Chapter 6

CRUSH SYNDROME

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DEFINITION AND HISTORY

Crush means crush as word meaning. Crush Syndrome was first observed in muscular pain, weakness and brown urine in 1910. Meyer-Betz described a clinical syndrome including dark brown urine, muscle pain and weakness in 1911(Meyer-Betz, 1911). In the 1914-18 war, it was defined as a renal lesion after local muscle damage by German authors (Frankethal, 1916). It was later identified in the mining sector (McClelland, 1941). In during II. World War, the full scope of renal failure after crush was first recognized as an entity by Bywaters and Beall in 1941 in the victims of the blitz of London. Bywaters and Beall observed the survivors who were trapped in the legs, that shock to responded the transfusion,swelling in the stuck extremities, and renal failure following uremia (Bywaters & Beall, 1941). They stated that the degree of injury, hemoconcentration degree, oliguria and blood urea elevation determine the prognostic factors. They reported that excessive crushing of the muscles could progress to a process that could result in death if a fast and effective treatment was not performed. They described the syndrome as open. Crush syndrome is a systemic symptom of muscle damage after direct trauma or ischemia-reperfusion injury. Signs and symptoms are tense, edematous, painful muscles, dark tea, shock and acidosis. Acute renal failure often develops.

Crush syndrome often occurs after trauma. It is also called traumatic rhabdomyolysis. Trauma is the most common cause of death after earthquakes. However, its importance in clinical practice is not always appreciated. Multiple tissue and organ damage occur due to the severity of trauma. Crush injury and rhabdomyolysis, which may occur in patients with major muscle injury

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metabolic acidosis and reduce hyperkalemia. When the urine output starts with fluid replacement, it is recommended to make difficult alkaline groove because it helps the movement of myoglobin from tubules. But excessive alkalinization has calcium phosphate deposition in soft tissues, worsening of hypocalcemia, and volume overload (Sanadgol, & et al., 2009). Mannitol may be added to the treatment. Mannitol increases the glomerular pressure and draws fluid from the muscle and interstitial tissue. It increases the urine flow and prevents the myoglobin from blocking the tubules. Mannitol is discouraged in anuric patients. If renal failure, acidosis and hyperkalemia develop, dialysis begins. Dialysis is continued until the kidney function improves.

Necrotic tissues need to be debrided. In patients with hand compartment syndrome after closed crush injury, the diagnosis can be made by clinical examination and intra-compartmental measurement. Hand swelling and palpation are sensitive. Severe pain may not be present in all patients. In standing crush injury, standing compartment syndrome may occur. Long term contractures, deformity, weakness, paralysis and sensory neuropathy are found. Compartment pressure> 30 mmHg fasciotomy is performed (Fulkerson, Razi & Tejwani 2003). In patients with compartment syndrome due to crush injury, in the absence of neurovascular compromise, a trial of mannitol therapy should be instigated, but a specialist opinion should be sought early (Better, 1999). Theoretically advantageous measure is amputation of a crushed limb to prevent crush syndrome. However, there is no evidence to support the use of amputation as a prophylactic measure to prevent crush syndrome. Reports from the literature suggest that even severely crushed limbs can recover to full function. If the limb is literally hanging on by a thread, or if the patient's survival is in danger due to entrapment by a limb, amputation should be considered and appropriate expert advice sought.

To conclude, crush syndrome is a major cause of mortality in the victims. However, the number of deaths due to crush syndrome can be decreased by appropriate management.

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