L'Hôpital de Montréal pour enfants
The Montreal Children's Hospital

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Crush Syndrome

Trauma Rounds

Jan 26, 2009

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Outline

- Terminology
- Historical descriptions
- Pathophysiology
- Features of crush syndrome
  - Shock, electrolyte problems, renal dysfunction, compartment syndrome, etc
- Management
- Outcomes

Objectives

At the end of this session, the learner will be able to:

- Define and describe the pathophysiology of crush syndrome
- Describe the features of crush syndrome
- Outline a management plan for a patient with crush syndrome

Terminology

- Crush syndrome
- Traumatic Rhabdomyolysis
- Bywater's syndrome

<u>Definition:</u>

- Severe systemic manifestation of trauma and ischemia involving soft tissues, principally skeletal muscle, due to prolonged severe crushing.

Terminology

Criteria?

- Crushing injury to skeletal muscle
- Sensory and motor disturbances to the compressed limbs -swollen and tense
- Myoglobinuria and/or hemoglobinuria
- Peak CK >1000 U/L
- Renal problems (oligouria, renal failure)

Settings

- Severe beatings
- Improper operative positioning
- Alcohol, drug intoxication→ immobilization
- MVAs
- Pneumatic antishock garment (PASG)
- Prolonged seizures
- Mine collapses
- Multistory building collapse
  - Up to 40% incidence
- War
- Earthquakes
  - eg Mamara earthquake, 1999
- Landslides
- Electrical injuries

Early descriptions

- WW1: Dr. Myer Betz: “muscle pain, weakness and brown urine” (1910)
- WW2: Dr. Eric Bywaters after the London blitz, BMJ 1941. “crush injury”

Early descriptions

- 1941, Bywaters:
  - "...in the renal tubules, degenerative changes and casts containing brown pigment"
- 1943, Bywaters and Stead
  - identified myoglobin as the brown pigment
  - Suggested Rx=
    - heat to the loins
    - volume resuscitation
    - alkalinization of urine
    - caffeine for diuresis

Our case

- 18 month old female
- Grandmother pushing her in her stroller
- Grandmother is hit by the bus mirror
- Child falls from stroller
- Bus runs over her legs
- Urgences Santé transfer to MCH

Our case

- What do you prepare?
- Who do you call?
- What do you do?

Epidemiology

- Earthquakes: > 2-5% buried in rubble
- Up to 50% of Mamara victims developed crush syndrome (Donmez et al, Iskit et al, 2001)

Pathophysiology

- Lysis of skeletal muscles and release of cellular contents (K, P04, myoglobin)
- Mechanical stress opens Ca channels with an influx in Na, Ca, fluid and neutrophils
- Main issue is muscle reperfusion with systemic effects of the toxins

