The Crush Syndrome

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Outline

- Discuss crush injuries and the Crush Syndrome
- Define treatment
- Discuss the treatment and management mangled extremities
- Discuss vascular injury and assessment
- Case discussions

The Crush Syndrome is the presence of localized crush injury with systemic manifestations: incidence 2-15%
Crush Injury is compression of body parts causing localized muscle damage
- bombings, industrial accidents, building collapse, earthquake tornadoes
Crush Injury
Muscle ischemia and Necrosis from Prolonged Pressure (Local effects)

Crush Syndrome (Systemic Effects)

- Fluid Retention in Extremities (third spacing)
- Myoglobinuria
- Metabolic Abnormalities (electrolytes)
- Secondary Complications
- Hypotension
- Renal Failure
- Cardiac Arrhythmias
- Compartment Syndrome

Crush Injuries
- Injuries typically associated with disasters that include muscle injury, renal failure and death
- Man made-war and natural-earthquake
- Earthquakes 3-20\% of crush injuries
- Building collapse up to 40\% of extricated victims
- Vehicular Disaster
- Terrorist Acts- Oklahoma City, 9/11
- Systemic manifestations of muscular cell damage resulting from pressure of crushing

Crush Injuries
- Recognized after the Messina earthquake of 1909 and during WWI by German MDs
- First described in the English literature by Bywaters and Beall in 1941
  - Several patients who were crushed during WWII during the Blitz over London.
  - All patients died from renal failure despite resuscitation

Br Med J 1941:427-432

The Crush Syndrome
- Characteristic Syndrome the results in rhabdomyolysis, myoglobinuria, ARF.
- Three criteria
  - Involvement of muscle mass
  - Prolonged compression 4-6 hrs. but can be < 1 hr
  - Compromised local circulation

Gonzalez, D Crit Care Med 2005 33. No 1(Suppl)
Causes of Mortality after Untreated Crush Injury

- **Immediate:**
  - Severe head injury, traumatic asphyxia, and torso injuries

- **Early:**
  - Hyperkalemia, hypovolemic shock

- **Late:**
  - Renal failure, coagulopathy, and hemorrhage, sepsis

Clinical Manifestations

**Crush Syndrome**

- **Hypotension:**
  - Massive 3rd spacing
  - Shock contributes to renal failure
  - Third spacing can lead to compartment syndrome

- **Renal Failure**
  - Rhabdomyolysis releases myoglobin, K, P04, Cr, into circulation
  - Myoglobinuria leads to renal tubular necrosis
  - Release of electrolytes from ischemic muscle cause metabolic abnormality

Clinical Manifestations of the Crush Syndrome

- **Metabolic Abnormalities:**
  - Ca flow intracellularly through leaky membranes causing systemic hypocalcemia
  - K is released from muscle causing systemic hyperkalemia
  - Lactic Acid is released from ischemic muscle into systemic circulation, causing metabolic acidosis
  - Imbalance between K and Ca cause cardiac arrhythmias-acidosis makes it worst
Clinical Manifestations
- Electrolyte Disturbances
  - Hyperkalemia, Hypocalcemia, Hyperphosphatemia, Metabolic acidosis
- Renal
  - Renal vasoconstriction due to shock
  - Pigment toxicity due to myoglobin
  - ATN
  - Luminal obstruction
  - Acute Renal Failure

Indicators of Severity
- CPK elevation correlates with renal failure (RF) and mortality
- Risk of mortality and renal failure increased with CK over 75,000 U/L
- Other suggested counting limbs crushed
  - one limb is 50,000 U/L
- Crush one limb - RF 50%, two - RF 75%, three - 100%

Oda J et al; J Trauma 1997;33:507-512

Crush Syndrome Pre-Hospital
- Coordinate time of release with rescue personnel
- Mass casualty scenarios should be discussed with personnel
- Airway secured and protected from dust
- Adequate oxygenation
- Maintain body temperature
- Rapid transit to a trauma center
- Intravenous fluids, cardiac monitoring

