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Pathology #2

مجموعة التفريغ السريع

MSS

Subject: *Bone Tumors*

Done by: *See the last page*

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Date: *Monday, September 30, 2013*

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Inflammatory Conditions

♣♣Osteomyelitis:

- Inflammation of the bone and the marrow cavity.
- Usually blood -borne infection.
- Most serious types are:

1. Pyogenic osteomyelitis (Bacterial osteomyelitis)
2. Tuberculous osteomyelitis (more chronic)
3. Syphilitic osteomyelitis.
   - it is almost extinct.
   - can be treated very easily.

1) Pyogenic Osteomyelitis:

✓ **Caused by** pyogenic bacteria:

- Staph. Aureus, E. coli, Salmonella.

- Mixed infection. (It is more frequent in people with sickle cell anemia)

✓ **Sources:**

1- Hematogenous (The most common)

2- Direct extension

If there was an adjacent inflamed focus, it will extend to the bone.

3- Direct trauma. (Fractures)

Fractures are of two types:

a- Open

The skin is open, and the bone protrudes to the outside. In this case the bone can easily get infected.

b- Closed

4- Iatrogenic (resulting from the activity of doctors).

For example:

If a patient came with a broken bone and it was treated surgically by implanting metals, nails, plates or artificial joints, this will make the bones prone to infection and osteomyelitis.

- ✓ **Mainly affects metaphysis** of long bones, but it can occur elsewhere in the body. (If someone had an ear infection or a paranasal infection it may extend to the bones of the skull)
- ✓ **In Infants**: The infection of the bone extends to the Epiphyseal plate and get to the joint, leading to the destruction of the joint. (this is not seen adults who stopped growing)
- ✓ **Clinical picture and diagnosis:**

- Acute: Fever, bone pain, leukocytosis, increased Basal Metabolic Rate.
- Chronic: Low grade fever, pain, abscess formation, discharging sinuses.

Discharging sinuses:

A tract that drains from the bone through a pathway, and it opens to the outside to discharge the abscess.

- **Diagnosis :**
  - Xray : Destructive lytic lesion with new bone formation
  - Blood & sinus discharge culture
- **Treatment :**

Appropriate antibiotic & surgical debridement.

Debridement: is the removal of dead tissue surrounding the infected part (specially dead bone).

Pathology:

- Acute, subacute, chronic stages.

The first stage :

- Neutrophil infiltration, early bone necrosis (because the inflammation leads to thrombosis in the vessels of the infected region → **ischemic changes → bone necrosis → sequestrum formation**)

A sequestrum is a piece of dead bone that has become separated during the process of necrosis.

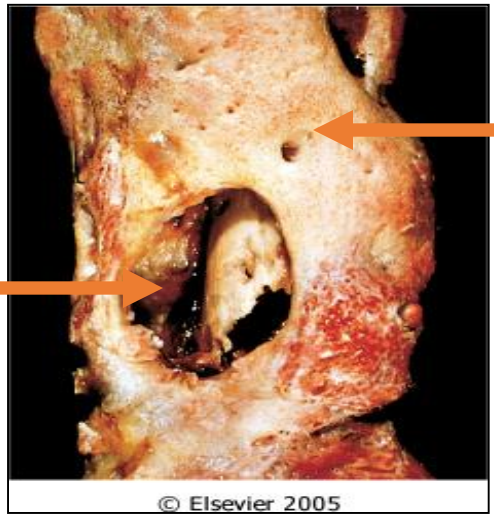
- The inflammation in the medullary cavity will Spread to the periosteum forming the subperiosteal abscess which can rupture into the soft tissue → draining sinus

The chronic stage:

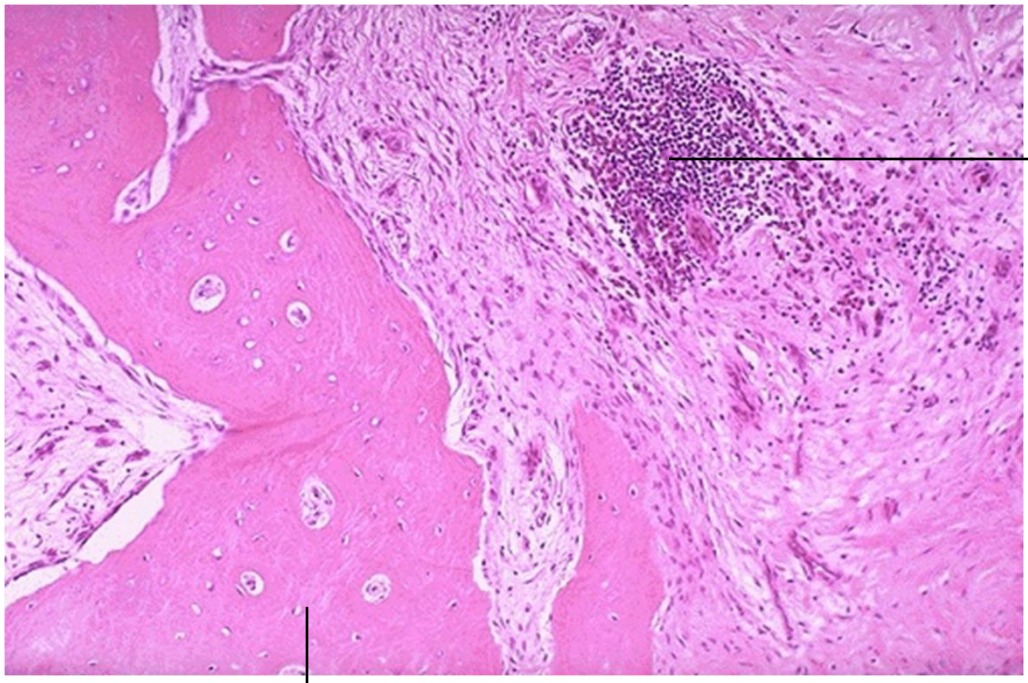
- The neutrophils will decrease in number and we will start to see plasma cells and lymphocytes.
- At this stage the bone marrow is more fibrotic with few amounts of proper marrow elements and a lot of plasma cells.
- Plasma cells are a good indication of chronic osteomyelitis.
- During the chronic phase, the reactive new bone formation will be initiated in order to repair the damaged bone. The repair is not done properly ,this leads to the formation of the involucrum.

Involucrum is a layer of new bone growth outside existing bone seen in pyogenic osteomyelitis. It results from the stripping off of the periosteum by the accumulation of pus within the bone, and new bone growing from the periosteum.

- (Brodie's Abscess): a chronic abscees of bone that may be filled with pus or sterile fluid by time.



Osteomyelitis



Collection of inflammatory cells

The bone trabeculae : it is very thick because it was irregularly deposited in response to the inflammation.

- This is NOT Pajet's disease, because the irregular broad trabeculae with disorganized cement lines in a mosaic pattern is not seen here.

The complication of osteomylitis :

☼ **P**athological fracture ..😐
☼ **S**eptic arthritis ..
☼ **B**acterimia & septicemia ..
* **E**ndocarditis >> specially if you have damaged valves already (more likely to get infection) .
☼ **A**myloidosis >> (there are different types of amyloidosis one is ***p***rimary & one is ***s***econdary to other conditions & any long standing chronic inflammatory process may end up by deposition by **amyloidosis**) ..
☼ Chronic skin sinus >> sinus doesn't heal because there is continuing inflammation that you may have at that sinus >>
☼ **S**quamous cell carcinoma of the skin, bcz here we have nonhealing so continuing regeneration .. ☺

2) Tuberculous osteomyelitis :

❄ ***M*ay follow pulmonary or GIT tuberculosis & some times military but usually secondary ..**
❄ ***M*ost common in immunosuppressed ..**
❄ ***H*ematogenous or direct from nearby focus …**
❄ ***M*ay be solitary or multicentric …**
❄ ***U***sually **long bones affected & vertebrae if you have tb CNS or vise versa (affected vertebrae then you will have CNS tb ..**
❄ ***P*athology is a caseating granulomatous reaction destroying the bone & if it's in the vertebrae it'll collapse .. !**

Pott's disease :

♦ **Tuberculous osteomyelitis affecting the vertebrae → KIFOSIS,SCOLIOSIS**

>> (Hunchback) ..

♦ **May drain into soft tissue & adjacent psoas muscle → PSOAS ABSCESS**

♦ **Therapy: Antituberculous therapy, but difficult to treat ..** ☹

♣♣ *joints :*

1) Inflammatory Disorders :

1- Infectious arthritis :

Septic- Acute :Staph.,Strep.,Gonococci…..etc

Any joint, inflammation with suppuration ..

