The Development of Acute Renal Failure and ARDS in Crush Syndrome

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Abstract

We conducted a retrospective analysis of two patients with acute renal failure (ARF) and adult respiratory disease syndrome (ARDS) caused by crush injury. Both patients had been extracted from buildings that collapsed in the 1999 Marmara (Turkey) earthquake. Though ARF is the most common complication of crush syndrome, ARDS is rare, when sepsis and multiple organ failure can be prevented.

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Key words: *Crush syndrome, renal failure, ARDS*

Introduction

An earthquake that registered a magnitude of 7,4 on the Richter scale struck the western part of Turkey, (Marmara) on 03.02 am in August 17, 1999. The earthquake killed more than 17.000 and injured 60.000 people. Under such a scenario, an epidemic of crush syndrome caused by continuous and prolonged pressure on the body would be expected (1,2). Crush syndrome, not only develops in disasters as earthquakes but also seen after bombings, mine accidents, and train accidents. Significant soft tissue injury predisposes the patient to multiple complications including: hypoxemia, compartment syndrome, rhabdomyolysis, electrolyte and acid base imbalances, coagulopathy, and renal failure (3,4,5). Acute renal failure is a well known complication of the crush syndrome with muscle trauma or compression, though other organ failures can be prevented (6,7,8).

Case 1

A 22-year-old man had been crushed under a collapsed building for 10 hours. On admission he was conscious and physical

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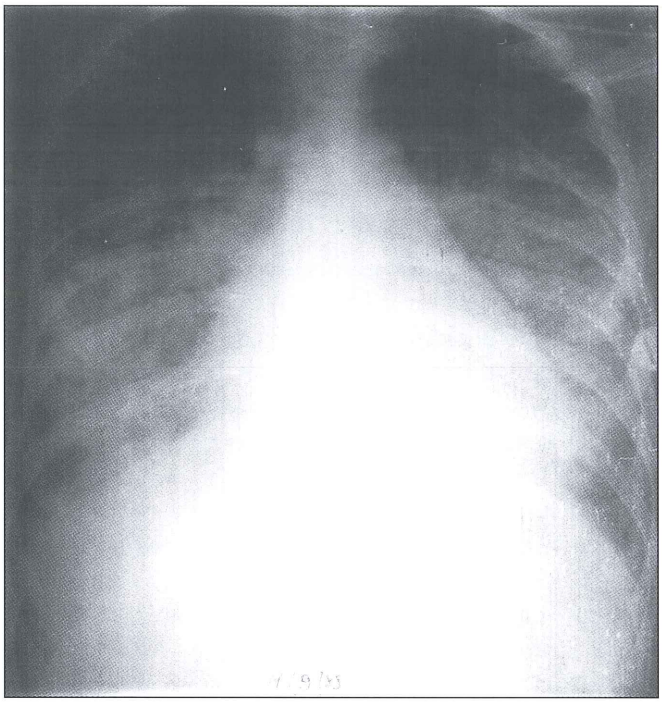

Figure 1: *Chest x-ray of the first case: diffuse nonhomogeneous infiltration with costodiaphragmatic blurring.*

examination revealed wide-spread slight bruises and superficial abrasions on his right lower limb and left upper limb. The leg and arm were swollen and patchy areas of hypoesthesia or anesthesia were present. There was no pattern of sensory loss attributable to denervation of a particular nerve or ischemia attributable to occlusion of a particular vessel. Radiological examination of the extremities revealed a normal bone structure. Laboratory studies showed: hemoglobin=8.9 g/dL, white blood cell count= 13 300 /mm^3, blood urea nitrogen (BUN)=155/dL, serum creatinin=6.0 mg/mL, Na=141mEq/L, K=5.9 mEq/L, Ca=7.8 mmol/L, lactate dehydrogenase=222.6 IU/L, creatine phosphokinase (CPK)=36 000 IU/L. The patient was diagnosed as having ARF due to crush syndrome. On the 10^{th} day, he complained of dyspnea with bilateral rhonchi on physical examination and chest x-ray showed diffuse nonhomogeneous infiltration with costodiaphragmatic blurring (figure 1). Thorax CT revealed bilateral nonhomogeneous air-space consolidation with pleural effusion (Figure 2). On blood gas analysis pH was 7.35, $PaCO_2$ was 56.1 mmHg, PaO_2 was 38.7 mmHg, and HCO_3 was 30.5 mmol/L. Echocardiography (ECHO) revealed a normal left ventricular function. Measured mean pulmonary arterial pressure (PAP) was 22 mmHg. The patient was diagnosed as having respiratory distress syndrome and mechanical ventilation was started. On the 7^{th} day of the mechanical ventilation the patient died because of severe hypoxemia and infection.

