



Crush Injury and Extremity Compartment Syndromes

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Abstract

Purpose of Review The purposes of this review are to define crush injury and crush syndrome and describe how it relates to extremity compartment syndrome. This review will also describe surgical interventions required once compartment syndrome has been identified and discuss the outcomes and complications of both timely and delayed surgical management.

Recent Findings The management of crush syndrome has evolved to push aggressive fluid resuscitation as early as feasible, including into the field. Developments regarding compartment syndrome are primarily exploring new tools to facilitate the early identification of compartment syndrome.

Summary Two important and clinically relevant sequela of a crushing traumatic injury are crush syndrome and compartment syndrome. The outcomes of both syndromes are greatly improved with early recognition and treatment. There are several different clinical and diagnostic ways to identify compartment syndrome that can be tailored to the clinical setting; however, surgical intervention to release the compartments should be performed immediately once there is a high suspicion as waiting can lead to devastating consequences.

Keywords Crush syndrome · Compartment syndrome · Crush injuries

Introduction

Crush syndrome was initially described during the Blitz of London. The syndrome was characterized after observing several patients who had no external signs of injury succumb to acute renal failure, anuria, proceeding to hypotension, and eventually death [1]. The understanding of crush syndrome has grown overtime mostly through clinical experience during times of war and disasters [2]. Crush syndrome often coincides with compartment syndrome and outside of natural disasters and wartime these syndromes occur concurrently in patients that suffer of multi-system trauma [3]. In this review, we will discuss the underlying pathophysiology of crush

syndrome and compartment syndrome and describe the current diagnostic and treatment algorithms of these syndromes.

Crush Syndrome

The association between crush injuries and kidney failure was first documented by Bywaters and Beall in 1941, who had extensive exposure to patients crushed by falling debris during the bombing of London [1]. Additional experiments during WWII developed our first understanding of this syndrome [1, 2, 4–7].

Contemporary definitions describe crush injury as any injury caused directly by crushing pressure. Crush syndrome is the systemic manifestations of this injury. The syndrome is characterized by shock, hyperkalemia, hypocalcemia, metabolic acidosis, renal failure, and often compartment syndrome. The associated renal failure is multifactorial and origin as several causes coexist including hypovolemia and the liberation of numerous nephrotoxic substances from injured muscle cell. Shock in these patients is also multifactorial, many cases are confounded by concurrent hypovolemia, potentially due to dehydration or hemorrhage, and a large portion of the intravascular volume is lost to fluid shifts. Electrolyte

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abnormalities such as hyperkalemia can result in negative inotropy, and potentially fatal dysrhythmias [8, 9].

Considering the environment in which these injuries often occur, considerations of scene safety, mass casualties, and triage must all be born in mind. On an individual patient level, however, the most critical component of the treatment is early and aggressive resuscitation to euvolemia with crystalloid when a mechanism consistent with crush syndrome is identified.

Gunal et al. demonstrated decreased need for dialysis and improved survival with early implementation of IV fluid resuscitation among earthquake victims when compared against historical outcomes [10]. Consensus guidelines emphasize early resuscitation, even prior to extrication if plausible [3]. Tourniquet use to prevent dissemination of the liberated cellular components is not supported by the literature nor recommended [3].

After arrival to the hospital setting and appropriate evaluation in compliance with Advance Trauma Life Support protocols, patients should have an electrocardiograph performed to evaluate for evidence of dysrhythmias. Blood gas and electrolyte levels should be assessed and serially checked as often as six times daily with de-escalation once stabilized. Similarly, serum creatinine kinase should be followed as a corollary to muscle injury. Levels exceeding 5000 U/L are associated with a 20% chance of developing acute kidney injury [11]. It is important to continue aggressive fluid resuscitation to euvolemia with a goal of maintaining urine output over 200 ml/h. There are adjuncts to crystalloid resuscitation which have been used both historically and in contemporary treatment regimens including bicarbonate and mannitol administration. There are currently conflicting data regarding the benefit of bicarbonate infusion [12], though it is often included in treatment algorithms in order to alkalinize the urine to a pH greater than 6.5. This minimizes myoglobin precipitation, cast formation, and subsequent tubular obstruction [11, 13]. This theoretical benefit has not been demonstrated in any randomized controlled trial.

