EFORT open reviews

Aseptic (avascular) bone necrosis in the foot and ankle

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- Aseptic necrosis may be defined as a group of diseases that have bone necrosis as a common denominator. They usually appear in the epiphyses and in the carpal and tarsal bones. They generally appear during a growth period and principally at those skeletal points subjected to particular stress.

- In Müller–Weiss disease in the advanced stages, talonavicular-cuneiform arthrodesis, with or without back foot correction, is the best surgical option.

- In Freiberg–Kohler disease, treatment can be conservative and we can maintain the head of the metatarsal by performing a joint debridement of the metatarsophalangeal joint with removal of loose bodies. The lateral upper and lower faces of the distal extremity of the metatarsal are resected, preserving the joint cartilage that in its centre portion is always healthy. The osteophyte border that may be present in the phalanx is resected.

- Most frequently, avascular necrosis (AVN) of the talus is a sequel to talar fractures, with the possibility that the AVN increases with the severity of the trauma and the damage associated with the already precarious blood supply of the talus.

- The surgical treatment used for sesamoid AVN is partial excision of the affected bone.

Introduction

Aseptic necrosis may be defined as a group of diseases that have bone necrosis as a common denominator. They usually appear in the epiphyses and in the carpal and tarsal bones. They generally appear during a growth period and principally at those skeletal points subjected to particular stress. There is great conceptual confusion revealed in the large number of names used, such as ischaemic necrosis, avascular necrosis, aseptic necrosis, osteonecrosis, idiopathic necrosis and bone infarction. Also, with the specific names according to the anatomical location (Perthes disease, Freiberg disease, Kienböck's disease etc.), differentiation between necrosis in adults and children, and distinction between osteonecrosis and dissecting osteochondritis. Many of these necroses were described first by radiologists.[1,2] We present here the most frequent osteonecroses in the foot and ankle.

Müller–Weiss disease

Necrosis of the navicular in children was first described by Kohler in 1908. It was named 'Kohler's first bone disease' to differentiate it from Kohler's second disease and Freiberg–Kohler disease which affects the heads of the second and third metatarsals. Ischaemic necrosis of the tarsal scaphoid in adults was initially described by Müller (1928) and Weiss (1929),[3–5] who described its radiological appearances of condensation and fragmentation. Müller–Weiss disease (MWD) is a navicular dysplasia that almost always develops during childhood and becomes symptomatic during adulthood. However, over the years, no osteonecrotic changes have been found in histological studies. In this disease two facts are important: a mechanical overload of the navicular together with its position in the medial arch of the foot and the vascular factor. There is not complete agreement in terms of the aetiopathogenesis of MWD. Delay in ossification can occur as a generalized or localized developmental disorder, with a force distribution pattern in the lateral half of the bone.

Another cause could be trauma, or it may develop spontaneously. Stress fractures are an attractive hypothesis, not only because of their similarity in the complementary tests, but also because together with acute trauma they are the first group of mechanisms that can compromise intra-osseous vascular flow. Many other aetiological factors can occur such as abnormal bone development in Kohler disease. Also, osteoarthritis, and

systemic metabolic and autoimmune diseases that increase the risk of osteonecrosis could be the cause.

Maceira and Rochera have so far presented the largest MWD series and proposed a combination of late ossification of the navicular tarsus and the abnormal distribution of force as a cause of MWD.[6] The disease begins in most cases insidiously with vague pain in the back of the foot. In the clinical phase, there is pain over the tarsal scaphoid. Peri-navicular arthritis has been the main feature of most studies, but clinical symptoms can be attributed to the main pathomechanical change which is hindfoot varus. Patients often complain about the functional instability of the lateral ankle and report pain around the peroneal tendons.

The clinical examination may show a normal, high or low-arched foot. In addition, because the navicular tuberosity is prominent on the medial side of the midfoot, simple inspection can give a false impression of hindfoot valgus.[7] Subtalar mobility is reduced. In loading radiographs, we find dorsal osteophytes in the medial tarsal joint[8] and dorso-lateral fragmentation of the navicular (Fig. 1). Non-surgical treatments should always be tried first for MWD because they do not compromise future surgical treatment. In the majority of cases, surgical treatment becomes necessary. So far, no 'gold standard' surgical treatment has been agreed. A wide variety of surgical options have been described. The indication for surgery is the severity of symptoms rather than the severity of the deformity. The foundations of surgical intervention are: symptomatic degenerative joint arthrodesis to relieve pain, restoration of the plantar vault and the medial longitudinal arch of the Meary–Tomeno axis.

