The foot in systemic disease: management of the patient with rheumatoid arthritis or diabetes mellitus

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Abstract

The foot is commonly affected in systemic diseases such as rheumatoid arthritis and diabetes mellitus. Treating patients who suffer with foot pathology secondary to systemic diseases requires a multidisciplinary approach, following the principles outlined within this review. There is little high level evidence in this field, such as prospective controlled clinical trials, hence much of what we know and practise is based upon the expert opinion of key individuals in specialist centres, to whom we owe a great debt.

Keywords acquired; arthritis; diabetes mellitus; foot; foot deformities; foot ulcer; rheumatoid

Introduction

The foot is commonly affected in systemic diseases such as rheumatoid arthritis (RA) and diabetes mellitus, and musculoskeletal manifestations of these conditions frequently present to the orthopaedic surgeon. This article aims to provide the reader with a grounding in the principles of assessment and treatment of the rheumatoid or diabetic foot.

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Patients with severe systemic disease require a contemporary multidisciplinary approach to their management. An holistic assessment is always important; never more so than in these cases, where the patient's co-morbidities and other musculoskeletal ailments must be carefully considered. Excellent communication with the other healthcare professionals within the team is critical to the patient's care. These include physicians, vascular surgeons, podiatrists, orthotists, occupational therapists and physiotherapists.

Good decision-making begins with a thorough history and examination. It is important to establish the patient's capacity for activity and their reasonable physical aspirations, and to ask about exercise tolerance, such as stair climbing, so as to assess the patient's cardiovascular fitness. A detailed history of the patient's previous operations and interventions must be taken. A drug history is vital, as patients may well be on medications such as antiplatelet therapy, steroids or immune modifying drugs, which may have to be stopped or modified peri-operatively. Finally, the physical examination must be thorough, specifically assessing the patient's vascular and neurological status and the condition of their skin and soft tissues. Inevitably, such consultations will take longer than average, but time invested at the beginning of care results in improved decision-making subsequently. Managed well, these patients can have highly satisfying outcomes and are likely to be very grateful. Conversely, poor decision-making can be disastrous for the patient and difficult to salvage.

The foot in rheumatoid arthritis

Rheumatoid arthritis (RA) is a systemic polyarthropathy that affects small or larger joints in 1–2% of the population. It affects women more commonly than men and can occur at any age, but most commonly in the third to fifth decades. In our region, a ten-year prospective cohort study of 1154 patients newly diagnosed with RA showed that 27% went on to have orthopaedic surgery.[1] In the same study, 12% had major joint replacement within a mean of four years from diagnosis, which illustrates the aggressive nature of the disease. The diagnostic criteria was updated by the American College of Rheumatology (ACR; previously known as American Rheumatism Association) and European League Against Rheumatism (EULAR).[2] This requires confirmed synovitis of one joint or more, with absence of alternative explanation for the synovitis, and achieving a score of six or greater out of ten from domains including numbers and location of the involved joint(s), serological abnormality, elevated acute phase response and symptom duration. These criteria replaced the previous set from 1987, which were felt to lack sensitivity in early disease.[3]

The exact aetiology is unclear, but Rheumatoid Disease seems to be a T-cell mediated inflammatory response. Numerous intracellular messengers are implicated in the inflammatory cascade, but in particular the cytokines Interleukin-1 (IL-1) and Tumour Necrosis Factor-a (TNF-a) are highly important in the disease process. TNF-a increases chondrocyte secretion of matrix metalloproteinases, which break down extracellular matrix. Mononuclear cells are the primary cellular mediators of tissue destruction in RA. More recent evidence also demonstrates the influence of osteoclasts on joint destruction and synovitis.[4]

History

Poorly localized forefoot pain is a common early symptom of RA. In these early stages there may be synovitis and swelling that limits walking and footwear choices. The diagnosis at this point can be difficult to make and the surgeon should have a low threshold for further investigation. As the disease progresses, other larger joints may become involved and the patient may report deformities of the forefoot such as hallux valgus, hammer or claw toes and dislocation of the metatarsophalangeal joints (MTPJs). Midfoot and hindfoot deformities often occur later in the disease evolution and can become very severe. Overall, the lifetime risk of foot involvement in patients with RA is 90%. Most patients prioritize pain first, followed by deformity and difficulty fitting footwear. It is very important to establish the patient's main concern; it is also vital to ask about and examine the rest of the limb, as it is generally advisable to address proximal pain and deformity in the hip or knee prior to any foot and ankle deformity.

Examination

As with most orthopaedic examinations, simple detailed observation can reveal a wealth of information. Overall limb alignment with the patient standing and the knee exposed is essential, as they may have deformities of the knee from the RA. The patient's gait should be studied. Typically, the gait velocity is slower with reduced stride length. There may be longer double limb periods and prolonged heel contact to avoid loading a painful forefoot. Other compensatory actions may also become apparent. Assessing a patient's shoes for wear will also often give clues about abnormal gait patterns and contact areas as well as the suitability of the footwear. Hindfoot position and its relationship with the forefoot should be assessed as planovalgus deformity is common in RA. Double and single stance heel rise as well as examination in recumbency should be performed in assessing tibialis posterior tendon function. One should remember that inability to perform a single stance heel rise is not necessarily due to tibialis posterior tendon pathology; it can also be observed in patients with painful midfoot arthritis, for example, or it can simply be associated with the muscle weakness seen in older patients. One should assess the gastrocnemius −soleus complex for tightness, and if present, then further assess by the use of the Silfverskiold test to differentiate which part of the complex is tight. The tibialis posterior tendon should be carefully palpated along its course for tenderness and swelling suggestive of tenosynovitis and an increased risk of tendon rupture. The ankle and subtalar joints should be examined for range of movement and evidence of synovitis or instability. Gentle passive pronation and supination of the midfoot in conjunction with palpation of the midfoot joints is sensitive for identifying synovitis here. One needs to be more delicate than usual when examining these joints, as if there is an acute flare of the RA then they will be very sore. In the forefoot there may be severe hallux valgus deformity and dorsal subluxation or dislocation of the lesser toes at the MTPJ level, with consequent metatarsalgia. In earlier disease there may be synovitis without established deformity, evidenced by swelling and pain on palpation, translation and the grind test.

Attention must be focussed on any skin pressure areas for threat of ulceration. The neurological and vascular status of the lower limb should always be documented. If ulcers are established, it is useful to photograph and document them, so as to allow for the assessment of size, character and subsequent response to treatment. A useful adjunct for neurological examination is a 5.07 Semmes-Weinstein monofilament, which applies a standardized 10 g of pressure to the skin. When sensation to this is lost, there is risk of neuropathic ulceration. Neuropathy in RA can be peripheral due to vasculitis, compressive from radiculopathy due to lumbar spine disease or, most worryingly, due to cervical myelopathy as a result of atlantoaxial instability. This diagnosis must not be missed, and when present it requires urgent investigation and onward referral.

Investigations

If the diagnosis of RA has not already been made then plain radiographs of the hands and feet may show characteristic periarticular osteoporosis and erosions. Weight-bearing plain X-rays are also useful for monitoring disease progression over time. In the very early stages MRI is useful for detecting synovitis and effusions. Laboratory tests should include full blood count, erythrocyte sedimentation rate and C-reactive protein. Rheumatoid factor is positive in about 80% of patients with RA. Antinuclear antibody may be positive in Juvenile RA. The differential diagnosis is often with other sero-negative arthropathies, such as Systemic Lupus Erythematosus, Reiter's Syndrome and psoriatic arthritis. The patient should be referred to a rheumatologist if newly diagnosed with an inflammatory arthropathy, where more detailed immune and genetic profiling can be performed as necessary.

