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Crush Syndrome Associated with Thoracic Compression and Crushing of Both Lower Limbs: A Case Report

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Abstract **Case Report**

This case report details the management of a 27-year-old male who sustained severe crush injuries involving thoracic compression and bilateral lower limb crushing after being trapped between shipping containers for over four hours. The patient presented in hemorrhagic shock with multiple fractures, extensive soft tissue damage, and a large left-sided pneumothorax. Initial management focused on stabilization, including fluid resuscitation, blood transfusion, and pain management. Surgical intervention involved wound debridement and external fixation of bilateral femur fractures. The patient developed acute kidney injury requiring hemodialysis, but ultimately recovered with improved renal function and the application of skin flaps for wound closure. This case highlights the complex multi-organ injuries associated with crush syndrome and the importance of aggressive early management to optimize patient outcome. The case emphasizes the need for prompt stabilization, surgical intervention, and close monitoring for complications like acute kidney injury.

Keywords: Crush syndrome, traumatic compression syndrome, rhabdomyolysis, acute kidney injury, multiple trauma, thoracic injury, compartment syndrome, external fixation, hemodialysis.

INTRODUCTION

Traumatic compression syndrome is usually associated with muscular crush injuries which occur during burials, commonly known as crush syndrome. This syndrome was first described by Bywaters and Beall during the bombing of London during the Second World War in 1941. This syndrome encompasses a wider range of injuries than just muscular injuries, such as thoracic and abdominal compressions. It is responsible for a significant morbidity and mortality rate, hence the importance of rapid treatment at the scene of the accident. Crush syndrome most often occurs in the context of serious accidents such as road traffic accidents, accidents at work or disasters [1]. In this article, we report a case of crush syndrome in a young patient following an accident at work with double compression of the chest and both lower limbs.

PATIENT AND OBSERVATION

A 27-year-old patient with no previous medical history was crushed in the chest and both lower limbs after being squeezed between two shipping containers for more than 4 hours. After a lengthy extrication effort, the patient was transferred by a non-medical ambulance to the emergency department of the university hospital centre. On admission to the emergency department, the patient was found to be agitated, pale and in a state of haemorrhagic shock, with a blood pressure of 60/40 mmHg and a heart rate of 150 beats/min. Neurological examination revealed an agitated patient with equal and reactive pupils, with no signs of localisation. Pleuropulmonary examination revealed a polypneic patient with 32 cycles/min, 85% pulse oxygen saturation, snowy crepitation in the left hemithorax on palpation, a soft abdomen and an unremarkable pelvis. Examination of the limbs revealed a large open wound with loss of substance on the anterior aspect of the left thigh, a circular wound with loss of substance on the right knee, and active bleeding with abolition of peripheral pulses in both lower limbs [Figure 1].

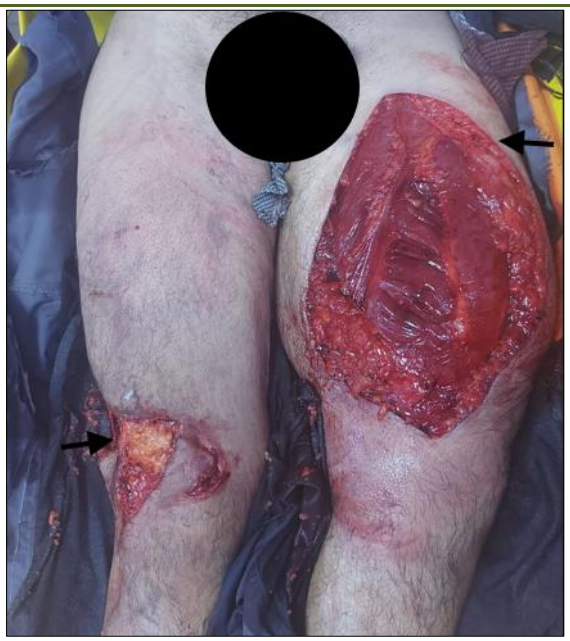

Figure 1: crushing of both lower limbs with loss of skin substance

After monitoring, the patient received oxygen therapy, two large-bore peripheral venous lines, massive vascular filling with 1 litre/hour crystalloids and an infusion of noradrenaline, a tourniquet applied to the right thigh with alternative compression and decompression, and a blood transfusion of 04 packed red blood cells, 03 fresh frozen plasma, 01 g of calcium gluconate, 01 g of tranexamic acid, antibiotic therapy with protected amoxicillin, analgesia with titrated morphine, paracetamol and nefopam, and biological sampling with a request for blood.