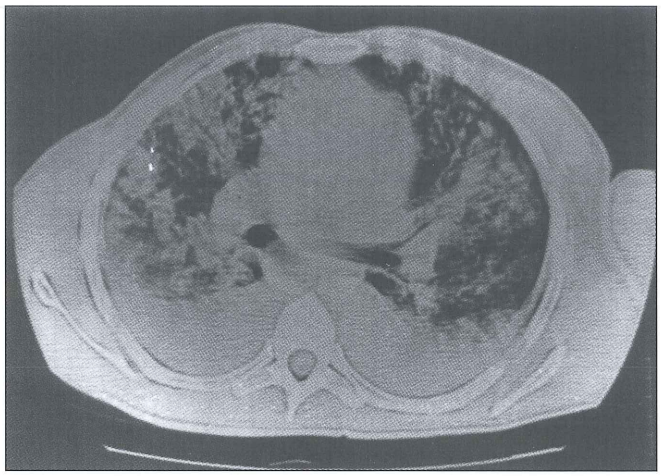

Figure 2: *Thorax CT of the first case: bilateral air space consolidation with pleural effusion.*

Case 2

A 22-year-old man had been crushed under a collapsed building for 12 hours. On admission he was conscious and physical examination revealed motor weakness of the left lower limb. Peripheral pulses were all intact and edema was present. Radiograms showed no bony lesions. Laboratory studies showed; haemoglobin=9.3 g/dL, white cell count= 11 000/mm^3, platelets=292 10^3/mm^3, blood urea nitrogen=157 mg/dL, serum creatinine=6.3 mg/mL, Na=138 mEq/L, K=6.7 mEq/L, lactate dehydrogenase=1091 U/L, creatine phosphokinase=27 000 IU/l. Urinary output was less than 200 mL/h. The patient was diagnosed as having