Medical therapy

Medical treatment of RA has advanced dramatically over the past two decades. A recent UK study found a shift in treatment strategy from sequential monotherapy to the use of disease modifying anti-rheumatic drugs (DMARDs) as add-on therapy, triple combination or in conjunction with anti-TNF therapy.[5] A decline was also found in the cumulative incidence of intermediate-type surgery, such as operations performed on the hand, wrist and forefoot over a 25-year period, whereas a similar trend was not observed in major orthopaedic surgery such as hip or knee replacement.

First-line treatment now involves non-biological therapy with methotrexate, a cytotoxic drug. Its usage has increased from 1% of patients in the late 1980s to 70% of patients in 2006 to 2011.[5] This is given weekly, orally, and is generally well tolerated. Intra-articular and systemic steroids may also be given initially, and the aim is to aid control of the disease while the slower DMARDs take effect. Steroid therapy can then be reduced over time with the aim that no patient should remain on steroids for longer than two years. Bone protection in the form of calcium supplementation and bisphosphonates are also used in combination with systemic steroid therapy to prevent osteoporosis. Disease activity is measured with validated scoring systems such as the DAS28. According to the National Rheumatoid Arthritis Society (NRAS), a 'treat to target' principle is currently in use to monitor treatment efficacy, with a score of 2.6 and 3.2 indicating remission and low disease activity respectively. If a patient scores equal or greater than 5.1 on two occasions with the DAS28, despite having undergone trials of two DMARDs, then biological treatment can

be started with anti-TNF drugs such as Infliximab and Etanercept. These drugs are highly effective in 60–70% of patients and can induce remission within a few weeks, sparing patients further joint damage.

Publications from the National Institute for Health and Care Excellence found that incremental cost-effectiveness ratios range from £14,132 to £23,821 per Quality-Adjusted Life Year (QALY) when using biological treatment in conjunction with methotrexate for RA patients who previously had inadequate response to conventional DMARD treatment. If anti-TNF therapy is not successful, other biological agents that block inflammatory cytokines may be tried, such as Rituximab and Abatacept. Current advice is that methotrexate may be continued in patients undergoing surgery, whilst anti-TNF therapy should be discontinued 2–4 weeks prior to surgery, due to an increased risk of both minor and major infection.

Despite such advances in medical therapies, the need for orthopaedic intervention is still substantial.[5] Many patients have a long disease history and already have established destructive arthritis at presentation. In addition, many newly diagnosed patients will not respond to DMARDs or may find the side effects intolerable. Steroids lose potency with time, and generally patients relapse if DMARDs are discontinued. It is therefore vital that orthopaedic surgeons keep abreast of medical developments and understand the point at which surgical treatment should be considered in such patients.

Non-operative treatment

Most patients with RA in an orthopaedic clinic will have exhausted conservative treatments. It is, however, vital to enquire about and consider the use of orthotics as this can be a very useful adjunct. Steroid injections can be effective in joints with active synovitis. Larger joint such as the ankle can be injected in the clinic, whilst smaller joints or tendon sheath steroid injections may be better targeted under X-ray or ultrasound control respectively. Local anaesthetic and steroid injections can be useful both diagnostically and therapeutically. A targeted injection that palliates symptoms for three months, whilst disappointing for the patient, is an extremely useful diagnostic tool that helps isolate which joint causes the pain in a group of patients in whom many joints may be diseased.

Surgical treatment

Surgery is often required to alleviate pain and correct deformity. The aim is to create a painless, stable, plantigrade foot. We shall consider the forefoot, midfoot and hindfoot and ankle in turn.

Forefoot reconstruction: careful pre-operative planning must take account of the severity of the deformities and their correctability, as well as the condition of the articular surfaces. One should remember that assessment of the hindfoot and midfoot is important and concurrent excessive hidfoot valgus or varus must be assessed and addressed with orthoses or surgery if planned forefoot intervention is to be successful. Gastrocnemius tightness also contributes to increased forefoot loading and can be addressed with physiotherapy or a gastrocnemius slide. Achilles tendon lengthening is occasionally needed if soleus is also contracted. Whilst we discuss procedures separately, it is very common to combine first ray procedures with lesser metatarsal osteotomies and lesser toe correction as a whole forefoot correction to achieve an optimum result. Coughlin showed highly satisfactory results in 47 feet with severe hallux valgus deformity secondary to RA treated with 1st MTPJ arthrodesis.[6] Additional forefoot reconstruction was carried out with excision of the lesser metatarsal heads for metatarsalgia and proximal interphalangeal joint (PIPJ) fusion for hammer toes. Excellent or good results were reported for 45 out of the 47 feet. In all feet, the hallux took part in weight bearing on Harris-mat pressure studies post-operatively. This type of forefoot reconstruction has traditionally been considered the gold standard in RA.

Hallux valgus and 1st MTPJ arthritis – whilst hallux valgus is common in the general population, a number of factors make it much more prevalent in RA. Synovitis of the first MTPJ causes ligamentous laxity and subsequent instability. Inactivity and pain, with or without peripheral neuropathy, causes wasting of the intrinsic muscles of the foot, which may contribute to widening of the forefoot. Subtalar disease often results in excessive pronation, which in turn loads the medial column and drives the hallux into valgus. The subsequent malalignment of the flexor and extensor tendons, which sublux laterally, tends to exacerbate the deformity and accelerate progression. Conventional hallux valgus re-alignment procedures can be used in patients with mild to moderate hallux valgus in the presence of well-preserved articular surfaces and relatively inactive disease. However, in the absence of well controlled disease, with clinical evidence of synovitis, or if there is radiologically advanced joint damage, then arthrodesis of the first MTPJ is generally favoured. Attention must also be focused on any concurrent metatarsalgia, and this should be addressed at the same time. Post-operatively, the patients may weight bear in a rigid post-operative shoe. Barouk recently reported good clinical results in a series of 55 feet using a Scarf osteotomy for hallux valgus in the rheumatoid foot combined with Weil's osteotomies for the lesser metatarsals.[7] Good correction was maintained at two years in 95% of feet and only one patient required revision to arthrodesis. However, given the chronic nature of RA, longer follow-up is needed to validate joint preserving surgery, which is known to be less successful in RA patients as they are still prone to ligamentous and capsular destruction, leading to recurrence.

Severe hallux valgus or hallux valgus associated with secondary joint damage or significant bone loss is best treated by arthrodesis. The joint surfaces are prepared and osteophytes resected. The bone quality is often very poor. The aim is to fuse the 1st MTPJ in 10–15° of valgus and 10° of dorsiflexion from plantigrade (20–30° dorsiflexion relative to the 1st metatarsal). Rather than focussing on exact measurements, it is most helpful to simulate loading of the foot with a flat surface intra-operatively, and the hallux pulp should just make contact with the surface. Either a single oblique lag screw and dorsal low profile plate or crossed lag screws are used for compressive, stable fixation. If there is significant bone loss, cancellous bone graft from the proximal tibia can be used to augment the fusion, or if a more structural graft is required one may use tri-cortical bone from the iliac crest and a small foot locking plate for enhanced stability.

Other 1st MTPJ procedures – The Keller excision arthroplasty has been used extensively in the past in patients with RA.

However, it may be associated with higher rates of transfer metatarsalgia.[8] We have used the Keller procedure as a salvage operation in the rare cases of failed 1st MTPJ arthrodesis, with reasonable outcomes. Arthroplasty of the 1st MTPJ remains an attractive proposition, particularly as it is so successful in other joints affected by rheumatoid arthritis. Silastic joints have the advantage of being constrained; however, the results have generally been poor due to silicone-induced synovitis in up to 72% of cases, cystic osteolysis and recurrence of pain.[9] Currently, there is insufficient evidence to recommend arthroplasty of the 1st MTPJ in these patients. Arthrodesis of the 1st MTPJ remains the gold standard in this patient population.

