Crush Syndrome Case Report and Literature Review

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Abstract
Extensive muscle crush injury culminating in a crush syndrome or traumatic rhabdomyolysis is often lethal unless treated actively on war footing. The causes of death in crush syndrome include hypovolemic shock, hyperkalemia, hypocalcemia, metabolic acidosis, acute myoglobinuric renal failure and the acute muscle – crush compartment syndrome. The literature is divided on the treatment, while some surgeons advocate early surgery others maintain, late surgery is best. We report a case of young man with crush syndrome presenting 10 hours after he was entrapped under his car following road traffic accident. An attempt has been made to review the literature on this topic.

Case Presentation
Twenty one years old Omani male patient presented to accident and emergency with history of road traffic accident and getting entrapped under the car for more than 10 hours, until he was extracted and shifted to nizwa regional referral hospital. Patient had no past history of any medical illness nor was he on any medication.

On admission, patient was fully conscious, oriented and vital signs were normal. His chest, abdomen, pelvis and spine examination was normal.

There was diffuse swelling of right shoulder, arm with inability to move the right upper limb, He had only shrugging movements of shoulder. Neurologically power around shoulder was G1, and around elbow, wrist G0, sensations were diminished below elbow. Radial pulsation was well felt, crepitus around clavicle was felt from underlying fracture right clavicle. Plain radiograph of right shoulder revealed fracture of the clavicle.

In the right lower limb there was mild swelling of the leg and ankle, with intact distal pulsations and associated foot drop but painless, full range of movements around hip, knee, joints with intact cutaneous...
sensations. No bony crepitus was felt.

The left lower limb was diffusely edematous in both thigh and leg compartments with deep abrasions and skin discoloration around the medial aspect of knee lower fourth lateral aspect of thigh. The abrasions were not communicating with the knee joint or deeper tissues. Both thigh and leg compartments were tense with absent ankle and toe movements, capillary refill in toes was less than 02 seconds. Cutaneous sensations were impaired below knee joint. Doppler study of the vascular system of the left lower limb was reported by radiologist as “Normal arterial and venous flow within the thigh. Mildly compromised arterial inflow within leg and foot with totally absent venous flow probably due to compression by swollen muscles of leg compartment”.

Patient was admitted under the care of orthopaedic surgeons in the intensive care unit. He was seen physician, nephrologist and general surgeons. Initial serum biochemistry results were as follows: serum urea 9.7 mmol/l, creatinine 342 μmol/l, and potassium 4.2 mmol/l. The biochemistry changed within hours of admission and repeat sample after few 3 hours revealed rising serum urea 15.8 μmmol/l, creatinine 430 μmmol/l, potassium 8.2 mmol/l and uric acid 602 mol/l, ALT 282 UI/l, AST 643 IU/l, CK 226960 U/L (N 0-190), serum myoglobin 1358 μg/l, urine myoglobin 144 μg/l.

The patient on admission was transfused immediately intravenous saline at the rate of one litre per hour along with monitoring of CVP, arterial pressure and urinary output. This was followed by continuous intravenous infusion of 500 ml, normal saline alternating with 500 ml 5% dextrose at the rate of one litre per hour. In addition patient was given 50 meq/l of sodium bicarbonate and calcium resonium enema. The patient however remained anuric till evening, with rising serum potassium, so patient had haemodialysis and immediately after that patient was taken for fasciotomy of left thigh and leg as the swelling of both thigh and leg compartment was increasing along with compromised arterial flow in leg compartment. On fasciotomy it was found that there was superficial necrosis of the muscles of lateral compartment of thigh, anterior and peroneal compartment of leg with lesser degree of necrosis noted in deep posterior compartment of leg (Fig. 1 and 2). Patient continued to be anuric and was put on continuous initially twice daily and later once daily haemodialysis.

He was started on appropriate antibiotics according his culture sensitivity report. The fasciotomy wound continued to be regularly dressed under all aseptic measures. During this time there was no change in the patients neurological status of left lower limb, right upper limb and right foot drop. On 12th day of admission, patient was transferred to tertiary care centre for plastic surgery care of his fasciotomy wounds and further evaluation of neurological deficit in particular in his right upper limb. During his stay in tertiary centre patients condition remained stable.
deteriorated and he went into gram negative septicemia and was advised to have amputation of left lower limb, for which the patient and relatives refused and instead took patient against medical advice abroad for treatment, where patient died three days after arrival due to severe sepsis.

Discussion

Crush syndrome is the systematic manifestation of muscle cell damage resulting from pressure or crushing [1]. The severity of the condition is dependent on the magnitude, duration of compression force and the bulk of muscle affected. It is however not dependent on the force applied [2].

The earliest description of crush syndrome and injuries in the English literature is by Bywaters and Beall in 1941 [3], after several patients who had been trapped under the rubble of buildings bombed in London, blitz died of acute renal failure. It has been described since then in numerous settings as earthquake, mining accidents, road traffic accidents and recently as a result of terrorist explosions. It is however worth noting that it can occur from non traumatic aetiologies following crush by patients own weight after intoxication or stroke [4].

