FREIBERG'S INFRACTION, COMPRESSION FRACTURES AND OSTEOCHONDRITIS

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INTRODUCTION

Freiberg's infraction is the fourth most common avascular necrosis affecting an articular surface. It usually affects the second metatarsal, but may occur in any of the lesser metatarsals (1). The onset of the disease is usually after the age of 13 with a normal range between 12 and 18 years of age (2). Subclinical manifestations usually occur during adolescence. Symptoms may not appear until degenerative changes occur within the joint in adulthood. A predilection for females exists with a ratio of 3 to 1 (3).

The metatarsal bones are each ossified from two centers; one for the body and one for the head of the second, third, fourth, and fifth metatarsals. The first metatarsal varies in that an ossification center exists in the body and the base. Ossification begins in the center of the body or shaft about the ninth week and extends distally. The center for the base of the first metatarsal appears about the third year; the centers for the heads of the lesser metatarsals appear between the fifth and eighth year. The epiphysis ultimately ossifies to the metaphysis between the ages of 19 and 20 (1).

ETIOLOGY

Freiberg first described the deformity in 1914, referring to the lesion as an infraction or incomplete fracture of the metatarsal head resulting from trauma. He attributed the etiology of this condition to the poor mechanics of the affected joints, relating the onset of pain to such conditions as pes valgo planus, or elongated second metatarsal. In 1924, Koehler described the etiology as unknown, but of a nontraumatic nature similar to Panner's disease. McMaster (4) in 1978, while researching hallux rigidus, described a chondral or osteochondral impingement lesion of the metatarsal head produced by the pressure of the proximal phalangeal base. He suggested that this process might also account for the Freiberg lesion found in lesser metatarsals.

Gauthier and Elbaz in 1978 (5) suggested that the joint changes were the result of subchondral fatigue fracture. They theorized that necrosis was caused by the absence or loss of vascular connections to the subchondral bone beneath the cartilage. They commented on the preferential location of the infraction on the dorsal part of the second metatarsal head but could not explain how the fatigue fracture occurred in this location (6). Wiley and Thurston (7) in 1981, on the basis of an injection study, found that in two of six cadavers the second metatarsal arteries supplying the epiphysis were absent. The second ray is supplied by branches from the first and third rays. Stress studies revealed that the second metatarsal bore more load than the third or fourth metatarsals (8).

Currently the etiology of Freiberg's infraction is attributed to interruption of a precarious blood supply to the epiphysis. This has been termed epiphysitis (8-10). This may cause a micro fracture at the junction of the metaphysis with the epiphyseal plate depriving the epiphysis of adequate circulation. The microfracture at the epiphysis may be related to trauma, infection, or endocrine disturbances. Hormonal changes cause an alteration in osteogenesis making the cells very susceptible to minor trauma or ischemia. Steroid therapy may lead to osteopenia which may increase the tendency to develop microfractures (11). It is reasoned that steroids aggravate this tendency of microfractures and impairs healing causing worsening of the process.

Trauma may be induced by abnormal weight distribution across the epiphyseal plate and the metatarsophalangeal joint. Salter (12) found a congenitally short first metatarsal to exist in patients with Freiberg's infraction. The added trauma to the epiphysis causes aseptic necrosis and may result in altered shape. Continued stress insures loose bodies and crepitus. Degenerative arthritis will eventually develop. The degenerative process may continue for 2 to 3 years followed by a regenerative process that results with osseous hypertrophy of the bone.

Hulbert (13) found that renal transplantation due to chronic renal disease may result with a complex mixture of osteomalacia, osteitis fibrosa, osteosclerosis and osteoporosis. Their results indicated that in 120 recipients of renal transplants, 21 patients developed major skeletal

disease confirmed radiologically. Avascular necrosis occurred in 17 patients, one of which affected the second metatarsal.

One or all of the mechanisms described may result in a change in shape of the epiphysis and may produce incongruity of the joint surface and potential degenerative osteoarthritis, with metatarsal shaft hypertrophy (14). The precise etiology of the changes that occur at the epiphysis remains unclear.

TERMINOLOGY

The terminology used to describe Freiberg's infraction is vast to say the least. The problems with terminology reflect the changing theories of etiology and the pathological process. Freiberg's infraction has been defined as an osteochondrosis of the metatarsal heads, specifically the second metatarsal (1, 14-17). Osteochondrosis refers to disorders of enchondral ossification of epiphyseal growth during childhood (18). This osteochondrosis actually refers to the process of interruption of blood supply, bone death, or osteonecrosis followed by some form of bone regeneration. Freiberg's infraction is a process of osteochondrosis found in the metatarsals.

Osteonecrosis refers to avascular necrosis or aseptic necrosis of bone. Bone death can occur without signs of inflammation or sepsis. This usually results from the loss of blood supply to a particular area of bone (19). Osteonecrosis is only a step in the process of osteochondrosis. It can also be found in many other disease states such as osteochondritis dissecans or osteomylitis. Osteonecrosis is not synonymous with Freiberg's infraction. It merely represents a step in the process of osteochondrosis.