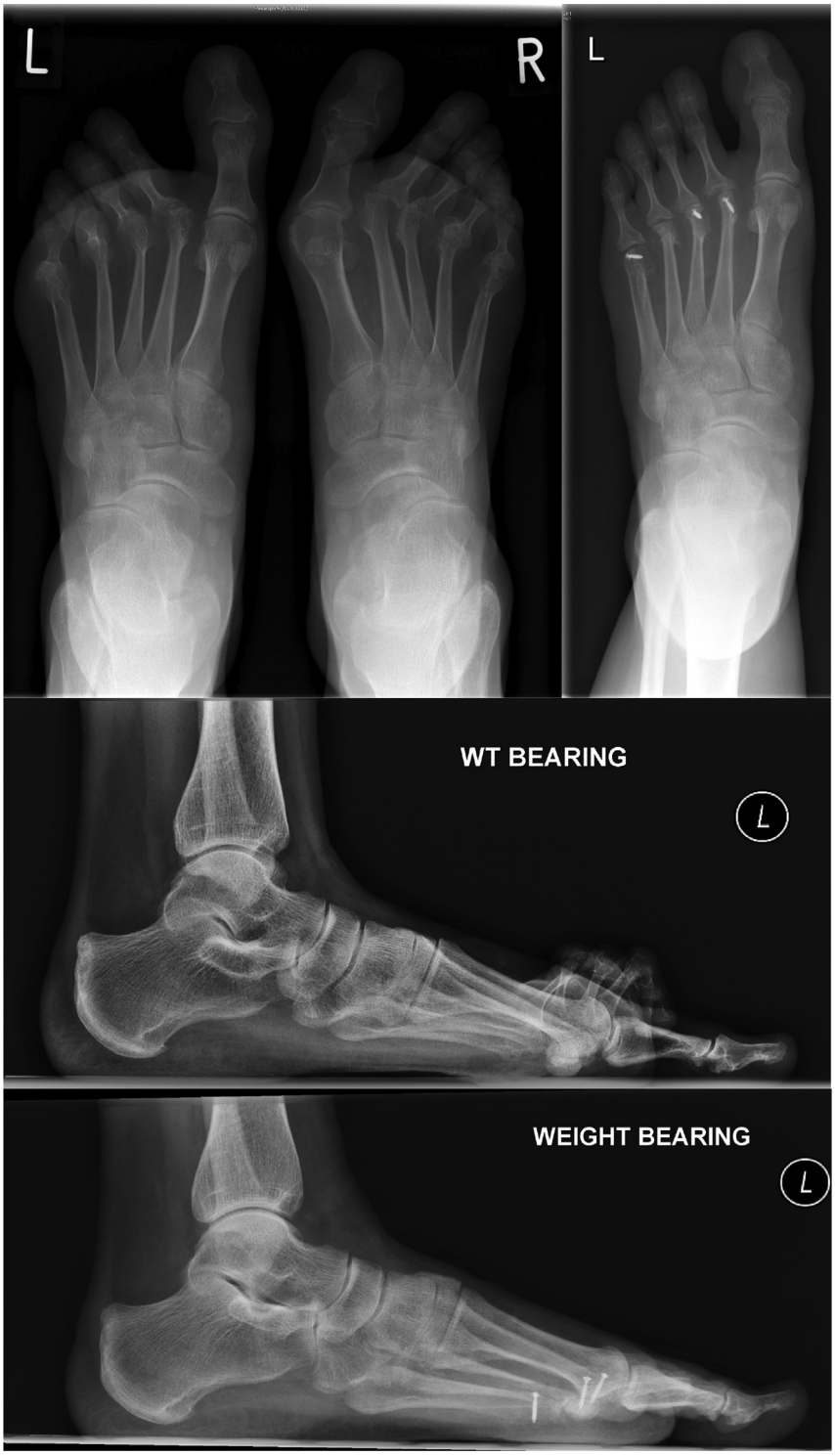
Metatarsalgia and MTPJ dislocation − a combination of synovitis and subsequent ligamentous laxity with or without rupture of the plantar plate pre-disposes the MTPJs to dorsal subluxation and then dislocation. This is combined with relative weakness of the intrinsics compared to the long flexors and extensors. As the MTPJ subluxes dorsally, the interossei and lumbricals, which act together to flex the MTPJ and extend the PIPJ, lose mechanical advantage. This perpetuates the problem and can rapidly turn a correctable deformity into a fixed one. The lesser toes play little part in load bearing and propulsive gate once the MTPJs are subluxed. In addition, dorsiflexion of the toes causes distal migration of the fat pad. Both factors pre-dispose patients to increased pressure under the exposed metatarsal heads, resulting in metatarsalgia with callosities on the plantar surface. In the normal foot, the proximal metatarsals form a transverse arch, but distally should come to lie in a horizontal plain. On an AP radiograph the metatarsals should form a parabolic cascade, with the second metatarsal longer than the first and subsequent metatarsals being progressively shorter than the second. The aim of surgery is two-fold. The first is to correct any abnormality of the normal metatarsal cascade and offload the symptomatic metatarsal heads. The second aim is to reduce the dislocated MTPJs, and in so doing bring the lesser toes back into weight bearing, therefore sharing load and reducing pressure under the metatarsal heads.

When the lesser MTPJ anatomy is reasonably well preserved, we favour the Weil osteotomy. This osteotomy is intra-capsular, with the cut parallel to the ground, allowing the metatarsal head to slide proximally. The osteotomy is fixed with a small twist-off screw and any overhanging dorsal bone spur is excised. This shortens the metatarsal and decompresses the MTPJ. In combination with extensor tendon lengthening and PIPJ fusion, the dislocated MTPJs can often be reduced. A good example of this type of reconstruction is demonstrated by Clinical Case 1. Bolland reported the results of forefoot reconstruction in 26 feet, combining 1st MTPJ fusion with Weil's metatarsal osteotomies.[10] Excellent or good results were achieved in 88% of cases, with a mean modified AOFAS score of 72/100 (34−90). The first MTPJ arthrodesis union rate was 92% at 26.2 months. There was a 12% rate of recurrent metatarsalgia or callosities. Similarly, Bhavikatti et al. reported a series of 49 patients who had Scarf osteotomy of the first metatarsal and Weil's osteotomy for the lesser metatarsals, with an improvement in mean AOFAS score from 39.8 pre-operatively to 88.7 at final follow-up.[11]

The Weil's osteotomy is not without complications. Scar contracture, stiffness and elevation of the toe are fairly common. Long-term stiffness occurs in as many as 20%.[12] There can also be avascular necrosis and recurrence of the deformity. Despite these potential problems, however, the Weil osteotomy remains a useful technique. In severe, destructive arthritis, when the metatarsal head is too damaged to be preserved, excision arthroplasty, such as described by Coughlin, is used.[6]

Lesser toe deformity − Hammer toe refers to hyperextension at the MTPJ, flexion at the PIPJ and extension at the DIPJ. In clawed toes there is hyperextension at the MTPJ and flexion at both the IPJs. These two deformities are common in RA and cause painful callosities over the dorsum of the PIPJs due to rubbing from shoes. If associated with moderate or severe hallux valgus, the toes may cross over or under the hallux, resulting in a painful deformity and significant problems with shoe fitting. Occasionally, lesser toe deformities are addressed individually but it is far more common that they are addressed as part of a more extensive forefoot correction. If shortening of the lesser metatarsal is also undertaken, this may correct a flexible toe deformity adequately. If there is residual fixed deformity then osteoclasis, where the joint and bones are manipulated straight, may adequately complete the correction, or alternatively PIPJ fusion may be considered. When undertaking PIPJ fusion, we use a transverse elliptical incision centred over the dorsal aspect of the PIPJ. The extensor tendon is divided and the joint is opened. The collateral ligaments are released and a fine saw blade is used to remove the subchondral bone. The remaining joint surface is debrided and prepared, with shortening as required, and the PIPJ is then reduced and stabilized with a 1.6 mm K-wire, which is removed in clinic at 4−6 weeks.