A wide variety of surgical procedures have been described:

- isolated talonavicular arthrodesis (Figs 2, 3 and 4),
- internal fixation of the navicular,
- talonavicular-cuneiform arthrodesis (TNC),[9]
- triple open or arthroscopic fusion (Figs 5 and 6),
- pantalar arthrodesis,

- complete excision of the affected bony area and the tarsal reconstruction of the diseased navicular and medial spine with femoral head allograft,
- Achilles tendon lengthening and calcaneal osteotomy.

Fig. 2 Talonavicular arthrodesis in Müller–Weiss disease.

Fig. 3 Severe talonavicular affectation in Müller–Weiss disease.
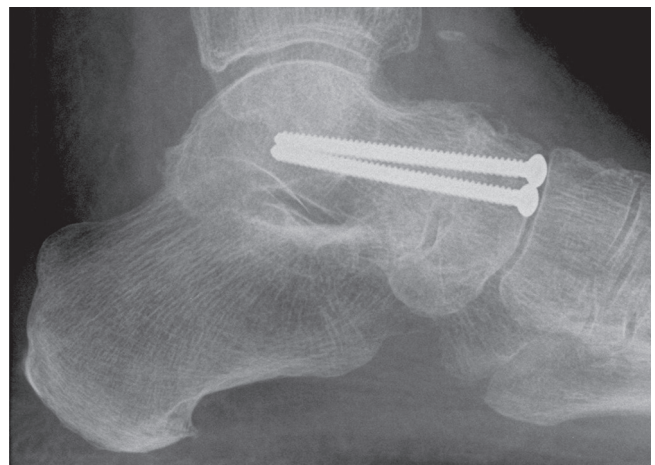

Fig. 1 Müller–Weiss disease.

Fig. 4 Isolated talonavicular arthrodesis.

Fig. 5 Müller–Weiss disease treated with talonavicular-cuneiform arthrodesis, with medial bone graft.

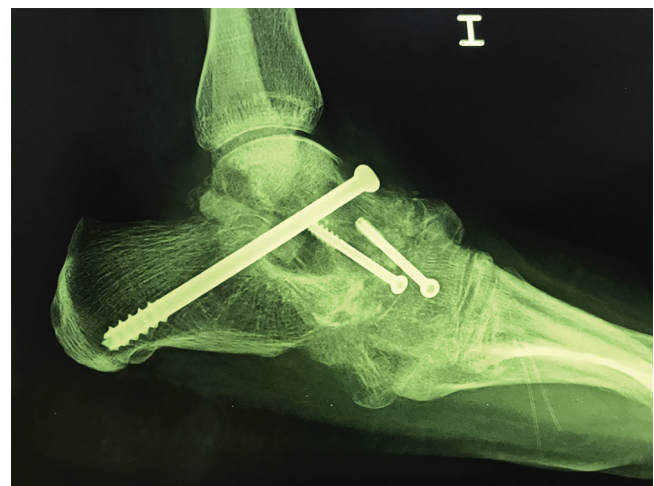**Fig. 6** Müller–Weiss disease with navicular fragmentation and subtalar arthritis.

Fig. 7 Treatment of subtalar and talonavicular arthrodesis.

We believe that in advanced stages, talonavicular-cuneiform arthrodesis, with or without hindfoot correction, is the best surgical option.[5] Classically, we perform this by a medial incision, following the arch of the foot, exactly in the line in which the characteristics of the skin change from dorsal to plantar. The head of the talus, navicular and cuneiform are freed from the intermediate joints and the salient portions of the navicular are removed in the dorsal region. We prepare a channel that runs from the talus to the cuneiform and a bone graft is placed (Fig. 7). Nowadays we perform an arthrodesis with screws and plates and cancellous graft. Our experience with this arthrodesis in the medial column of the foot gives good results in our patients.

