FIRST METATARSAL DISTAL OSTEOTOMIES: ASEPTIC NECROSIS OR HALLUX LIMITUS

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INTRODUCTION AND PURPOSE

The purpose of this paper is to help differentiate postoperative aseptic necrosis from postoperative hallux limitus. According to our previous studies and a survey conducted by the Podiatry Institute, aseptic necrosis of the first metatarsal head is rarely encountered. The overall incidence of aseptic necrosis at this institution is much less than 1%. Previous investigators have reported an incidence of aseptic necrosis following distal first metatarsal osteotomies ranging from 4% to 40%. The experiences of those at the Institute and the identification of factors which reduce the frequency of aseptic necrosis have been previously reported in *Reconstructive Surgery of the Foot and Leg - Update '89.*

Idiopathic and iatrogenic hallux limitus receive significant attention in the literature with regard to pathogenesis and correction. However, the incidence of this condition is not clearly demonstrated. On the other hand, the incidence and pathophysiology of aseptic necrosis has been detailed. It is the author's belief that one explanation for this is the lack of a classification scheme for iatrogenic hallux limitus. One of the goals of this study is to stimulate interest and attempt to establish a classification for the phases of postoperative hallux limitus based on pathophysiology. In addition, comparisons will be made between the two aforementioned conditions to encourage discussion and to determine if similiarities may be noted.

DEFINITION AND INCIDENCE

Hallux limitus in its broadest definition refers to a limitation or restriction of motion of the first metatarso- phalangeal joint (MPJ). It may be as simple as a momentary inhibition of joint motion or as extensive as total absence of joint motion or hallux rigidus. Hallux limitus has been described in the literature by a number of terms including dorsal bunion, metatarsus primus elevatus, hallux limitus, hallux rigidus, hallux dolorosus, and hallux flexus. (Fig. 1) The incidence of postoperative hallux limitus is not well represented in the literature. However, its correction is widely described in both the podiatric and orthopedic journals.

Aseptic necrosis is a process represented by an initial loss of blood supply to the bone with resultant bone death. Necrotic bone is then gradually replaced by a more viable osseous material. Aseptic necrosis is commonly known as avascular necrosis, osteo necrosis, and ischemic necrosis. (Fig. 2)

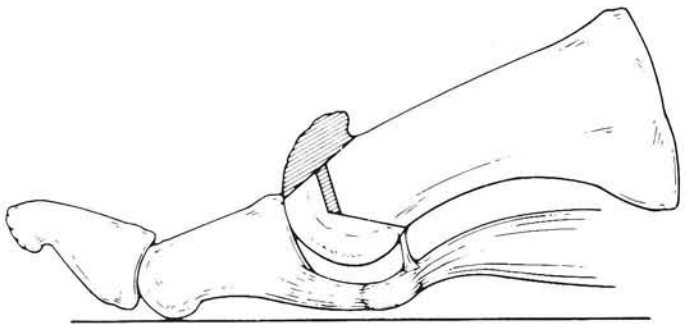

Fig. 1. Modified cheilectomy in combination with Green-Waterman osteotomy for correction of hallux limitus.

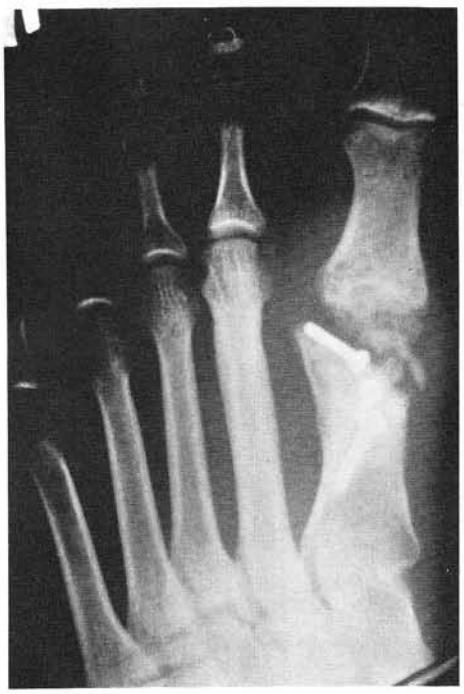

Fig. 2. Aseptic necrosis of the first metatarsal head following metatarsal osteotomy for correction of hallux valgus.

