MUSCULOSKELETAL BLOCK

LECTURE ONE: HEALING OF BONE FRACTURES

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MOTOR VEHICLE ACCIDENTS (RTA)

Trauma due to motor vehicle accidents is of major clinical and social importance. Damage inflicted is related to several factors, the most important of which are speed of travel, restraint, and protection from impact. There are three main types of injury caused by RTA:

1] **Injuries caused by suddent deceleration**. When a body is accelerated and then suddently brought to a stop, the resulting internal stresses may cause severe damage.

- The aorta may be transected, leading to sefere internal bleeding.
- The brain may sustain internal tearing of white matter tracts.

2] **Injuries caused by direct trauma**. These occur when a body impacts on parts of a vehicle or with road surfaces. There may be:

- Lacerations to face and hands from windshield glass.
- Fracture of sternum and ribs from impact with steering column.
- Fracture of legs from collapse of car frame, or from impact of car on a pedestrian.
- Contusional damage and laceration of liver, spleen and lungs.

- Contusions of brain and fracture of neck from impact damage to head.

3] **Injury secondary to impaired cardiorespiratory function**. Blood loss, unconsciousness and interruption of the airway are common in victims of trauma and lead to secondary damage.

- Brain is extremely vulnerable to hypoxia, developing neuronal death.
- Kidneys may develop tubular necrosis.

Bone is composed of specialized collagen (osteoid), which is mineralized by the deposition of hydroxyapatite

Bone is composed of a collagen-containing extracellular matrix (osteoid) synthesized by osteoblasts, which is mineralized by calcium-containing salts. There are two main patterns of bone deposition.

In normal lamellar bone the osteoid collage is deposited in a mechanically strong, parallel stratified pattern. The collagen is deposited in a direction dependent upon the maximal stresses to which the bones will be exposed, giving the maximum strength for the minimum bone bulk.

In normal woven bone, the osteoblasts deposit osteoid collagen in a haphazard pattern. With its random arrangement of osteoid collagen

fibers, this woven (spongy or trabecular) pattern is far less efficient and much weaker than lamellar bone with a greater tendency to fracture under stress.

Bone is constantly being refashioned by osteoblastic new bone formation and osteoclastic removal of old bone

Osteoclasts are highly specialized cells capable of removing bone; they are multinucleate giant cells derived from the monocyte-macrophage series. Combined activity of osteoblasts and osteoclasts can produce reshaping (remodeling) of bone in order to meet new directional stresses. Unbalanced pathological increase in osteoclastic activity leads to destruction of bone, a feature of some metabolic bone diseases. The control of osteoblast and osteoclast activity and hence the balance between bone formation and destruction in normal remodeling is not completely understood, but many factors play a role like parathyroid home (PTH), vitamin D and calcitonin.

BONE FRACTURE

Bone fractures heal by granulation tissue formation and fibrous repair, followed by new bone formation in the fibrous granulation tissue.

Caused by physical trauma, bone fracture is one of the most common abnormalities of bone. The degree of fracture can very widely, from a simple crack in the cortical bone to a complex multiple fracture with fragmentation and displacement of the bone pieces, associated with severe damage to the surrounding soft tissues and sometimes exposure of the bone fragments to the exterior through a large gaping wound (open "compound" fracture).

Bone fracture healing

When a bone breaks, **haematoma** (blood clot) forms around the broken ends of the bone. Healing occurs through organization of this haematoma by **granulation tissue**. However, this process is modified because the granulation tissue contains proliferating cells derived from the periosteum and endosteum which differentiate into chondroblasts and osteoblasts that lay down new cartilage and immature (woven) bone. The result is hard tissue called **callus** surrounding and joining the broken ends of the bone. Bony union occurs when new bone produced in the callus links the bone fragments together. The cartilage and woven bone are gradually replaced by **lamellar bone** (the type found in mature bone). This new bone can be **remodeled** so that the anatomy can return to close to normal. However, the fracture bone fragments must be aligned and

placed together (apposed) for optimum healing to occur, otherwise the callus may not adequately bridge the gap betweemn them, resulting in permanent deformity.

Delayed or abnormal healing of fractures can lead to non-union in which the fractured bone ends do not join by bone

For proper fracture healing to take place, it is essential that the fractured bone ends be in close apposition, that the fracture is immobilized and that the patient's healing capacity is adequate.

Among the factors that detrimentally affect healing of bone is **poor blood supply** to the affected area. This is particularly important in certain areas such as the scaphoid bone in the wrist and the neck of the femur, both of which can be associated with **avascular necrosis** of fracture fragments. **Poor general nutritional status** (particularly wehre there is protein malnutrition or vitamin deficiencies) and **poor apposition of the fractured bone ends** (e.g. whide displacement, entrapped viable soft tissue between the bone ends or excessive mobility) can also contribute to delayed or abnormal healing as can the presence of **foreign bodies** or large quantitis of necrosis bone, the presence of infection (particularly in open fractures) and corticosteroid therapy.

The aim of treatment in fractures is to ensure closure apposition of the bone ends, followed by firm immbolization so that the fractured ends cannot move during the formation of granulation tissue and callus.

When fractured bone ends are not closedly apposed, or if any of the above local complicating factors are present, ossification of the callus does not occur and the two bone ends are joined by **fibrous tissue** (fibrous ankylosis) which is unstable.

Fracture may occur with minimal trauma if the underlying bone is abnormal.

For normal bone to fracture, the causative trauma usually has to be severe. In contrast, trivial or weak trauma may cause fracture when the underlying bone is abnormal (**pathological fracture**).

Among the common abnormalities predisposing to pathological fracture are **osteoporosis** (particularly in the femur and verterbral column in the elderly), **osteomalacia** (the fractures are often small microfractures without displacement), **Page't s disease of bone** (the pagetic bone being structurally weak despite the increase in bulk), and **primary or metastatic tumor**. Metastatic carcinoma in bone is an important cause of pathological fracture. Bone metastases from carcinoma of the breast, bronchus, thyroid and kidney produce bone destruction (osteolytic

metastases), which predisposes to fracture. Primary tumors of bone or bone marrow, such as giant-cell tumor of bone and melanoma, also play a role, as do some non-neoplastic bone lesions such as bone cyst.

Congenital bone disorders can also predispose to pathological fracture. The most important disorder is osteogenesis imperfecta, in which multiple fractures occur, often in utero and in infancy with minimal trauma.