Mannitol has also been included in contemporary protocols as both a free-radical scavenger and as an osmotic [14, 15]. Much like sodium bicarbonate administration, there is no high quality evidence demonstrating improved outcomes. Initially, hyperkalemia should be pharmacologically temporized. Bartal et al. utilized a regimen including calcium carbonate, co-administration of insulin and glucose, and albuterol specifically to treat hyperkalemia [16•]. The administration of furosemide and sodium bicarbonate, whose potassium effects were secondary, additionally facilitated the excretion and intracellular storage of potassium respectively in their case series. The aggressive volume resuscitation regimen requires close monitoring of volume status to avoid pulmonary edema and strictly monitored intake and output [16•]. Even with aggressive and early treatment deterioration into renal failure is

common and clinicians should be vigilant for the development of classic indications for dialysis [17•].

Hyperkalemia

Hyperkalemia can be the result as the consequence of reperfusion injury. When the blood flow has been reduced and the abruptly reestablished, then the muscle breakdown products are released into the blood stream. Notably, the mechanism is believed to be the release into the bloodstream of muscle breakdown product, most notably myoglobin, potassium, and phosphorus will be increased since these are the products of rhabdomyolysis. The treatment for this is aggressive resuscitation, early diuresis, and protecting the cardiac membrane to avoid arrhythmias. Maintaining renal perfusion will allow for the clearance of these products. If the patient develops acute kidney injury, then it is important to consider dialysis to avoid further complications [17•].

Compartment Syndrome

Concurrent with the systemic manifestations of crush syndrome, the injured extremity begins to show external signs of injury when none may have previously been visible. Extreme swelling and the development of bulla are common but are also a late sign.

As edema worsens within the skeletal muscle tissues, pressure grows between the rigid fascial planes. Intracompartmental pressures often exceed perfusion pressure leading to muscle ischemia—compartment syndrome [18]. Without early and aggressive treatment, this will lead to additional rhabdomyolysis. Early and frequent monitoring for compartment syndrome facilitates early treatment. Patients with a reliable exam and sound mental status report increasing pain followed by paresthesia and numbness as compartment syndrome develops [19]. Late signs of compartment syndrome are the 6 “P’s”: pain, pallor, poikilothermia, paresthesia, pulselessness, and finally paralysis [20]. Considering patients at risk for compartment syndrome often have a limited physical exam and the consequences of delayed diagnosis and treatment are high, numerous methods have been developed to directly measure compartment pressures reliably when patients are unable to participate in serial assessment [21, 22].

Evaluation of Compartment Syndrome

The evaluation technique most readily available is serial examination, evaluating for worsening pain, especially out of proportion with known injuries, pain on passive motion,

paresthesia, and paresis. Although these individual physical findings are not sensitive for the diagnosis of compartment syndrome, when found in combination, the odds of compartment syndrome are 68% for pain and pain with passive stretch and approximately 98% when all four findings are present [19]. Without a patient reliably participating in this exam, the evaluation is limited to a clinician's subjective assessment of compartment tenseness. This subjective assessment was found to be unreliable across all levels of clinician experience [23]. Despite this evidence, a 2018 survey found 84% of orthopedic trauma attending respondents believed subjective assessment was an important and reliable tool for the assessment of compartment pressures [20]. In addition to physical examination, several techniques and tools have been developed to directly monitor compartment pressures.

Whiteside's technique uses a system where saline is injected into the muscle of concern and a manometer attached to the system reads the intracompartmental pressure. There are two lengths of tubing systems connecting the manometer to a syringe and needle via a stopcock [24]. The apparatus can be somewhat unwieldy; therefore, this usually requires two providers to obtain measurements. Depressing the syringe plunger or placing the needle in a tendon provides a falsely high reading therefore adequate training is required [25]. Whiteside's technique can be modified to utilize a large bore IV catheter and an arterial line pressure transducer with comparably reliability [21]. The most common method of measurement device is the solid-state transducer inter-compartmental catheter, or Stryker needle (Stryker Surgical, Kalamazoo, MI) [20]. This handheld intracompartmental pressure monitor by Stryker measures compartment pressure directly by inserting the needle into a compartment, injecting saline, and a digital reading from the manometer displays on the device [26]. The benefits of continuous vs intermittent pressure measurement have been discussed, but continuous measurement has not been demonstrated to improve outcomes [27] or decrease time to fasciotomy [28]. However, compartment pressures are measured; if the difference in pressure between the patient's diastolic pressure and measured compartment pressure, or delta P, is less than 30 mmHg, compartment syndrome is likely [22]. Historically, an absolute compartment pressure threshold of 30–45 was used as diagnostic criteria, but in a 2018 survey of orthopedic trauma surgeons, 71% of respondents now preferentially utilize delta P to diagnose compartment syndrome [20].

Studies have evaluated pulse oximetry as a monitoring device and found it to be inadequate in detecting tissue perfusion [29]. Additional techniques are under investigation, including a needle-based oxygen assessment device. This device may better measure perfusion without the technical limitations of the pressure-based monitoring devices which can be unreliable if clot or tissues plug the apparatus [30]. Laboratory-based screening techniques via assessment of glucose and

pH are also being explored, but further studies are needed to determine their viability [18, 31].