After stabilisation, a lesion assessment was carried out and revealed: Cerebral and cervical: Significant dissecting emphysema of the cervical soft tissues and the deep spaces of the face; Thoracic: Large left haemopneumothorax with a predominance of the gaseous component responsible for compression of the pulmonary parenchyma and pushing of the mediastinal elements to the right. Large pneumomediastinum. Small right pneumothorax. No pericardial effusion. No extravasation of PDC. A fracture of the posterior arch of the 4th and 5th left ribs. Fracture of a cortical bone of the body of the left scapula and fissuring of a left costo-sternal cartilage. Pneumorachy emphysema of the thoracic soft tissues [figure 2], Abdominopelvic: No traumatic lesion No pneumoperitoneum. No liquid peritoneal effusion. No extravasation of DCP. No fracture lines in the spine or pelvis. Presence of air in the left scrotum with dissecting emphysema of the abdomino-pelvic soft tissues, predominantly on the left. The radiological work-up showed open fractures of both femurs [figure 3].

The rest of the paraclinical work-up showed haemoglobin at 5.2 g/dl, thrombocytopenia at 100,000/mm, urea at 0.33 g/l, creatinemia at 18 mg/l, PT at 40%, creatine phosphokinase (CPK) at 8,000 IU/l and troponin at 1.85 ng/ml, kalaemia at 5 mmol/l, and calcaemia at 2.2 mmol/l.

The patient then underwent placement of a left chest drain with gentle wall suction and placement of a left internal jugular line, and a right radial arterial catheter and bladder catheter bringing back concentrated urine. The patient was admitted to the operating theatre: After general anaesthesia using crush induction, the patient underwent trimming of the musculocutaneous lesions and two external fixators were fitted to the two fractured femurs. The peroperative period was marked by slight haemodynamic instability, necessitating an increase in the dose of noradrenaline and the transfusion of 03 RBCs, 02 PFCs and 1 platelet unit, before being transferred to intensive care.

After gradual weaning of the vasoactive drugs and a follow-up chest X-ray, which showed the lung had returned to the wall, and a check-up with haemoglobin at 9g/dl, PT 55%, platelet count 90,000, the patient was extubated.

The course was marked by the onset of renal failure, with creatinine at 80 mg/l, urea at 1.5 g/l, kalaemia at 6 mmol/l and oliguria of 300 ml over 12 hours. The patient underwent 06 haemodialysis sessions.

Finally, the patient resumed diuresis with improved renal function, and the local condition of his left thigh and right knee improved following the application of a skin flap taken from the left leg and daily dressing changes [figure 4].

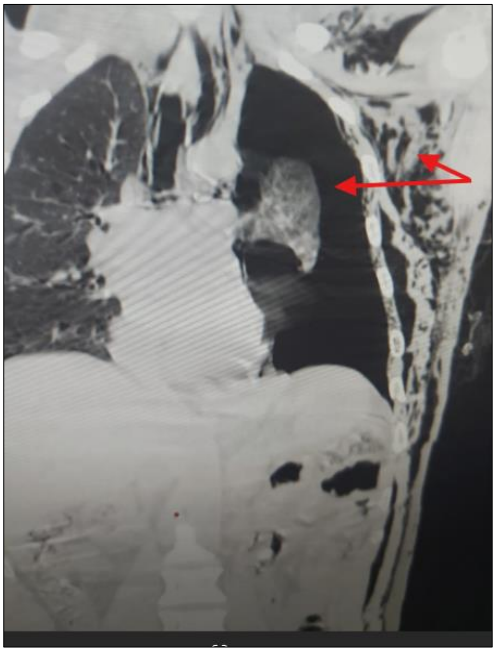

Figure 2: Compressive pneumothorax with mediastinal pneumo and diffuse cutaneous emphysema