Crush Syndrome Pre-hospital
- Establish two large bore IVs
- Administer 1-2 liter or LR prior to extrication
  - If prolonged infuse 1.5 liters/hr
  - Young and elderly be cautious about fluid overload
- Sodium Bicarb 2 amps prior to extrication
- Cardiac monitoring
- Pain control PRN
- Extricate
**Definitive Treatment**

- **Hypotensive:**
  - Massive fluid shifts
  - Hydration 1.5 liters/hour
  - Patient may gain massive amounts of weight in the resuscitation
  - Similar to Burn patients

- **Renal Failure:**
  - Prevent renal failure with adequate hydration
  - Maintain diuresis of 300ml/hr with IV fluids and mannitol (carefully)
  - Triage to hemodialysis as needed
    - May need 60 days of Rx
    - Should return to normal function

**Definitive Treatment**

- **Metabolic Abnormalities:**
  - Acidosis: administer IV sodium bicarbonate until urine pH reaches 6.5 to prevent myoglobin deposition
  - Hyperkalemia/Hypocalcemia: administer Ca, sodium bicarbonate, insulin/D5W, consider kayexalate
  - Cardiac arrhythmias monitor for cardiac arrhythmias and arrest and treat

- **Secondary Complications**
  - Monitor for compartment syndromes and do fasciotomies as needed
  - Treat open wounds with antibiotics, tetanus toxoid, and debridement
  - Monitor for pain, pallor, pulselessness, paresthesias, paralysis - ischemia
  - Observe all crush injuries - even those who look normal
  - Delays in hydration for more than 12 hours lead to renal failure
  - Definitive surgery - amputations as needed
**Perte’s Syndrome or Traumatic Asphyxia**

- Craniocervical cyanosis
- Subconjunctival hemorrhage
- Multiple petechiae
- Neurological symptoms
- Results from sudden severe compression of the thorax or upper abdomen or both
- Valsalva is necessary before crush
- Associated injuries pulmonary contusions, hemothorax or pneumothorax

In addition to the monitoring of the vital signs, more attention should be paid to observation of the blood pressures and changes of urine to make an early diagnosis of crush syndrome.

We believe that surgical intervention of earthquake victims should be considered for earthquake victims when the correct indications are strictly followed.

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**Aortic Crush Injuries vs. MVA**

- Increase risk of Rhadomyolysis, ATN and renal failure
- Tendency to develop lower risk aortic injuries than MVAs
- Both type of patient must be followed since they can progress
- High rate of mortality in missed injuries
Retrospective Cohort design of data from the NTDB 2007-2009

- Assessed the result from 222 Level I & II trauma centers of severely mangled extremities
- 1354 patients were analyzed and logistic regression done to assess factors associated with amputations
- 21% of patients underwent amputations in this study (9% early amputations)

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Factors correlated with amputation
- Presence of severe head injury AIS>3
- Presence of shock in the ED (BP<90)
- Limb injury type
- High energy mechanism of injury
- Age, comorbidities, and insurance status do not govern amputation rate
- Injury type is the most important thing

J of Trauma 2013;74:597-603

Blunt Arterial Injury Salvage Rates

- Have a high amputation rate due to associated soft-tissue and nerve injuries (the mangled extremity)
- These injuries may result in a non-functional limb in spite of a successful revascularization

Blunt Vascular Trauma

- Retrospective review at a Level I trauma center
- Jan 1995-Dec 2002 62 patients
- ISS >14.6, 93 vascular injuries, 66% hard signs, 95% had associated fracture
- Age, ISS, and MESS was significantly different between survivors and non-survivors
- Injuries to the upper and lower extremity
- Shunt were used in 18 vessels prior to repair
- Anteroposterior tibia artery most commonly injured
- Amputation rate was 18% 3X that for penetrating injury

Rozycki G et al., J Trauma 2003;55:814-824
Mangled Extremity

- Indications for Primary Amputation
  - Anatomically complete disruption of sciatic or posterior tibial nerves in adult even if vascular injury is repairable
  - Prolonged warm ischemia time
  - Life threatening sequelae
    - rhabdomyolysis

- Relative Indications for Primary Amputation
  - Serious associated polytrauma
  - Severe ipsilateral foot trauma
    - Loss of plantar skin/weight bearing surface
  - Anticipated protracted course to obtain soft-tissue coverage and skeletal reconstruction

Variables in Consideration of Limb Viability

- Skin/Muscle Injury
- Bone Injury
- Ischemia (time, degree)
- Type of Vascular Injury
- Shock
- Age
- Infection
- Associated injuries (pulmonary, abdominal, head, etc.)
- Comorbid Disease (peripheral vascular disease, diabetes mellitus, etc.)

