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Freiberg's disease in diabetes mellitus

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Abstract. We evaluated a total of 13 cases of Freiberg's disease in patients aged 47–77 years collected over an 8-year period. Seven were associated with diabetes mellitus and one with chronic renal failure; the remainder had no underlying disease. Atrophy of intrinsic small foot muscles secondary to neuropathy, which is prevalent in diabetes mellitus, may play a part in the development of Freiberg's infraction.

Key words: Freiberg's disease – Ischemic bone necrosis – Diabetes mellitus

Freiberg's disease is an ischemic necrosis of the metatarsal head. Although rare, it is the fourth most common type of avascular necrosis involving an articular surface [17]. This disease predominantly affects women; its usual onset is in adolescence. Freiberg's disease has been described in systemic lupus erythematosus, in patients taking steroids, and in patients sustaining unrelated trauma elsewhere in the foot [1, 2, 23]. The purpose of this paper is to report seven cases observed in diabetic patients during an 8-year period.

Materials and methods

We reviewed the charts and radiographs of 11 patients with Freiberg's disease at our institution. Their ages ranged from 26 to 77 years. Five had associated diabetes mellitus, one had chronic renal failure, and the others were without underlying disease. Subsequent review of foot radiographs of 120 diabetic patients yielded two more cases of Freiberg's disease. Affected patients, aged 48–77 years, had had diabetes for 9 or more years, and three had clinical symptoms of foot neuropathy (Table 1).

Discussion

Various patterns of bone involvement have been described in diabetic osteopathy of the foot [5, 7, 8, 12, 15, 19, 20]. Newman and Wagner have classified these bony lesions into those associated with osteoporosis, hyperostosis, osteolysis, or spontaneous subluxation/dislocation without bone destruction [16]. In an extensive review of the radiographs of 1501 diabetic patients, Geoffrey et al. found that exostoses occurred in 36%, osteoporosis in 12.2%, geodes in 3.7%, sclerosis in 4%, articular lesions in 5.1%, and destructive lesions in 3.9% [8]. The prevalent exostoses observed by these authors may be related to the aging process. The wide radiographic range of metatarsal bone destruction included total resorption of the distal part of the bone, the "sucked barley sugar" appearance, the "pencil point" deformity, and other lesions. These bony involvements may be secondary to several possible causes including infection, repetitive trauma, vascular insufficiency, neurotrophic or neuropathic changes, and metabolic alterations. Friedman et al. noted that 15 of 22 patients with severe diabetic neuropathy had metatarsal head and neck bone lesions, manifested as irregular destruction and occasional small punctate areas of bone resorption [7]. Freiberg's disease has not been mentioned in these or other reports.

Clinically Freiberg's disease develops 3–4 times more frequently in women than in men during late childhood or adolescence. Both feet are involved equally; only one lesion is usually found in a foot. Bilateral involvement is rare. The second metatarsal is affected in 68%, the third metatarsal in 27%, the fourth in 3%, and the fifth is only rarely involved [4]. In the acute phase, local pain, tenderness, and swelling are reported; later during osteonecrosis and repair, patients are often asymptomatic.

Table 1. Characteristics of patients studied

Case	Age (years)	Sex	Duration of disease (years)	Neuropathy
1	61	F	13	+
2	72	M	15	–
3	77	F	9	–
4	48	F	18	+
5	53	M	10	–
6	61	M	12	–
7	51	F	10	+

