Open Tibia Fracture with Compartment Syndrome

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Compartment syndromes in the leg associated with closed fracture of the tibia are well documented. However, the existence of compartment syndrome in open fractures of the tibia has received little recognition. It is generally believed that open fractures adequately decompress the compartments and hence prevent the accumulation of pressure. The goals of this paper are to emphasize that open tibia fractures can produce compartment syndromes and that the associated compartment syndrome greatly increases patient morbidity.

MATERIALS AND METHODS

During the period between January 1975 to January 1979, 104 open tibia fractures were treated at the University of Texas Health Science Center in San Antonio, of which six developed compartment syndromes involving all four compartments of the lower leg. During the same period, 411 closed tibia fractures were treated, five developing compartment syndromes.

Initial treatment for fractures consisted of intravenous administration of antibiotics, debridement and irrigation of the wound, followed by immobilization with the Hoffman external fixation device or a long leg cast until clinical and roentgenographic union was evidenced.

The diagnosis of compartment syndrome was based on the following clinical signs: (1) pain disproportional to injury; (2) palpably swollen compartments; (3) pain on passive stretching of the involved muscles; (4) diminished simple touch perception; (5) decreased strength of the involved compartment muscles; and (6) hypesthesias or anesthesia in the sensory distribution of nerves in the involved compartment. Distal pulses were palpable unless there was an associated vascular injury.

In each case, a four-compartment fasciotomy was executed either through combined anterolateral and posteromedial incisions or a single posterolateral incision. All wounds were initially left open. Closure was later performed by the delayed primary technique or by skin grafting. Five of the six patients were examined postoperatively for joint range of motion, sensory and motor function, drainage, and fracture union. One patient had changed residence after initial fracture care and was unavailable for personal interview.

RESULTS

Our study included six patients (five men, one woman) ranging in age from 18 to 42 years (mean, 25 years). In the five patients contacted, time from injury to follow-up was 16 months (range, eight to 25 months). No patient had a previous history of injury to the lower extremity.

Of the six fractures, four were sustained in motorcycle accidents, and two on impact with an automobile. The open wounds were small, measuring an average of 4 cm in length (range, 2 to 7 cm) and would correspond to Grade II injuries. The fibular fractures were segmental and displaced in four cases; the tibia fractures comminuted in five (Table 1). Initial examination re-
TABLE 1. Wound Debridement and Fasciotomy

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex/Age</th>
<th>Tibia Fx</th>
<th>Fibula Fx</th>
<th>Time Injury to Onset Sx</th>
<th>Time: Initial Debridement to Onset of Sx</th>
<th>Time: Onset Sx to Fasciotomy</th>
<th>Signs and Sex</th>
<th>Follow-up Time and Findings</th>
<th>Fx at F/U (Union, Nonunion, Osteomyelitis)</th>
<th>Fasciotomy</th>
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<tr>
<td></td>
<td></td>
<td>Fibula: Segmental and displaced</td>
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<td></td>
<td>DPN and SPN sensation</td>
<td>DPN, PT Motor:</td>
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<td>Pain on passive PF</td>
<td>ankle, toe</td>
<td>DF ankle and toe PF Ankle ROM:</td>
<td>Active: 0°DF-20°PF Passive: 10°DF-30°PF Clawing of toes-Yes</td>
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<td></td>
<td></td>
<td>L: Swollen tense lateral compartment</td>
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<td>L: peroneal strength</td>
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<td>P: JPTN sensation</td>
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<td>Painful passive DF toes</td>
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<tr>
<td>2</td>
<td>F/32</td>
<td>Tibia: M/3</td>
<td>Comminuted</td>
<td>2 hr.</td>
<td>5½ hr.</td>
<td>5½ hr.</td>
<td>A: Weak EHL and EDL</td>
<td>F/U: 8 mos. Sensation:</td>
<td>Union: no Combined</td>
<td>Fibulectomy: no Bone graft: no Delayed union: 8 mos., post Fx Osteomyelitis: yes</td>
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<td>Fibula: Segmental and displaced</td>
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<td>DPN and SPN sensation</td>
<td>DPN</td>
<td>Motor:</td>
<td>ankle, toe, DF Ankle ROM:</td>
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<td>Pain on passive PF</td>
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<td>Fibula: Segmental and displaced</td>
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<td>DPN and SPN sensation</td>
<td>DPN, PTN, SN Motor:</td>
<td>ankle, toe</td>
<td>DF, ankle PF</td>
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<td>L: Very weak peroneals; JSN sensation.</td>
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</table>

