which is suggestive of Morton's neuroma (Briggs, 2006; Bhatia and Thomson, 2020). Morton's neuromas are most commonly found in the third webspace followed by the second webspace. This condition is most often seen in middle-aged women (Kay and Bennett, 2003). Ultrasound and magnetic resonance imaging can be both be used to help identify Morton's neuromas but clinical identification remains key (Briggs, 2006).

Systemic conditions

Rheumatological (rheumatoid arthritis and psoriatic arthritis) and neurological conditions (Charcot–Marie–Tooth disease) can contribute to a patient's metatarsalgia by their inflammation or deformities resulting in increased load of the metatarsal heads (Espinosa et al, 2010; Besse, 2017).

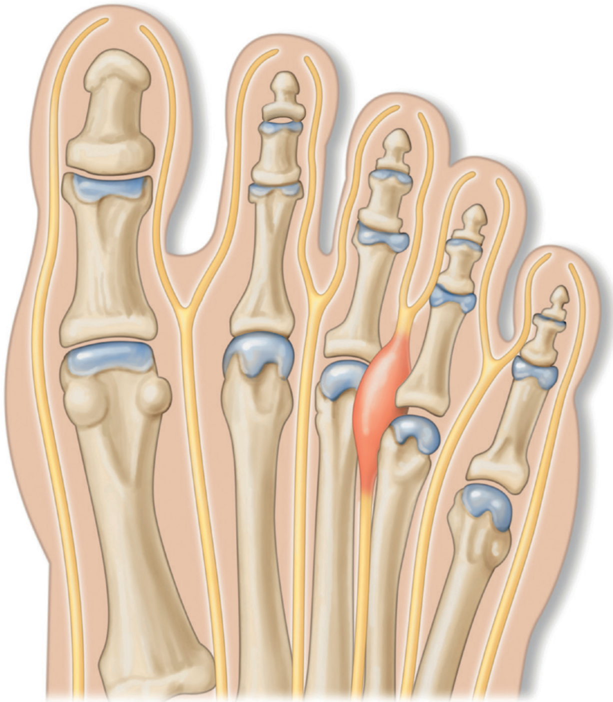

Figure 3. Morton's neuroma.

The autoimmune inflammatory process caused by rheumatoid arthritis affects the joint synovium, resulting in severe joint destruction which causes foot deformity. As well as the intra-articular effects of rheumatoid arthritis there are significant soft tissue complications, including synovitis, bursitis, tendonitis, fasciitis, vasculitis and neuritis. The combination of these intra- and extra-articular manifestations can cause the patient to develop metatarsalgia (Abdo and Iorio, 1994).

Charcot–Marie–Tooth disease is an inherited peripheral neuropathy which affects the upper and lower limbs. There is distal muscle wasting and weakness with sensory loss which develops slowly over time. The muscle weakness and imbalance lead to the patient developing foot deformities, including forefoot cavus, clawtoes, hindfoot varus and ankle instability. The alterations to the foot anatomy caused by Charcot–Marie–Tooth disease result in overloading of the metatarsal heads (Laurá et al, 2018).

Iatrogenic metatarsalgia

A patient can develop metatarsalgia following forefoot surgery; this is becoming more common as the volume of forefoot surgery increases. Hallux valgus corrective surgery can cause the first metatarsal to become shortened or excessively elevated. Subsequently, this can cause effective defunctioning of the first ray and load to be transmitted to the lesser metatarsals. Lesser metatarsal osteotomy can cause metatarsalgia at the operated ray or adjacent rays. This can be the result of excessive shortening, depression or elevation of the osteotomy or whether there is non- or malunion of the osteotomy. Restriction of extension at the metatarsophalangeal joint post-surgery can also result in symptoms (Espinosa et al, 2010; Besse, 2017; Chahal et al, 2020).

Management

Once sufficient clinical assessment and investigation has been performed then treatment can be considered (Figure 4). Conservative measures should be tried initially for up to 6 months before surgical intervention is considered, despite limited high level evidence to support conservative measures (Espinosa et al, 2008, 2010; Besse, 2017; Lopez and Slullitel, 2019; Chahal et al, 2020).

