



Pathology

Doctor 2017 | Medicine | JU | MSS

Number >>

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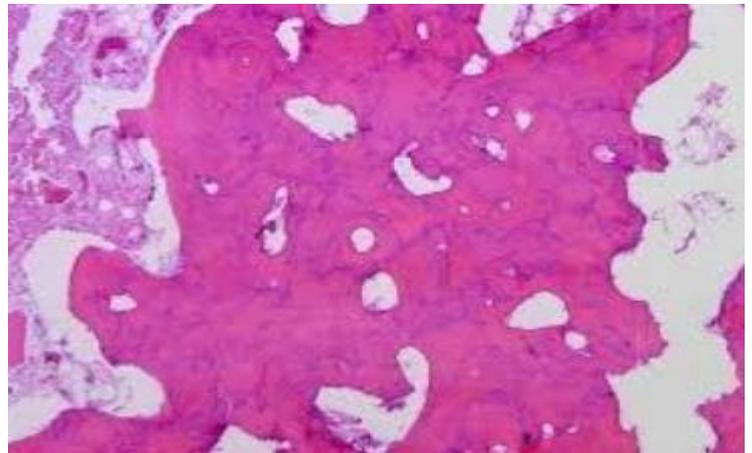
1st system - MSS



Paget Disease: Osteitis Deformans

- It is badly formed bone structure; can be divided into 3 different phases:
 - Lytic: bone is being resorbed at a high rate.
 - Mixed: bone resorption and bone formation.
 - Sclerotic: bone is being built at a high rate.
- Most important thing to know; unknown cause
 - There are geographic variations.
 - There is a genetic predisposition.
 - There is an acquired form (environmental).
 - 50% of familial and 10% have mutations of the SQSTM1 gene
 - Causes increase in RANK expression, and decrease in OPG expression.
 - Bottom line; multifactorial cause.
- **Clinical features:**

If a patient presents with clinical features of Paget, and the histology or radiograph comes up like this, you *must* be able to make a diagnosis. This bone is *not normal*. As you can see almost all of it is woven, not lamellar, indicating a problem with the homeostasis or balance of osteoclasts and osteoblasts.



Another picture that may show up on the exam is this. As you can see there are many areas that are radio dense and radiolucent, which hints at Paget.

Look and compare the two pubic rami. The one in the picture is *much* less white than the opposite side.



- Here are some clues that point you in the direction of Paget disease:
 - Paget is polyostotic in 85% of cases; more than 1 bone affected (rarely 1 bone); and 15% monostotic.
 - Axial skeleton is more likely to be affected (vertebra, hip, prox. Femur).
 - Most are mild and asymptomatic; some pain which is caused by micro fractures.
 - Multiple compression fractures (especially near the vertebral bodies) can pinch a nerve and cause pain.
 - Severe forms can affect the skull and cause *Leontiasis ossea* or *platybasia*.
 - Other symptoms or risks are 2ry osteoarthritis, fractures, and osteosarcoma (only effects 1% of patients).
- **Diagnosis:**
 - X-ray.
 - **High serum alkaline phosphatase**, normal Ca and PO₄.

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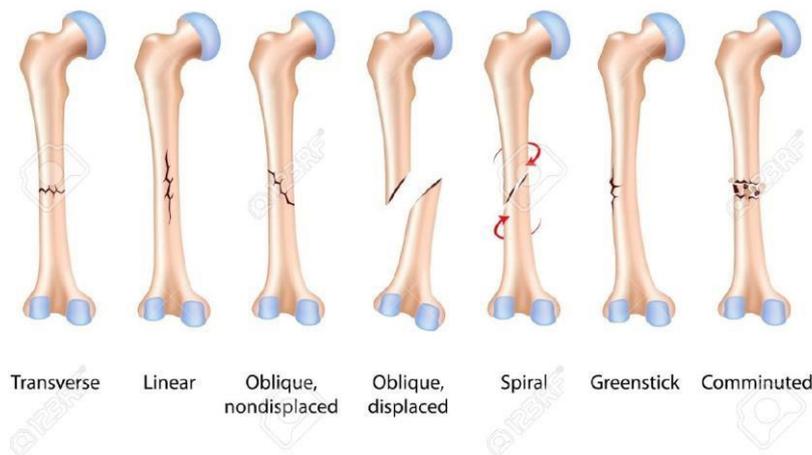
Fractures: #'s

Defined as: loss of bone integrity due to mechanical integrity and/or diminished bone strength (due to disease). ○ Most common pathology of bone.