Wound debridement and fasciotomy simultaneously
1 *Insulin-dependent diabetic

2 *Extensive soft tissue infection resulted in debridement of peroneal (sup. and deep) nerves

Wound debridement with fasciotomy later
3
|---|------|-----------------------------------------|-------|------|--------|--------------------|--------------|---------------------|-------------------|-------------------|-------------------------------|------------|----------------|-----------------------------|---------------------|------------------|----------------------|------------------|

**Notes:**
- **M =** male
- **F =** female
- **M/1 =** mid-third
- **D/3 =** distal third
- **EHL =** extensor hallucis longus
- **EDL =** extensor digitorum longus
- **DPN =** deep peroneal nerve
- **SPN =** superficial peroneal nerve
- **PTN =** posterior tibial nerve
- **SN =** sural nerve
- **DF =** dorsiflexion
- **PF =** plantarflexion

**Abbreviations:**
- **EHL** = extensor hallucis longus
- **EDL** = extensor digitorum longus
- **DPN** = deep peroneal nerve
- **SPN** = superficial peroneal nerve
- **PTN** = posterior tibial nerve
- **SN** = sural nerve
- **DF** = dorsiflexion
- **PF** = plantarflexion
vealed no evidence of sensory deficit in the posterior tibial, sural, superficial or deep peroneal nerves. Due to pain from the comminuted tibial fractures, individual muscle strength was not gradable. However, no leg muscles were found to be totally lacking in motor function (Table 1).

Patients’ charts were reviewed to determine the time of onset of the compartment syndrome. In two cases, the compartment syndrome was diagnosed prior to debridement (Table 1). The average time between injury and onset of symptoms was two hours; the average time from onset of symptoms to debridement–fasciotomy was six hours. In the other four cases, the diagnosis of compartment syndrome was made after initial wound debridement and fracture reduction. In none of these patients was the wound closed primarily. The average time from debridement to the development of symptoms was 4.75 hours (Table 1). Fasciotomy was accomplished within eight to 17 hours (averaging 12.5 hours after the onset of symptoms).

Prior to fasciotomy, all patients were noted to have a tense, swollen leg with marked pain on muscle palpation in all compartments, and diminished sensation of the superficial and deep peroneal nerves. Five patients had decreased sensation in the posterior tibial nerve and four in the sural nerve (Table 1). Muscle testing revealed extensor hallucis longus and extensor digitorum communis function to be weak or absent in all cases. Marked weakness of the toe flexors was evidenced in five cases. Although muscle strength had not been gradable on initial examination, progressive weakness in these muscles was evident. Muscle pain on passive plantar flexion of the toes was present in all six cases, whereas pain on passive dorsiflexion was present in only four. Review of patient records revealed that serial neurologic examinations were inadequate in three cases.

Vascular surgery consultation was obtained in all six cases (Table 1). Only one patient had early loss of palpable pulses; arteriography demonstrated vessel spasm with no evidence of disruption.

When last seen, all six patients demonstrated absent dorsiflexion of the ankle and toes; five patients evidenced weakened plantar flexion. Superficial and deep peroneal sensation was decreased or absent in all six cases, posterior tibial nerve sensation was decreased but protective in five, and sural nerve sensation was diminished in four.

The mean length of hospitalization was 25 days (range, 14 to 42 days). An average of six surgical debridements per case were required (range, two to 13). The Hoffmann external fixation device was employed in five of six cases, and long-leg cast immobilization in one. Patellar tendon-bearing casts of orthoplast were employed after adequate soft tissue stability developed.

