Crush Syndrome

The ‘Diseases Database’ defines crush syndrome as:

“Severe systemic manifestation of trauma and ischaemia involving soft tissues, principally skeletal muscle, due to prolonged severe crushing. It leads to increased permeability of the cell membrane and to the release of potassium, enzymes and myoglobin from within cells. Ischaemic renal dysfunction secondary to hypotension and diminished renal perfusion results in acute tubular necrosis and uraemia.”

It is also known as Bywaters’ syndrome. Crush syndrome was first described by Bywaters in the British Medical Journal in 1941 after the London Blitz.

Pathophysiology

- Crush injury can follow prolonged continuous pressure on muscle tissue. Crush injury can lead to crush syndrome.
- Ischaemia reperfusion (when the pressure is released from the crushed limb) is the main mechanism of muscle injury in crush syndrome. There is traumatic rhabdomyolysis.
- Muscle injury causes large quantities of potassium, phosphate, myoglobin, creatine kinase and urate to leak into the circulation.
- Myoglobin levels in the plasma are normally very low. If a significant amount of skeletal muscle is damaged (>100 g), excess myoglobin is filtered by the kidneys and can cause renal tubular obstruction and renal damage: the excess myoglobin is nephrotoxic.
- Intravascular volume depletion and renal hypoperfusion, combined with myoglobinuria, result in renal dysfunction.

Crush syndrome is characterised by:

- Hypovolaemic shock (due to sequestration of water in the injured muscle cells).
- Hyperkalaemia (release of cellular potassium by the injured muscle cells).

This can also lead to:

- Metabolic acidosis (release of cellular phosphate and sulphate by the injured muscle cells).
- Acute kidney injury.
- Disseminated intravascular coagulation (DIC).

Epidemiology

Crush syndrome has been described in numerous settings, most commonly after earthquakes, during war and after explosions that have caused buildings to collapse. It is also seen following industrial accidents, such as those occurring in mining and after road traffic accidents. The incidence of crush syndrome has been reported as 2% to 15% in all trauma patients and it can be as high as 30% in earthquake victims.

Presentation

The key clinical features of crush syndrome are:

- Crushing injury to a large mass of skeletal muscle.
- Sensory and motor disturbances in the compressed limbs, which subsequently become tense and swollen. The limb/body part may be pulseless.
- Myoglobinuria and/or haemoglobinuria, which may make the urine tea-coloured quite early on.
- There may be oliguria with profound hypovolaemic shock.
- Nausea, vomiting, confusion and agitation may occur as consequences of disturbed body chemistry: urea, creatinine, uric acid, potassium, phosphate and creatine kinase are elevated. There may also be hypocalcaemia.

Initial management

- Check for safety of self and others.
- The patient must be assessed in keeping with the usual criteria for assessing a severely injured person.
- Assessment of ‘Airway, Breathing and Circulation’ should be carried out.
- Monitor vital signs and oxygen saturation level.
- Administer oxygen through a non-rebreather mask.
- Assess limbs using the '5 Ps' - pain, paraesthesia, paralysis, pallor and pulselessness - to estimate extent of ischaemic injury.
Attention should be given to life-threatening injuries.
Venous access should be obtained as early as possible, ideally before the trapped limb is freed and decompressed.
Preserve body heat.
Prior to release, consider an arterial tourniquet if compression has been less than 30 minutes. Apply a tourniquet if compression has been more than 30 minutes.
In the adult, a saline infusion of 1,500 ml/hour should be initiated during extrication. Early, vigorous hydration (≥10 litres/day) helps preserve renal function. 
Because of the very high risk of acute kidney injury, a catheter should be inserted at an early stage and urine output monitored.
Because of the need to maintain fluid balance, a central venous line is usually required.
Analgesia should be provided. Entonox® may be more suitable than IV or oral analgesia due to the dangers of hypotension.

Investigations

Blood tests
These should include:

- U&Es, including potassium.
- Creatinine.
- Calcium (there may be hypocalcaemia).
- Phosphate.
- Creatine kinase (rhabdomyolysis has been defined as total creatine kinase levels 5-10 times above normal in a patient with typical symptoms and/or risk factors).[7]
- Uric acid (may be raised).
- FBC and clotting studies (to look for evidence of DIC).
- LFTs (may show hepatic dysfunction).
- Blood gases.

Other investigations
- Urine dipstick for myoglobin (but this is only positive in 50-80% of cases of rhabdomyolysis so a negative dipstick does not exclude it).[8]
- ECG may show changes secondary to hyperkalaemia.
- The usual assessment for trauma, including X-rays, should be performed.
- Assessment of compartment pressures (see ‘Complications’, below).

Further management

Medical[3, 6]
- Urine output should be maintained at 300 ml/hour until myoglobinuria has ceased.[9]
- A forced mannitol-alkaline diuresis may help to protect the kidneys against damage from myoglobin and may reduce the risk of hyperkalaemia. Mannitol protects the kidney by enhancing renal perfusion and may reduce muscle injury as well.
- Urinary alkalisation with sodium bicarbonate may help to prevent acute kidney injury.
- Hyperkalaemia will need treatment.
- Hypocalcaemia does not generally need treatment.
- Renal dialysis may be needed.
- DIC will need treatment with fresh frozen plasma, cryoprecipitate and platelets.

Surgical[4]
It may be necessary to amputate crushed limbs. Amputation at an early stage may prevent crush syndrome.

Complications[4]

- Hyperkalaemia and infection are the most common causes of death. Hyperkalaemia can lead to arrhythmia and arrest.
- Infection is a major cause of death in disaster zones.
- Acute kidney injury can occur.
- Compartment syndrome can occur because of the uptake of fluid into muscle cells contained within a tight compartment. Fasciotomy is useful in reducing muscle damage from compartment syndrome.[10] It should be done early.
- DIC can occur with massive tissue damage.[2]
- Creatine kinase levels peak within 24 hours and should then decrease by 30-40% per day. Serial measurements will be needed. If levels continue to elevate, consider ongoing muscle injury or compartment syndrome.[11]

Prognosis

- Adequate fluid support improves prognosis.[12]
- The mortality rate for crush syndrome following the earthquake in northern Turkey in 1999 was 15.2%.[13] However, rates in subsequent quakes have varied and it is thought that many factors may affect survival, such as hampered rescue and transport, destroyed medical facilities, availability or not of sophisticated therapeutic options and the method of construction of the collapsed buildings.
• Time under the rubble does not have an adverse effect on outcome but this may be because those who survive have been less severely injured. It has been recommended that recovery of survivors should continue for at least five days.\(^\text{[14]}\)
• Anyone who has been buried under rubble for a length of time will be dehydrated and hence more susceptible to renal damage.

**Prevention**

• In any major disaster, adequate triage must carried out to identify those in need of urgent attention. This may have a marked effect on morbidity and mortality.\(^\text{[15]}\)
• Adequate rehydration reduces the risk of acute kidney injury in crush syndrome.
• In acute kidney injury, peritoneal dialysis may be life-saving. To this end, the Renal Disaster Relief Task Force (RDRTF) has offered support for renal problems in the aftermath of several disasters - eg, the Marmara earthquake (1999) in Turkey, the Bam earthquake (2003) in Iran and, more recently, the earthquake in Eastern Turkey (2011).\(^\text{[16]}\)

**Further reading & references**

  3. Reperfusion Injury/Crush Injury; Wheeless' Textbook of Orthopaedics
  5. Crush Injury; University of California Medical School, 2013

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