Hindfoot and midfoot reconstruction: changes in the hindfoot and midfoot tend to occur with longer duration of disease and therefore present later. They are involved in approximately two-thirds of patients with chronic RA. The main driver of deformity appears to be a combination of subtalar synovitis, causing ligamentous disruption (especially the spring ligament), in conjunction with dysfunction of the tibialis posterior tendon. There is pronation at the subtalar joint with hindfoot valgus and loss of the medial arch. Synovitis of the naviculocuneiform or tarsometatarsal joints exacerbates the problem and can lead to a prominence of the medial midfoot bones. In severe cases there can be complete lateral dislocation through the transverse tarsal joint, with callosity over the exposed head of the talus. Severe deformities can be challenging to treat, with a high potential risk of serious complications. Operations need to be a la carte, and may combine forefoot correction at the same sitting. Important considerations when correcting severe hindfoot deformities include proximal limb alignment or deformity, the relative presence or absence of ankle arthritis or deformity, and the condition of and tension in the soft tissues. The aim of surgery is to bring the heel back under the tibiotalar joint and to restore the medial arch. It is also important to restore the shape of the foot to facilitate shoe fitting. Finally, it is important to explain to patients that there is a significant investment of time and energy with these procedures, as the total time spent in a cast or protective boot can be up to 12−16 weeks, and full recovery can take up to 18 months or so. With careful planning and surgery good results can be achieved. Gougoulias and Lampridis recently reported a series of 30 patients who underwent midfoot arthrodesis with a 93% union rate, and 90% of patients reported their outcome as good or excellent.[13]



Case 1 This young female presented with severe forefoot deformity secondary to rheumatoid arthritis, sparing the left 1st MTP joint. Surgical correction of the left forefoot involved extensive soft tissue release, Z-lengthening of all extensor tendons, Weil's osteotomies and PIP joint fusions, to achieve a very satisfactory correction. This is best appreciated on the lateral radiograph, where it can also be seen that the plantar fat pad has been restored under the metatarsal heads on these weight-bearing films. A similar correction including a 1st metatarsal scarf osteotomy is planned for the right side.

Tibialis posterior dysfunction: in Stage 1 tibialis posterior dysfunction there is pain along the course of the tendon and there may be some weakness but a single stance heel raise is usually possible. In RA this dysfunction is caused by active tenosynovitis, which can be treated with open or arthroscopic decompression of the tendon and synovectomy followed by orthotic management. Surgery usually improves the patient's symptoms and can prevent tendon rupture, but patients rarely

present to an Orthopaedic Surgeon at this early stage. More commonly, we see patients with Stage 2 or 3 disease, which represents a spectrum through weakness of the tendon and a correctable valgus hindfoot to fixed hindfoot valgus with abduction of the transverse tarsal joint and Achilles tendon contracture. If patients fail six months of orthotic management, we then consider surgical correction. If the hindfoot and forefoot deformity is still passively correctable then generally the deformity can be treated without fusing joints. First, we perform a medializing calcaneal osteotomy, usually using a minimally invasive burr, with the patient in the lateral position. The osteotomy is fixed with one or two large cannulated screws. Then, with the patient supine we perform tendon reconstruction with a flexor digitorum longus transfer (FDL). The pathological posterior tibial tendon is excised and the spring ligament repaired. FDL is harvested at the knot of Henry and passed plantar to dorsal through a 5 mm drill hole in the navicular tuberosity. Long-term results for FDL transfer and calcaneal osteotomy for Stage 2 disease have been reported by Chadwick et al.[14] They showed that 87% of patients were pain-free with good function at a mean follow-up of 15.2 years, albeit not in an exclusively RA population.

If the hindfoot valgus is fixed but the forefoot varus is less than 10° and there is no abduction of the transverse tarsal joints, then an isolated subtalar arthrodesis with correction of the heel position may be sufficient. If, however, there is also abduction at the transverse tarsal joints, then a triple arthrodesis (i.e. subtalar, talonavicular and calcaneocuboid joint arthrodesis) is required. Knupp et al. published long-term follow up of 24 triple arthrodesis procedures for RA.[15] There was satisfactory fusion in all cases and 100% patient satisfaction; however, in 15 out of 24 feet there was advancement of midfoot arthritis and in ten cases there was progressive ankle arthritis.

Ankle arthritis: if there is ankle arthritis and deformity at this joint in conjunction with subtalar arthritis, a pantalar fusion is required. A curved lateral incision between the superficial peroneal and sural nerves is used to access the ankle and subtalar joints ± the calcaneocuboid joint. The distal fibula is resected, and cancellous bone is harvested from it to augment the fusion. Fibula resection significantly reduces the tension in the contracted lateral soft tissue following correction of the deformity, facilitating wound closure. An additional anteromedial incision is required if the talonavicular joint is to be included in the fusion. A locked tibiotalocalcaneal nail augmented by additional cannulated screw fixation of the triple joint complex is preferred over lateral plate fixation, as it is difficult for patients with RA to be fully non-weight bearing, and therefore the more stable nail construct is favoured. Clinical Case 2 demonstrates a severe hindfoot and ankle deformity correction.

Ankle replacement is an attractive option where the range of movement of the tibiotalar joint can be preserved. Previous studies on first generation total ankle replacement (TAR) reported high complication rates.[16–18] However, more recent studies found good outcomes at medium term follow-up.[19–21] Currently, the 3rd generation TARs utilize uncemented fixation into bone with mobile bearing polyethylene inserts. Wood et al. reported a series of 100 modern TARs and found comparable results to other total joint replacement.[22] Similar to other joint replacement procedures, complications including problems with wound healing, infection and osteolysis were also encountered in TAR. A systematic review and meta-analysis by Zaidi et al. drew similar conclusion from 7942 TARs in 58 studies, with a survivorship of 89% at 10 years, and the AOFAS score improved from 40 to 80, although the authors caution against the quality of studies contributing to the current evidence.[23]

The diabetic foot

Diabetes mellitus is a syndrome of chronic hyperglycaemia caused by insulin deficiency or resistance. It affects well over 100 million people worldwide and the incidence of type 2 diabetes is rising in parallel with the increase in obesity. The World Health Organisation predicts a diabetes epidemic, with 300 million people likely to suffer from the disease by 2025. Diabetes causes damage to numerous organs and body systems. A number of mechanisms contribute to organ damage in diabetes, including glycosylation of proteins in peripheral nerves, activation of the pyolol pathway and disruption of the microvascular circulation. The two main diabetic problems presenting to orthopaedic surgeons are ulcerations and Charcot neuroarthropathy. Often the two may co-exist, with Charcot deformity posing a major ongoing ulceration risk.