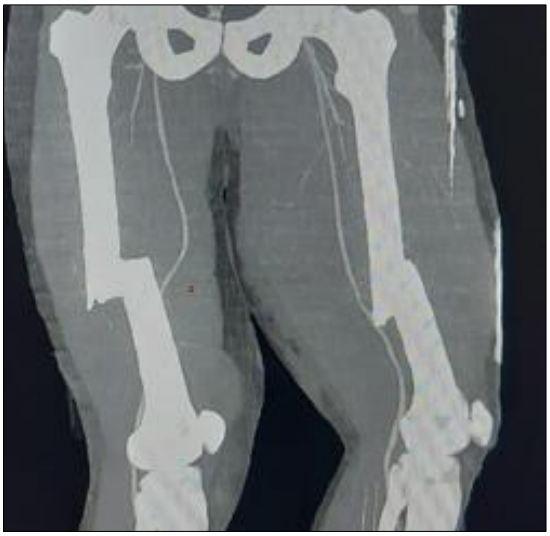

Figure 3: Standard X-ray showing two displaced fractures of both femurs

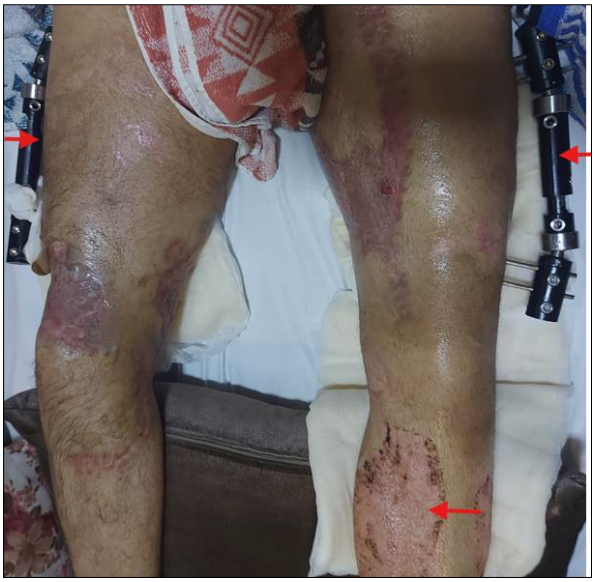

Figure 4: Image showing two external fixators and improvement of the local condition by application of a skin flap

DISCUSSION

Traumatic compression syndromes cover a wide range of injuries. In addition to muscle injuries, they include syndromes associated with thoracic and abdominal compression. Depending on their location, these compressions lead to different mechanisms of vital distress [2]. Compression syndromes found in everyday practice are often the result of prolonged immobilisation leading to rhabdomyolysis. The mechanisms may be interlinked, as in the case of our patient, who suffered a crush injury, hypovolaemia and haemorrhagic shock.

From a pathophysiological point of view, compression of the thoracic floor can lead to serious lesions, rapidly fatal in the presence of high kinetic energy. These injuries generally correspond to those observed in closed thoracic trauma, affecting either the chest wall or the internal organs. When prolonged compression is accompanied by obstruction of the upper airways, a traumatic asphyxia syndrome, also known as Perthes syndrome, may occur. This syndrome is manifested by capillary ruptures causing petechiae and cyanosis of the face. To limit complications, it is essential to remove thoracic compression as soon as possible. Appropriate management of the airways and respiratory system must be considered, including, if necessary, interventions such as exsufflation of a pneumothorax or the introduction of mechanical ventilation.