Another option of treatment proposal by Monteagudo and Maceira is Calcaneal osteotomy is a calcaneal osteotomy incorporating a wedge and lateral translation.[10,11]

A complication in Müller–Weiss arthrodesis is nonunion of the talonavicular joint. In these cases we perform a bone graft with new osteosynthesis (Figs 8, 9 and 10).

Freiberg disease

Dr. Albert H. Freiberg first described avascular necrosis (AVN)[12] of the second metatarsal head in 1914. The disease appears more frequently in women than in men, in the ratio of 4:1, and the disturbances usually start between 16 and 20 years of age. Sometimes the injury is found incidental to a radiological examination made for another reason. It is not infrequent that the process shows up years later when, as a consequence of Kohler's second bone disease, osteoarthritis of the metatarsophalangeal joint appears.

Clinically, patients complain of pain over the head of the metatarsal associated with weight-bearing. Radiographs reveal a sclerotic and flattened metatarsal head and the development of loose bodies within the second metatarsophalangeal joint (MTP). The classic location of this disorder is the second metatarsal (68%), although it can also be found in the other minor metatarsals, most commonly the third (27%) and then the fourth (3%). The head of the fifth
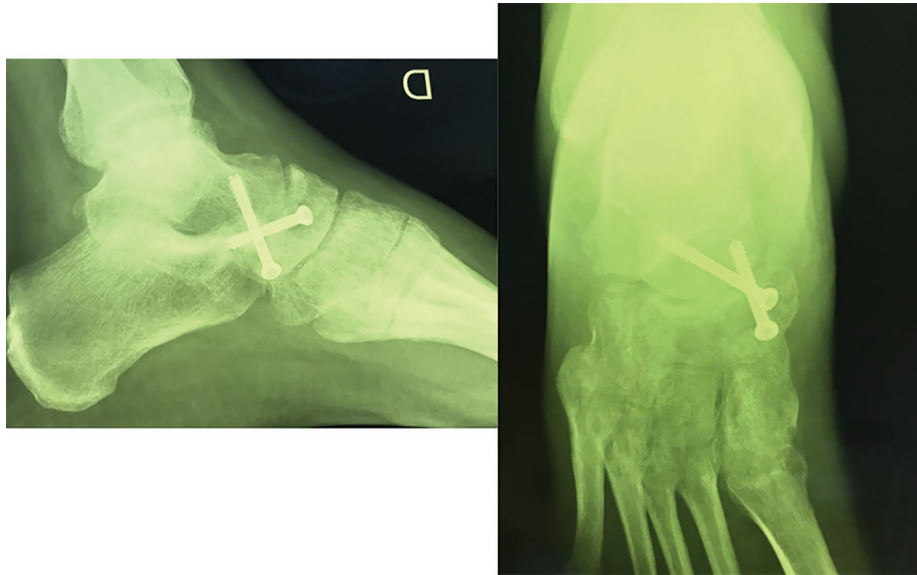
Fig. 8 Isolated talonavicular arthrodesis, after six months. Nonunion had occurred, therefore we decided to perform a new arthrodesis.
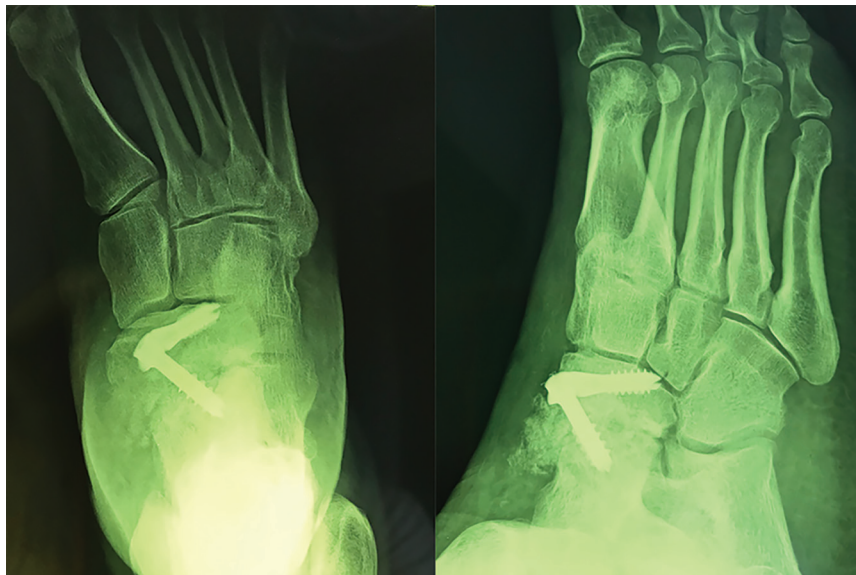

Fig. 9 Bone graft and new talonavicular osteosynthesis.

metatarsal is rarely involved. Cases of multiple metatarsal and bilateral involvement have also been reported.