Pathophysiology

Vascular compr

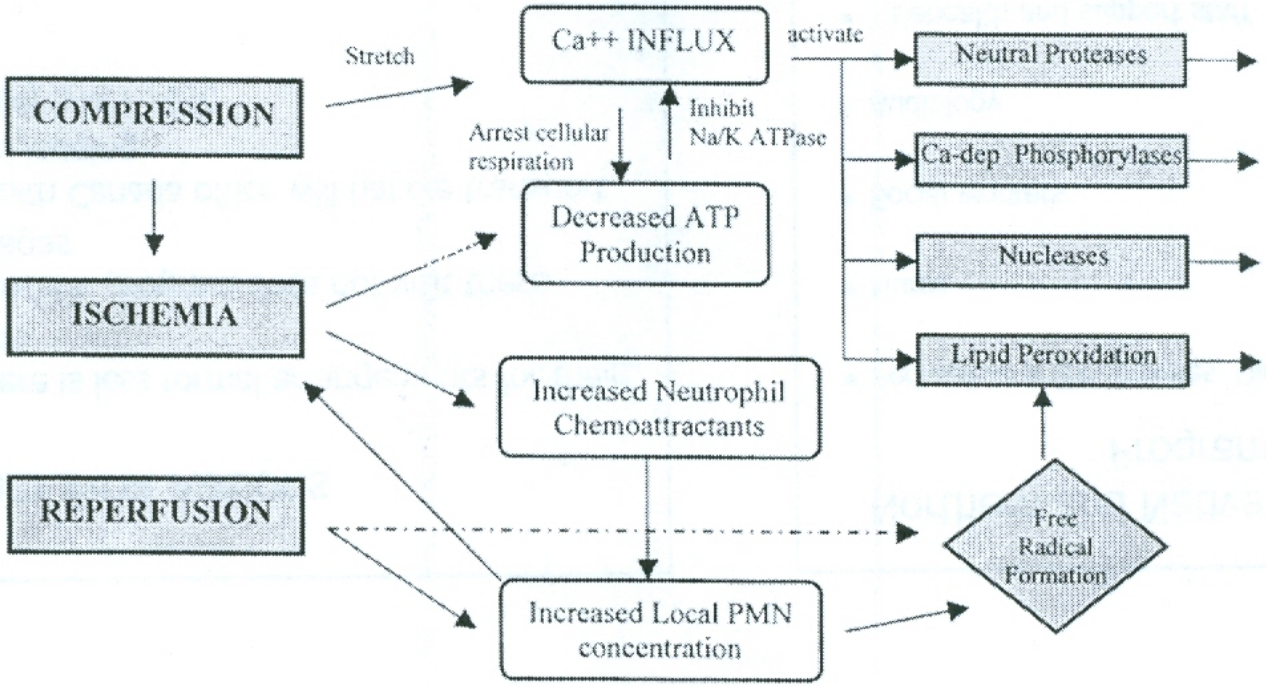


Fig. 1. The pathogenesis of rhabdomyolysis.

R
H
A
B
D
O

Malinoski, et al. 2004

Pathophysiology: reperfusion

Table 1
Intracellular contents released during rhabdomyolysis and their effects

Agent	Effect
Potassium	Hyperkalemia and cardiotoxicity, provoked by hypocalcemia and hypovolemia
Phosphate	Hyperphosphatemia, worsening of hypocalcemia, and metastatic calcification
Organic acids	Metabolic acidosis and aciduria
Myoglobin	Myoglobinuria and nephrotoxicity
Creatine kinase (CK)	Elevation of serum CK levels
Thromboplastin	Disseminated intravascular coagulation

Crush Time

- >1h likely to result in crush syndrome
- Reported after 20 minutes
- Can tolerate up to 2h warm ischemia
- 4-6 hours→ anatomical, functional changes
- >6 hours→ muscle necrosis

Clinical Evaluation

- History
  - SAMPLE Hx
  - Estimated crush time
- Physical Exam
  - External signs of trauma
  - Compartment syndrome (more later): temperature, colour, pulses, motor, sensory

Features of Crush Syndrome

- Shock
- Electrolyte disturbances
- Renal Failure
- Compartment Syndromes
- Other

Shock

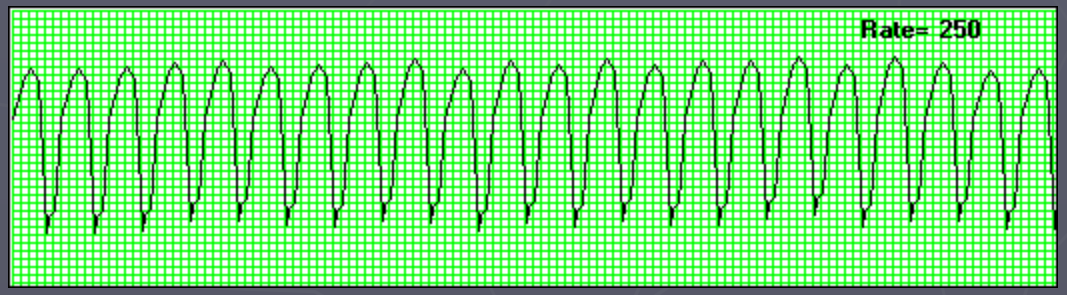
- #1 cause of death day 0-4
- Capillary leak→ volume loss
  - Up to 12L into the involved extremities (A. Blalock)
  - Global 3rd spacing

Case scenario

- A 15 year old male extracted from his collapsed apartment building
- He was trapped for 6 hours
- Decreased level of consciousness and unable to protect his airway
- You do a rapid sequence intubation

Case scenario

- The monitor alarms and you see:



WHAT HAPPENED?