Osteochondritis dissecans described by Lehman and Gregg (10) is a clinical entity whose hallmark is development of an osteochondral fracture involving articular cartilage and the underlying subchondral bone. There is no particular relationship to an epiphysis or growing bone with this term. Freiberg's infraction is not present in osteochondritis dissecans. The distal metatarsal can be affected by two processes involving osteonecrosis as a step: 1) osteochondritis dissecans involving osteonecrosis of subchondral bone or 2) Freiberg's infraction involving osteonecrosis of the epiphyseal plate. With an accurate understanding of terms, the physician can better understand the disease processes affecting bone.

CLASSIFICATIONS

Gauthier and Elbaz (5) describe four anatomic evolutions of Freiberg's infraction.

Stage 1. Osteonecrosis without deformation
Stage 2. Deformation by crushing of osteonecrosis
Stage 3. Gradual cartilaginous tearing
Stage 4. Arthrosis

This staging system is didactic in nature and poorly adaptable clinically. The procedures recommended by the authors only include recommendations for end stage disease. These procedures include only osteotomies and arthrotomies.

Smillie proposed a pathological staging of Freiberg's disease (18). Stage one is a fissure fracture developing in the ischemic epiphysis. Stage two presents with the contour of the articular surface being altered by collapse of the central portion after bone resorption. Stage 3 is described as the central portion sinks into the head after further resorption leaving projections on either side. The plantar articular cartilage remains intact. Stage four presents as a loose body separates after the plantar isthmus of cartilage gives way. The dorsal and lateral projections fracture. The final stage, stage five, shows a flattening deformity and arthritis. Smillie related his staging scheme to treatment approaches. These will be covered later in the text.

Omer (19) has described Freiberg's in three classifications. The first stage demonstrates thickening and edema intra-articular and periarticular of the soft tissues. The adjacent metaphysis may demonstrate minimal osteoporosis with coarse trabecular patterns. This suggests trauma as an influential factor, with reactive edema of the articular tissues and active hyperemia of the metaphysis (19).

The second stage consists of irregularity of the contour of the epiphysis and a thinning of the subcortical zones of rarefaction. There are irregular zones of bony condensation which appear within the epiphysis. On roentgenogram the epiphysis may demonstrate complete fragmentation. Blood vessels within the epiphysis are incompetent secondary to thrombosis or microfractures of the trabeculae. The chondrocytes of the epiphysis that receive nutrition by diffusion from the joint fluid are deprived as a result of the edematous pressure from chronic synovitis. Microfractures may extend to the subchondral plate, increasing the metabolic breakdown of cartilage into the marrow spaces (19).

The third stage represents the period of repair, with gradual replacement of the necrotic tissue. Within the epiphysis, there is peripheral ingrowth of capillaries and cellular differentiation of basic mesenchymal cells to replace or envelope cancellous bone fragments. The necrotic bone loses its structural support and, if a large area is involved, there is associated compression and flat-

tening of the articular surface (19). One must remember to compare contralateral films in determining the extent of repair.

CLINICAL PRESENTATION

Symptoms are usually vague, with patients describing pain at the extremes of motion, with the onset of symptoms occurring between the ages of 10 and 15 (1). The acute inflammatory stages subside where there is periarticular edema with a slight increase in temperature on palpation. The patient will usually limp on the affected limb, with symptoms subsiding with protected mobilization. Symptoms reappear between the third and fifth decades of life. By this time there is severe hypertrophy and deformity at the second metatarsal head resulting from attempts to repair the earlier epiphysitis.

Active and passive motions may be severely limited. Gross changes that may accompany the deformity include bony lipping, which may tend to fuse the joint (1). Joint mice may be present, increasing the pain. In the later stages, the proximal phalanx is frequently involved in the total deformity.

Other deformities described by Thompson and Hamilton (9) may be present. Subluxation of the proximal phalanx, when there has been stretch of the plantar capsule, may occur dorsally at the metatarsophalangeal joint in toe-off, but not in static stance (20). This abnormal motion causes irritation, synovitis, capsulitis, and pain. The etiology is attributed to the long second metatarsal and disproportionate weightbearing on the second metatarsal head in toe-off. The physical sign which is diagnostic of this deformity is termed the Lachman test. This is very similar to that of the Lachman knee test. The test is graded by how much the proximal phalanx can be uncovered vertically. Normal being that the metatarsophalangeal joint cannot be manipulated so that the base of the proximal phalanx cannot be displaced vertically.

Occasionally, dislocations of the second metatarsophalangeal joints are present without hallux valgus. Most patients with chronic dislocation reveal a history of painful joint, usually at the subluxation stage. Pain may also be present at the dorsum of the toe where contact of the toe box of the shoe strikes the proximal phalanx.

