Case Report

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Supraspinatus and infraspinatus compartment syndrome following scapular fracture

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ABSTRACT

Acute compartment syndrome occurs when pressure within a confined fascial space rises to a level impairing microvascular perfusion to surrounding tissues.[1-7] The majority of the reported literature is based on lower extremity compartment syndrome, but any muscle group within an osteofascial compartment has the potential to develop compartment syndrome. We report a case of a 64-year-old male who developed an acute compartment syndrome of both the supraspinatus and infraspinatus after sustaining a severely comminuted scapula fracture. Diagnosis of compartment syndrome was made after intracompartmental pressure measurements of the supraspinatus and infraspinatus revealed pressures within 30 mmHg of the diastolic blood pressure, prompting emergency decompressive fasciotomy. At final follow-up, the examination revealed full shoulder strength with near-full range of motion. There were no signs of sequelae from compartment syndrome at any point. Few case reports describe compartment syndrome of the periscapular fascial compartments. However, these cases were either retrospectively diagnosed[8,9] or diagnosed via magnetic resonance imaging (MRI) findings and lab values.[9,10] Surgical management of acute compartment syndrome of the supraspinatus has been reported in only one other case.[10] To our knowledge, we report the only case of a patient with acute compartment syndrome of both the supraspinatus and infraspinatus compartments treated with emergent decompressive fasciotomy. Due to the devastating complications and functional loss of a missed diagnosis of compartment syndrome, a high index of clinical suspicion for developing compartment syndrome must be maintained in every fracture setting, regardless of anatomic location or rarity of reported cases.

Key words: Supraspinatus, infraspinatus, compartment syndrome, scapula fracture

INTRODUCTION

Compartment syndrome has been defined as any condition in which pressure build-up within closed osteofascial space compromises tissue circulation and function.[1-7] The resultant ischemia can quickly cause irreversible muscle and nerve damage leading to musculotendinous contractures and sensorimotor deficits.[7] Urgent diagnosis and surgical treatment with decompressive fasciotomy is the key to optimizing functional outcome.[1,5,8] Diagnosis of compartment syndrome heavily depends on clinical presentation and becomes increasingly difficult in patients who are unconscious, intoxicated, or have altered mental status. Measurement of compartmental pressures using various devices has been shown to help facilitate diagnosis in such patients.[2,4]

Compartment syndrome of the upper extremity has been extensively described and reported in the hand, forearm, and upper arm, with the forearm most commonly affected.[8-11] A literature review returned only 4 other case reports termed compartment syndrome of the periscapular fascial compartments of the supraspinatus or infraspinatus.[12-14] Some authors suggest that the rarity of upper extremity and shoulder region compartment syndrome may be due to

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the blending of fascial communications seen between the infraspinatus, deltoid, pectoralis, and brachial fascia, which may help to dissipate pressure build-up.[9-11]

To our knowledge, we report the only case of a patient with a comminuted scapular fracture that developed an acute compartment syndrome of both the supraspinatus and infraspinatus, diagnosed via intracompartmental pressure measurements and treated with emergent decompressive fasciotomy.

CASE REPORT

A 64-year-old male involved in a motor vehicle collision presented with left shoulder and facial pain. Examination revealed a swollen, soft shoulder [Figure 1] with scapular and acromioclavicular (AC) joint tenderness and painful motion. X-rays showed a comminuted scapular body and spine fracture extending to acromion base with AC joint widening [Figures 2a, b and 3]. The patient's airway later became compromised secondary to facial fracture swelling, requiring intubation.

Later, evaluation revealed increased swelling and tenseness over the left scapula. Supraspinatus and infraspinatus compartment pressures were measured at 49 mmHg and 32 mmHg, respectively, with a diastolic blood pressure of 43 mmHg.

Fifteen hours after presentation, emergent fasciotomy was performed.