Heals with antibiotics, or fibrosis & calcification..

Tuberculous: Complication of pulmonary or miliary Granulomatous inflammation with caseation ..

Destruction of cartilage & deformity..

More in children & spine .. ☹

2- Rheumatoid Arthritis :

☞ **Female>Male, usually at middle age > (can occur in younger & not usually starts at old age) , Etiology ? unknown ..!**

☞ Autoimmune process →T cell & macrophage reactions in genetically predisposed patient → producing TNF → tissue damage & synovial proliferation..

☞ Systemic disease with involvement of small joints of hands & feet, mainly proximal interphalyngeal joints , but later any joint can be affected ..

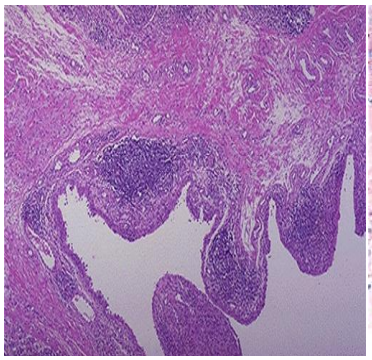
☞ Joints usually starts at Stiff swollen red joints specially in the morning , later it will lead to deformity, & fusion of joints (ANKYLOSIS) .

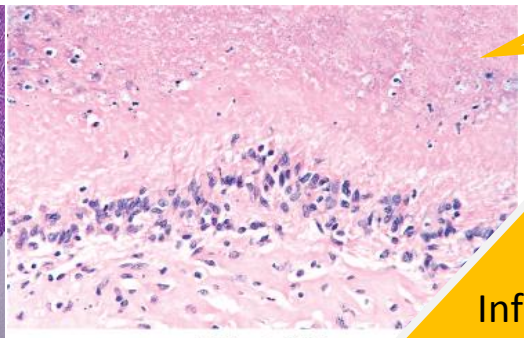
Pathology :

💣 Acute & chronic inflammatory cells, lymphoid follicles in hypertrophied synovium, extends into joint ..

💣 Later, synovium + inflammatory cells → 'PANNUS'(which contain a granulatous tissue) → fibrosis → Joint destruction..

💣 There you will find "Rheumatoid nodules' " in joints & soft tissue ..





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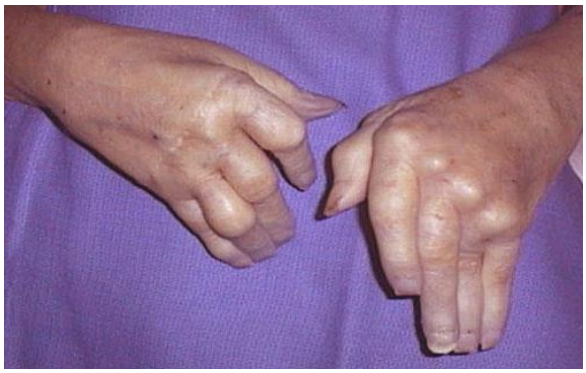
Rheumatoid nodule

Inflammatory cells and fibro blasts , these elongated cells are fibroblsts

💣 Serological tests : Hypergammaglbulinemia & Rheumatoid Factor Positive (IgM or IgG auto AB that bind to Fc portion of IgG → immune complexes deposited throughout the body creating the damage basically in the soft tissue of the body " *the collagen* " . ☺



This is the rheumatoid nodule , and this is the result , you can see the joint , the proximal part of the joint , very thick , swollen , and has led to fusion here , and very short index finger , very deformed , we do get it here but more in the west.

2) Degenerative & Metabolic Diseases:

1- Osteoarthritis :

*not osteomyelitis

- is an extremely common disease
- part of it is an aging process
- occurs in both sexes
- **primary** degenerative disease of cartilage, usually occurs >65 years.
- **Secondary** in younger patients it can occur for example in somebody who falls and has damaged joints , this may lead to osteoarthritis later on:

* any trauma , obesity because of the weight bearing , and any bone deformity more likely to be involved .