At six months’ follow-up, fractures had not healed in any of the five patients (Table 1). Fibular ostectomy, to encourage tibial union, was performed in two of the five cases, of which one healed without further surgical intervention. One patient required a posterolateral bone grafting 13 months postoperatively and subsequently healed by 19 months. All five of these cases were characterized by significant soft tissue loss and exposed bone. Chronic osteomyelitis and small draining sinuses developed in four of these cases.

Five cases had fasciotomy of the four compartments of the lower leg using combined anterolateral and posteromedial incisions. One patient had decompression through a single posterolateral incision. Delayed wound closure of the fasciotomy incisions was performed in all cases. Soft tissue loss from necrosis was severe in four cases, requiring multiple debridements in the operating room, subsequent whirlpool debridement, and eventual skin coverage with split-thickness grafts. In four cases where large areas of muscle necrosis had occurred, requiring subsequent debridement, return of ankle motion was negligible. None of the three patients with a follow-up of more than one year had active dorsiflexion past neutral
Four of the six patients now have some degree of active plantar flexion. Two patients developed equinus contractures of the foot and required heel elevation for satisfactory gait; both had significant anterior compartment injuries and were unable to actively dorsiflex the foot. None of the six patients was noted to have varus deformities of the forefoot. Clinically, clawing of the toes was evident in three of the six patients.

DISCUSSION

Volkmann, in his report of a case involving a contracture in the leg, drew attention to the development of ischemic injury following trauma as early as 1872. The key etiologic factor of ischemic injury is the development of increased tissue pressure inside closed fascial-osseous compartments, hence, "compartamental syndromes." Burton demonstrated that if tissue pressure rises, or arteriolar pressure decreases, the transmural pressure difference decreases. Blood flow decreased until, at a critical tissue pressure, there is no transmural difference, and arterioles close. This causes prolonged ischemia and muscle anoxia which in turn promote the formation of histamine substances that dilate the capillary bed and increase endothelial permeability. This increases intramuscular edema and swelling (expansion is limited by the fascial envelope), and causes a rise in tissue pressure. Thus a vicious ischemia–edema cycle develops which will ultimately result in necrosis of all elements in the involved compartment.

DIAGNOSIS

This study illustrates that, even in the presence of an open fracture, development of compartment syndromes is possible. Physical examination, including neurologic and vascular tests, immediately on arrival at the emergency room is essential for documenting a developing compartment syndrome.

The identification of the "patient at risk" will aid in early diagnosis. The minimal size of the open wound may be misleading; indeed, the small wounds may be the reason that these fractures do not spontaneously decompress. Comminution of tibial diaphyseal fractures implies a great amount of energy absorbed at this fracture site. In addition, the association of displaced, segmental fibular fractures in four of our six cases stresses the presence of high energy trauma. Displacement of the fibular fracture confirms disruption of the interosseous membrane with associated severe soft tissue injury (Figs. 1A and 1B). This allows the fracture hematoma to extend into the posterior and lateral compartments with resultant increased pressure. A displaced fibula fracture can result in peroneal artery laceration, another cause of increased compartment pressure (Fig. 1B). We conclude that a displaced, comminuted fibula fracture should suggest high energy trauma and pos-
possible compartment syndrome in patients with open tibia fractures. (Fig. 2).

Several methods have been advocated to accurately determine interstitial fluid pressure and ultimately diagnose compartment syndromes in the clinical setting. However, tissue pressures were not used in our study. It was decided that increasing pain in muscle groups that was exaggerated with stretching, a definite loss of sensation, and a decrease in muscle strength as noted by the physician were sufficient to establish the diagnosis in these cases. In all six of our patients, results from the initial sensory examination were normal. Muscle strength, although initially ungradable due to pain at the fracture site, had decreased prior to fasciotomy.

It is recognized that diminished arterial pulses are seen late in the development of compartment syndromes. Rarely does the tissue pressure rise high enough to completely obliterate the pulses. One patient in our study had early loss of palpable pulses; however, an arteriogram revealed vascular integrity. We believe that the absence of pulses should be an absolute indication for arteriography.