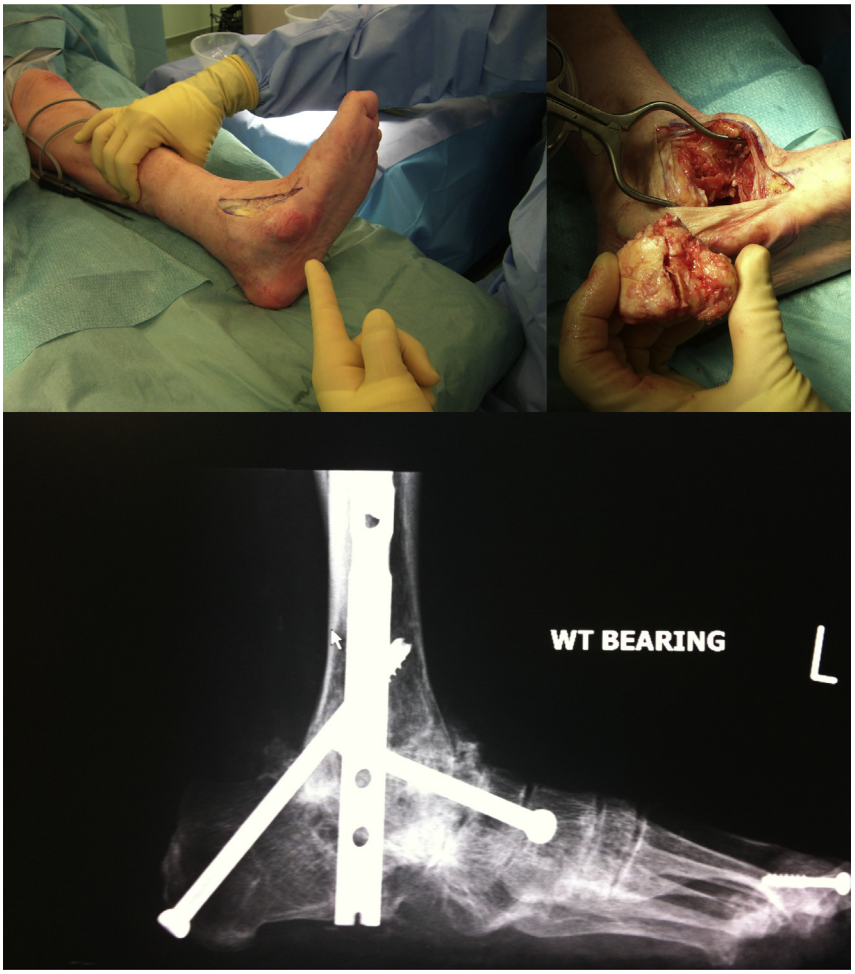
History

Much of orthopaedic practice focuses on relieving pain, whereas in diabetic patients the main aim is to reduce risk to the limb. A general medical history is required, and direct questions should be asked to establish the degree of end-organ damage, such as retinopathy, nephropathy and ischaemic heart disease. It is important to ask how long ulcers have been present and whether patients have already been hospitalized with diabetic foot complications. The aim is to develop an idea of the degree of risk posed by a patient's foot problems and the available treatment options.

Examination

Careful attention must be paid to footwear, which can often be a cause of problems in the diabetic patient with neuropathy. Neurological assessment should include recording sensation to either a 10 or 4.5 g Semmes-Weinstein monofilament. Saltzman showed that loss of sensation to a 4.5 g Semmes-Weinstein monofilament in a single point under the first metatarsal is sensitive in predicting risk of ulceration.[24] Neuropathic ulcers can be very deep, and if painless can be gently probed in clinic. Absent ankle reflexes are an early sign of neuropathy and correspond to increased risk of ulceration.[25]

Peripheral pulses should be palpated and if absent or reduced, ankle brachial pressure indices (ABPI) should be recorded. An ABPI from 0.8 to 1.0 implies no significant ischaemia. An ABPI between 0.5 and 0.8 warrants further investigation with duplex ultrasonography and onward referral to a vascular surgeon. An ABPI of less than 0.5 implies critical ischaemia and should trigger an urgent vascular opinion. Calcification of the vessels is common in diabetes and may give a falsely normal or high reading.



Case 2 This patient with rheumatoid arthritis presented with a severe planovalgus foot with dislocation through the talonavicular joint. The surgeon is pointing to the talar head, which is completely dislocated, and the patient had been walking on this prominence. The foot had effectively dislocated laterally through this level. Removal of most of the talus and the distal fibula was necessary, to bring the foot back underneath the leg. This excised bone was then used as autograft as part of a tibiocalcaneal fusion, including a tibionavicular fusion.

An ABPI of above 1.3 implies incompressible calcified vessels. The clinician must be aware of other signs of ischaemia in this situation and look for reduced skin temperature, sluggish capillary refill and absent leg hair.

Investigations

The main challenge in the diabetic foot is to distinguish neuroarthropathy from infection. This is particularly important if there is an ulcer overlying the affected bone or joint. Plain X-rays are useful to monitor progress. Typically, osteopaenia is more generalized in Charcot joints and more localized in osteomyelitis. MRI can be useful for detecting abscesses but will not generally prove reliable in distinguishing aseptic neuroarthropathy from osteomyelitis. Scintigraphy with Technetium-99 has a 91% sensitivity for osteomyelitis but only 54% specificity.[26] Schauwecker et al. compared scans with Indium-111 labelled white cells, technetium-99 and gallium-67 in 57 patients with Charcot joints and osteomyelitis.[27] Indium labelled white cells were 100% sensitive in detecting acute osteomyelitis and 60% sensitive in chronic osteomyelitis, with a specificity of 96%. Whilst labelled white cells are more specific for infection, during the acute stages of a Charcot joint there is inflammation in response to micro-fractures, which can result in false positive scans. The gold-standard investigation to diagnose osteomyelitis is bone biopsy and culture but this is not often practical. All infected wounds should be swabbed and swab results discussed with a microbiologist to target appropriate antibiotic therapy.

Good diabetic control is vital when treating wounds and should be monitored by measuring glycosylated haemoglobin (HbA1C). Dyslipidaemia must be treated and it is often prudent to involve a dietician in the patient's care early in order to optimize healing potential. Wukich et al. showed in a Level 1 study that the risk of surgical site infection was 7.25 times higher in patients with complicated diabetes versus non-diabetic controls, but the risk in patients with controlled diabetes was not significantly higher than controls. Peripheral neuropathy and a HbA1C of greater than 8% were independently associated with surgical site infection.[28]

The diabetic ulcer

Approximately 3–4% of the diabetic population have foot ulcers or deep infection, and 15% of diabetics will have foot ulcers in

their lifetime, neuropathy being the main risk factor. Diabetic foot ulcers precede 85% of non-traumatic lower limb amputations. Ulceration is a serious event, associated with a 5-year risk of mortality of around 40%.[29] Increased age, male gender, peripheral vascular disease and renal disease are associated with a higher risk of death.

Aetiology of the diabetic ulcer: sensory neuropathy is the most common cause of ulceration in the diabetic foot due to the loss of normal protective mechanisms. Neuropathy affects 50% of patients who have had diabetes for 10 years. The ulcer may start due to acute trauma or as a result of repetitive micro-trauma, such as from ill-fitting footwear. Plantar ulceration is most often found over bony prominences and reflects intermittent excessive pressure with neuropathy. Dorsal ulceration is almost always caused by constant pressure from poorly fitting shoes.

Autonomic neuropathy is common in diabetics and results in a dry, scaly and often swollen foot. If the skin cracks, bacteria may enter the soft tissues. Nail growth is abnormal in autonomic neuropathy and can pre-dispose to ingrowing toenails and paronychia.

Motor neuropathy in diabetic patients can cause intrinsic muscle weakness and subsequent imbalance between these and the extrinsic muscles of the foot, which predisposes to hallux valgus and lesser toe deformities. Hammer and claw toe deformities increase plantar pressure under the metatarsal heads, pre-disposing to ulceration. A combination of motor and sensory neuropathy may also result in subtle abnormalities of gait, which increases pressure on bony prominences such as the medial sesamoid of the 1st MTPJ. Diabetic patients are pre-disposed to contracture in the gastrocnemius–soleus complex. This limits dorsiflexion at the ankle and increases plantar pressure in the forefoot during standing and walking, predisposing to plantar ulcers. Contracture of the gastrocnemius–soleus complex should be clinically assessed using the Silfverskiold test.

Atherosclerosis is more common in patients with diabetes and tends to be more localized to the infra-popliteal vessels in diabetics compared to the general population. Ischaemia on its own rarely causes ulcers; however, in combination with other factors such as neuropathy and pressure it can increase the risk of diabetic ulceration by a factor of nine.[30] Ischaemia also significantly impairs healing of established ulcers and creates a relatively anaerobic environment in which infection can thrive.