Compression of the abdominal wall can cause respiratory distress due to functional impairment of the diaphragm. This incompetence may result from direct injury to the muscle or from hyperpressure in the abdominal compartment. Diaphragmatic rupture may also occur, leading to displacement of abdominal organs into the thoracic cavity (hernias), which reduces lung compliance. Abdominal compression can act as a temporary tourniquet, limiting the immediate effects of post-traumatic internal bleeding. However, removal of this compression may lead to haemorrhagic collapse. In a patient with impaired consciousness, abdominal hyperpressure is an aggravating factor, increasing the risk of regurgitation and inhalation. These complications require rapid and appropriate management to minimise the serious consequences [3]. Our patient underwent thoracic drainage with optimal analgesia and did not present any abdominal lesions

Crush syndrome is a clinical and biological syndrome linked to the destruction of striated muscle fibres with systemic release of their contents. On a cellular level, there is a cellular energy deficit due to a reduction in adenosine triphosphate (ATP) reserves, resulting in failure of the Na/K ATPase pump, which is responsible for a massive influx of sodium and H2O, leading to cellular oedema, On the other hand, the failure of intracellular calcium pumps will be responsible for an accumulation of cytoplasmic calcium leading to prolonged contracture with activation of phosphorylases and destruction of the cell membrane, as well as the release of free radicals and cytokines, which will aggravate inflammation and cellular oedema. At the level of the muscle compartment: the latter becomes inextensible with an increase in intra-muscular pressure, which aggravates the ischaemia leading to the onset of mixed oedema, major fluid sequestration with effective hypovolaemia (vicious circle). On reperfusion, the massive release of myoglobin will saturate the haemoglobin binding sites, and the release of potassium presents a major risk of rhythm disorders aggravated by hypovolaemia, hypocalcaemia and hyperphosphaemia, and the release of lactic acid and other organic acids precipitates the onset of metabolic acidosis, Tissue thromboplastin will be responsible for coagulation

disorders with a picture of disseminated intravascular coagulation, free radicals and inflammatory mediators may aggravate the systemic inflammatory response syndrome or even a syndrome of multivisceral failure, the release of creatinine phosphokinase (CPK) considered non-toxic but represents an element of diagnosis and severity, in the end renal damage occurs by three mechanisms: Tubular obstruction due to tubular precipitation of myoglobin, direct toxicity of myoglobin degradation products and renal hypoperfusion due to hypovolaemia and intra-renal vasoconstriction [4, 5].

Clinically, the main lesions are dermabrasions, ecchymoses and skin detachments. The patient may present with closed or open fractures. Nerve lesions are secondary to ischaemia, stretching or sectioning; they mainly involve the radial and sciatic nerves. The large vascular trunks are frequently affected when limbs are crushed, often as a result of stretching or comminuted fractures, particularly in the proximal humerus, elbow or knee. Clinical signs associated with muscle damage and rhabdomyolysis (fatigue, pain) are generally masked by disturbances in consciousness, and urine becomes dark due to the presence of myoglobin. Muscle oedema is responsible for compartment syndrome, with signs of ischaemia and sensory-motor disorders. The crush syndrome is accompanied by general symptoms, with signs of hypovolaemia, oligoanuria, shock, neuropsychological disorders, hyperventilation linked to metabolic acidosis and arrhythmias linked to hyperkalaemia. Consumption coagulopathy and ARDS may also occur due to the release of free radicals during reperfusion**.** Our patient presented with skin, muscle and bone lesions associated with haemorrhagic shock.

Biologically, there is an increase in muscle enzymes such as myoglobinuria, which can be detected by a urine dipstick. This is often transient and is not essential to the diagnosis (especially if there is anuria), as well as an increase in CPK, the most sensitive indicator of muscle damage, with a late peak between 24 and 36 h. Its value varies between 1000 IU/l and 16,000 IU/l, and may exceed 100,000 in the most severe cases. An increase in other enzymes of muscular origin (LDH, ASAT) is possible, and on the other hand there are ionic disorders such as hyperkalaemia, which is precocious and may be massive, resulting from the release of injured muscle cells and the fall in glomerular filtration rate in a hypovolaemic patient, Hypocalcaemia is secondary to intracellular Ca accumulation and a reduction in intracellular ATP, and the occurrence of metabolic acidosis aggravates hyperkalaemia and causes aciduria responsible for intratubular precipitation. Other biological disturbances may occur, such as hyperphosphatemia, hyperuricemia and hypoalbuminemia secondary to capillary leakage in the injured muscle [6].