Electrolyte Problems

- Hyperkalemia
  - Largest stores of K in skeletal muscle
  - #2 cause of death (D1-4)
- Hyperphosphotemia
  - Worsens hypocalemia
- Hypocalcemia
  - Influx of ca into muscle tissue

Tumour lysis- like

- **Recipe for arrhythmia =** acidosis + hyperkalemia + hypocalcemia

Renal failure

- up to 35% with crush syndrome →RF
  - **Donmez et al**, 2001: 20/40 crush syndrome, 7/20 (35%) RF, 4/7 dialysis
  - **Iskit et al,** 2001: 15/33 crush syndrome, 10/15 RF, 2/10 dialysis
- 3-50% associated mortality
- Accounts for 5-7% of ARF in the USA

Renal failure

- Pathophysiology
  1) ATN from decreased renal perfusion
  2) Myoglobin
     - Binds Tamm-Horsfell protein and precipitates→ casts→ tubule obstruction (worse if aciduria)
     - Stimulates free radical formation
     - Lipid peroxidation→ destruction of phospholipid bilayer

Renal failure

- Clinical presentation
  - Tea-coloured or “motor oil” urine
  - Positive urine dip for blood, few RBC on microscopy
  - Urine or serum myoglobin

Compartment Syndrome

= elevated interstitial pressure in a closed fascial compartment (P>30 mmHg)

Compartment P > capillary perfusion pressure

- Pressure relative to diastolic
  - <20-30mmHg consider faciotomy
- Vascular compromise, myoneural damage, tissue hypoxia

Compartment Syndrome

- 5 Ps
  - pain **with passive stretch**
  - Pallor
  - Paralysis
  - Paresthesia
  - pulseless
- Children ↑ risk: smaller spaces, less elastic

Other problems

- DIC-release of thromboplastin
- Pulmonary
  - Diaphragm weakness→ resp failure
  - Fractures, pneumo/hemothroax, contusion
  - Fat embolism
  - Pneumonia
  - ARDS

Management

Usual **trauma Rx**, plus...

- A
- B * avoid succhinylcholine
- C *early, aggressive fluid resuscitation
  - * No Ringer's lactate (contains K)
  - * trauma labs + *CK, Ca/P04/Mg, urine dip, serum/urine myoglobin, uric acid
  - *remove contaminated IV lines placed in field

Management

- D
- E *evaluate for compartment syndrome
- F *insert foley early to monitor U/O

Management- electrolytes and arrhythmia

- CLOSE electrolyte monitoring!
- Avoid giving K in IV fluids

- <u>Hypocalcemia</u>
  - Only treat for hyperkalemia
  - Risk of metastatic calcification

Management- electrolytes and arrhythmia

- Hyperkalemia
  - Calcium
    - May be ineffective (binds to P04)
    - Metastatic calcifications
  - Treatments causing cellular shifts- ineffective
    - Eg Insulin/glucose
  - May need hemodialysis earlier

Management- renal failure

- **Fluid resuscitation**
  - Improve renal perfusion pressure
  - Prevent contraction alkalosis with aciduria
    - Angiotensin II
    - Aldosterone
  - Better et al. 1979: 7 men, 7 ARF, 7 deaths. 1982: 8 men, 7 early fluids, 7 no ARF

Management- renal failure

- **Alkalinization**
  - Bicarb +/- acetazolamide
  - Improves clearance
  - Prevents precipitation
  - Reduces free radicals
  - Goal pH >6.5
  - Benefit: Rx of ↑K

Urine pH	Percent precipitated
6.5	4
5.5	23
5.0	46
<5.0	73

Gonzalez et al, 2005.

Management- renal failure

- Lasix (pros vs cons)
- Mannitol (pros vs cons)
- Renal replacement therapy
  - Support only
  - Myoglobin is not dialysable
- NEW!
  - Antioxidants (glutathione, vit E)

Management: Radical surgical procedures

- Crush Syndrome
  - Fasciotomy does not improve outcome (consensus only)
  - Amputation:
    - May not change outcome
    - Expedite extraction
- Compartment Syndrome
  - Fasciotomy?

Another talk...

Management

- DIC
  - Usual management

Management- Prehospital

- Remove compressive forces
- Risk to rescue personnel
- Start fluid resuscitation during extrication
  - Positive effect on survival (Better et al, Israel studies 1979 and 1982)
  - Donmez et al: n=40 in Mamara earthquake, 8 got fluids at the scene→ none developed RF
- Transfer to a dialysis-capable facility

Management

Anticipate and prevent complications

Prevention

- Disaster planning
  - Rescue availability
  - Personnel availability
  - Dialysis availability

Outcomes

Outcome

- Crush **time** DOES NOT predict outcome
- Renal and cardiac complications:
  - **Magnitude** of pressure
  - **Size** of compressed area
    - Rule of thirds (Ringer AG Jn, 2004) analogous to the rule of 9s for burns