Immune deficiency is also a feature of diabetes. Glycosylation of immune proteins inhibits their function and neutrophil performance is impaired. The result can be infection with micro-organisms that would not usually be pathogenic in a healthy person.

Deformity of any type in the diabetic foot may lead to increased pressure areas whilst weight bearing or wearing shoes. While many orthopaedic surgeons may be understandably cautious about operating on foot deformities in diabetic patients, such procedures may in fact be necessary to prevent future complications due to ulceration.

Classification of diabetic ulcers: The Wagner classification was developed in the 1970s by Meggitt and Wagner, and is widely used to describe diabetic ulcers (Table 1).[31] The Wagner classification has been adapted to separate out the depth of the ulcer from the degree of ischaemia. This avoids confusion in certain areas, for example where there is limb threatening ischaemia but only a shallow ulcer. The depth-ischaemia classification proposed by Brodsky is a little more complex but perhaps more useful clinically.[32] Brodsky also suggested treatments, which correspond to the various grades of both ulcer depth and ischaemia (Table 2). A further refinement of Brodsky's depth-ischaemia classification has been developed by The Diabetic Foot Center at the University of Texas San Antonio.[33] They included the presence or absence of infection and showed greater association with risk of amputation and likelihood of ulcer healing than the original Wagner classification.

Treatment of diabetic ulcers: patients with Grade 0 ulcers (or more accurately the patient at risk of ulceration) must be carefully advised and followed up. A podiatrist or chiropodist should look after the patient's feet on a regular basis to take care of nail cutting and pad pressure areas, and to advise on footwear. Patients should be advised to keep their feet warm and not to smoke.

Grade 1 and 2 plantar ulcers can largely be treated with local debridement, either in clinic or in theatre, and total contact casting with antibiotic therapy in the case of superficial infection. Casting relieves pressure areas and shares up to 30% of the patient's weight. Total contact casts should avoid excessive padding and should incorporate the toes, preventing excessive dorsiflexion of the MTPJs. Bony prominences should be padded with felt or foam. The first cast should be changed at a week, as swelling subsides. Subsequent cast changes may occur every two weeks. Overall, healing rates are reportedly between 70% and 100%. Frigg et al. showed primary healing rates of 85% in an average of 4.2 months with total contact casting for Grade 1 and 2 ulcers.[34] However, 56% of patients suffered recurrent ulcers after healing, despite custom-made orthopaedic shoes and foot care advice. In approximately a third of recurrent ulcers, significant deformity was a contributor and these patients underwent corrective surgery. Nearly two-thirds of recurrences could be treated again with total contact casting. In addition to recurrence, 14% of patients acquired new ulcers from poorly fitting casts. This is the main complication of total contact casting but can largely be treated by changing the cast frequently and meticulous wound care.

An alternative to a total contact cast is a pneumatic walking boot. Faglia et al. recently reported similar efficacy to total contact casting; however, as such devices are removable compliance can be an issue.[35] A number of studies have, however, shown greater success and more rapid healing with total contact casts versus off the shelf devices. For this reason, total contact casting was still recommended over and above removable pneumatic boots in a recent review concerning offloading the diabetic foot.[36] We discuss both options with patients and make decisions on a case by case basis. Significant deformity is a relative contra-indication to offloading with a pneumatic walking boot.

Gentle probing of a wound can detect Grade 3 ulcers if bone is felt. If there is significant deep infection the patient should be admitted for intravenous antibiotics. The aim is to down-grade the ulcer with surgical debridement and adequate antibiotic therapy, at which point total contact casting may then be used to promote healing. If the ulcer is particularly resistant to treatment,

The Wagner classification of diabetic ulcers

Grade 0	Skin intact, erythema, at risk
Grade 1	Superficial ulcer of skin or subcutaneous tissue
Grade 2	Ulcers extend into tendon, bone, or capsule
Grade 3	Deep ulcer with osteomyelitis, or abscess
Grade 4	Gangrene of toes or forefoot
Grade 5	Midfoot or hindfoot gangrene

Table 1

the vascular supply should be investigated even in the presence of palpable foot pulses and normal ABPIs. Partial amputations may be necessary in established osteomyelitis that fails to respond. The involvement of a microbiologist and an adequate number of microbiological specimens is vital for successful treatment of Grade 3 ulcers.

Correction of deformity is vital in the non-healing diabetic ulcer where deformity is felt to be the major cause of increased pressure. Some general principles apply. Incisions are preferably made through a separate, healthy part of the skin near the ulcer rather than through the ulcer itself. Dorsal incisions are favoured. The opportunity should be taken to debride the ulcer, which is best left open to drain. The surgical incision can be closed. After surgery careful pressure relief and dressing care is required to promote ulcer healing by secondary intention. Careful attention should be paid to glycaemic control.

Chronic toe ulceration is often secondary to hammer or claw toe deformities and is most often treated with amputation. Most ulcers of the hallux are plantar-medial and may be associated with pronation of the digit and hallux valgus. If hallux ulcers are resistant to offloading then resection of the medial condyles of the phalanges, either side of the interphalangeal joint is often successful. If the joint is infected, excision arthroplasty is a better option.

Ulceration beneath the first metatarsal head is common and is often caused by pressure under the medial sesamoid. Ulcers in this area can rapidly progress to Grade 3 with osteomyelitis and therefore relatively aggressive surgery to excise the medial sesamoid is indicated. This may be performed through a standard medial approach to the 1st MTPJ. Attention should also be paid to any contribution to overloading of the forefoot by tightness in the gastrocnemius–soleus complex, and an Achilles or gastrocnemius lengthening undertaken when necessary.

Ulceration under the lesser metatarsal heads may be treated with dorsiflexion osteotomies at the base of the metatarsal, such as the BRT procedure, which offloads the metatarsal head and avoids osteotomy or metalwork close to the ulcer. Excision of the metatarsal head is indicated for established osteomyelitis. Ulceration under the fifth metatarsal head may be treated with condylectomy. If this fails, excision of the distal third of the bone can be performed through extension of the same incision.

A number of studies have shown that Achilles tendon lengthening can be effective in treating plantar forefoot ulcers associated with contracture of the gastrocnemius–soleus complex, and the technique is growing in popularity. Mueller at al were able to show reduced rates of ulcer recurrence after Achilles lengthening and total contact casting versus total contact casting alone (15% vs 49% at seven months).[37] If the tightness is mainly in the gastrocnemius muscle, the authors always favour gastrocnemius lengthening over Achilles lengthening due to the reduced need for immobilization post-operatively.

During follow-up, ulcer healing should be documented by taking measurements and photographs as well as radiographs to

Brodsky's depth ischaemia classification of diabetic ulcers

Grade	Definition	Treatment
Depth Classification		
0	At-risk foot, no ulceration	Patient education, accommodative footwear, regular clinical examination
1	Superficial ulceration, not infected	Offloading with total contact cast (TCC), walking brace, or special footwear
2	Deep ulceration exposing tendons or joints with or without superficial infection	Surgical debridement, wound care, offloading, culture-specific antibiotics
3	Extensive ulceration with exposed bone and/or deep infection or abscess	Debridement or partial amputation, offloading, culture-specific antibiotics
Ischaemia Classification		
A	Not ischemic	
B	Ischemia without gangrene	Non-invasive vascular testing, vascular consultation if symptomatic
C	Partial (forefoot) gangrene	Vascular consultation for bypass or angioplasty, partial amputation if unable to re-vascularise
D	Complete foot gangrene	Vascular consultation, major extremity amputation

Table 2

determine progression or resolution of osteomyelitis. Once healed, patients must be educated to prevent recurrence and provided with good footwear. They should be followed up by a podiatrist or chiropodist and their primary physician. A multidisciplinary approach to these problems is important to achieve optimal results.

