

## Compartment Syndrome in Tibial Plateau Fractures: Educational Corner

Amirhossein Karim<sup>1</sup>, Salar Baghbani<sup>1</sup>, Yousef Fallah<sup>2</sup>, Ehsan Pendar<sup>2</sup>, Seyyed Hossein Shafiei<sup>1,2,\*</sup>, Mohammadreza Golbakhsh<sup>3</sup>

<sup>1</sup> Resident, Department of Orthopedics, Sina Hospital, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran

<sup>2</sup> Assistant Professor, Department of Orthopedics, Sina Hospital, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran

<sup>3</sup> Associate Professor, Department of Orthopedics, Sina Hospital, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran

\*Corresponding author: Seyyed Hossein Shafiei; Department of Orthopedics, Sina Hospital, School of Medicine, Tehran University of Medical Sciences, Tehran, Iran.  
Tel: +98-9126113990, Email: dr\_hshafiei@yahoo.com

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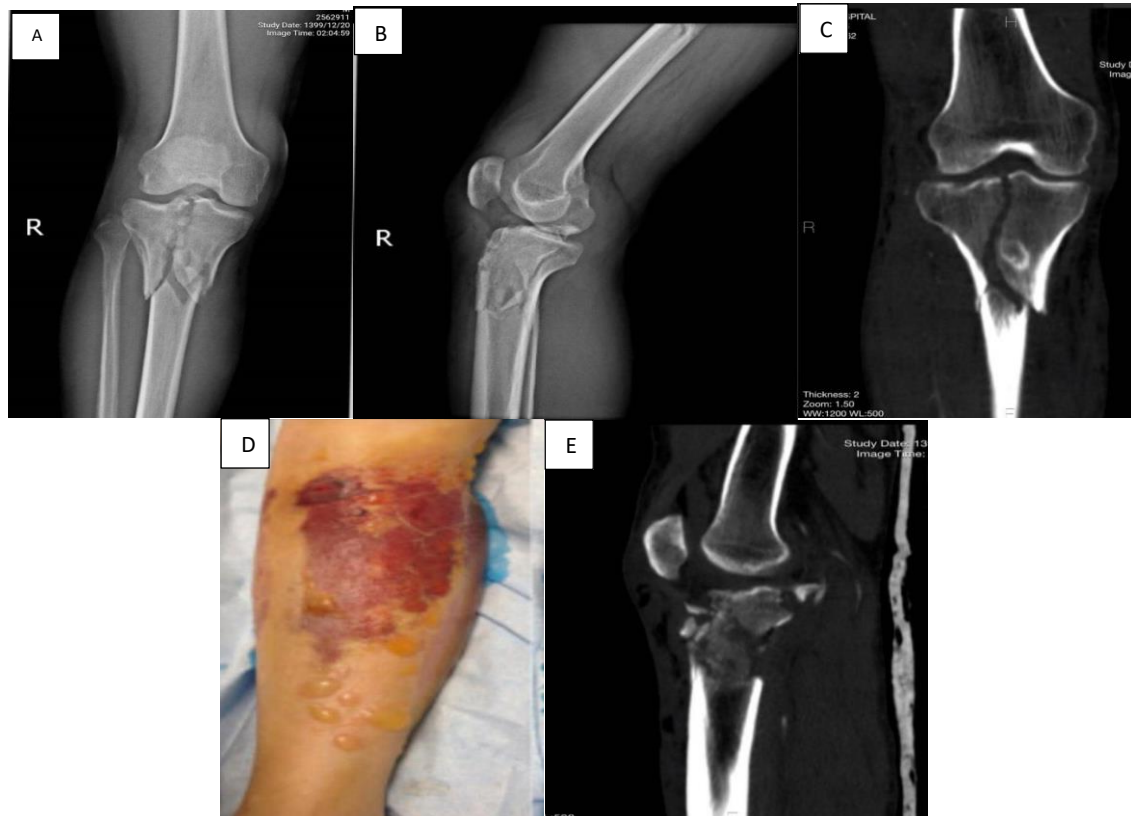
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### Background

Tibial plateau fractures consist 1% of all fractures and are one of the most challenging intraarticular fractures to fix as an orthopedic surgeon (1, 2). Several classification systems have been developed to categorize the tibial plateau fractures and until recently, Schatzker, Hohl and Moore, and AO classifications have been the most popular systems among the 38 classification systems introduced so far (3-9). Most recently, new classifications have been introduced based on the computed tomography (CT) scan evaluations and the core idea is dividing tibial plateau

biomechanically to three main columns (9, 10).

