CHAPTER 37

COMPLICATIONS OF THE KALISH OSTEOTOMY: Avascular Necrosis With Joint Replacement

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Avascular necrosis is generally described as the death of any tissue secondary to an absent blood supply. Following surgical manipulation, infarcts may occur in the first metatarsal bone. Histologically, one observes a central zone of cellular death, surrounded by successive zones of ischemic injury, active hyperemia, and normal tissue.[1] With increased physical stress to the first metatarsal head, collapse may result if the ratio of resorbed bone becomes greater than the re-ossified bone. Subchondral bone plate collapse may result in articular surface collapse and possible arthritic complications of the first metatarsophalangeal joint. Differential diagnosis of avascular necrosis most commonly includes osteomyelitis, septic arthritis, Charcot changes, delayed union, or nonunion.

Anatomically, the first metatarsal head is supplied by intrinsic and extrinsic vessels. The extramedullary vessels are concentrated dorsally and laterally.[2] The extrinsic blood supply to the first ray is derived from three major arteries: the dorsalis pedis, medial plantar, and lateral plantar. The intrinsic blood supply to the first metatarsal head and neck area is derived from the periosteal arterial system, the periarticular metaphyseal and epiphyseal arteries, and the nutrient arteries to the distal first metatarsal diaphysis.[3] Lateral soft tissue releases (fibular sesamoid release, adductor tenotomy, lateral capsulotomy) may disrupt the extramedullary vessels, whereas osteotomies through the distal one-third of the first metatarsal may potentially disrupt the intramedullary vessels.[4,5]

The incidence of avascular necrosis after distal first metatarsal osteotomies is low, with variable rates reported. Meisenhelder's analysis of 54 such osteotomies revealed a combined avascular necrosis rate of 4%.[6] Rates can vary depending on the source of those reporting. Meier and Kensora indicated a 20% avascular necrosis rate in Chevron osteotomies without adductor release, and a 40% rate for those with adductor release.[5] In contrast, Williams reported on 64 osteotomies recently, without a single case of avascular necrosis.[7]

The reliability of these reported studies depends heavily on the criteria for arriving at the diagnosis, compliance of patients, surgical technique, type of procedure performed, and health status of patients at the time of surgery.

Detection of avascular necrosis is difficult considering most cases are asymptomatic. Radiographically, avascular bone appears more dense than the surrounding bone. One would possibly see different levels of radiolucency or fragmentation on x-ray. The end result could be arthritic changes of the first metatarsophalangeal joint.

Wilkinson reported that only 10% of postoperative bunionectomies showed evidence of avascular necrosis on radiographs, whereas 50% of these cases showed evidence on magnetic resonance imaging (MRI).[8] The literature consistently reports MRI-detectable lesions as common, although its clinical significance appears limited. Patients with evidence of avascular necrosis on MRI and radiographs are almost always asymptomatic. Occasionally, joint involvement causes clinically significant complications as described in the following case report.

CASE REPORT

The authors report the case of a 45-year-old female who developed avascular necrosis following a Kalish osteotomy. This patient initially presented to the clinic with a complaint of a painful first metatarsophalangeal joint of the left foot. She also had complaints involving a painful 2nd hammertoe, and

interspace pain that was unresolved following neuroma surgery eight years previous.

Surgery was subsequently performed on this patient's left foot including excision of neuromas from the 2nd and 3rd interspaces, arthroplasty of the 2nd toe with K-wire stabilization, and a Kalish osteotomy bunionectomy (Figs 1A, 1B). Normal postoperative recovery was apparent at the six month follow-up visit.

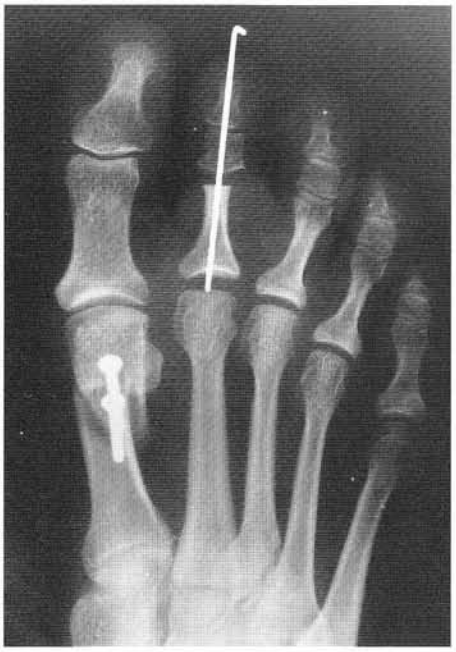

Figure 1A. Kalish osteotomy, immediate postoperative dorsoplantar radiograph.