The incision extended over the scapular spine similar to a modified Judet incision.[15,16] Upon release, the supraspinatus and infraspinatus compartments showed good muscle escape, beefy red coloration, adequate bleeding, and contracture with stimulation [Figure 4]. Elevated serum myoglobin levels prior to fasciotomy were 2705 ng/ml, which declined to 1221 ng/ml and 630 ng/ml on postoperative day #1 and #2, respectively. The combination of scapular spine, acromion base, and AC joint separation prompted plans for open reduction internal fixation (ORIF),[17] which were delayed until hospital day #13 due to pneumonia [Figure 5]. Twenty-week post-op examination revealed active shoulder motion of 50° requiring removal of the AC plate and manipulation under anesthesia. Final 10-month follow-up revealed full strength and near-full flexion and abduction with complete radiographic bony healing. There were no signs of sequelae from compartment syndrome at any point.

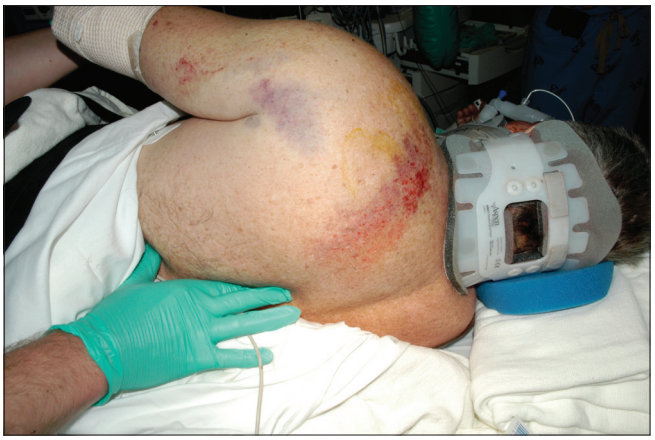

Figure 1: Posterior view of left shoulder showing extensive swelling, ecchymosis, and abrasion

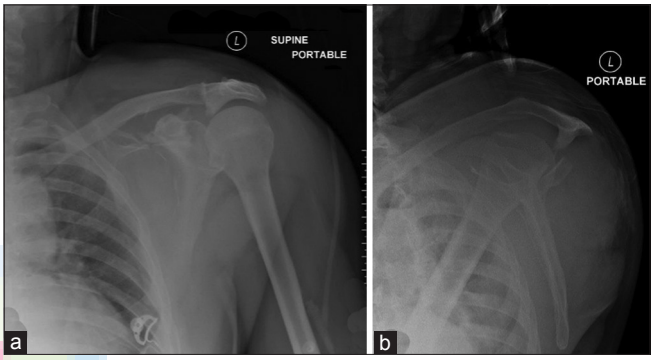

Figure 2: (a and b) AP and Lateral plain radiographs of Left shoulder injury demonstrating AC joint widening, acromion base fracture, scapular spine and body fracture

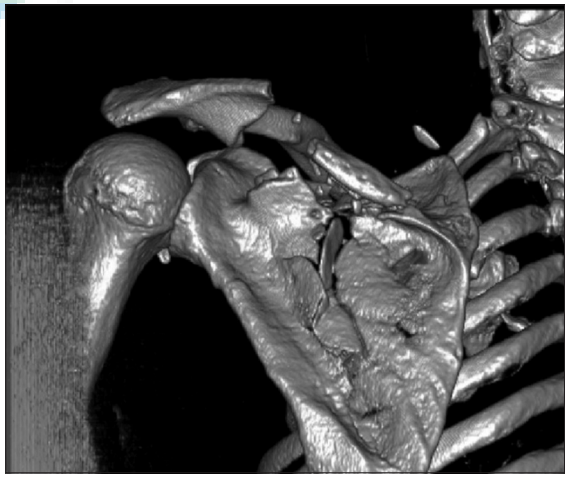

Figure 3: 3D reconstruction CT scan of left scapula showing segmental fracture of scapular spine and extensive comminution of scapular body

DISCUSSION

To our knowledge, we report the only case of a patient with a comminuted scapular fracture that developed an acute compartment syndrome of both the supraspinatus and infraspinatus, diagnosed via intracompartmental pressure measurements and treated with emergent decompressive fasciotomy. The diagnosis of compartment syndrome is frequently a clinical diagnosis based primarily on findings of pain out of proportion and pain on passive stretch of involved muscles.[1-7] The unconscious or obtunded patient at risk of compartment syndrome presents a difficult challenge for the physician in that these findings are either unreliable or unobtainable. The use of additional diagnostic modalities of intracompartmental pressure monitors has been found to be useful in such patients.[2,4,6] The unfamiliarity and rarity of