Tibial plateau fractures occur in both high and low energy traumas and therefore may appear as innocently undisplaced fractures or catastrophic as in accompanying knee dislocations and vascular injuries leading to amputation (8, 11-13). During the trauma episode, a remarkable amount of energy is delivered to soft tissue, resulting in a wide range of injury, among which acute compartment syndrome (ACS) is one of the most challenging issues (14) (Figure 1). Altogether 12% of tibial plateau fractures lead to compartment syndrome, this figure rises up to 53% in high grade Schatzker subgroups (15-19).



**Figure 1.** (A, B) Anterior-posterior and lateral radiographs of unstable, displaced, high-energy bicondylar tibial plateau fracture. (C, D) Coronal and sagittal computed tomography (CT) cuts. (E) Associated with swelling and blistering of the surrounding soft tissues

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Young men with long bone fractures are statistically the most group in danger for experiencing ACS, however it may occur in soft tissue injuries whose bones are intact. This highlights the role of soft tissue edema and bleeding, which disrupts the perfusion in an injured limb and begins the process ending in ACS (20). The increased swelling on the one hand and inelasticity of the myofascial membrane on the other hand transmit the pressure to veins and lead to venous hypertension, which can be evaluated manually or mechanically. During the process, cellular lysis rises the interstitial fluid accumulation by osmotic pressure change and eventually ends in microvascular collapse (20-22). Within 120 minutes of this chain of entities, myonecrosis may take place and after 6 to 8 hours irreversible ischemia may result in permanent neurovascular damage (23-25).

Emergent fasciotomy is necessary in even doubtful conditions to prevent unappealing complication, but the fasciotomy itself makes troubles in the definite treatment of the fracture by possibly limiting the available approaches to the bone (26-28).

The objective of this study was to assess the relation between key demographic, injury-related, clinical, and radiographic factors in patients with tibial plateau fractures and the subsequent development of ACS. Diagnostic challenges, management options, and surgical complications of ACS were also reviewed in this study.

#### **What Are the Main Risk Profiles for ACS?**

General surgeons have a traditionally developed idea that prefers a remarkable rate of false positive appendicitis just to avoid the following morbidities with missed patients. Here we feel the same happens with orthopedic surgeons and ACS. As a first line physician, it is unforgivable to miss or neglect the initial occurrence of ACS (29). Some potential indicators for ACS in tibial plateau fractures have been identified as higher Schatzker and AO/OTA grades, raised tibial widening ratio (TWR), increased length of fracture, concurrent fibular fracture, increased femoral displacement ratio (FDR), and muscle diameter behind the plateau (15, 16). In this regard, the risk profile for ACS following the tibial shaft fracture have been revealed to be younger ages, male gender, mechanism of injury, trauma with higher force, and the existence of open fracture (30, 31). In this regard, increasing the likelihood of ACS in men aged lower than 29 years has been reported (32-34). It has been also revealed that occurring simultaneous knee dislocation or non-contiguous tibia fracture can be other main determinants for extended musculoskeletal damage. It causes high-energy traumatic events that may ultimately predict ACS development (35, 36).

#### **What Are the Common Mechanisms of Injury?**

Tibia plateau fractures result from a variety of injury mechanisms as mentioned above, but obviously a direct impact to the knee joint is necessary to maintain the disruption of the articular line (36, 37). Knee joint is strengthened by several intra and extra articular anatomic structures, which regarding the tibial plateau fracture, the lateral collateral ligament in the lateral side and medial collateral ligament in the medial side play the most important role to prevent any subluxation of the joint in the coronal plane (9).

Mostly, valgus or varus impactions to an extended knee accompanying an axial force is the cause of a fracture. Valgus impaction results in lateral tibial plateau fracture or Schatzker type 1, 2, and 3. Type 1 usually happens in young aged patients with a better bone quality

and type 2 and 3 are expected to happen in minor bone densities which cannot stand the axial force. Recently, some studies, have mentioned the very rare occurrence of the second Schatzker type (38-40).

Applying energy in the anterior-posterior direction imposes a lot of pressure on the bent knee, injuring the posterior aspect of tibial plateau (6, 8).

Medial tibial condyle is stronger than the lateral, so fractures consisting the medial side as in Schatzker 4, 5, and 6 represent a higher amount of energy and have a greater rate of soft tissue injury. If a knee dislocation takes place, rotational injuries co-exist and ruptures of the intra and extra articular structures such as menisci and the collateral ligaments are possible (34).