The incidence of aseptic necrosis following distal osteotomies of the first metatarsal, as reported in the literature, varies significantly. Meisenhelder, Harkless, and Patterson (1984), in a review of 54 procedures, reported 2 cases of aseptic necrosis following distal osteotomies of the first metatarsal resulting in an overall incidence of 4%. The average time elapsed prior to follow-up was 41 months, with the longest being 78 months and the shortest 16 months. Radiographs were obtained to determine the presence of aseptic necrosis in these cases.

Meier and Kenzora (1985) reported a higher incidence of 20% in 60 patients following Chevron osteotomy of the distal first metatarsal. The incidence of aseptic necrosis increased to 40% when an adductor tenotomy was performed in combination with the Chevron osteotomy. Overall, the results of this study demonstrated an incidence of aseptic necrosis of 18% in 13 of the 72 cases presented.

Preliminary investigations at the Podiatry Institute over the last ten years reveal an incidence of aseptic necrosis of much less than 1%.

ETIOLOGY AND PATHOPHYSIOLOGY

The etiology of iatrogenic hallux limitus subsequent to distal metaphyseal osteotomies is not well understood. However, most investigators agree that the development is multifactorial. There are identifiable iatrogenic and postoperative factors that can lead to this condition. Iatrogenic factors include extensive capsular dissection, excessive resection of bone, lengthening of the first metatarsal, and dorsal elevation, or excursion of the capital fragment. Postoperative factors include prolonged immobility of the joint after surgery, poor compliance with range of motion exercises of the metatarsophalangeal joint, and extensive fibrosis of the capsule due to internal bleeding or other causes. There are a number of reports of sesamoid apparatus immobility secondary to fibrosis of the flexor complex with resultant hallux limitus. Suffice it to say the sesamoid apparatus should glide smoothly as the great toe is placed through its full range of motion.

Some investigators have postulated that the shape of the first metatarsal head is a contributing factor in the development of hallux limitus or hallux rigidus. Brahm (1988) presented a retrospective study on the shape of the first metatarsal head and its relationship to disorders of the first metatarsophalangeal joint. Depending on the biomechanical forces and the stresses placed upon the first metatarsophalangeal joint postoperatively, hallux limitus may occur as a later sequalae. The hypermobile first ray may tend to progress to recurrent hallux valgus or hallux limitus. The stable first ray with a more squared head may manifest with excessive compressive stress to the joint resulting in a similar reduction of motion.

Regardless of the cause of restricted joint motion, most investigators agree that early recognition and appropriate treatment is necessary in preventing the long term effects or progression of hallux limitus.

The etiology of aseptic necrosis is not clearly understood or adequately described in the literature. Most investigators agree that aseptic necrosis is primarily a condition which results from an ischemic episode to that particular portion of bone. Subsequent to ischemia there is actual bone death or necrosis. In time the necrotic bone is replaced with living material. The degree of resultant deformity depends on the forces exerted on the first metatarsophalangeal joint during this process. Early recognition and appropriate treatment is essential in preventing progression to more advanced sequalae. Potential problems are the development of degenerative arthritic changes, hallux limitus, and hallux rigidus. In advanced cases of aseptic necrosis complete loss of the first metatarsal head with an accompanied shortening of the great toe can occur. Once present, controlling abnormal biomechanical forces postoperatively at the level of the first metatarsophalangeal joint is essential in preventing progression of the deformity.

Aseptic necrosis of subchondral bone has also been attributed to the following: Thermal injuries, radiation therapy, metabolic disorders such as Gaucher's disease, hematologic disorders such as aplastic anemia, polycythemia , or sickle cell anemia. Long term use of corticosteroids and even nitrogen emboli have been implicated in aseptic necrosis. The use of power instruments may also have a profound influence.

Table 1 summarizes the comparison of the etiology of iatrogenic hallux limitus and iatrogenic aseptic necrosis.