Charcot's neuroarthropathy

Jean Martin Charcot described neuroarthropathy due to syphilis in 1868. Currently, however, diabetic neuropathy is by far the most common cause of this destructive arthropathy, with the foot and ankle being the most commonly affected sites. The prevalence of Charcot's neuroarthropathy is estimated as being between 0.12% and 1.4% of the diabetic population. In the UK that correlates to as many as 25 000 individuals. The incidence appears to be rising although it is difficult to determine if this is a true reflection of more disease or simply better awareness of neuroarthropathy as a complication of diabetes. Either way, it is clear that neuroarthropathy presents a significant burden on health resources and in particular the foot and ankle surgeon.

Aetiology of the charcot joint: the pathophysiology of neuroarthropathy is not entirely understood and it is highly probable that a number of processes contribute to joint destruction. The **neurotraumatic theory** suggests that arthropathy is the effect of continuous micro-trauma and overuse or misuse of the joint due to impaired pain transmission and proprioception. A normal person would stop as a protective response due to pain. In patients with Charcot, the protective feedback of pain is absent, so they continue walking and the damage continues, resulting in arthritis and deformity. Occasionally the process may be initiated by a single traumatic episode such as an ankle fracture which, despite standard treatment, may go on to delayed union, late displacement, collapse and subsequent Charcot deformity. This theory is simple and makes sense in the foot; however, it does not adequately explain neuroarthropathy in non-weight bearing joints such as the elbow or shoulder.

The **neurovascular theory** suggests that autonomic dysfunction leads to increased blood flow to the joint via arteriovenous shunting. This hyperdynamic circulation leads to bone resorption, via increased osteoclastic activity. Additionally, there is evidence of an inflammatory component in neuroarthropathy, with raised levels of the cytokines TNF-α and interleukin-1, although causality has not been established. Neuroarthropathy in diabetes is a strange condition as it does not correspond well with severity or duration of neuropathy or type of diabetes, as one might reasonably expect. This feature serves to highlight the multifactorial aetiology of a Charcot joint.

Clinical assessment: clinical presentation of a Charcot joint is varied. Presentation types can be thought of as acute or chronic. Acute cases may present with a fracture, dislocation or rapidly developing deformity. Marked erythema, warmth and swelling can give the appearance of septic arthritis or cellulitis. Elevation of the limb reduces the erythema, as this is largely dependent, and distinguishes a Charcot joint from cellulitis. A careful history may reveal a traumatic event. Despite the limb by definition being neuropathic, there is pain in up to half the cases, although it is not always in proportion to the degree of joint destruction.

Chronic Charcot joints present more often with deformity: classically, widening of the foot, collapse of the longitudinal arch and even a rocker-bottom appearance. There may be ulceration over bony prominences, particularly the plantar aspect of the mid-foot. Vascularity should be assessed although it is often adequate. Footwear and risk of ulceration are prime concerns.

Classification of the charcot foot: Eichenholtz described three distinct stages in the pathophysiological process of developing a Charcot joint.[38] The first stage is characterized by acute inflammation. Radiographs show fracture and fragmentation of the bone associated with osteopaenia and eventual collapse of the joint. In the second stage there is coalescence with new bone formation. Clinically, the foot returns to a normal colour and temperature. In the third stage there is consolidation and healing with persistent deformity. A simple aide-memoire for the reader for this classification is that the stages are similar to those of Avascular Necrosis. A modification of Eichenholtz's classification adds an initial stage zero, in which there is warmth and erythema without radiological changes. It is supposed that early intervention here may prevent progression to destructive arthropathy.

Brodsky has described an anatomical classification to describe the common patterns of neuroarthropathy in the foot and ankle.[32] Type 1 Charcot joints are the tarsometatarsal joints and naviculocuneiform joints of the midfoot. This type of midfoot collapse is the most common pattern in diabetics with neuroarthropathy and often causes a fixed planus or even rocker bottom deformity. Type 2 disease involves the triple joints of the hindfoot i.e. the subtalar, talonavicular and calcaneocuboid joints. Charcot changes here result in valgus collapse of the hindfoot. Type 3A is rarer and involves the ankle joint. This may often relate to trauma such as a fracture or severe ligamentous injury. Varus or valgus collapse may occur and there is risk of ulceration of the malleoli. Type 3B feet are those that develop a pathological fracture of the tubercle of the calcaneus. Trepman added two more categories to this classification.[39] Type 4 disease involves a combination of patterns and type 5 deformity is in the forefoot alone.

Non-operative treatment of the charcot foot: the aim of treatment is to prevent deformity and minimize risk of ulceration. Patients with a Charcot foot are at increased risk of amputation, quoted at 7%. This dramatically increases to 28% if ulceration is present.[40,41] A secondary aim is to keep patients mobile until the third stage of the disease, in which there is bony healing and consolidation. It is vital to explain to patients at the outset that this process may take months or even years. Immobilisation in a full contact cast, rest, non-weight bearing and elevation are key in the acute stage. As the swelling can rapidly subside, frequent cast changes are initially required to prevent rubbing and ulceration as the less swollen foot becomes mobile inside the plaster.

Once the acute stage has settled and the patient has the clinical and radiological features of Eichenholtz Stage 2 disease, they can be converted into any number of offloading devices. We have used pneumatic walking boots with some success in more minor deformity. In severe deformity, custom-made thermoplastic ankle-foot orthoses, such as a Charcot Restraint Orthotic Walker (CROW), are indicated. After consolidation, the patient may

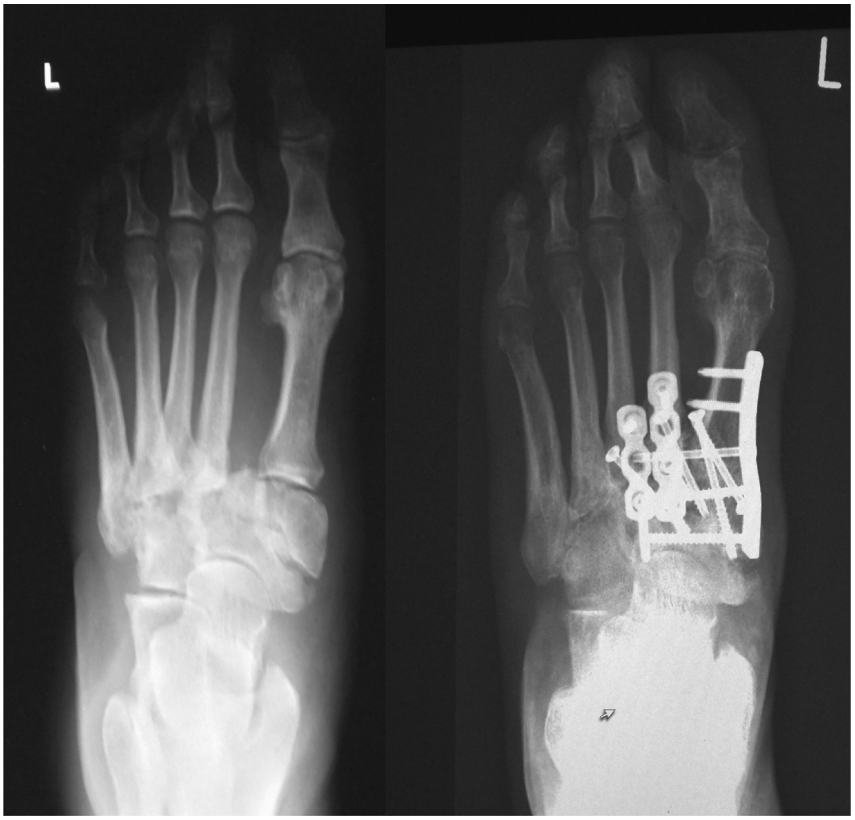
return to an accommodative shoe with a wide toe box and soft upper, with a custom-made orthotic insole.