Radiographically, sclerosis has been the earliest change observed in the disease (10, 21). The epiphysis becomes fragmented, irregular, and flattened. The distal end of the metatarsal exhibits marked flattening and is wide at the articulating surface. There is thickening of the metatarsal shaft later in the progression of the disease. The metatarsal head may be fragmented in chronic, untreated cases, with the osseous trabeculae becoming thinned out and replaced by granulation tissue.

Treatment

Conservative therapy early in the disease process can consist of customary non weight-bearing cast immobilization, aspirin, non-steroidal anti-inflammatory medication and elevation (22). Physical therapy can be initiated within 3 to 4 weeks after the initial casting. Accommodative orthotics can be constructed with the possible use of injectable corticosteroids with a local anesthetic to give temporary relief. Due to the stress of weightbearing, the period of protected mobilization must last at least 2 to 3 years. If this therapy does not relieve symptoms, then the prognosis for a painfree joint is guarded. Degenerative changes within the joint may eventually necessitate surgical repair (2).

Smillie (8) suggested that the described pattern of management of Freiberg's infraction presents a logical approach to the disease in its various stages. Stages 1 and 2 relief from weightbearing by the use of crutches or weight-relieving orthosis have not resulted in satisfactory results.

Smillie, in the early stages 1 and 2, restores the contour of the head of the metatarsal by inserting a dorsal graft of cancellous bone. The purpose of this graft is to support the weakened subchondral bone to a more normal contour. In the late stages, 3-5 management should be tailored to the cause of symptoms. If there is no joint pain on forced movement with symptoms due to pressure metatarsalgia, an appropriate osteotomy may be os assistance. The use of nonfixated osteotomies is not recommended by the Podiatry Institute. If there is severe arthrosis and joint pain, then a silicone joint replacement arthroplasty is considered. If a metatarsal head has already been excised and the patient presents with pressure metatarsalgia beneath the remaining heads, Smillie recommends implant arthroplasty to replace the excised head. Metatarsal head osteotomies of the adjacent metatarsals are used as needed to relieve metatarsalgia. Of interest is Smillie's lack of attention to the use of digital stabilizations to aid in the control of lesser metatarsalgia.

Smillie's total of 25 patients, 14 women and 11 men between the ages of 8 and 48, were classified into stages. Eleven patients were classified into early stages (one and two) and 14 were classified into the late stages (23). Follow up of the stages one and two, eight patients were clinically and radiographically normal, one was clinically normal, and two had pain running and wearing high heeled

shoes. At follow up of the latest stages (3 to 9 years) eight patients were fully active and symptom free. Six were symptom free while standing and walking, but had pain with running.

Thompson and Hamilton (9) described four classifications. Their classification scheme is based on the resultant metatarsal deformity following the complete progression of the process to osteochondrosis. The scheme is useful clinically as it assumes all conservative measures have been applied. Type one is more theoretical than actually clinically notable and involves a transient lesion which heals with no loss of articular cartilage or degenerative osteophytes around the joint. No surgical treatment is required for this self limited process. However, the patient should be protected with reduced activity, wearing a lower heel, and a metatarsal pad. Type two comprises a larger vascular insult with the articular cartilage being preserved. The symptoms are related to proliferative degenerative osteophytes surrounding the metatarsophalangeal joint. Treatment is limited to debridement of the osteophytes and synovectomy. Type three represents the most severe involvement. It results in articular destruction as well as proliferative degenerative changes. Surgical treatment consists of a DuVries arthroplasty (24) of the metatarsophalangeal joint including the plantar condyles of the metatarsal head. Type four is rare and represents epiphyseal dysplasia in that more than one metatarsal head is involved. Surgical treatment should be tailored to the stage at each metatarsal head.

Miller, Lenet, Sherman (14), and Bordelon (25) discuss joint replacement with a Swanson silastic flexible hinged toe implant. The author's objectives of implant arthroplast include adequate range of motion, adequate stability in the sagittal and transverse planes, mechanical advantage of the joint, resistance to lateral or rotational stress, resolution of pain, cosmetic improvement via maintenance of normal length, and minimal tissue reaction. The silastic hinged implant also allows for greater sagittal plane motion via pistoning effect of the stems within the intramedullary canals. These reports represented only two clinical cases, only one a pediatric patient. Further study is needed on the use of silastic implantation in the adolescent. To date a life expectancy of lesser metatarsophalangeal joint implants is unknown.

To overcome the use of silicone implant in the pediatric patient, Kehr (12) described an interesting combination of arthroplasty and capsulorrhaphy. He describes a procedure where a dorsal U-shaped flap is used through the dorsal apparatus of the metatarsophalangeal joint. An arthroplasty is performed along with resection of all hypertrophied bone on the medial, lateral, and plantar aspects of the metatarsal head. The U-shaped dorsal flap is then directed plantarward and sutured to the plantar joint capsule.

Historical research indicates that the etiology of Freiberg's infraction is unknown. We do know that the process of osteochondrosis involves osteonecroses for which there is no known cure but only treatment dependent on the end stage presentation. The surgical procedures used tend to be salvage procedures combining joint arthroplasty and capsulorrhaphy with joint implants reserved for late degenerative changes in adults.

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