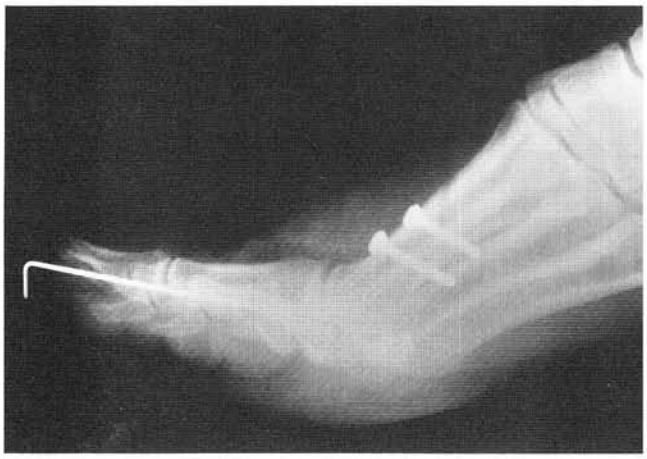

Figure 1B Kalish osteotomy, immediate postoperative lateral radiograph.

At 17 months following surgery, the patient had developed a hallux varus deformity. Radiographs revealed increased density of the first metatarsal head and subchondral collapse of bone consistent with avascular necrosis (Figs. 2A, 2B).

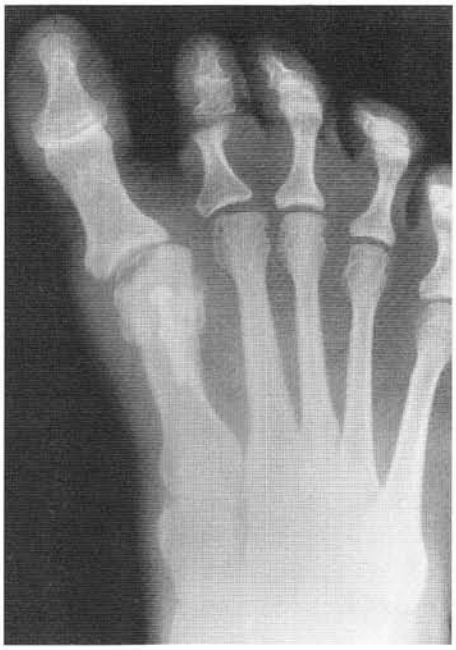

Figure 2A. Dorsoplantar radiograph at 17 months following Kalish osteotomy. Note the subchondral collapse and joint space narrowing leading to hallux varus deformity.

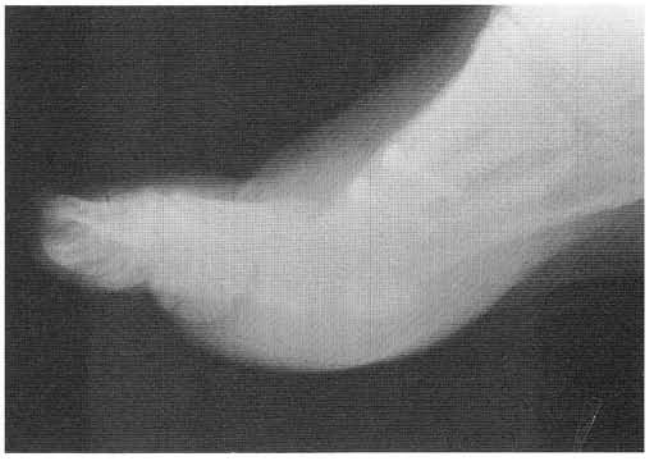

Figure 2B. Lateral radiograph at 17 months postoperative.