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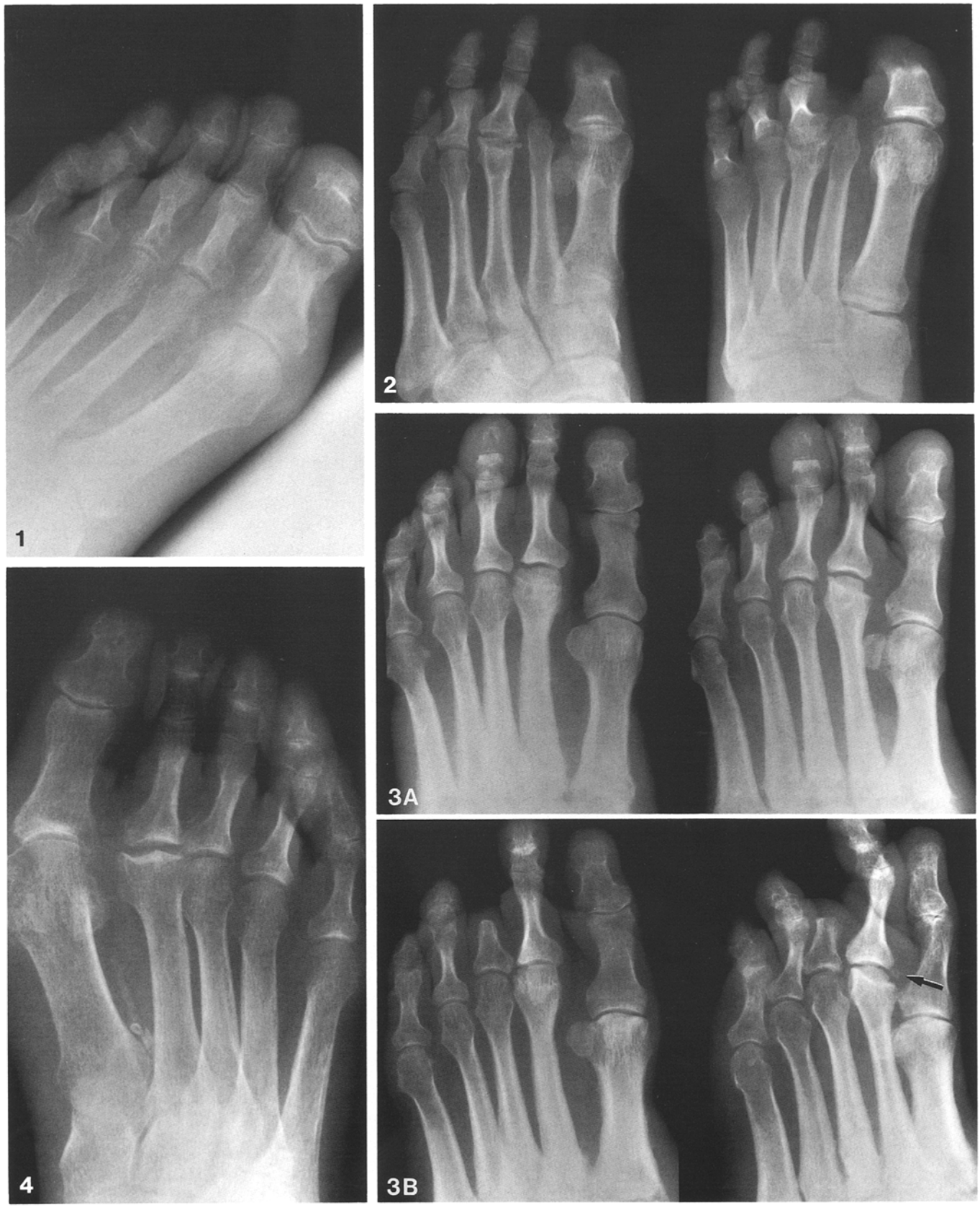

Figs. 1–4. See next page for corresponding captions

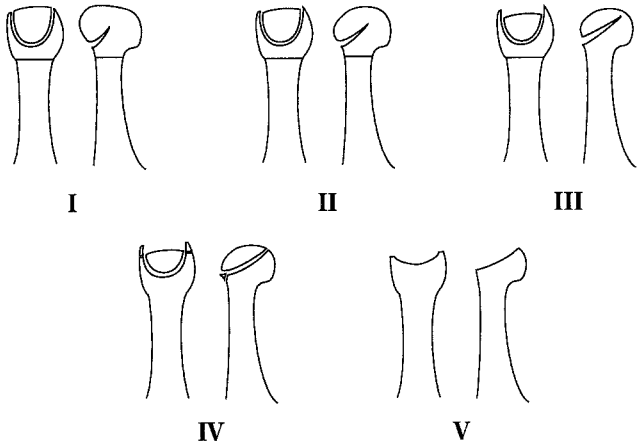

Fig. 5. Pathological staging of Freiberg's disease (after [6])