#### **Problems Related to Injury Diagnosis**

Delay in injury diagnosis is a major determinant for poor prognosis. Despite limb-threatening nature of ACS, early detection of this complication has always been difficult. However, different approaches have been proposed for the diagnostic management of this complication (41-43); however, late and incorrect diagnosis of the injury has had irreparable consequences for the patient and the treatment staff (44). However, some clinical evidence has been consistent with the diagnosis. For instance, worsening pain along with passive stretching of the involved muscle and paresthesia in the path of the sensory dermatomes can help in the definitive diagnosis. Overall, a combination of clinical manifestations has not been sensitive for diagnosing or screening ACS (26). Early detection will inevitably be associated with a reduction in morbidity (45, 46). Although it has been emphasized that ACS is an orthopedic emergency, there is always a delay in evaluating and diagnosing patients and therefore designing a treatment plan (46). However, delay in diagnosis can be reduced by continuous assessment of intramuscular pressure (IMP) (46, 47), however IMP monitoring as a helpful method for ACS detection remains controversial and even some studies refuse its helpfulness in pressure monitoring (47). Moreover, single pressure measurements are not clinically applicable and in fact, assessing the trend of pressure changes will be essentially useful in diagnosis. In this context, it has been shown that detecting upward trend in ACS or falling perfusion pressure can be specific for definitive diagnosis of ACS (48). Some authors pointed continuous pressure monitoring as a gold standard for timely diagnosis of ACS. In this approach, by using "a threshold for fasciotomy related to the perfusion pressure (intramuscular pressure within 30 mmHg of the diastolic blood pressure for two consecutive hours or more)", the sensitivity for ACS diagnosis reaches to 94% (49).

Besides, it should be considered that ACS misdiagnosis may be caused by some underlying confounders such as concomitant nerve injuries leading to loss of sensation. Such factor particularly in older affected adults or unconscious or regionally anaesthetized patients is very misleading to diagnose ACS. But in conscious patients, the onset of pain commensurate with the severity of the injury, which is not usually cured through anesthesia, can be a reliable and applicable indicator of ACS (49, 50). With all these descriptions, diagnosis of this complication in unconscious or intubated patients is difficult and becomes more problematic in the patient with communication problems and concomitant nerve injuries with loss of sensation. Nevertheless, the diagnostic value of IMP monitoring remains questionable (29).

### Treatment Approaches for ACS

Immediate surgical fasciotomy is followed by releasing the skin and muscle fascia to elevate the volume of muscle compartment in order to decrease pressure of compartment in the treatment fundament (51). Despite the pointed surgical approach, the gold standard for ACS treatment, in some affected cases, conservative measures can be the first-choice approach. In the latter approach, musculoskeletal regimens including resting, using nonsteroidal anti-inflammatory drugs (NSAIDs), strengthening and orthotic therapy could be helpful. In cases with no improvement within 6 to 12 weeks of conservative treatment, surgical treatment is indicated (52). Lower leg fasciotomy approaches have been also recommended in the literature. In this regard, the approach of "2-incision, 4-compartment" could be therapeutically efficient (53). Creating a linear longitudinal cut in the incision between the anterior crest of the tibia and fibula can facilitate accessing the anterior and lateral muscle compartments through the intermuscular septum. For more access to the pointed part, another incision can be created about 2 cm posterior to the palpable aspect of the tibia. Before appearing tissue necrosis, fasciotomy should be performed as soon as possible. In this regard, reviewing the literature indicates efficacy of early fasciotomy and in contrast, potential complications of late fasciotomy (38). Even unnecessary fasciotomy can be more appropriate than late fasciotomy, because the latter action provides an opportunity for serious complications (myonecrosis or rhabdomyolysis), delaying recovery, and even loss of limb function (40).

### Complications of Surgical Fasciotomy

In spite of high effectiveness of treatment with fasciotomy in ACS treatment, some related complications exist. These additional measures should be considered to repair delayed wound closure, skin grafting, fixing cosmetic problems, improving persistent pain, repairing local nerve injuries, eliminating muscle weakness, and improving chronic venous insufficiency (54-57). To reduce infection risk following tibial plateau fractures, staged management with spanning external fixator followed by definitive fixation is frequently considered, while prior to such an approach, the infection rate was estimated to be even more than 80% (37).

The likelihood of the infection problems is closely linked to damaged soft tissue, extreme traction exposure, as well as prolonging surgical procedure. Moreover, time of fixation related to closing the fasciotomy seems not to be affecting the incidence of infection, soft tissue enveloping before fixation is highly recommended to minimize the risk for infection (58). Additionally, despite compartment release in essential with the aim of salvaging the extremity as well as preventing muscle necrosis, opening up a large area of the limb to reduce risk of in-hospital infection is recommended (59).

### Conclusion

Given the life-threatening nature of ACS, prompting action to diagnose and determine the best treatment approach for it is essential to achieve the best prognosis. In this regard, timely fasciotomy, repair of bone fractures, relieving pressure on adjacent soft tissue, as well as repair of possible treatment-induced lesions such as local nerve damage and wound healing, lead to improve treatment outcome and reduce post-treatment infection rate that ultimately reduce debilitating post-procedural complications.

### Conflict of Interest

The authors declare no conflict of interest in this study.

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