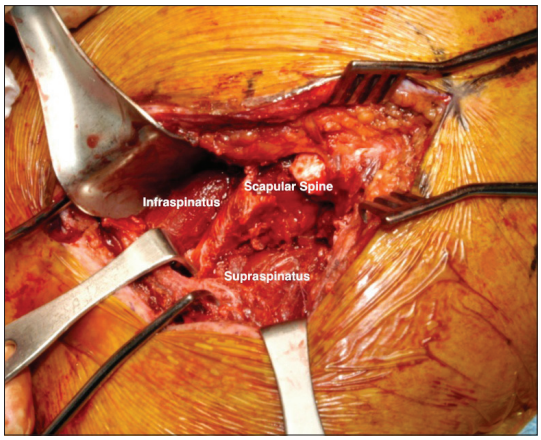

Figure 4: Intraoperative view after fascial release of the infraspinatus and supraspinatus

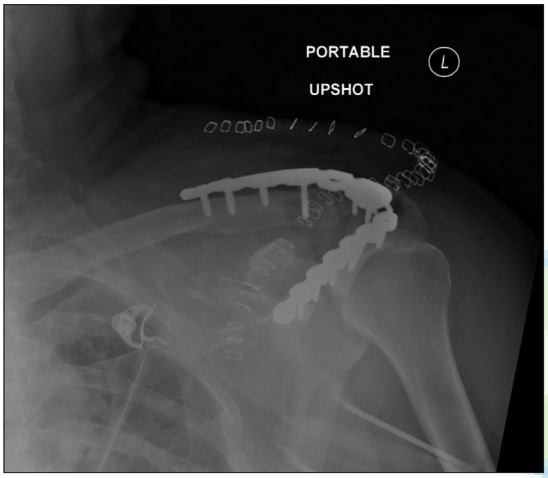

Figure 5: Postoperative AP shoulder plain radiograph after scapular spine and AC joint fixation

reported scapular compartment syndrome requires adherence to thorough physical examination, a high index of suspicion, and when necessary, the utilization of compartment pressure monitoring to prevent missed diagnosis.

In 1992, Landi *et al.* reported the first two cases of scapular compartment syndrome.[12] The first case described a patient with extensive necrosis of the infraspinatus encountered during exploration of a non-healing pressure ulcer. The authors attributed this finding to possible ischemia from unrecognized compartment syndrome. The second case described a patient with a scapular fracture with increasing pain and swelling. Intracompartmental pressure measurements revealed an absolute pressure of 75 mmHg, which continued to decline on subsequent measurements. Fasciotomy was cancelled due to dissipating pressures and resolving symptoms. Follow-up demonstrated full motion, and absence of pain with no sequelae of an untreated compartment syndrome.

The diagnosis of compartment syndrome based on absolute pressure measurements alone has been challenged in several studies and its acceptance has diminished.[6,18,19] Prayson *et al.* challenged accepted thresholds and formulations for diagnosing compartment syndrome.[19] Their study measured compartment pressures in 19 lower extremity fractures, all lacking clinical signs of compartment syndrome. Ninety-five percent of patients had at least one compartment measurement >30 mmHg and 84% had measurements within 30 mmHg of diastolic pressure. No patient developed sequelae for untreated compartment syndrome. To our knowledge, there is no similar study to that of Prayson *et al.* involving baseline upper extremity fracture compartment measurements. Further study may be useful to help guide treatment and evaluate injury location as a possible variable in the development of compartment syndrome.

Compartment syndrome is not only the result of an increased volume within a fixed space, but rather the body's inability to compensate for a change in the microvascular pressure gradient within a compartment. Once pressures have exceeded capillary perfusion pressure, the ability to exchange oxygen and clear waste is hindered. The resolving clinical signs and lack of sequelae despite the elevated compartmental pressures as seen in Landi *et al.*'s case further substantiates that compartment syndrome is not purely related to an increase in volume but involves other confounding variables.