External mechanical pressure destroys the volume regulation of myocytes, whose cytoplasm is negatively charged and is hyperosmotic compared with the extracellular fluid. By disrupting the impermeability of the sarcolemma, extracellular cations and fluid flow down the electromechanical gradient into sarcoplasm, over whelming the capacity of the cationic extrusion pump and leading to swelling of the myocytes. Consequently mechanical muscle crush injury causes gross edema that may incarcerate much of the extracellular fluid and cause hypovolaemic shock within hours of injury [5]. The local manifestation is acute muscle crush compartment syndrome, which develops rapidly in and around the crushed muscle, as reperfusion syndrome, and which appears immediately after the extrication of trapped victim and the consequent removal of crushing force. An ominous chain of events then unfolds as the crushed vasculature allows the rapid seepage of fluid and plasma proteins in to the dead muscle that is sheathed within its inelastic fascial compartment. In addition intracellular potassium ions released through the incompetent sarcolemma of the dead muscle cells surge into the general circulation at this reperfusion stage and can lead to hyperkalemic cardiac arrest. This can occur rapidly as soon as two hours after extrication [5]. Also at this stage nephrotoxic myoglobin released from the disrupted muscle cells floods the circulation. In addition other substances creatinine kinase, urate and lactic acid levels are also elevated. Free radicals such as superperoxide and hydroxyl cause further damage to cell membrane. This serves to promote platelet aggregation, microvascular clotting, anoxia and continuing overall detrimental process [6-8].

It is not possible to miss the diagnosis of crush injury if the history is known and by limb inspection. The limb after extrication is not usually painful and often is numb with peripheral pulse usually present unless the patient is in shock. Direct arterial injury is very rare in closed crush injury. Swelling appears rapidly, causing acute compartment syndrome and within hours the limb becomes turgid and brawny. The crushed skin is bruised and discolored but remains intact. Pain develops gradually and becomes excruciating as the intracompartmental pressure rises [5].

The clinician should be aware of the fluid requirements indicated in early management of this condition to counter life threatening hyperkalemia, hypocalcemia and to prevent myoglobinuric renal failure. Massive fluid transfusion should commence as soon as intravenous access is gained, preferably while the victim is still trapped [1, 5].

The local treatment of crush syndrome is controversial. Two school of thoughts are there about the management of local injury. Fasciotomy is expected to reverse the muscle necrosis by improving circulation and reducing the load of muscle lysis products, harmful cytokine production and free oxygen radicals [6]. Another school of thought suggests that fasciotomy should not be performed in crush injury cases and the treatment should be conservative as fasciotomy makes the injured muscle more vulnerable to infection which can later endanger patients life [4, 9]. Finkelstein et al [10] in their series of cases of crush injury presenting 36 hours after injury who underwent fasciotomies had significant mortality and this made them change their treatment to supportive care for acute renal failure and late reconstructive procedure as sequelae of infection are more grave than muscle contracture. Mubarak et al [11] also have reported no improvement in final outcome in patients of crush syndrome after fasciotomy.

In a scenario of mass casualties as during earthquakes the debate about fasciotomies is still not settled, Huang et al [12] reported high infection and amputation rates in their fasciotomies cases during Chi-Chi earth-
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sever. Sever et al. are of opinion not to undertake fasciotomy unless clear objective indications are present such as increased intracompartmental pressure [13]. In an earthquake disaster because of increased patient overload and the chaotic situation, fasciotomies carry a higher risk of infection and can result in improper wound care with resultant sepsis and mortality. However Duman et al. [14] from their experience of earthquake victims in Turkey recommend fasciotomy whenever there is suspicion of compartment syndrome as they feel decision making in mass casualty is very difficult especially in absence of sophisticated diagnostic methods.

Recent literature about cases of crush syndrome from compression of single limb have reported good results with early fasciotomy [15, 16], but these are isolated case reports. Fasciotomy is recommended within six hours of development of compartment syndrome [17], but is not indicated when the muscle is already dead, a situation which invariably exists in acute muscle crush compartment syndrome. There is no place for prophylactic fasciotomy. Intracompartmental pressure can be reduced without need for fasciotomy and risking infection by use of intravenous hypertonic mannitol [18], although it is contraindicated in presence of renal failure. Conservative management in form of hyperbaric oxygenation reduces edema and floods tissues with oxygen dissolved in extracellular fluid. Strauss and Harr noted in the a series of patients of compartment syndrome who were treated with hyperbaric oxygen, none required fasciotomy [19].

Conclusion

Crush syndrome is on increase world wide. Increasing road traffic accidents in this part of globe more and more patients present with picture of crush injuries which can lead to high morbidity and mortality if not recognized early and appropriately managed. Surgeons are still, divided on the local management of the crush limb. The recent consensus on accumulated evidence is that fasciotomy is indicated when muscle death is threatened or when the distal pulse is absent after direct, major arterial injury provided systemic hypotension have been excluded.

It is contraindicated in patients with closed acute muscle crush injury as it increases the danger of fatal sepsis and does not improve the outcome for limbs, nor for the kidneys. Also if fasciotomy is done for benefit of doubt, then it should be repeated every 48 hours by radical excision of dead tissues. One must remember that the only definitive way to differentiate dead muscle from living muscle is by electrical stimulation, change of muscle color is not enough.

References


