Crush injury & compartment syndrome

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Crush injury

• Rhabdomyolysis caused by continuous & prolonged pressure on the limbs
• Results in a death of a large amount of muscle tissue
• Usually follows severe beatings, disasters such as collapse of mines, earthquakes, bombings, electrical injuries (lightning strikes, high voltage power lines)
Pathogenesis of muscle injury
Pathogenesis

• Muscle damage from electrical injuries:
  ❖ direct muscle injury by the electrical current
  ❖ high temperature generated
  ❖ Coagulation of blood vessels
Crush syndrome

- Systemic manifestation of crush injury
- Occurs when the cellular contents of damaged muscles are released into the circulation
- Characterised by:
  - hypovolemic shock,
  - electrolyte disturbances
  - acute kidney injury
- Carries a high mortality & morbidity rate
Crush syndrome

• Hypovolemia
  ❖ leakage of plasma into the intestinal spaces of traumatised extremities
  ❖ most common cause of death during the first 4 days of crush injury
• Hyperkalemia
  ❖ second most common cause of early death, with its associated cardiotoxicity
  ❖ Provoked by hypocalcemia & hypovolemia
Crush syndrome

- **Hypocalcemia**
  - due to influx of Ca into the affected muscle tissue
- **Hyperphosphatemia**
  - can worsen the hypocalcemia by depressing the levels of 1,25dihydrocholecalciferol
- **Increased tissue thromboplastin**
  - can lead to DIC
- **Organic acids**: resulting in metabolic acidosis
Crush syndrome

• Acute kidney injury
  ❖ 4-33% of patients with rhabdomyolysis
  ❖ Present in up to 1/3 of patients despite adequate resuscitation and prophylaxis against myoglobinuric AKI
  ❖ 3 underlying mechanisms implicated:
    ✓ Decreased renal perfusion
    ✓ Cast formation with tubular obstruction
    ✓ Direct toxic effects of myoglobin on the renal tubules
Crush syndrome-AKI

• Decreased renal perfusion
  ❖ Hypovolemia
  ❖ Release of vasoconstrictors in the presence of myoglobin:
    ✓ **Endothelin-1**: constrict the afferent & efferent arterioles, thereby reducing the GFR
    ✓ **platelet activating factor**: produced in the mesangial & glomerular cells
Crush syndrome-AKI

- **Cast formation & tubular obstruction**
  - myoglobin precipitates with uric acids in the presence of aciduria forming casts
  - casts obstruct urine flow with a resultant transtubular leakage of glomerular filtrate
Crush syndrome

• Direct toxic effect of myoglobin
  ❖ Free-radical –mediated renal injury
  ❖ At the urinary ph of<5.6, myoglobin dissociates into ferrihaemate & globin
  ❖ Ferrihaemete has a nephrotoxic effect:
    ✓ Iron catalyses the formation of free radicals which leads to lipid peroxidation
    ✓ Heme group induce peroxidation during redox cycling between its different oxidation states
Diagnosis

- Clinically-painful, swollen extremities with a background history of trauma
- Exclude compartment syndrome
- Dark, tea coloured urine, that is dipstick positive
- Blood: CK and myoglobin levels
Prognostic measures

• Predict likelihood of renal failure
• Both serum CK & myoglobin levels represents the severity of muscle injury

• **Creatinine kinase**: preffered because:
  ✓ Not cleared as quickly as myoglobin, thus a more sensitive marker of muscle injury
  ✓ Readily available and less expensive
  ✓ Therefore, should be used to screen patients with suspected rhabdomyolysis
• Recommendation: suspected rhabdomyolysis:
  ✤ Serum CK levels:
  ✓ >20 000- treat for rhabdomyolysis
  ✓ <20 000-repeat 8 hourly, until 3 consecutive values decline
Treatment-crush injury

• Primary objectives:
  ✓ To achieve hemodynamic stability and prevent volume overload
  ✓ To achieve a urine output >300ml/hr
  ✓ To maintain urine ph between 6 & 7
  ✓ To keep serum ph < 7.5
Treatment- forced alkaline diuresis

• **Fluid resuscitation**: preferably started at the site of injury, until the patient is euvoletic

• **Mannitol**:
  ✓ Osmotic diuretic, promoting an increase in urine output and the washout of tubular myoglobin
  ✓ Volume expander
  ✓ Effective hydroxyl-free radical scavenger
  ✓ **Side effects**:  
    ▪ volume overload in patients with marginal cardiac function & established AKI  
    ▪ Can produce a hyperosmolar state & electrolyte derangement
Treatment

• Sodium bicarbonate
  ✓ Alkalinize the urine
  ✓ Decrease cast formation
  ✓ Lessen the direct toxic effects of myoglobin
  ✓ Can ameliorate the systemic acidosis

• MYOGLOBIN REGIME (FORCED ALKALINE DIURESIS):
  0.45% SALINE + 75ML 8.5% NAHCO3 + 250ML MANNITOL TO RUN AT A RATE OF 166ML/HR
Treatment

• Saline diuresis vs forced alkaline diuresis
  ✓ Retrospective study: n=24
  ✓ To determine the benefit of forced alkaline diuresis over that of saline diuresis
  ✓ Results: none of the patients on saline diuresis developed renal dysfunction
  ✓ Limitations: low degree of muscle injury (average CK: 2750U/L)
  ✓ This study neither supported nor negated the use of a forced alkaline diuresis
  ✓ Therefore a prospective study is recommended in this regard
Treatment