- genetic causes that affect basically the hips , knees , hands , intervertebral joints with swelling and decrease mobility

Pathology :

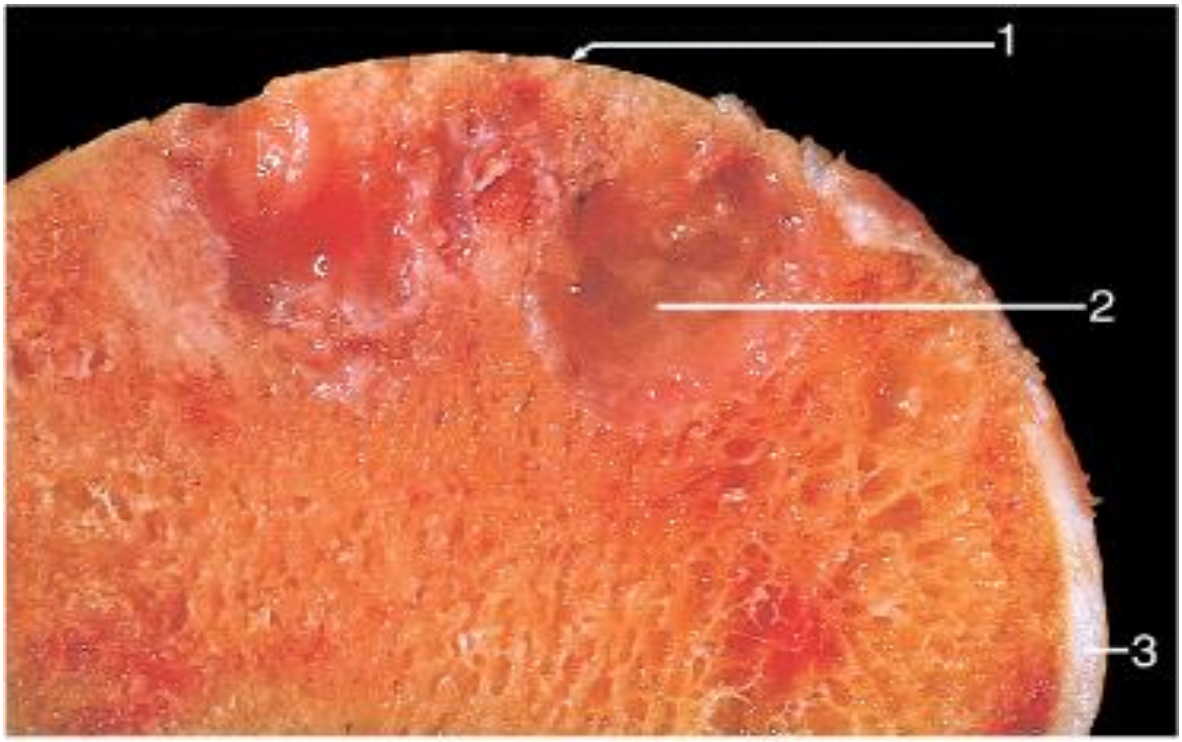
* Basically it's a disease of chondrocytes , they proliferate and "drink water" (water gets into cells so they die) , this will lead to dilution and decrease in proteoglycans (part of structure of cartilage) so there will be cracking (يتفطر) in cartilage.

* Some of the cartilage will be lost which will expose the

underlying bone , this process is called **Bone eburnation** "to expose a part of the bone due to destruction of overlying cartilage .