Early in the disease, radiographic changes include subtle flattening, areas of increased radiodensity, and cystic lesions of the metatarsal head with widening of the metatarsophalangeal joint space. This widening is secondary to the infraction of subchondral trabeculae with collapse of the articular surface [21, 27] (Fig. 1). On bone scan, high resolution magnification images obtained with pinhole collimation may show a photopenic infarcted area surrounded by a very active revascularized collar [13]. As the disease progresses, one may see osteochondral fragmentation, with progressive flattening and sclerosis of the metatarsal head, and increased cortical thickening of the adjacent metaphysis and diaphysis of the bone [21] (Figs. 2, 3). Later radiographic changes may include premature closure of the growth plate, intraarticular osseous formation and deformity, and enlargement of the metatarsal head with secondary degenerative joint disease [21, 27] (Fig. 4).

Three of the six original cases reported by Freiberg had a definite history of trauma, and subsequent reports by other authors have also supported a traumatic etiology [3, 6, 25, 28]. The second metatarsal is most commonly involved; it is usually the longest and is subjected to the greatest reactive ground forces during ambulation [6, 13]. The higher incidence in female patients may be related to the use of high-heeled shoes which place the phalanges in the dorsiflexed position relative to the metatarsal heads, thereby causing a retrograde pressure on the metatarsal heads [14, 24]. The primary lesion is a superficial fissure involving the dorsal aspect of the metatarsal head. This fissure causes disruption of the precarious epiphyseal vascular supply, leading eventually to ischemic bone necrosis with subsequent repair [4, 25, 26, 29]. Five stages of anatomic changes have been described (Fig. 5, Table 2).

Table 2. Pathological staging of Freiberg's disease (modified from [6]

Stage 1:	A fissure fracture develops in the ischemic epiphysis.
Stage 2:	Bone resorption with the central portion beginning to sink into the head, resulting in alteration of the articular surface.
Stage 3:	Continued resorption with the central portion sinking further into the head, resulting in peripheral projections.
Stage 4:	Fractures of the lateral and dorsal projections as well as the plantar isthmus of articular cartilage.
Stage 5:	Flattening, deformity, and arthrosis.

Neuropathy, a frequent complication of diabetes mellitus, may play a role in Freiberg's disease observed in diabetic patients. Neuropathy is reported in approximately 12% of diabetic patients at the time of diagnosis, and the prevalence increases with the duration of the disease to nearly 50%–60% after 25 years [11, 26, 30]. Motor weakness involves primarily the most distal intrinsic muscles of the hands and feet and occurs late in the course of the disease [11]. The neurogenic atrophy of intrinsic foot muscles which normally provide a tonic counterbalance to the more proximal foot flexors and extensors leads to chronic flexion of the metatarsophalangeal joints. This causes the toes to be drawn into a cocked-up position called "claw toes" [11, 30]. As a result, the toes no longer participate in the distribution of load on the foot, and weight-bearing is shifted to the now uncovered metatarsal heads; thinning and atrophy of the normal fat pads ensue [30]. Thick calluses may form over the exposed metatarsal bony prominences and protrude from the plantar surface. These cause a further shift of weight-bearing to the metatarsal heads, with subsequent stress microfractures, vascular injury, and, eventually, avascular necrosis.

In summary, diabetic patients are subject to a variety of musculoskeletal disorders secondary to neurotrophic, vascular, and metabolic alterations [9–11, 18, 22, 30]. Through these harmful effects, diabetes mellitus may be a predisposing factor in the development of Freiberg's disease.

Fig. 1. Stage II: Flattening of the articular surface with subchondral bony sclerosis of the second metatarsal head. The widening of the metatarsophalangeal joint space is secondary to the infraction of subchondral trabeculae with collapse and compression of the articular surface

Fig. 2. Stage IV: Flattening of the third metatarsal head with subchondral bony resorption and an adjacent small, round intraarticular osseous fragment

Fig. 3. Stage IV. **A** Sinking of the central articular portion into the deformed second metatarsal head with cystic formation. **B** Eight months later, sinking of the central articular portion into a lesser deformed metatarsal head is evident, probably secondary to bony remodeling and healing. Note the formation of an intraarticular osseous body (*arrow*)

Fig. 4. Stage V: Flattening of the articular surface with subchondral bony sclerosis of the second metatarsal head and widening of the metatarsophalangeal joint space. Note the deformity and enlargement of the metatarsal head

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