Cause of death

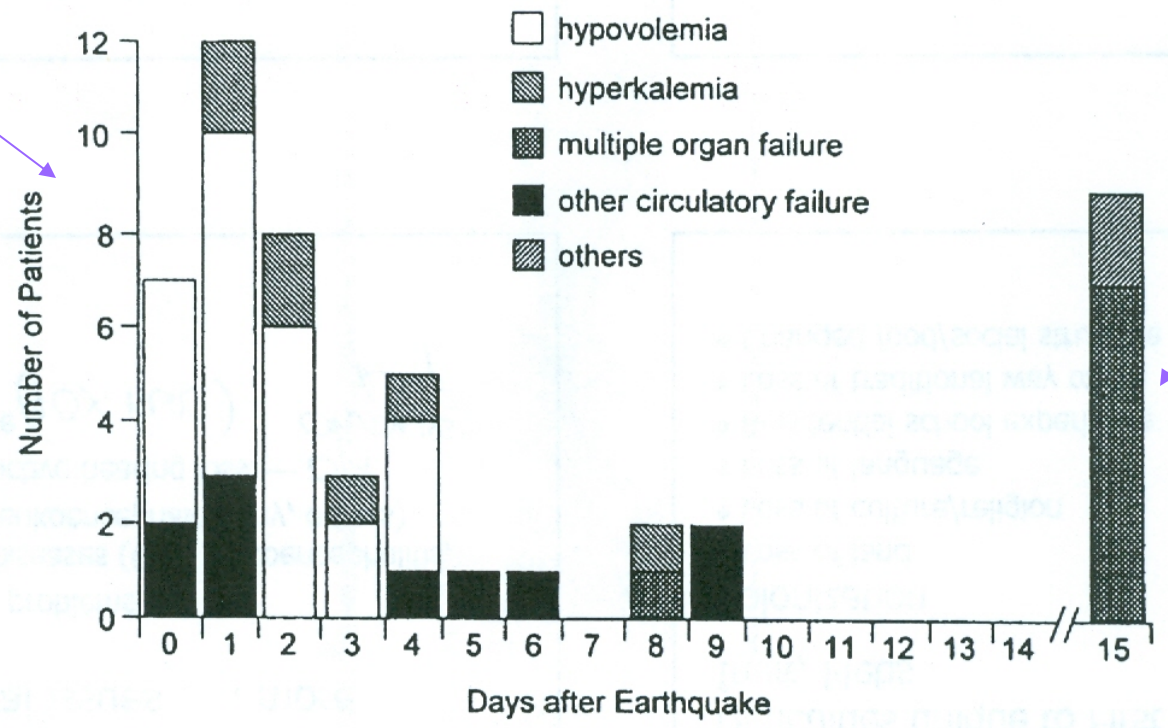
- Shock #1 (day 0-4)
- Hyperkalemia #2 (day 0-4)
- Renal failure (day 3-7)
- Infection (later)

- Mortality in Mamara = 15.2%
- Mortality double other trauma patients

(Kobe, Japan earthquake)

Cause of death:
Hanshin-Awaj earthquake

Hypovolemia
Hyperkalemia



sepsis

Fig. 2. The causes of death in 50 patients with the crush syndrome following the Hanshin–Awaj Earthquake. Deaths from hypovolemia and hyperkalemia were the most common in the early period while sepsis leading to multiple organ failure was responsible for most of the late deaths. (*Adapted from* Oda J, Tanaka H, Yoshioka T, et al. Analysis of 372 patients with crush syndrome caused by the Hanshin–Awaji Earthquake. J Trauma 1997;(42):470–6; with permission.)

Oda et al, J Trauma, 1997

Predictors of Poor Outcome

- **Peak CK level**
  - Most sensitive marker of ARF
  - >100 000 predicts dialysis, death
  - >20 000→ monitor closely (**ICU)**
- Oligouria
- Raised Cr
- Low platelets
- Low albumin
- Hypotension
- High K
- High body temperature
- Amputations
- Abdominal or thoracic trauma

Back to our case

- What do you prepare?
- Who do you call?
- What do you do?

Take home points

- Above all else, treat shock early and aggressively
- Anticipate other problems:
  - renal failure
  - electrolyte disturbances
  - compartment syndrome
- Need to work as an interdisciplinary team to achieve good outcome

Suggested reading

- Gonzalez D. Crush Syndrome. Critical Care Medicine V33(1) supp, Jan 2005, S34-41.
- Malinoski D, Slater M and Mullins R. Crush injury and rhabdolyolysis. Crit Care Clin 20 (2004) 171-192.