Classification Systems

- Mangled Extremity Syndrome Index (MESI) - 9 variables
- Predictive Salvage Index (PSI) - 4 variables
- Mangled Extremity Severity Score (MESS) - 4 variables
- Limb Salvage Index (LSI) - 7 variables
- NISSSA scoring system (Nerve Injury, Soft Tissue Injury, Skeletal Injury, Shock, Age of Patient Score) - 6 variables
- Hanover Fracture Scale (HFS) - 12 variables
### Vascular Injury – Non-invasive tests

#### “Soft signs”
- Large stable hematoma
- Prior significant bleeding
- Possible nerve damage
- Proximity (<3cm, used to angio but only 10 – 20% pos)

Physical exam and API – arterial pressure index (ABI) <0.9, perform Duplex, color flow – to decide whether to angio, observe, OR

Mandatory exploration not needed

*Richardson, JD et al., Arch Surg 122:878, 1987*
Vascular Injury – Surg vs Angio

- “Hard signs” – absent or dec distal pulse, pulsatile bleeding, expanding hematoma, bruit
- Depends on surgeon whether explore and repair or request angio – usually do study first if multiple (gsw), blunt trauma (pre-ortho), or possible vascular intervention

Vascular Injury – Surg vs Angio

“Hard signs”
- absent or decreased distal pulse-no brainer usually
- pulsatile bleeding
- expanding hematoma
- bruit

Depends on surgeon’s experience whether explore and repair or request angio, study requested for penetrating (gsw), or possible vascular intervention

Patient with suspected vascular injury

Resuscitation

“Hard” signs

Observe

API<0.9

Positive

Duplex

Negative

AGRAM

Clinical Follow-up

Surgical Exploration

Soft signs

API>0.9

Positive

AGRAM

Clinical Follow-up

Temporary Vascular Shunt
Definitive Vascular Repair

Mechanisms of Arterial Injury

- Patient experiences – blunt, penetrating or combined mechanism (fx/dislocation)
- Artery experiences – crush, shear, stretch, laceration, shock wave
- Artery responses – spasm, tear (partial or complete transection), bleeding with hematoma, intimal injury, thrombosis
- Angio shows – narrowing (spasm or extrinsic compression), intimal injury (flap, dissection), extravasation, pseudoaneurysm, AVF, abrupt cut-off, intraluminal thrombus

Train Accident
Mangled Extremity Management

- Involves a determination of both the feasibility (restoring viability) and advisability (restoring function) of salvaging the limb
- Should be a coordinated effort of the trauma, orthopedic, vascular and plastic surgeons starting at the initial evaluation of the patient
Goal of Early Management of Extremity Injury in the Polytrauma Patient:

Stop the Ongoing Injury

- Release compartments
- Reduce dislocations
- Debride open wounds
- Stabilize long bones
- Restore vascular flow

Crush Injury Signs and Symptoms - Summary

- Compression in excess of 60 minutes
- Involvement of large muscle mass
- Absent pulse and capillary refill return to distal limb
- Pale, clammy, cool skin
- Usually absence of pain in affected region
- Onset of shock
- Consider prior to extraction

The Crush Syndrome Treatment Summary

- Radical Surgery
- Fluid resuscitation
- Alkalization
- Mannitol Diuresis
- Compartment Syndromes must be treated
  - Pain, Pallor, Pulselessness, Parathesias and Paralysis

Conclusions

- Difficult situations in patients with multiple injuries
- Patients with crush injuries are complex
- Have to make a decision early if it is a salvageable extremity
- Amputation takes courage
- Multidisciplinary approach is the best when the patient initially presents