The incidence of renal failure in crush syndromes varies between 31 and 52%. Patients are usually oligo-anuric. The main risk factors for renal failure are delay in management, severe metabolic acidosis (bicarbonates < 17 mmol/l), low haemoglobin, myoglobinuria and CPK above 8500 U/l. Our patient presented with metabolic acidosis, hyperkalaemia, elevated CPK and renal failure [7].

Treatment of traumatic rhabdomyolysis should be initiated immediately at the scene of the accident, even before the victim is released. Once stabilised, the patient should be transferred in a medical ambulance to a hospital equipped with an intensive care unit capable of performing extrarenal purification if necessary. The main therapeutic objectives include correcting hypovolaemia and hyperkalaemia, preventing renal failure and coagulation disorders, and stabilising polytrauma-related injuries. Close monitoring is essential, including the following parameters: heart and respiratory rates, invasive arterial pressure, heart rate, oxygen saturation, central venous pressure, body temperature, capillary blood glucose, muscle compartment pressure, urinary pH (maintained above 6.5) and hourly urine output (target 200 to 300 ml/h or 3 ml/kg/h) [8].

Treatment requires: Two large-bore peripheral venous lines, massive vascular filling, started in the pre hospital phase, using warmed 0.9% saline. An initial flow rate of 10 to 15 ml/kg/h is recommended, followed by 500 ml/h over the first 24 hours, with a total volume of up to 10 to 20 litres per day. Colloids such as hydroxyethyl starch (HES) can be used in the event of shock. However, lactated Ringer's lactate is contraindicated because of its potassium content. In the event of hyperkalaemia, urgent treatment is required, includingalkalinisation, diuretic therapy, generally with 20% mannitol (1 to 2 g/kg over the first four hours), and administration of calcium chloride in the event of electrical signs. Finally, succinylcholine should be avoided during general anaesthesia, as it may increase hyperkalaemia [9]. In the presence of renal failure associated with hyperkalaemia, severe acidosis and volume overload, extrarenal purification becomes essential. The use of loop diuretics is not recommended because they acidify the urine. Mannitol, on the other hand, offers several advantages: it acts as a diuretic, increases vascular volume, improves cardiac and urinary output, and reduces pressure in the muscle compartments. However, because of the risk of nephrotoxicity and hyperosmolarity, its dose should not exceed 200g per day. Its use is contraindicated in anuric patients. Alkalinisation by infusion of sodium bicarbonate aims to maintain a urinary pH above 6.5, thereby preventing precipitation of myoglobin in the renal tubules. This alkaline therapy is generally continued for around three days, the time required to eliminate the myoglobin. Other management measures include prevention of hypothermia, effective multimodal analgesia and oxygen therapy by mask to correct hypoxia. Mechanical ventilation may be required in

situations of shock or severe hypoxia, or in the presence of cerebral or thoracic lesions associated with polytrauma. In the event of persistent hypotension despite vascular filling, the early use of vasoactive drugs is essential. If injuries to the cervical spine are suspected, a cervical collar should be fitted. Limb fractures should be immobilised, and bleeding wounds treated with a pressure dressing. The use of a pressure tourniquet should be the exception, reserved for major arterial haemorrhage, as it worsens muscle ischaemia. Management of open trauma requires appropriate antibiotic therapy and anti-tetanus prophylaxis in accordance with current recommendations. Blood transfusions, which are often necessary, are aimed at achieving a haemoglobin target of between 9 and 10 g/dl. Systematic treatment of hypocalcaemia is not justified, except in cases of hypocalcaemia associated with a haemostasis disorder [10]. Some studies suggest that hyperbaric oxygen therapy may improve the hypoxia associated with crush syndrome [11]. Surgical management of crushed limbs generally involves three stages: Stabilisation of the fracture, often using an external fixator due to skin and muscle damage. Repair of vascular lesions, often using bypass grafts. Offloading aponeurotomies to treat compartment syndrome, characterised by intra-compartmental pressure in excess of 30 mmHg and signs of vascular and nerve damage. Although limb amputation is a last resort, it remains a complex decision requiring multidisciplinary consultation between surgeons, anaesthetists, the family and, if possible, the patient himself. In our patient's case, external fixators were used to stabilise the fracture, and the loss of skin substance was treated with a skin flap.

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