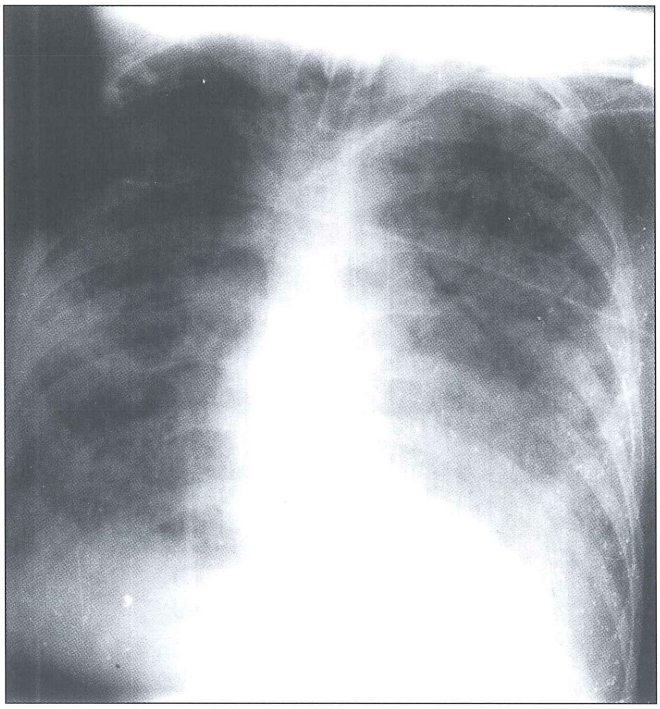

Figure 3: *Chest x-ray of the second case: diffuse nonhomogeneous infiltration.*

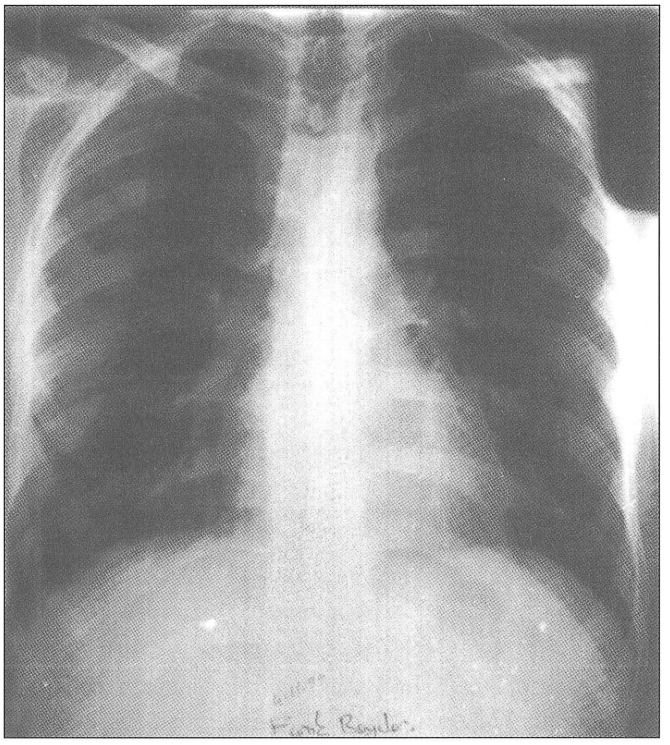

Figure 4: *Control chest x-ray of the second case.*

ARF due to crush syndrome. On the 10th day, tachypnea and symptoms of respiratory distress appeared. Blood gas analysis revealed a pH of 7.47, PCO_2 of 26.4, PO_2 of 41.8 mmHg, HCO_3 act=19.0 mmol/L. He was given O_2 with a fraction of inspired oxygen (FIO_2) of 6 L/min, but hypoxemia persisted. On physical examination, marked ronchi and bilateral late inspiratory crackles were present. Chest x-ray (A-P) showed diffuse infiltration (Figure 3). ECHO revealed a normal left ventricular function. Measured mean PAP was 18 mmHg. The patient was diagnosed as ARDS. He was ventilated mechanically nearly for four weeks and after a successful weaning, he was discharged from the hospital on the 45th day of therapy. The control chest x-ray was normal (Figure 4).

Discussion

Crush syndrome is a form of traumatic rhabdomyolysis, which occurs as a consequence of prolonged continuous pressure on the limbs (1,2). It reflects the disintegration of muscle tissue and the influx of myoglobin, potassium and phosphorus into the circulation (9,10). The syndrome is characterized by a reduction in circulating plasma volume, hypotension and hemoconcentration (9). Diagnosis usually depends on high index of clinical suspicion in the appropriate clinical setting, followed by confirmatory findings (11). The presence of myoglobin in the urine and elevated muscle enzyme activity in the serum (skeletal muscle creatine kinase, aldolase, carbonic anhydrase III) are diagnostic (11). However, clinically significant traumatic rhabdomyolisis may occur without grossly visible pigment in the urine (4,11). Other laboratory findings are hyperkalemia, hypocalcemia, hyperphosphatemia and hyperuricemia (4,5,6,7,10,11).