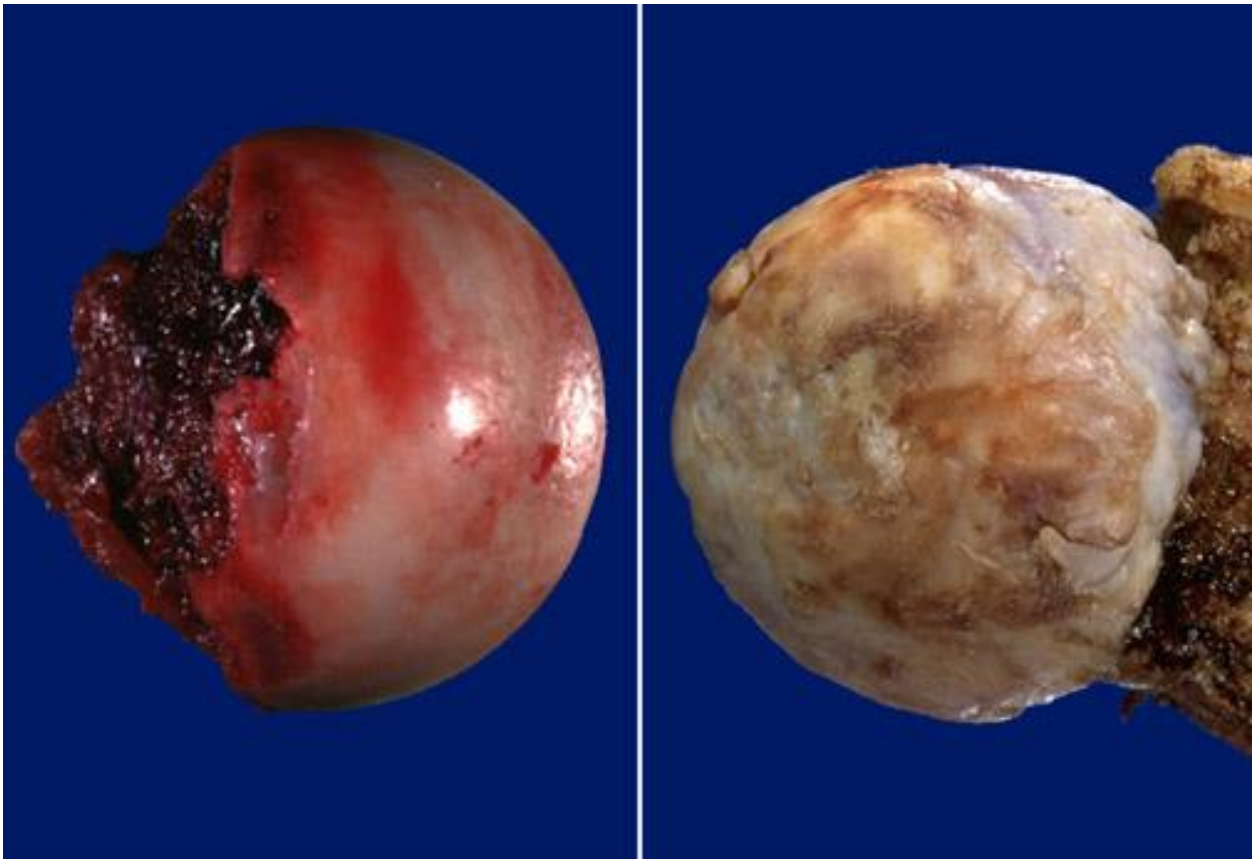
* The cartilage itself because it's cracked it will show like a minor fracture , what is called loose cartilage bodies "joint mice" ; broken peaces of cartilage in joint space , and the joint will accumulate fluid

* The gaps made by these cracks and fractures are filled with synovium grows into it , and forms a small cyst , and surrounding this there will be bone production ; boney outgrowths which are called **osteophytes** , this is specially seen in the spine because if you take an X-ray you will see they are protruding and may produce pressure on the spinal nerves and backache . and **Occasional fibrous Pannus that contain more granulation tissue , this is not an inflammatory process** but a degenerative process .



Severe osteoarthritis with small islands of residual articular cartilage next to exposed subchondral bone. *1*, Eburnated articular surface. *2*, Subchondral cyst. *3*, Residual articular cartilage.

In the picture above:
"this is the head of the femur , this is the exposed part , this is a cyst (2) , and this here is a residual normal cartilage (3) , compare this to this ! it is gone in the cyst.



And look at the head of this femur , the one to the right is smooth and normal , and the other one is very rough . this is the main indication for patients who have hip replacement "

Clinical:

- Morning stiffness
- Pain & swelling in joints
- Progressive deformity in joints, but no ankylosis .
- Spinal root compression due to involvement of vertebra by osteophytes

Treatment :

Analgesics, Joint replacement.

2) Gout (النقرس ، داء الملوك) :

Is the Accumulation of Uric Acid in soft tissue & joints leading to **acute arthritis** this will change into chronic arthritis , and crystals will be deposited in the kidney , and you will get renal complication

*there are two types of Gout

1- primary

Which is what people know about , **high uric acid level in middle aged men** more than women .