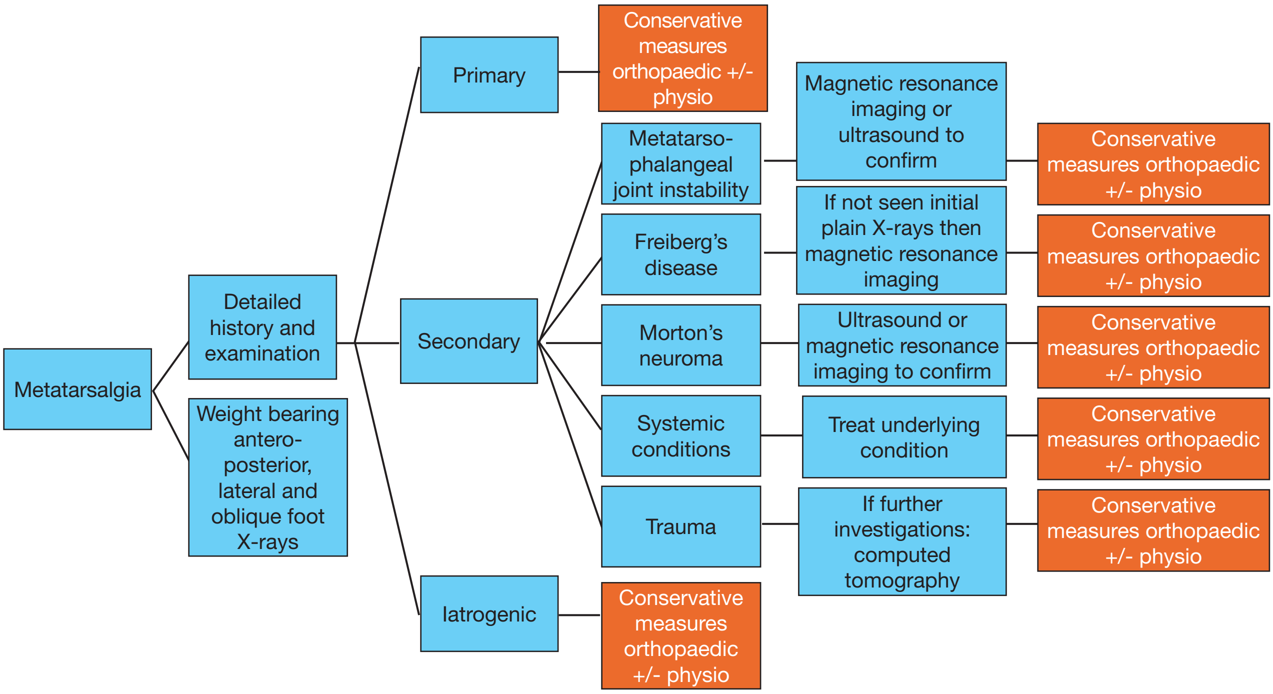

Figure 4. Initial management algorithm for metatarsalgia.

Conservative treatment

Although there is limited high level evidence to support it, a large number of patients will achieve sufficient results with conservative treatment. This involves explanation of their condition, simple footwear advice, stretching exercises for gastrocnemius–soleus tightness, footwear modifications including rocker bottom or broad toe box shoes. Debridement of plantar keratosis and specifically targeted and image-guided steroid injection therapy for conditions, such as Morton's neuroma and others, can also be performed as a diagnostic test as well as to provide the patient with symptomatic relief (Espinosa et al, 2008, 2010; Besse, 2017; Chahal et al, 2020).

Surgical treatment

If conservative management fails, surgery can be considered. Surgical management can be complex and require multiple procedures. The principle is to correct the offending pathology while improving the weight distribution across the metatarsals (Espinosa et al, 2010). Procedures can include soft tissue procedures, such as plantar plate repair, tendon transfers or lengthening, and excision of Morton's neuroma, or bony procedures involving metatarsal osteotomy (including shortening, lengthening, rotating, elevating and plantar flexing), fusions and condylectomy (Espinosa et al, 2010; Chahal et al, 2020). Decisions regarding surgical management should be made on an individual basis depending on the patient's symptoms, pathology and expectations.

Conclusions

Metatarsalgia has many different causative pathologies, and a detailed clinical history and examination is key to identifying the underlying cause. Treating metatarsalgia can be complex but conservative measures should be advised for at least 6 months before considering surgical intervention. When surgery is performed, it should aim to correct the pathology, alignment, weight-bearing axes and balance the load across the metatarsals evenly.

Author details

[1]Department of Trauma and Orthopaedics, East Lancashire Hospitals NHS Trust, Blackburn, UK

[2]Department of Radiology, East Lancashire Hospitals NHS Trust, Blackburn, UK

Key points

- Metatarsalgia is a symptom and not a diagnosis.
- Detailed and focused clinical history and examination can provide most of the information required to identify the potential cause of a patient's metatarsalgia.
- Iatrogenic metatarsalgia is increasing with large number of patients undergoing forefoot surgery, for example secondary to hallux valgus correction.
- Conservative measures should be tried for at least 6 months before consideration of operative intervention.
- When required surgical interventions should be considered on an individual basis.