The origin of this disease has been explained by many hypotheses, but most authors attribute this injury to microtrauma or to injuries due to overload. Another possible and likely cause is that the blood supply of the epiphysis of the second metatarsal depends almost exclusively on a small vessel that flows alongside the insertion of the joint capsule. Women of 15 or 16 years of age used to wear shoes that compressed the forefoot, especially between the heads of the metatarsals. This is enhanced in those patients who have a second metatarsal longer than the first. Following this external mechanical cause, arterial spasm occurs, which is responsible for the lack of blood supply to the epiphysis and causes the disease. Confirming this concept, Nagura has also proved that this disease never appears among Japanese people, who wear wide shoes or sandals.[13]

In general, it is accepted that the condition is multifactorial, including trauma, foot mechanics and arterial insufficiency. There are several systemic risk factors identified for Freiberg disease, including hypercoagulability, systemic

Fig. 10 Final result, consolidation at 11 months.

Fig. 11 Kohler II disease.

lupus erythematosus and diabetes mellitus, but the research around them is scarce. In addition, the disease is likely to have a genetic component, because Freiberg disease and other osteochondrosis have been reported in identical twins.[14]

Despite all this, the cause surrounding Freiberg disease remains unclear. The typical presentation is a female teenager with pain and often a slight oedema starting at the site of the dome of the foot. On palpation, tenderness is located over the head of the second metatarsal that worsens with weight-bearing and walking. Often the disease rarely causes problems and can even pass unnoticed, being a casual radiological finding. On examination, the second MTP joint may be swollen, there is pain on palpation due to synovitis and there may be hyperkeratosis under the affected metatarsal head. Crepitus and loose bodies can be palpable. The range of motion is reduced. In anteroposterior loading radiographs, a gradual collapse of the metatarsal head with loose intra-articular fragments is seen (Fig. 11).

As with other osteochondroses, early treatment generally focusses on symptom relief and prevention of deformity. Non-surgical treatment may include oral analgesics, activity modification, protected weight load, braces, and footwear modifications. In cases with persistent symptoms, especially in patients with marked osteoarthritis, surgery is required.

Various procedures have been recommended. Conservative procedures involve retaining the head of the metatarsal by performing a joint debridement of the metatarsophalangeal joint and removal of loose bodies. The lateral, upper and lower faces of the distal extremity of the metatarsal are resected, preserving the joint cartilage

which in its centre portion is always healthy. The osteophyte border that may be present on the phalanx is resected. Another treatment is a wedge dorsiflexion osteotomy of the metatarsal head (Fig. 12). The advantages of an osteotomy include the maintenance of the patient's own tissue to restore the cartilage surface to the main weight-bearing portion of the joint.[15] Cartilage replacement with a chondral graft may consist of autogenous osteochondral graft, osteochondral allograft or chondral allograft. A portion of the distal femur can be used both to fill the bone void associated with late-stage Freiberg

Fig. 12 Kohler II disease, treatment with dorsal wedge osteotomy. Evolution at 13 years.

disease and to replace poor articular cartilage. The resection of the metatarsal head can improve joint pain, but the amount of shortening caused predictably produces unacceptable long-term results. In addition, excision of the head makes any future reconstructive options extremely difficult.

Avascular necrosis of the talus

This can appear in different ways. In spontaneous cases without any known aetiology, as described by Mouchet in 1925,[16] it presents with pain in the ankle. On taking a radiograph, it is surprising to find a severe deformity, with the body of the talus having a greater than normal density or a condensed appearance. But AVN of the talus is most frequently a sequel to talar fractures, with the possibility that the AVN increases with the severity of the trauma and the damage associated with the already precarious blood supply of the talus,[17] which loses its muscular insertions. Its blood supply comes primarily from the dorsalis pedis and posterior tibial arteries and secondarily from branches of the peroneal and small periosteal vessels.[10] In addition to post-traumatic causes, occasionally there may be non-traumatic causes ranging from alcoholism, steroid use, dyslipidaemia or an idiopathic cause.