TREATMENT

In five cases, fasciotomy was performed through two incisions. The two posterior compartments were released through the medial incision, and the anterior and lateral compartments through the lateral incision (Fig. 1A). Mubarak and Owen recommend that both incisions be 15 cm long; however, we feel that the fascia should be released through the entire leg length. Incisions shorter than 15 cm may result in inadequate decompression. Gaspard has shown that with massive swelling the skin itself may become constricting. In one patient, we released all four compartments through a single posterolateral incision as described by Matsen. Epimysiotomy, sometimes necessary to decompress tight, individual muscles, was utilized in four cases in this series.

Due to the swelling which occurs after fasciotomy, all six cases required split thickness skin grafting to cover the gaping wounds.

Fibulectomy–fasciotomy and decompression of all four compartments through a lateral approach has been recommended; however, we think that the presence of an intact fibula may be necessary for later posterolateral bone grafting. The peroneal and posterior tibial arteries are more vulnerable in this approach and, if injured, could cause further ischemia.

Duration of hospitalization is usually less than two weeks for uncomplicated open tibia fractures. No patient in this series was discharged earlier than 14 days; the majority required more than three weeks of treatment. The most important factor prolonging
Compartment Syndrome

Problems

Ischemic Necrosis

Harmon and Guinn13 and Whitesides et al.32 found that 90% of muscle fibers evidence injury after eight hours of ischemia. Sheridan and Matsen99 have reported 92% residual functional impairment after 12 hours of ischemia. In our series, ischemia for a duration longer than six hours led to irreversible changes and permanent necrosis of tissues. Our poorest results occurred in the four patients where fasciotomy was performed after the initial debridement (there was an average delay of 12.5 hours from onset of clinical symptoms to fasciotomy). Patients evidenced marked sensory deficit and a total active motion at the ankle of less than 20°. In all patients, active dorsiflexion of the toes or ankle was absent, and plantar flexors either weak or absent. Such losses can be secondary to direct muscular injury at the time of fracture or to ischemic necrosis postfracture. Inability to document the degree of muscle weakness in a fractured limb makes it difficult to determine the cause of postfracture motor weakness.

Neurologic Deficit

Functional neurologic changes occur after 30 minutes of ischemia, and irreversible changes after 12 to 24 hours.16,24 Although permanent sensory changes resulted in all six patients in this series, they were more severe in the four who evidenced ischemia 12.5 hours (average time) before decompression. Of the other two, one case had severe involvement of the anterior and lateral compartments in which both the superficial peroneal and deep peroneal nerves were necrosed and subsequent debridement required. In all patients, plantar sensation was sufficient to prevent ulceration.

Open Wounds

The effects of creating large open wounds in grossly unstable tibial fractures should not be taken lightly. The posteromedial incision employed to decompress both posterior compartments may have contributed to the morbidity rate in this series. In open fractures, the anteromedial skin is often compromised, and fasciotomy in this area may lead to ad-
ditional skin necrosis. Moreover, if the skin incision is too long, much of the soft tissue support is lost. The muscles arising from the posterior tibia will tend to "hang" and often pull away from the tibia. This results in further compromise of bone circulation and exposure of more bone as was seen in four of the five patients who had a double incision fasciotomy. In the one patient who had decompression performed through the posterolateral approach, soft tissues did not tend to "hang" off the tibia, exposed bone was not a problem, and union occurred in 16 months without difficulty. We have since used this approach on three patients and have found it to be much easier to manage.

Skin coverage of swollen, edematous muscle and soft tissue becomes preeminent after limb viability is assured. Of greater importance is coverage of bone at the fracture site. We found necrosis of skin and muscle at the level of the fracture site to be present, particularly in the four cases with prolonged ischemia from the compartment syndrome. In this situation, multiple operative debride-
ments were necessary to remove all nonviable tissue before a granulating bed capable of accepting a skin graft could be established. Since the tibia is subcutaneous in most of its diaphyseal portion, debridement of the anterior compartment leaves most of the lateral aspect of the tibia exposed. Hence, exposed bone was drilled to encourage granulation tissue formation as a base for later skin grafting. After soft tissue healing and bone coverage were achieved (usually by six weeks), the Hoffmann device was discontinued and full weight bearing was commenced. Once marked stability was regained, an orthoplast gaiter was employed until union occurred.