There are some basic histological changes around the first metatarsophalangeal joint following hallux valgus surgery which may cause iatrogenic hallux limitus or hallux rigidus. These histological changes include traumatic synovitis, the formation of subchondral cysts, and cartilage erosion or fragmentation. Early recognition and appropriate treatment by eliminating abnormal biomechanical forces around the first MPJ are essential in an attempt to prevent propagation of the deformity.

TABLE 1

IATROGENIC HALLUX LIMITUS	IATROGENIC ASEPTIC NECROSIS
Not clearly understood.	Not clearly understood.
Extensive capsular dissection and/or excessive remodeling of bone.	Most agree following initial loss of blood supply to bone with resultant bone death followed by gradual replacement of dead bone by living bone. (Fig. 3)
Poor range of motion exercises of the 1st MPJ post operatively.	
Excessive immobility of the 1st MPJ.	
Long first metatarsal metatarsus elevatus with compressive forces at mtpj	Other known causes include: 1. Physical causes 2. Radiation 3. Metabolic 4. Hematological - Embolic 5. Excessive use of corticosteroid.
Excessive hypermobility of the first ray with functional limitation of dorsiflexion.	
Adhesions or arthrodesis of the sesamoid apparatus.	
Flattening of the shape of the metatarsal head causing excessive compressive stress on the first MPJ.	

The classification of hallux limitus may be defined in clinical terms as mild, moderate, or severe. Pathomechanical factors have been alluded to earlier in the paper and include:

a. A long first metatarsal
b. The effects of hypermobility on the distal aspect of the first metatarsal head with functional limitation of dorsiflexion.
c. The effects of metatarsus primus elevatus with its associated jamming of the proximal phalangeal base into the head of the metatarsal.
d. Adhesions or arthrodesis of the sesamoid apparatus.
e. The shape of the metatarsal head

The pathophysiology of aseptic necrosis is not well defined. However, the pathology and pathogenics are well correlated with readily identifiable radiographic changes. Salter (1970) described the four phases of aseptic necrosis. Although discussed in regard to other osteochondroses, these findings are applicable to those changes seen in the first metatarsal head affected by postoperative aseptic necrosis. The four phases of development in aseptic necrosis are often gradual and subtle and may be reversed depending on the biomechanical stresses placed upon the joint.

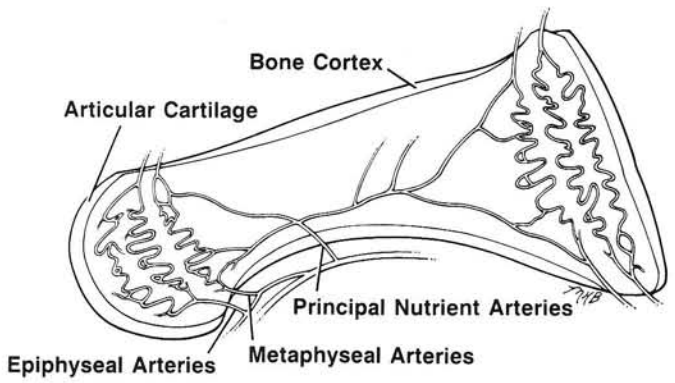

Fig. 3. Anatomical design of vascular supply of the first metatarsal.

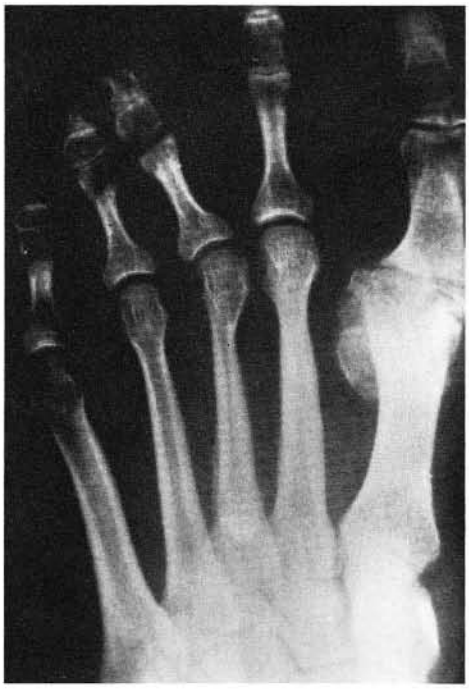

Fig. 6. Phase of bone healing characterized by early and late degenerative arthritic changes.

