CAUSES OF SCI

1. Road accidents > 50% (80% in Kenya)
2. Assault
3. Falls
4. Sports injuries (e.g. diving in rugby)

5 6.5% SCI die before reaching hospitals

4 0.7% die within two years of injury

MECHANISM

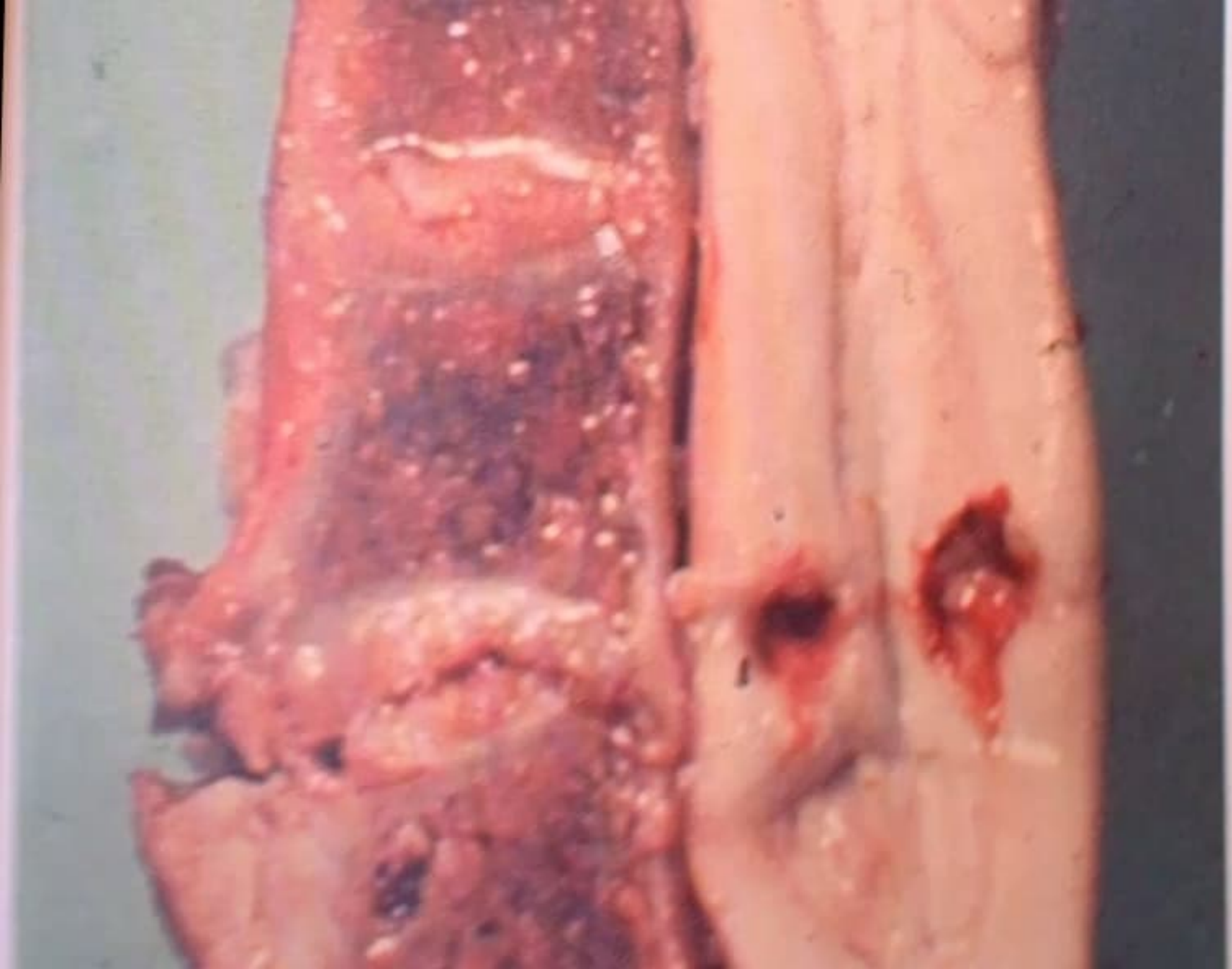
- Mechanical impact causes primary neural injury:

a. Neuropraxia- spinal shock

b. Neurotemesis

c. Axonotemesis- complete paralysis.

- The impact, compression and contusion causes immediate damage to nerve cells, axonal rails and blood vessels.



Spinal injury models show:

1. Increased tissue water content
2. Increased lactate levels
3. Decreased extra cellular calcium levels
4. Decreased tissue oxygenation
5. Decreased pyruvate
6. Decreased ATP concentration

- These are features of ischaemia, hypoxia, uncoupled oxidative phosphorylation and aerobic glycolysis

Spinal injury models show:

1. Increased tissue water content
2. Increased lactate levels
3. Decreased extra cellular calcium levels
4. Decreased tissue oxygenation
5. Decreased pyruvate
6. Decreased ATP concentration

- These are features of ischaemia, hypoxia, uncoupled oxidative phosphorylation and aerobic glycolysis

Explanatory theories:

- **Free radical theory:** Increase in O_2^- damage cell membrane
- **Calcium theory:** Influx of extra cellular Ca^{2+} which activates phospholipases, proteases and phosphatases. The net effect is disruption of mitochondrial activity (oxidative phosphorylation)
- **Opiate receptor theory:** Opiate antagonist have shown improvement
- **Inflammatory theory:** Increase in inflammatory substances like prostaglandins, leukotrenes, platelet activatory factors.