2-secondary

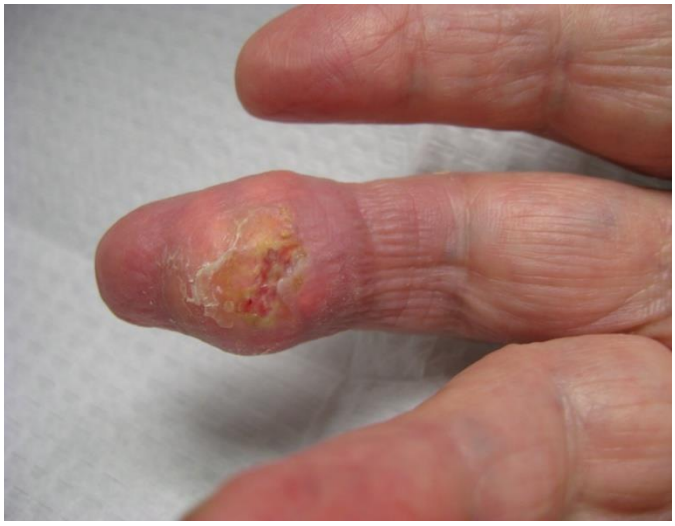
- **High nucleic acid turnover :** in people with leukemia when you give them therapy there will be break down of many cells that will lead to a high uric acid .
- **Renal failure :** because of the inability to expel it
- **Inborn error of metabolism** : leads to increase in uric acid.

Pathology:

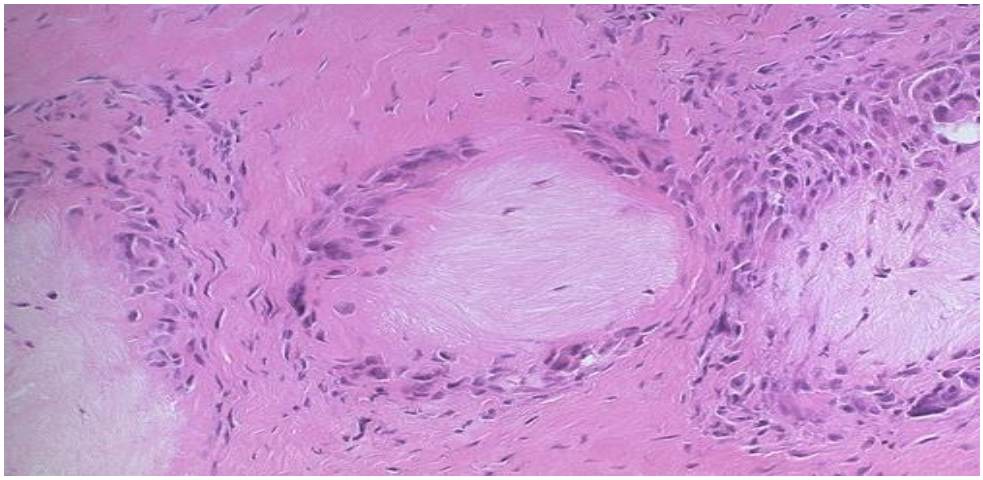
- Repeated attacks of ACUTE ARTHRITIS in small joints which becomes red and hot, with deposition of MONOSODIUM URATE crystals in joint tissue and soft tissue adjacent to it which leads to severe and inflammation .
- Repeated attacks leads to " Tophi"
  **Tophi are foci of urate crystals surrounded by inflammation with foreign body giant cell reaction due to the presence of the crystals . The crystals Can be seen on aspiration if you take the fluid and examine it under polarized light , under the microscope .
- Chronic Tophaceous arthritis will lead to joint destruction & deformity.

- Gouty nephropathy : with production of calculi" which is stoned in the kidney " and this will lead to inflammation and what we call ,

pyelonephritis ,,pelvis ,, cortex of the kidney so altimately you get damage of the kidney .



It's very common in the lobe of the ear and in the toes also like in the photo above . As we see it 's ulcerated later on . It looks like granuloma but the center is not degenerated collagen like in rheumatoid lesions , it's not caseating lesion .here in the center there are crystals that can be seen under polarized light , under the microscope .



3) Pseudogout (chondrocalcinosis)

It's very similar to gout but it's less severe also the deposit differ " in gout there is uric acid accumulation" .

- **Chondro calcinosis**
- Deposition of calcium pyrophosphate ** with similar pathology to gout
- Over the age of 50 "gout also " , often less severe.
- Acute, subacute, or chronic arthritis of knees, wrists, elbows, shoulders, and ankles.
- The Cause is unkown
- Some cases are inherited

* does not lead to ,as much joint deformity as, the other type " gout " .