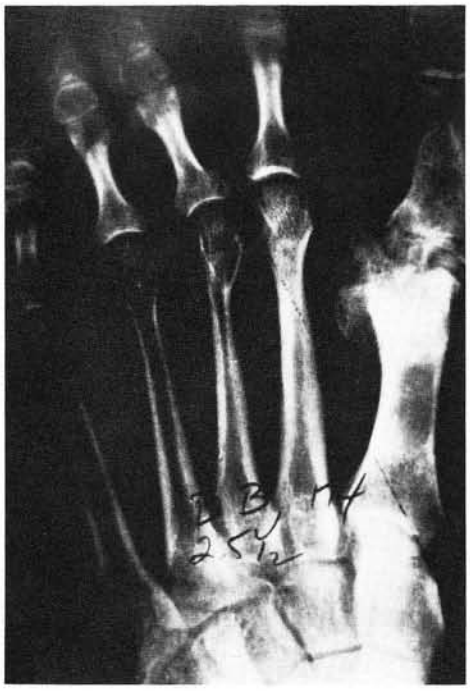

Fig. 7. Phase of deformity characterized by obvious residual deformity.

RADIOGRAPHIC FINDINGS

Hallux Limitus

Radiographic signs of hallux limitus are characteristic and often include the following:

a. Flattening of the metatarsal head.
b. First metatarsal head exostosis or osteopathic changes.
c. Irregular joint space and narrowing.
d. Osseous impingement with dorsiflexion of the great toe.
e. Early cyst formation and subchondral sclerosis.
f. Presence of a loose osseous body within the joint space.
g. Plantar articulation of the hallux on the metatarsal head in the presence of a metatarsus primus elevatus.

Aseptic Necrosis

Iatrogenic aseptic necrosis is well correlated with radiographic changes and divided into four phases:

Early Phase (Avascularity) - During the first phase the radiographs are usually normal. The radiographic density of bone remains unchanged since neither bone resorption nor bone formation can occur without a vascular supply. Nuclear bone scans during phase one frequently reveal little or no uptake within the first metatarsal head indicating necrosis. Rather than the entire head being involved, frequently only a portion of the metatarsal is involved in the development of aseptic necrosis.

Revascularization Phase With Bone Deposition And Resorption - Radiographs during phase two are characterized by bone deposition and resorption revealing increased radiodensity of the metatarsal head as new bone is laid down on dead bone. The new bone formation will be modeled into either a normal or abnormal shape depending on the biomechanical forces applied to the first MPJ during this stage. Nuclear bone scans during stage 2 reveal an increased uptake. This may involve only a portion of the metatarsal head or in more severe cases, the entire metatarsal head may be absorbed during the aseptic necrosis process. Many authors refer to this phase as the collapsed phase of aseptic necrosis.

Bone Healing Phase - Late degenerative arthritic changes are characteristics of phase three. Radiographs may reveal fragmentation and the formation of subchondral cysts with

joint narrowing indicating early signs of degenerative arthritis at the level of the first MPJ. Serial radiographs pre- and post operatively are important in providing an accurate diagnosis in these cases and determining the phase of aseptic necrosis.

Phase of Deformity - Radiographs during the phase of deformity are characterized by an obvious presence of a residual deformity. The deformity may demonstrate severe degenerative joint disease which will persist and may require additional surgical intervention.

PREVENTION

Table 2 presents the factors which help prevent the development of iatrogenic hallux limitus and iatrogenic aseptic necrosis following distal osteotomy of the first metatarsal for correction of hallux valgus.