The Trauma Committee of the American Orthopaedic Foot Society noted that avascular necrosis of the entire talar body occurs only if the body is extruded, if it loses soft tissue insertions or in cases where treatment is delayed for a long time.[18]

Radiographically, talar avascular necrosis manifests itself as an area of increased sclerosis in the talar dome that can also extend to the body with collapse of the subarticular surface and, in severe cases, with fragmentation of the talar body. Non-surgical management is reserved for cases diagnosed early, with the intention of preventing collapse until revascularization is complete. Patients with early asymptomatic lesions should be followed up frequently (every three to six months). We recommend a trial of conservative therapy for at least three months before surgery, even in early symptomatic AVN.

The results of central decompression in the treatment of early stage talar AVN have been extremely satisfactory, especially when the cause is not traumatic. This works by reducing intra-osseous pressure and improving revascularization in the necrotic area, similar to the idiopathic variant of osteonecrosis of the head of the femur. The use of vascularized and non-vascularized bone grafts has provided beneficial results in the treatment of talar AVN. All previous studies show that a vascularized bone graft can give satisfactory and predictable results with the preservation of the joints in patients even to stage III of the disease, and can be combined with arthrodesis in cases with OA and collapse. The fusion of the neck and head of the talus

with the distal anterior tibia (anterior sliding distal tibial osteotomy) usually with the removal of the talar body and the increase of the structural graft, is an alternative. This type of fusion has the advantage of preserving some subtalar movement, better cosmesis and footwear, as well as preserving limb length. Use of TAR (total ankle replacement) without cement in patients with talar AVN and secondary arthritis, although extremely limited, has not been encouraging. The propensity to progression of the AVN and the inevitable progressive collapse of the talar dome can eventually lead to subsidence of the talar component followed by loosening of the implant and instability.

Renander's disease

Sesamoid osteonecrosis was described in 1924 by Renander.[19] It is a rare condition and can affect any sesamoid, the medial being more frequent. Various names have been used to describe this process, these include sesamoiditis, osteomalacia, sesamoid insufficiency, aseptic necrosis, osteochondritis, and others. The aetiology is in relation to an overload of a sesamoid, due to differing causes:

- In women wearing high-heeled shoes the first metatarsal is forced to be vertical and places the big toe in extension, thus compressing the sesamoid under the metatarsal.
- In cases of cavus foot, in which pronation of the forefoot is almost constant, the first metatarsal becomes more vertical, thus compressing the sesamoids.
- Overload is frequent in many sports and especially in dancing which involves standing on the ball of the foot.

It occurs in the form of selective pain under the head of the first metatarsal, especially in women, between the ages of 9 and 17, in relationship to sports microtrauma. In general, the disease can be classified into four phases:

1. The initial phase is characterized by mild pain without obvious radiological findings – detectable by magnetic resonance imaging (MRI).
2. The static phase where the pain is persistent and the radiological findings become persistent.
3. The phase of joint involvement in which microfractures and joint incongruities appear that will favour the development of osteoarthritis.
4. Finally, the osteoarthritic phase, characterized by a decrease in the arcs of movement, bone deformity and surface irregularity.

Radiologically, sclerosis and irregularity of the affected sesamoid are usually seen. The MRI scan can be useful in the early stages. The differential diagnosis should be made from the bipartite sesamoid. The diagnosis is made by

requesting axial or Walter-Müller radiographs to observe the sesamoids below the head of the first metatarsal.

Conservative treatment consists of placing a plantar support that unloads the affected area, and the prescription of analgesics. If the first metatarsal is vertical, an extension osteotomy of its base can give excellent results. Another surgical treatment used for sesamoid AVN is partial excision of the affected bone, being careful to leave a small portion of plantar bone, which is embedded in the rest of the cartilage. Only in selected cases do we resect all the sesamoid.

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LICENCE

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