Conflicts of interest
The authors declare that they have no conflicts of interest.

References

Abdo RV, Iorio LJ. Rheumatoid arthritis of the foot and ankle. J Am Acad Orthop Surg. 1994;2(6):326–333

Besse JL. Metatarsalgia. Orthop Traumatol Surg Res. 2017;103(1):S29–S39

Bhatia M, Thomson L. Morton's neuroma – current concepts review. J Clin Orthop Trauma. 2020;11(3):406–409. https://doi.org/10.1016/j.jcot.2020.03.024

Briggs PJ. Morton's neuroma. Br J Hosp Med. 2006;67(2):68–71. https://doi.org/10.12968/hmed.2006.67.2.20464

Cerrato RA. Freiberg's disease. Foot Ankle Clin. 2011;16(4):647–658. https://doi.org/10.1016/j.fcl.2011.08.008

Chahal GS, Davies MB, Blundell CM. Treating metatarsalgia: current concepts. Orthop Trauma. 2020;34(1):30–36. https://doi.org/10.1016/j.mporth.2019.11.005

Doty JF, Coughlin MJ. Metatarsophalangeal joint instability of the lesser toes. J Foot Ankle Surg. 2014;53(4):440–445. https://doi.org/10.1053/j.jfas.2013.03.005

Espinosa N, Maceira E, Myerson MS. Current concept review: metatarsalgia. Foot Ankle Int. 2008;29(8):871–879. https://doi.org/10.3113/FAI.2008.0000X

Espinosa N, Brodsky J, Maceira E. Metatarsalgia. J Am Acad Orthop Surg. 2010;18(8):474–485. https://doi.org/10.5435/00124635-201008000-00004

Hsu RY, Barg A, Nickisch F. Lesser metatarsophalangeal joint instability: advancements in plantar plate reconstruction. Foot Ankle Clin. 2018;23(1):127–143. https://doi.org/10.1016/j.fcl.2017.09.009

Kay D, Bennett GL. Morton's neuroma. Foot Ankle Clin. 2003;8(1):49–59. https://doi.org/10.1016/S1083-7515(03)00004-4

Laurá M, Singh D, Ramdharry G et al. Prevalence and orthopedic management of foot and ankle deformities in Charcot–Marie–Tooth disease. Muscle Nerve. 2018;57(2):255–259. https://doi.org/10.1002/mus.25724

Lopez V, Slullitel G. Metatarsalgia: assessment algorithm and decision making. Foot Ankle Clin. 2019;24(4):561–569. https://doi.org/10.1016/j.fcl.2019.08.006

Maas NMG, van der Grinten M, Bramer WM, Kleinrensink GJ. Metatarsophalangeal joint stability: a systematic review on the plantar plate of the lesser toes. J Foot Ankle Res. 2016;9(1). https://doi.org/10.1186/s13047-016-0165-2

Maceira E, Monteagudo M. Mechanical basis of metatarsalgia. Foot Ankle Clin. 2019;24(4):571–584. https://doi.org/10.1016/j.fcl.2019.08.008

Nery C, Coughlin MJ, Baumfeld D, Mann TS. Lesser metatarsophalangeal joint instability: Prospective evaluation and repair of plantar plate and capsular insufficiency. Foot Ankle Int. 2012;33(4):301–311. https://doi.org/10.3113/FAI.2012.0301

Nery C, Coughlin M, Baumfeld D et al. How to classify plantar plate injuries: parameters from history and physical examination. Rev Bras Ortop. 2015;50(6):720–728. https://doi.org/10.1016/j.rbo.2014.05.006

Trnka HJ, Lara JS. Freiberg's infraction: surgical options. Foot Ankle Clin. 2019;24(4):669–676. https://doi.org/10.1016/j.fcl.2019.08.004

Valisena S, Petri GJ, Ferrero A. Treatment of Morton's neuroma: a systematic review. Foot Ankle Surg. 2018;24(4):271–281. https://doi.org/10.1016/j.fas.2017.03.010

Wax A, Leland R. Freiberg disease and avascular necrosis of the metatarsal heads. Foot Ankle Clin. 2019;24(1):69–82. https://doi.org/10.1016/j.fcl.2018.11.003