Operative treatment of the charcot foot: there is much greater interest now in the operative management of the Charcot foot. The indications for surgery are severe uncontrolled deformity, ulceration, infection and persistent pain. Two main surgical strategies exist. The first is excision of bony prominences, with or without debridement of ulcers and infected bone. The second strategy is re-alignment and arthrodesis to reconstruct the shape of the foot in order to relieve pressure and facilitate shoe fitting.

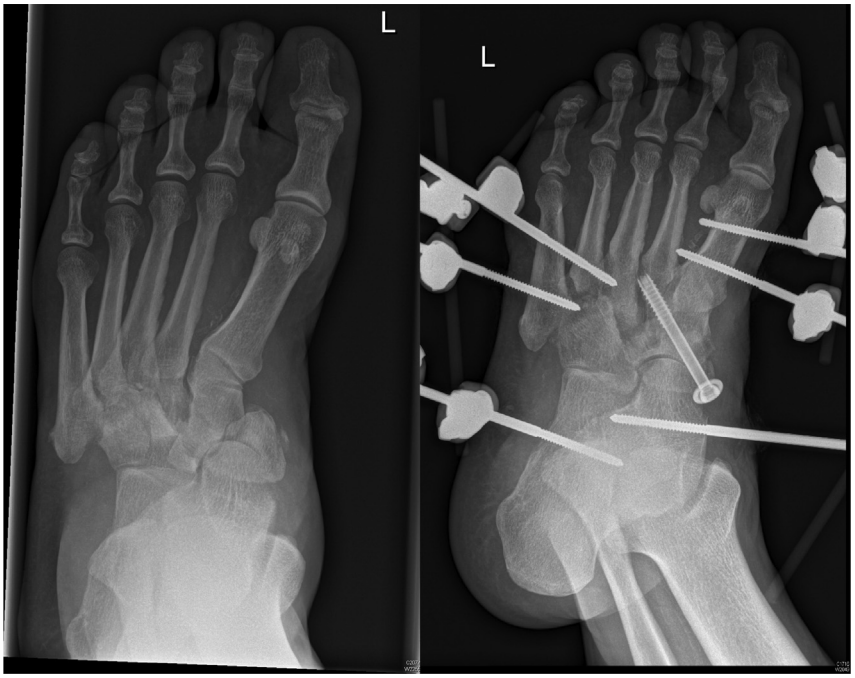
Exostectomy is similar to procedures used for chronic ulcers. Various principles should be applied. Incisions should be longitudinal and away from the pressure areas if possible. Plantar incisions should be avoided. Careful soft tissue dissection should be carried out, raising full thickness flaps as a single layer to expose the bone. Attempts should be made to diagnose infection pre-operatively, as bony resection needs to be more extensive to treat osteomyelitis. The patient may also require prolonged intravenous antibiotic therapy post-operatively. Bony resection should leave smooth rounded surfaces and try to minimize the risk of creating a new pressure area. Post-operatively, wounds should be offloaded with padded casts and non-weight bearing.

Reconstructive surgery for Charcot, comprising deformity correction and arthrodesis, is almost always performed in the 2nd or 3rd stages, when oedema has resolved and skin temperature has returned to within 1–2 °C of the non-affected side. A number of studies report good outcomes of ankle and hindfoot fusion using intramedullary hindfoot nails and screws. Siebachmeyer et al. achieved 100% limb salvage in 20 patients treated with hindfoot nails for severe Charcot deformity.[42] Principles of surgery include skin incisions and wedge resection of bone on the convex side of the deformity in order to de-tension the soft tissues for closure. Standard joint preparation is performed. Multiple biopsies are taken for microbiology. Strong fixation is required using the largest nail diameter possible and the maximum number of locking bolts. There are a wide variety of hindfoot-specific nails available and there may possibly be a role for additional design features such as HA coating of locking bolts, to reduce loosening.

Midfoot correction and arthrodesis utilizes similar surgical principles, with, however, an even more vulnerable soft tissue envelope. Sammarco introduced the concept of the *superconstruct* for midfoot reconstruction.[43] The principles include extension of the fusion beyond the zone of injury to include joints that are not affected, using the strongest device possible. Intramedullary screws, locking plates and plantar plating are all advocated. Correction also involves aggressive bone resection to shorten the extremity, which facilitates both reduction of deformity and soft tissue closure. Clinical Case 3 demonstrates an example of a Charcot midfoot reconstruction.



Case 3 AP X-ray of diabetic patient with a painful swollen foot after what was thought to be an acute Lisfranc injury; however, the history revealed a more insidious onset, and on clinical assessment there was neuropathy. We protected this patient in a diabetic boot and waited three months for the Charcot process to move into Stage 2, before then undertaking reconstruction of the foot. A good result was obtained using a strong construct, but the patient required boot protection for six months after surgery before moving into custom-made shoes. A cautious approach is required in such cases.



Case 4 This patient presented in a very similar manner to the patient in Case 3. However, there was much more severe deformity, with abduction through the midfoot and loss of the longitudinal arch. Therefore, in this case the decision was taken to perform acute correction through a small medial incision with lag screw fixation of the main fracture. External fixation was then used to stabilize the medial and lateral columns. This patient is still undergoing treatment. The foot is settling quickly and at eight weeks post-operatively the patient was transferred into a full contact cast, and he is now being monitored for consolidation. It is hoped that this strategy will minimize the deformity, reduce the risk of ulceration and save the patient from a major future reconstruction.

The success of intramedullary nailing in the hindfoot has prompted significant interest in intramedullary midfoot fusion bolts to reconstruct the medial column. However, despite sound biomechanical principles, a number of authors have reported unsatisfactory clinical results with this technique.[44,45] A biomechanical study showed a trend towards a stiffer construct using a relatively thin plantar plate versus large intra-medullary screws.[46] There is a trend toward combination fixation, utilizing compression screws and strong locked plates. The fixation must be durable as these patients may have a prolonged time to union and may inadvertently bear weight due to lack of protective sensation. Casting and non-weight bearing is often routinely recommended for six months, and sometimes longer.

An alternative surgical strategy is to utilize minimally invasive techniques and external fixation. External fixators may be used as static devices to maintain correction or as dynamic devices to gradually correct a large deformity over time, and a number of authors report success with this technique.[47] One additional advantage is that external fixators can be applied in the acute stages in a very rapidly progressing deformity to correct and stabilize the foot more powerfully and with less risk to the soft tissues than a full contact cast, as illustrated by Clinical Case 4. Disadvantages include pin site infections, which are much more common in the diabetic population, but which may not impair long-term outcomes.[48] Also, there have been a number of reports of tibial stress fractures with the use of circular frames.[47,49]

All surgery for severe Charcot should be considered limb-saving and a potential alternative to amputation, especially if patients have already suffered ulcerations. Treatment is long, involved and possibly more costly than amputation, in the short-term.[50] Diabetic patients, however, have little physical reserve and often function poorly due to the increased energy demands of mobilization after amputations. This major reconstructive surgery must only take place within institutions with dedicated orthopaedic specialists as part of a wider multidisciplinary team including diabetologists, diabetic podiatrists, plaster technicians, vascular and plastic surgeons and vascular interventional radiology.

Summary

Treating the orthopaedic problems of patients who have systemic diseases requires a multidisciplinary approach, following the principles outlined in this article. There is very little high quality research in this field in the form of properly controlled clinical trials, and much of what we know owes debt to the experience and dedication of key individuals and centres who specialize in treating these demanding foot pathologies.

Ethical approval

Ethical approval was sought from The Guy's and St Thomas' Hospital Research and Ethics Committee (REC). After preliminary questioning, formal endorsement was deemed

unnecessary due to the intrinsic nature of the study and lack of patient involvement or patient data in any form.

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Contribution statement

I confirm that all authors have contributed directly to this study and the resulting paper. Contributions have included conception, planning, data collection, analysis, direct authorship, editing and supervision. ◆

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