Classification of fractures:

- Simple: skin is intact.
- Compound: communicates with overlying skin (bone is coming out).
- Displaced: ends are not aligned.
- Stress: repetitive pressure causes fracture (more common in axial skeleton).
- Greenstick: soft bone fracture (bone bends, not breaks; in kids).
- Pathologic: fracture caused by abnormal bone (tumors, Paget, osteoporosis).

Types of Bone Fractures



- Spiral: often seen in kids, mother lifts her child by the arm, causing him to spin (in this case: spiral and greenstick).
- Comminuted: sever trauma, multiple pieces of bone broken off.

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Bone Healing:

Happens in stages, and is affected by several factors regarding the fracture, and the health of the patient

The bone healing process continues as follows in the case of a simple fracture in a healthy patient:

- First 24 hours: organized hematoma around fracture site.
- First 2 weeks: PDGF, FGF, and FGF- β are secreted to begin preparing the osteoblasts and osteoclasts for major remodeling.
- Weeks 2-3: bony callous forms (woven bone), which is the scaffolding of the new bone about to be mineralized.
- 3 weeks+: lamellar bone is reformed.

The scenario above is the best case, and as always some factors can affect the amount of time spent healing, as well as how perfect the healing will be.

- Factors impacting proper healing:
 - Compound/comminuted fractures: more trauma means more time spent healing, and the healing will never return the bone to its original strength.
 - Age: younger, healthier patients heal more quickly.

- Steroid/AI drug usage: remember, healing is an acute inflammatory reaction: drugs that affect inflammatory response will delay healing.
- Infection: more likely to happen in compound fractures, needs a surgery.
- Inadequate immobilization: delayed union = healing has started without the bones being in close proximity, non-union = bone has healed without touching, forming a gap in the bone.
- Pseudoarthrosis (false joint): a new joint has formed after a fracture has healed without a union.
- Malnutrition: delays healing process.

31:18

Osteonecrosis: Avascular Necrosis

Infarction of the bone or bone marrow (ischemic necrosis)

Caused by:

- Vascular injury/trauma, and vasculitis (mostly of large vessels)
- Steroids: underlying mechanism is unknown
- Alcoholism: higher risk for avascular necrosis
- Systemic diseases: such as sickle cell
- Radiation



The most common bone affected by avascular necrosis is the **femoral head**. The classic gross view of it is the triangular shape of the necrotic tissue (remember coagulative necrosis?).

There are multiple mechanisms that cause the ischemia which leads to necrosis: mechanical disruption (fracture), thrombotic occlusion, extravascular compression.

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Acute Osteomyelitis:

Inflammation of bone and bone marrow. The cause of inflammation is **almost always infectious**. If this type of infection produces puss, it is called **pyogenic osteomyelitis**. Osteomyelitis can become a complication of a systemic infection, but it frequently manifests as a primary, solitary focus of disease. **Any organism can cause osteomyelitis**. The most common culprits are *S. aureus* (80-90%!!!), *E. coli*, *Pseudomonas* and *Klebsiella* are more common in UTIs and IV drug abuse.

Acute Pyogenic Osteomyelitis:

Is caused by 3 different mechanisms ranked by their incidence:

1. Hematogenous spread (in children).
2. Extension from contiguous site (eg. Diabetic foot or compound #).
3. Direct implantation from surgery (iatrogenic).

In neonates, *H. influenza* and group B strep. are the more common causes of pyogenic osteomyelitis.

Patients with sickle cell disease are more susceptible to salmonella. When a patient comes who has sickle cell, and you think it may be osteomyelitis, cover *S. aureus* and salmonella (G- bacilli).

In half the cases no organisms are isolated, which means you continue with empiric treatment. This is due to an incomplete course or improper use of antibiotics

Most common in long bones. In adults the epiphysis and metaphysis can be affected, but in children, its either the epiphysis or metaphysis, but why? In children, if you remember there is an epiphyseal growth plate between the epiphysis and metaphysis, which is made of cartilage. Cartilage, as well know is avascular, which disrupts the main mechanism of osteomyelitic spread, through the blood.

Good Luck