Analysis of 372 Patients with Crush Syndrome

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To clarify clinical features and determine the severity in patients with crush syndrome from the Hanshin-Awaji earthquake, medical records of 6,107 patients hospitalized in 95 hospitals were reviewed retrospectively, and 372 patients with crush syndrome were identified. The major sites of crush injury were in the lower extremities (74%), the upper extremities (10%), and the trunk (9%). Pelvic fractures, limb fractures, and abdominal injuries were the most frequently associated injuries. Patients with trunk compression and/or with abdominal injury had a higher mortality rate. A total of 50 patients (13.4%) died. The causes of death within five days after the earthquake were hypovolemia and hyperkalemia. Peak serum creatine kinase (CK) concentration increased with the number of crushed extremities. Mortality and the risk of acute renal failure were highest in patients with CK concentration greater than 75,000 U/L.

Conclusion: The severity of crush syndrome can be estimated by both the number of crushed limbs and peak serum CK concentration, which reflects the extent of underlying muscle damage. These results help us to recognize the crush syndrome, evaluate its severity, and to consider indications for transferring the patients to unaffected hospitals for critical management.

Keywords: creatine kinase (CK); crush syndrome; hyperkalemia; hypovolemia; mortality; risk; severity; sites
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Diagnosis, Treatment, and Pathophysiology of the Crush Syndrome

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Crush syndrome with acute renal failure has been identified as a major medical complication that occurs among people who are trapped under the debris from earthquakes, bombings, and other events that result in entrapment. Traumatic rhabdomyolysis induced by ischemia reperfusion injury plays a crucial role, although the pathophysiology is not fully understood. Initially, prolonged limb compression may cause stretch myopathy as well as ischemic injury. Immediately following decompression of limbs, re-establishment of the microcirculation produces an adverse effect; reperfusion injury that promotes further ischemia, muscle swelling, and myonecrosis that leads to an acute compartment syndrome. In association with these local events during limb compression/decompression, a massive loss of extracellular fluid into the injured muscles and solutes leaking out of damaged muscles results in the development of systemic manifestation. Chief among these are hypovolemia and hyperkalemia which, synergistically, have a potential for early death, metabolic acidosis, shock, coagulopathy, and acute renal failure (ARF).

A history of prolonged limb compression trapped by heavy objects and a physical finding of limb paralysis following extrication should suggest the diagnosis of crush syndrome. The treatment consists of aggressive volume replacement followed by forced diuretic therapy, which may combat shock and correct the hyperkalemia. If ARF has occurred, regular hemodialysis is indicated. Although the surgical management of injured limbs still is controversial, suggestions were provided in this report.

Keywords: acidosis; acute renal failure; crush syndrome; hemodialysis; ischemia; myonecrosis; reperfusion; rhabdomyolysis; shock
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Radiographic Imaging and Histological Findings of Crush Syndrome

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Objective: To identify the radiographic imaging and histological findings of crush syndrome to better understand the pathophysiology of its development.

Radiographic findings: Seven patients injured in the Hanshin-Awa Earthquake underwent serial computerized tomography (CT) and magnetic resonance imaging (MRI) examinations of the injured extremities. In all patients, the CT showed persistent edema of the subcutaneous tissue until approximately one month after the injury. In four patients who developed acute renal failure, CT revealed transient high density areas in the muscle, which was confirmed histologically as calcification. An MRI performed 4 to 5 weeks after the injury revealed inhomogeneous high intensity areas in injured muscles on T2-weighted images in all patients, indicating prolonged edema within the muscles. The use of Contrast T1-weighted MRI localized the injured area more clearly than did CT.

Histological findings: Muscle tissue was taken by biopsy from six patients 40 to 50 days after their injury. In four patients, typical myopathic changes such as necrosis, regeneration, and inflammatory cell infiltration were observed. Neurogenic muscular changes, such as small angular fibrosis, also were observed in three patients. In five patients, calcium deposition was demonstrated by von Kossa staining.

Conclusions: These radiological and histological findings support the notion of calcium-related biochemical events intrinsic to crush syndrome and show that the mechanism of the injury is not simple, but sustained and complicated.

Keywords: calcifications; computerized tomography; edema; fibrosis; histology; imaging; inflammation; magnetic resonance imaging; myonecrosis; necrosis
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Clinical Presentations of Typical and Atypical Crush Syndrome

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