The relationship between the measured compartmental pressure and diastolic pressure gives a more accurate insight into the dynamic compartmental environment. This pressure differential value, as originally described by Whiteside *et al.*[6] as significant when absolute pressure measurement is within 30 mmHg of the diastolic pressure, has become more widely accepted as the diagnostic criterion compared to the use of an absolute pressure alone. As seen in our case, patients with hypotension have lower intravascular pressures and may have inadequate tissue perfusion even at mild increases in intracompartmental pressure.[2,6,7] A normotensive or hypertensive state in our patient may have been protective against rising compartment pressures.

Stahlfeld *et al.*[14] described a patient with severe shoulder pain that failed to respond to analgesics. The patient denied any history of trauma, but admitted intensive upper extremity weight lifting prior to a plane flight. Diagnosis of compartment syndrome was made based on magnetic resonance imaging (MRI) findings of an enlarged and edematous supraspinatus. Fasciotomy was performed with hypercontracted and necrotic myofibers shown on biopsy. EMG 15 months after insult demonstrated persistent injury to the supraspinatus with shoulder abduction limited to < 30°. Takakuwa *et al.* presented a similar case of a patient with shoulder pain after performing bayonet-thrusting exercises while wearing tight-fitting shoulder equipment.[13] The authors made a delayed diagnosis of compartment syndrome based on MRI findings of diffuse supraspinatus edema, elevated serum creatinine phosphokinase (CPK) and lactate dehydrogenase. The patient's symptoms improved with non-operative

Table 1: Literature review of supraspinatus or infraspinatus compartment syndromes

Author	Date	Mechanism	Diagnosis	Treatment	Reported outcome
Landi *et al.*[12]	1992	Case 1: Unconscious pt with prolonged pressure against hard surface	Case 1: Intraop biopsy with infraspinatus necrosis	Case 1: Muscle Debridement	Case 1: None reported
		Case 2: MVC, scapular fracture	Case 2: Absolute infraspinatus compartment pressure measurement	Case 2: Observation	Case 2: 26 month follow-up with full ROM, no pain
Takakuwa *et al.*[13]	2000	Fencing, tight shoulder equipment	MRI with enlarged supraspinatus, elevated CK and LDH	Rest, Ice, NSAIDS	Full recovery and strength at 11 months
Stahlfeld *et al.*[14]	2004	Weight lifting, flying	MRI with enlarged supraspinatus	Fasciotomy	EMG at 15 months showed persistent muscle and nerve injury; Abduction limited to <30 deg

treatment. Full recovery of motion and strength was seen 11 months after injury.

Similar MRI findings between Stahlfeld *et al.*'s and Takakuwa *et al.*'s patients were observed; however, the latter lacked uncontrollable pain and clinical sequelae of compartment syndrome, making the diagnosis of "severe overuse synfrome of the supraspinatus" , as described by Graves *et al.*,[20] a more likely diagnosis. Graves *et al.* made two diagnoses based on elevated creatinine kinase (CK) levels and MRI findings of marked swelling and high-intensity supraspinatus signaling, similar to those of Takakuwa *et al.*[13] and Stahlfeld *et al.*[14] Both patients showed good functional recovery and symptom relief with non-operative treatment. The authors chose to consider these cases "overuse syndromes with rhabdomyolysis" instead of compartment syndrome due to the absence of tense compartments, improved symptoms without fasciotomy, and lack of compartment syndrome sequelae.[20]

Four cases, termed either infraspinatus or supraspinatus compartment syndrome, have been reported in the English literature [Table 1]. However, several of these cases lacked clinical compartment syndrome signs of tenseness and swelling, were diagnosed via MRI and lab values, or were successfully treated non-operatively. As suggested by Graves *et al.*, a diagnosis of overuse syndrome may better suit some initial reported diagnoses of supraspinatus compartment syndrome, as nonsurgical management may be used to adequately treat overuse injuries of the supraspinatus that result in rhabdomyolysis and not compartment syndrome. To our knowledge, we have described here the first case of both supraspinatus and infraspinatus compartment syndrome and its successful surgical treatment with decompressive fasciotomy. Due to devastating complications and functional loss of a missed diagnosis of compartment syndrome, a high index of clinical suspicion for developing compartment syndrome must be maintained in every fracture setting, regardless of anatomic location or rarity of reported cases.

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