3) Tumor –Like Lesions in Joints :

1) **Ganglion :**

It has nothing to do with the nervous tissue , it involves degeneration in the tendon sheath and it will be incised mostly .

"Small cyst near tendon or joint"

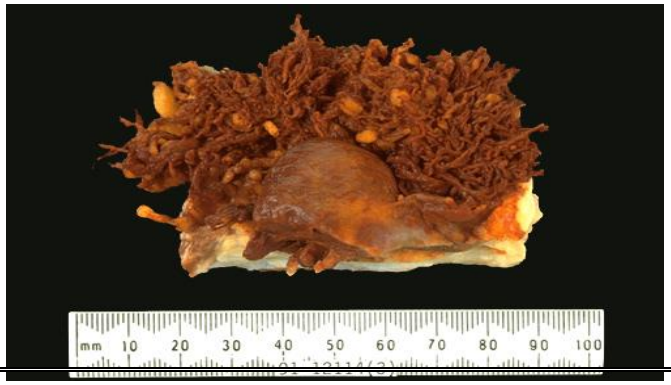
* you can actually see it as a small ball around the wrist , sometimes it moves , sometimes it disappears and it's symptomless .

** and if it's excised surgically , there is no lining .

2) **Synovial cyst** :

It's related to bigger joints .

There would be degeneration and Herniation of synovium" so there will have lining " through joint capsule at popliteal fossa , behind the knee ,and again can be removed surgically . it's also called **(Baker's cyst).**



Pigmented Villo-nodular Tenosynovitis
& Giant Cell Tumor of Tendon Sheath

It's not inflammation at all although it ends with "itis " , it's just a misnomer .

Pigmented Villo-nodular Tenosynovitis actually it's mainly around the knee joint but we can find it in another joints .

Acually when it's in the fingers , we call it **Giant Cell Tumor of Tendon Sheath** or tynosynovitis

- PVNS mainly around knee, benign but may be large & aggressivebecause it sometime reoccur . also we can sometime find mitotic figures BUT it's not malignant nor does it change to malignant
- GCT smaller around the tendon speciallyin the fingers , painless nodule & around tendon
- Cause ? Neoplasm ?
  *You may think it's malignancy but it’s probably reactive condition and it occurs ore in active people like football player … after they have knee injury and it progress.
- Both have similar morphology :

  spindle cells, histiocytes , pigment ….

  *here histiocytes takes pigment "because of hemorrhage , there will be hemosedrine there "

  So they are hemosedrine laden macrophages .

-villinodular due to the villi : result because of the proliferated synovial tissue , it becomes villus

- brown due to old hemorrhage and histiocytes are there to take up the hemosedrine .

- so it contains ordinary giant cells .

♣♣Tumors of Bone & Soft Tissue

Firstly

INTEGRATION of Main Relevant Points

- Good History

Remember that's very important to take good history and X-rays , so you can almost diagnose the nature of the tumor

- Age of patient
- Site of lesion
- Duration of lesion whether its new or related to trauma
- Presence or absence of PAIN in the lesion*

the Presenceof pain is more in expanded lesion than in a quiescent one. That presence for years and painless is not as suspension as a lumb that swelling which increasing in size and increasing in pain , all these are bad indications .

- PLAIN X-ray of lesion*
- CT of lesion*

Helps to know the nature of the tumor whether it's cystic , calcified …..

- MRI of lesion*

Helps to know the extend of the lesion . for example if there is malignant in he elbow , we need to know if it extends to the soft tissue nearby or not , because this affect the prognosis and the stage .

- Combined clinico-radiological-pathologic approach

You should be in clocse contact with the surgeon with Combined clinico-radiological-pathologic approach before you can properly diagnose a tumor .

*** the dr said ' I don’t want you to memories this table in slide#3 ' and she mentions some examples from BUT she added :

Remember there are many conditions in bone specially the inflammatory which produce a bone.

And osteosarcoma also producing bone . so sometime a chronic osteomyelitis may be diagnosed as osteosarcoma . so it’s very important to realize that not every new bone formation is neoplastic .It is very difficult to diagnose malignancy in fracture because fracture may produce very irregular bone and it's continuing and of course it can occur because the malignant tumor can fracture so you have repairing and neoplasm together .

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