Our cases were diagnosed as having ARF due to the crush syndrome depending on the history, clinical and laboratory findings. Both were the victims of a large disaster and transferred to our hospital with oliguria. We did not have the chance to examine myoglobin in the urine. In the first case, creatine phosphokinase level was determined as 36.000 IU/L, lactate dehydrogenase was 2246 IU/L and in the second case they were 27 000 IU/L and 1091 IU/L respectively. On admission, other laboratory findings of the first case that revealed ARF due to the crush injury were elevated level of BUN (157 mg/dL) and serum creatinin level (6.0 mg/dL), hyperkalemia (5.9 mEq/L), hypocalcemia (7.8 mmol/L) and hyperuricemia (13.0 mmol/L). In the second case laboratory findings revealed an elevated BUN (157 mg/dL), serum creatinine (9.0 mg/dL) and K (6.4 mEq/L) levels. Hypocalcemia (6.3 mmol/L) was also present. They were both given volume replacement with mannitol therapy and hemodialysis was initiated. Many reports on patients with ARF due to crush syndrome, conclusively recommended that once the systemic circulation has been stabilized and the presence of urinary output has been confirmed, forced mannitol alkaline diuresis therapy for prophylaxis against hyperkalemia and acute renal failure shoud be started, though there were conflicting results related to the mechanism (4,6,7,10,11,12). Our patients had hypocalcemia but as there was not a condition such as ventricular arrhytmias, the therapy was not required in accordance with the recommendations in the current literature (11). Development of parestesia or tense muscular compartments on physical examination should promt direct measurement of intracompartmental pressures (13,14). In various reports compartmental pressures above 30 mmHg were accepted as indication of fasciotomy (15,16). Reis et al., suggested that the only rational indication for performing a fasciotomy in a patient with crush injury of a limb, is absence of distal pulses following exposure to high compartmental pressure for several hours (17). In our cases, physical examination revealed an intact skin. Patchy areas of hypoestasia or anesthesia were present but the loss was not attributable to denervation of a particular nerve. Distal pulses were intact. Therefore, we preferred the conservative therapy and fasciotomy was not performed. Except for ARF, another organ failure is an uncommon complication, if other sequela such as sepsis or multiple organ failures can be prevented (8). In our two cases, respiratory distress occurred during the seventh and tenth day of the therapy. Both patients were on poliuric stage of ARF. ARDS was diagnosed according to the criteria established by the American European Consensus Conference on ARDS

(18). In the first case, blood gas analyses revealed a severe hypoxemia with compensated respiratory acidosis and though he was given oxygen, hypoxemia persisted. Chest x ray revealed bilateral nonhomogeneous patchy infiltrations with costadiaphragmatic blurring. ECHO revealed a normal left ventricular function. Measured mean PAP was 22 mmHg. Laboratory findings revealed hypoalbuminemia. Central venous pressure (CVP) revealed 12 cmH_2O, thus over-transfusion in ARF might not be responsible for the development of ARDS, and was thought to account for the pleural effusion. In the second case, blood gas analyses revealed a mixed pathology with severe hypoxemia. Though he was given O_2 at FIO_2 6 L/min, hipoxemia persisted. Chest x-ray revealed bilateral nonhomogeneous patchy infiltration. Cardiac pathology was excluded with ECHO. The measured mean PAP was 18 mmHg and the CVP was 10 cmH_2O.

Other clinical conditions such as fat embolism, oxygen toxicity, pneumonia and disseminated intravascular coagulation, which are common causes of ARDS, appeared to be improbable according to the clinical presentation of our patients. ARDS was diagnosed as a late complication of crush syndrome in our two cases as stated in the reports of Nishihara, and similar cases were mentioned in the report of Santelgela (5,8). The pathophysiologic process of the disorders associated with the crush syndrome is not fully understood, the injuries induced by ischemia and reperfusion is likely to be important in its development (8,9). Gastrointestinal tract has been emphasised as a potential effector of MOF due to crush syndrome through the release of several cytokines, particularly tumor necrotizing factor-α, when ischemia and reperfusion of the microcirculation occurred (9). The results of the many studies suggested that reactive oxygen metabolites may also play a major role in the injuries due to ischemia and reperfusion (9,17,19). We presume that these factors are the causes of ARDS in our cases. ARDS should be taken into consideration even when other clinical conditions such as embolism, oxygen toxicity, pneumonia and disseminated intravascular coagulation which are common causes of ARDS are prevented.

Though acute renal failure is the most common complication, ARDS is a rare but an important complication of crush syndrome which leads to an increase in mortality.

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