Treatment of Compartment Syndrome

The only available treatment for patients who have developed compartment syndrome is immediate fasciotomy (see Fig. 1). The incision through the entire length of the fascia allows for the swollen, edematous tissue to expand which lowers the compartment pressure and allows for tissue perfusion. Incomplete fasciotomy can prevent full expansion of the muscle and lead to continued compartment syndrome. Once the muscle compartment has been opened, identifying the 4 "C's" of muscle viability to determine if nonviable muscle needs to be debrided: color, contractility, consistency, capacity to bleed. A second look procedure to identify questionable muscle viability can be planned and the wounds should be dressed with wet to dry dressings or negative pressure vacuum device. The wounds can be closed once muscle viability has been confirmed, and be closed via delayed primary closure or skin grafting.

The lower leg is the most common area for extremity compartment syndrome and has four compartments: anterior, lateral, superficial, and deep posterior. All four are typically released in a fasciotomy since it is not common for only one compartment to be affected. This is performed by both medial and lateral incisions. The medial incision allows for release of the superior and posterior deep compartments. The medial incision is created 1 cm posterior to the tibial diaphysis and extended for 20 cm between the knee and the ankle. The incision is taken down to the fascia with care to preserve the saphenous vein. The superficial posterior compartment is released by incising the fascia overlying the gastrocnemius-soleus complex along its entire length, inferiorly to the Achilles tendon. The soleus muscle is then elevated off the tibia and the fascia of the deep posterior compartment is incised via a separate incision. Once these compartments have been released, a 20 cm incision over the lateral leg, between



Fig. 1 Fasciotomy

the tibia and fibula is created and taken down to the fascia. This will expose the anterior and lateral muscle compartments. The intermuscular septum is identified and incised transversely in the middle of the leg, which protects the peroneal nerve. The fascia is then incised superiorly and inferiorly at the ends of the transverse incision, opening the anterior and lateral compartments and creating an “H” incision. After inspection of all compartments and debridement of dead tissue, the wounds can be dressed.

The upper leg has three compartments, anterior, posterior, and medial. A lateral incision 20 cm in length is taken down to the fascia to expose the anterior compartment. The fascia is incised to allow for release of the anterior compartment. The lateral intermuscular septum is then identified and incised to allow for release of the posterior compartment. A medial incision from the thigh crease to halfway down the thigh exposes the fascia of the adductor compartment which is incised to allow release of the medial compartment.

The arm has two compartments, anterior and posterior. The anterior compartment is released via a 15 cm incision over the biceps. The fascia of the biceps is incised and retracted and the brachialis is identified and its fascia incised to allow for release. The posterior compartment is accessed via a 15 cm incision from the inferior acromion to the olecranon tip. The fascia is incised and the triceps muscle evaluated for viability.

The forearm has three compartments: mobile wad of three, volar, and dorsal. A volar incision from the antecubital fossa to the distal wrist crease releases the volar compartment. A carpal tunnel release can be performed to further release the compartment. A dorsal incision from just distal to the lateral elbow epicondyle to the wrist releases the dorsal compartment. Forearm compartment syndrome is unique in that isolated compartments can have compartment syndrome so individual muscles of the forearm must be inspected.

The consequences of delayed diagnosis and fasciotomy are severe, but complications are common even after timely treatment. Up to 20% of patients requiring surgical management of compartment syndrome have persistent ipsilateral neurologic deficits at 1-year follow-up, and approximately 10% report persistent pain [32].

Conclusions

Crush injury occurs during a natural disaster or traumatic event in which compression of an extremity leads to muscle swelling and neurological deficits. This can worsen to crush syndrome where systemic manifestations such as rhabdomyolysis, renal failure, and shock ensue. One such sequela of crush injury is compartment syndrome, where the muscle swells after injury or an underlying hematoma causes an increase in the compartment. As the pressure of the compartment increases, the capillary pressure which supplies the

muscle with oxygenated blood becomes overcome and the capillaries collapse and can no longer bring blood to the muscles. Initial signs include pain with passive motion and pain out of proportion to exam, while late findings include pallor, pulselessness, poikilothermia, paresthesia, and paralysis. While initial management of crush injury is medical with hydration and electrolyte monitoring, treatment for compartment syndrome is immediate operative intervention with release of the fascia encasing the muscular compartments.

Compliance with Ethical Standards

Conflict of Interest The authors declare no conflicts of interest relevant to this manuscript.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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