TREATMENT

A thorough understanding of the definition, incidence, etiology, pathophysiology, radiographic findings, and factors necessary to prevent iatrogenic hallux limitus and iatrogenic aseptic necrosis is essential in assuring the most optimal postoperative result following osteotomies of the first metatarsal. Appropriate early diagnosis and recognition of these disorders is imperative in order that appropriate treatment may be provided. The treatment, whether conservative or surgical, is aimed at controlling abnormal biomechanical forces postoperatively at the level of the first MPJ with the goal of preventing propagation of the deformity.

Table 3 outlines the conservative and surgical principles as they relate to the treatment of iatrogenic hallux limitus and iatrogenic aseptic necrosis.

TABLE 2

IATROGENIC HALLUX LIMITUS	IATROGENIC ASEPTIC NECROSIS
1. Anatomical dissection.	1. Anatomical dissection.
2. Proper tissue handling.	2. Proper tissue handling.
3. Early range of motion exercises post operatively.	3. Preservation of blood supply to maintain viability of the capital fragment.
4. Decreased capsular dissection around the first MPJ.	4. Preservation of adherent soft tissues of the lateral aspect of the first metatarsal.
5. Avoid excessive remolding of the 1st metatarsal head.	5. Maintenance of good bone apposition.
6. Preserve gliding mechanism of sesamoid apparatus.	6. Appropriate internal fixation of osteotomy.
7. Control hypermobility of first ray post operatively with orthotic devices.	7. Anatomical alignment of osteotomy.
8. Avoid excessive elongation of 1st metatarsal.	8. Appropriate patient selection and execution of surgical procedure.
9. Prevent metatarsus primus elevatus using proper hinge-axis guide for base wedge osteotomy.	
10. Avoid excessive flattening of the metatarsal head.	
11. Frequent irrigation of cartilage intraoperatively to prevent necrosis of the condrocytes.	
12. Appropriate patient selection and execution of surgical procedure.	

TABLE 3

IATROGENIC HALLUX LIMITUS	VS.	IATROGENIC ASEPTIC NECROSIS
CONSERVATIVE 1. Shoe modifications. 2. Orthotic devices. 3. Rocker bottom shoes to decrease amount of MPJ dorsiflexion at heel off. 4. Molded or orthopedic shoes.		*CONSERVATIVE* 1. Shoe modifications. 2. Orthotic devices. 3. Molded or orthopedic shoes.
SURGICAL 1. Cheilectomy. 2. Green-Watermann osteotomy. 3. Opening plantarflexory wedge osteotomy. 4. Biplane Austin-McBride. 5. Keller arthroplasty. 6. Implant arthroplasty. 7. Arthrodesis first MPJ.		*SURGICAL* 1. Bonegrafting - lengthening. 2. Step down osteotomy of adjacent metatarsals. 3. Implant arthroplasty. 4. Pan metatarsal head resection 1-5. 5. Arthrodesis first MPJ.

The above surgical procedures for correction of iatrogenic hallux limitus and iatrogenic aseptic necrosis have been previously described in detail in the Podiatry Institute publications. The reader is referred to previous annual updates on *Reconstructive Surgery of the Foot and Leg* as presented at the annual surgical seminar of the Podiatry Education and Research Institute in Atlanta, Georgia.

SUMMARY

Iatrogenic hallux limitus and iatrogenic aseptic necrosis have been compared in this paper. Continued research and investigation is necessary in order to better differentiate these disorders and establish preventative measures. It is hoped that this paper might stimulate further research and investigation.

In conclusion, it is possible that iatrogenic aseptic necrosis, if not treated will worsen with time and may develop into hallux limitus or hallux rigidus depending on the biomechanical forces exerted during gait. The converse, or hallux limitus developing into an aseptic necrosis is highly unlikely. It is extremely difficult in most cases to clinically differentiate between these two disorders particularly during the early phases of development. The late stages of aseptic necrosis with degenerative joint disease may actually develop into a hallux limitus or hallux rigidus. Serial radiographs preoperatively and postoperatively may help in providing an accurate diagnosis in these cases. In many instances, the development of aseptic necrosis may go unnoticed without serial x-rays until symptoms present in the late phases of deformity. As this comparison study illustrates, treatment varies considerably depending upon the degree and stage of deformity.

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