Skeletal Stability

The Hoffmann external fixation device obviated the problem of fracture instability. Since many patients were debrided on a daily basis early in the course of treatment, the external fixator simplified measures dramatically. We were unwilling to use internal fixation in the face of these grossly contaminated wounds.

Delayed Union

Nicoll\textsuperscript{21} notes that displacement, comminution, loss of bone, infection, and soft tissue wounds are the main determinants causing an increase in the rate of delayed or nonunion. In our study, union was delayed over six months in all patients with follow-up. This rate is greater than in most series whereby delayed union in open tibia fractures treated by external means varied from 10% to 33%.\textsuperscript{13} The amount of soft tissue injury and the fasciotomy itself were major contributors to delayed healing. In five patients, the bone ends were devoid of all soft tissue for up to 11/2 inches. This degree of soft tissue disruption about the fracture site must play a significant role in decreasing the vascularity of the bone ends.

Posterolateral bone grafting was done late in one case and proved successful. At present, one patient (Case 4) is under treatment with a transcutaneous electrostimulation unit. Two patients are still in orthoplast gaiters (Case 2 and Case 5).

Our approach to delayed union has been to perform a fibulectomy early (within six months), particularly if we can demonstrate hypertrophic callus or the possibility of a healed fibular fracture distracting the fracture site. Fibulectomy, a simple and effective method for enhancing compression at the fracture site, was successful in treating one of two cases in this series.

Infection

Patzakis and Harvey\textsuperscript{25} demonstrated that open tibia fractures treated with Cephalothin became infected in 2.3% of cases. We discontinued intravenous antibiotics (Cephalothin) early as we felt the presence of local commensal organisms would be preferable to more resistant strains. Although no patient in our series required treatment for an acute wound infection, 66% of our cases (four out of six) harbored infections
as evidenced by chronic osteomyelitis with draining wounds. This high incidence of osteomyelitis underlines the severity of the soft tissue and bone injuries.

**Functional Deficit**

Ankle stiffness and decreased range of motion occurred in all patients. One patient had passive range of motion of 10° dorsiflexion to 30° plantar flexion, but active motion from neutral to 20° plantar flexion. The other patients had less than 20° of active or passive total motion and two of these patients had fixed equinus contractures. One may ask whether or not internal fixation and early motion may have prevented the degree of ankle stiffness in our patients. Nicoll\(^1\) found that severity of soft tissue damage with fibrosis of muscles, ligaments, and fascial planes was more important than immobilization in causing residual stiffness. It is also important to emphasize that muscle strength improvement due to hypertrophy and regeneration, will continue for up to one year postinjury.\(^15\) This was encouraging to the patients in our series (two have been followed for less than one year).

In spite of the complications (nonunion, infection, limited range of ankle motion, and loss of muscle function) no amputations have been indicated. To our knowledge no other series of patients has been reported with compartment syndromes involving all four compartments in the lower extremity. However, Matsen and Clawson\(^17\) reported two cases of four-compartment syndrome, one of which had not recovered at two months and the other required amputation. This reinforces the fact that four-compartment syndromes are high-energy injuries. The prognosis for significant recovery, especially with delay in diagnosis, is poor.

**Summary**

In a series of 104 open tibia fractures, six patients developed compartment syndromes involving all four compartments of the lower leg, four of which developed after initial debridement and reduction. The presence of an open tibia fracture with a displaced, comminuted, fibula fracture should suggest the possibility of a developing compartment syndrome.

Clinical symptoms include: increasing muscle pain which can be exaggerated by stretching; loss of sensation; decrease in muscle strength; and palpably swollen compartments. Double fasciotomy incisions may lead to adequate decompression, but result in marked loss of soft tissue support for the fracture. To sustain stability, a single posterolateral incision is recommended for compartmental decompression.

**References**