Whatever the mechanism, there is a *hostile environment* for surviving neurons which were not directly injured. The *neurotrophic* and other growth factors can not support the surviving neurons. *Toxic molecules* from dying neurons further kill the neutrons.

Inhibition of axonal regeneration

These factors affect the above process:

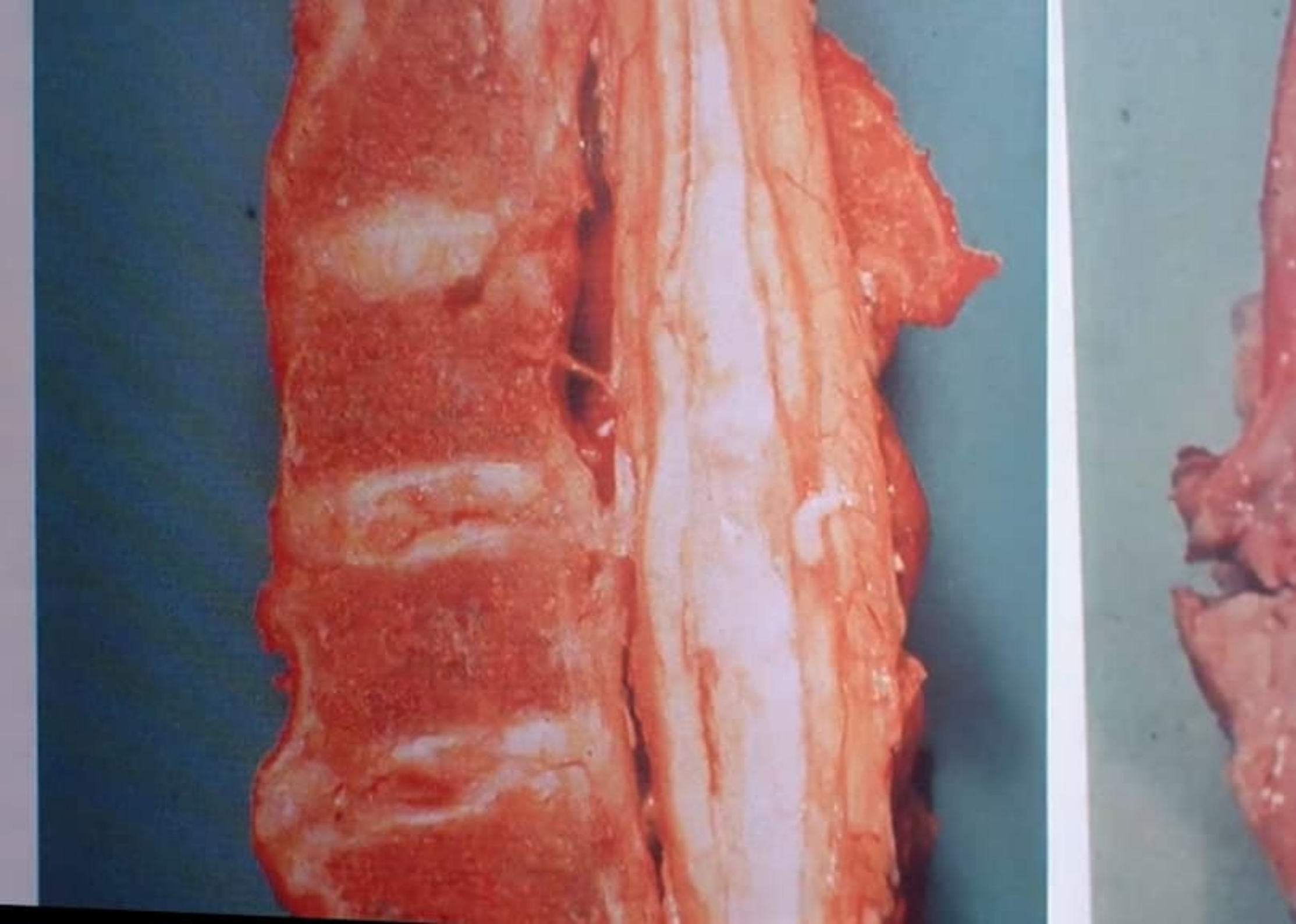
- Toxic molecules of dying cells
- Deleterious effect of immune system
- Astrocytic barrier
- Apoptosis

Histological Manifestation of ASCI

- Nectosis of central cord gray matter (hours)
- Followed by cystic degeneration (weeks)
- Development of scar

Management Principles

- Neuroprotection
- Inhibition of growth inhibiting proteins (and inhibition of astrocytic response).
- Provide molecular environment for surviving neurons (blocking apoptosis through Bcl-2)
- Delivery of appropriate trophic factors and stimulation of axonal growth.



HISTORY

- Weakness
- Transient paralysis
- History of electric shooting sensation distal to the site

PHYSICAL FINDINGS

- Pulse pressure dissociation
- Local crepitus on the spine, haematoma or local deformity
- Paralysis: - tetraplegia
  - paraplegia

- COMPLETENESS

-Sacral sparing:

  - rectal examination
  - anal wink

- LEVEL

-Radiological

-Clinical

  - Motor
  - Reflex
  - Sensory

- STABILITY

-Column concept

-Potential for neural injury