COMPLICATIONS OF FRACTURES

1. General

2. Local: Early and Late

General complications

1. Shock
   a. Hypovolemic or hemorrhagic shock.
   b. Septic shock.
   c. Neurogenic shock.

2. Fat embolism.

3. Pulmonary embolism.

4. Crush syndrome.

5. Multiple organs failure syndrome (MOFS).

6. Thrombo-embolism.

7. Tetanus.

8. Gas gangrene.

Local complications

Early

1. Visceral injury (the lung, the bladder, the urethra, and the rectum).

2. Vascular injury.


5. Haemoarthrosis.

6. Infection.

7. Gas gangrene.

8. Fracture blisters.

9. Plaster and pressure sores.

Late

1. Delayed union.

2. Non-union.

3. Malunion.

4. Avascular necrosis.

5. Growth disturbance.


7. Myositis ossificans.

8. Muscle contracture.


11. Joint instability.


GENERAL COMPLICATIONS

SHOCK

Three types of shock may complicate fractures

_Hypovolemic or hemorrhagic shock_

This type of shock is due to blood loss due to vascular injury. The vessels may be injured by the fracture pieces or in open fractures the vessels are injured by the same cause like in missile or bullet injury. In hypovolemic shock there will be reduction in the circulating volume causing reduction in venous return and cardiac output.

The patient usually: severely pallor, shivering, rigor, hypotensive and sometimes comatose.

Treatment by 1) control of hemorrhage (may require surgery), restoration of circulating volume (fluid and blood products).

CRUSH SYNDROME

*Occur in*

- Large bulk of muscle crushed
- Tourniquet left for TOO long

*What happened?*

1<sup>st</sup> theory = Compression released → acid myohaematin → enter the circulation → kidney → blocks the tubules → Renal failure and death.

*What we can see?*

Limb
- Pulseless
- Red
- Swollen

Renal
- Secretion diminished
- Low output uraemia
- Acidosis

Neurologically
- Drowsy → not treated → DEATH

*How to treat it?*

1<sup>st</sup> rule = Limb crushed severely (>6hrs) --------> AMPUTATION

How the amputation done?
- Above the compression or crushed injury
- Before compression is released

VENOUS THROMBOSIS & PULMONARY EMBOLISM

Commonest Complications of Trauma & Surgery

Most frequently
Calf
Less frequent in proximal of thigh & pelvis

Pulmonary Embolism
From Proximal of thigh & pelvis
Incidence=5% & Fatal = 0.5%

What cause DVT?
The primary cause in surgical
HYPERCOAGULABILITY of the Blood
due to activation of Factor X by Thromboplastin from damaged tissues
Thrombosis occurs→secondary factors are
  Stasis
  Pressure
  Prolonged immobility
  Endothelial damage
  Increase in no & stickiness of platelet

What are the high risk group?
1. Old people
2. Cardiovascular Disease
3. Bedridden patient
4. Patients undergoing hip arthroplasty

What we can see in DVT?
1. Pain the calf or thigh
2. Soft tissue tenderness
3. Sudden slight increase in temperature
4. Sudden increase in pulse rate
5. Homann’s Sign positive

How to diagnose DVT?
  Ascending venography (bilaterally)
  US scanning (detecting prox DVT)
  Radioactive iodine labelled fibrinogen(clot)
  Doppler technique (measure blood flow)

How about pulmonary embolism?
Difficult to diagnose =only minority have symptoms (chest pain, dyspnoe, heamoptysis)
So high risk patients should be examine for pulmonary consolidation
  X-ray
  Pulmonary angiography

How to prevent it?
Prophylactic treatment
Foot elevation
Graduated compression stockings
Exercise
Anticoagulant treatment
  Subcut low dose heparin 5000 units preops & 3/7 postops (but Cl in older patient→bleeding)
  Change to low molecular weight heparin (less likely to cause bleeding)

What is the treatment?
Localized DVT
  Elastic stockings
  Low dose subcut heparin (5000 unit)
More extensive DVT
  Bed rest
  Elastic stockings
  Full anticoagulation
    Heparin IV (10000 units 6 hourly)
    Continue for 5-7/7 with last 2/7 warfarin introduce

How to treat Pulmonary Embolism?
Cardiorespiratory resuscitation
Oxygen
Large dose heparin (15 000 units)
Streptokinase (dissolve clot)
Antibiotics (prevent lung infection)

TETANUS
What is Tetanus?
Tetanus organism live only in dead tissue→exotoxin→blood & lymph to CNS→anterior horn cell
Will develop
  Tonic clonic contraction
    Jaw and face (trismus and risus sardonicus)
    Neck and trunk
    Diaphragm and Intercostal muscle→spasm→ASPHYXIA

What is the prophylaxis?
Active immunization (tetanus toxoid)
Booster doses (immunized patients)
Non Immunized patients
  Wound toilet & antibiotics
  If wound contaminated → antitoxin

*Treatment for Tetanus*
IV antitoxin
Heavy Sedation
Muscle Relaxant drug
Tracheal Intubation
Controlled respiration

**GAS GANGRENE**
By clostridial infection (esp C.welchii)
Anaerobic with low oxygen tension
Produce toxins→ destroy cell wall→ tissue necrosis → Spreading

*The clinical features*
Within 24 hours
  Intense pain
  Swelling
  Brownish discharge
  Pulse rate increased
  Characteristic smell
  Little or no pyrexia
  Gas formation not marked
  Toxaemic→ coma→ DEATH

*How to prevent it?*
Deep penetrating wound should be EXPLORED
ALL dead tissue → completely EXCISED
Doubt about tissue viability→ left it OPEN
No antitoxin

*Treatment for gas gangrene*
The key = EARLY DIAGNOSIS
General measures (fluid, IV antibiotics)
Hyperbaric oxygen (limiting spread)
Decompression of wound
Removal of all dead tissue
Amputation (advanced case)
FAT EMBOLISM

Only minority patients with circulating fat globules will develop POST TRAUMATIC RESPIRATORY DYSFUNCTION

Source of fat emboli=bone marrow

Usually in MULTIPLE CLOSED FRACTURE

But other condition also reported (burns, renal infarction, cardiopulmonary operation)

How can we detect it?

Usually young adults with LL fracture

Early warning signs (72 hrs. of injury)
  Rise in temperature and pulse rate

More pronounced case
  Breathlessness
  Mild mental confusion
  Petechia (chest & conjuntival fold)

Most severe case
  Marked respiratory distress → coma → ARDS

How to treat it?

Mild case
  Monitoring of blood PO2

Signs of hypoxia
  Oxygen

If severe
  Intensive care with sedation and assisted ventilation
  Swan ganz Catheterization (monitor cardiac Fx)
  Fluid balance
  Supportive
    Heparin-thromboembolism
    Steroids-pulmonary oedema
    Aprotinin-prevent aggregation of chylomicrons