• Loop diuretics: acidifies the urine thus precipitating the direct toxic effect of myoglobin

• Antioxidants: glutathione, vitamin E analogs, but still experimental
Treatment of renal failure

• Renal replacement therapy
• Indications:
  ✓ Intractable hyperkalemia and acidosis
  ✓ Refractory to volume expansion
  ✓ Refractory to bicarbonate administration
• NB: hypocalcaemia must not be corrected unless there is a danger of a hyperkalemic arrhythmias since most of it will be deposited in the injured muscles
Treatment

• Fasciotomies
  ✓ not recommended as it is associated with high mortality and morbidity in these patients
  ✓ Complications: - recurrent bleeding - infection
  ✓ Should only be carried out if it is open injury, where aggressive radical debridement is performed
Compartment syndrome
Compartment syndrome

- Condition in which a raised pressure in a closed osteofascial compartment reduces capillary perfusion below a level necessary for tissue viability
- It is both a life & limb threatening condition requiring emergency treatment
- Can affect virtually every muscle group, but it is common in the forearm & leg
Surgical anatomy

- Forearm compartment
  - 3 fascial compartments: superficial flexor, deep flexor & extensor compartments
  - Deep flexor is more prone to develop increased intra-compartmental pressure
Forearm compartment

Normal Forearm

Nerves
Blood vessels
Ulna
Radius

Cross-sectional view through the forearm
Surgical anatomy

- Leg compartments
  - Four compartments:
    - Anterior compartment
    - Peroneal compartment
    - Superficial posterior compartment
    - Deep posterior compartment
  - Anterior and the deep posterior compartments are most commonly affected (author’s experience: all four compartments are usually simultaneously involved to a greater or lesser degree)
Leg compartments
Aetiology

• Orthopaedic factors
  ✓ Fractures of tibial shaft, distal radial & ulnar
  ✓ Closed therapeutic interventions (traction, cotton padding, plaster cast)
  ✓ Intramedullary nailing

• Vascular causes
  ✓ Ischemic-reperfusion injury
  ✓ Hemorrhage (trauma, bleeding tendencies)
  ✓ Phlegmasia caerulea dolens
Aetiology

• Iatrogenic
  ✓ Vascular puncture in anticoagulated patients
  ✓ Extravasation of IVI fluids
• Soft tissue injury
  ✓ Crush injury
  ✓ Burns (circumferential burns with the contraction of the eschar)
  ✓ Snake bites
  ✓ Frost bites
Pathophysiology

- Increased interstitial swelling
- Increase in interstitial pressure
- Partial or complete collapse of the venules & capillaries
- Increased hydrostatic pressures
- Compromised blood flow
- Hypoxia of nerves & muscles
- Tissue necrosis
- Resolution by fibrotic tissue scar
Pathophysiology

• Factors predisposing to development of compartment syndrome
  ✓ Reduced perfusion pressure: hypotension, elevation of the limb
  ✓ Tendency to oedema: reduced plasma oncotic osmoytic pressure, increased venous pressure
  ✓ Traction
  ✓ Intact fascial envelope
Clinical features

• Pain (severe & out of proportion to the apparent injury)
• Pain on passive stretch
• Swollen & tense compartment
• Progression of the above over a short time period
• Paraesthesia (esp. loss of two point discrimination)
• Pallor & pulseness (usually with vascular injury)
• Paralysis (late symptom)
Diagnosis

- Clinical
- Measuring intra-compartmental pressure:
  - Normal resting pressure: 0-8 mmHg
  - Differential pressure between diastolic pressure & ICP within 10-30 mmHg of the diastolic pressure: threshold for performing a fasciotomy
Treatment

• Fasciotomy
  ✓ Forearm: volar fasciotomy preferred but concurrent dorsal incisions may be done
  ✓ Leg: single or double incision fasciotomy, with or without fibular excision
  ✓ To decompress adequately all the compartments of the leg requires a longer incision of between 12 & 20cm
Fasciotomy incisions-forearm

Forearm Fasciotomy Incisions

Dorsal

Volar
Fasciotomy incision-leg
Complications-fasciotomy

- Wound infection
- Bleeding
- Impaired sensation around the margins of the wound, with a tethered scar
- Poor calf muscle function
- Chronic venous insufficiency
- NB: complications are common in delayed fasciotomies
Other treatment modalities

- Hyperbaric oxygen therapy
  - Reduces oedema & muscle necrosis
  - Hyperoxygenation increases both the supply of oxygen & causes vasoconstriction, which in turn reduces capillary blood pressure & transudation

- Mannitol
  - Reduces postischemic oedema & muscle necrosis
    (experimental)
Conclusion

• Crush injury & compartment syndrome carry a high mortality & morbidity unless treated adequately and earlier

• Ischemic-reperfusion injury is a fundamental pathogenic mechanism for both crush injury & compartment syndrome

• CK should be the marker used for screening

• Mainstay of treatment for crush injury is to restore intravascular volume & prevent crush syndrome
Conclusion

• Mainstay of treatment in compartment syndrome is decompression of the affected compartment through fasciotomy

• Fasciotomy should preferably be done within 6 hrs of injury
References

• Brown C, et al, journal of trauma 2004
Thank you!