What's new in Osteoarthritis in 2019?

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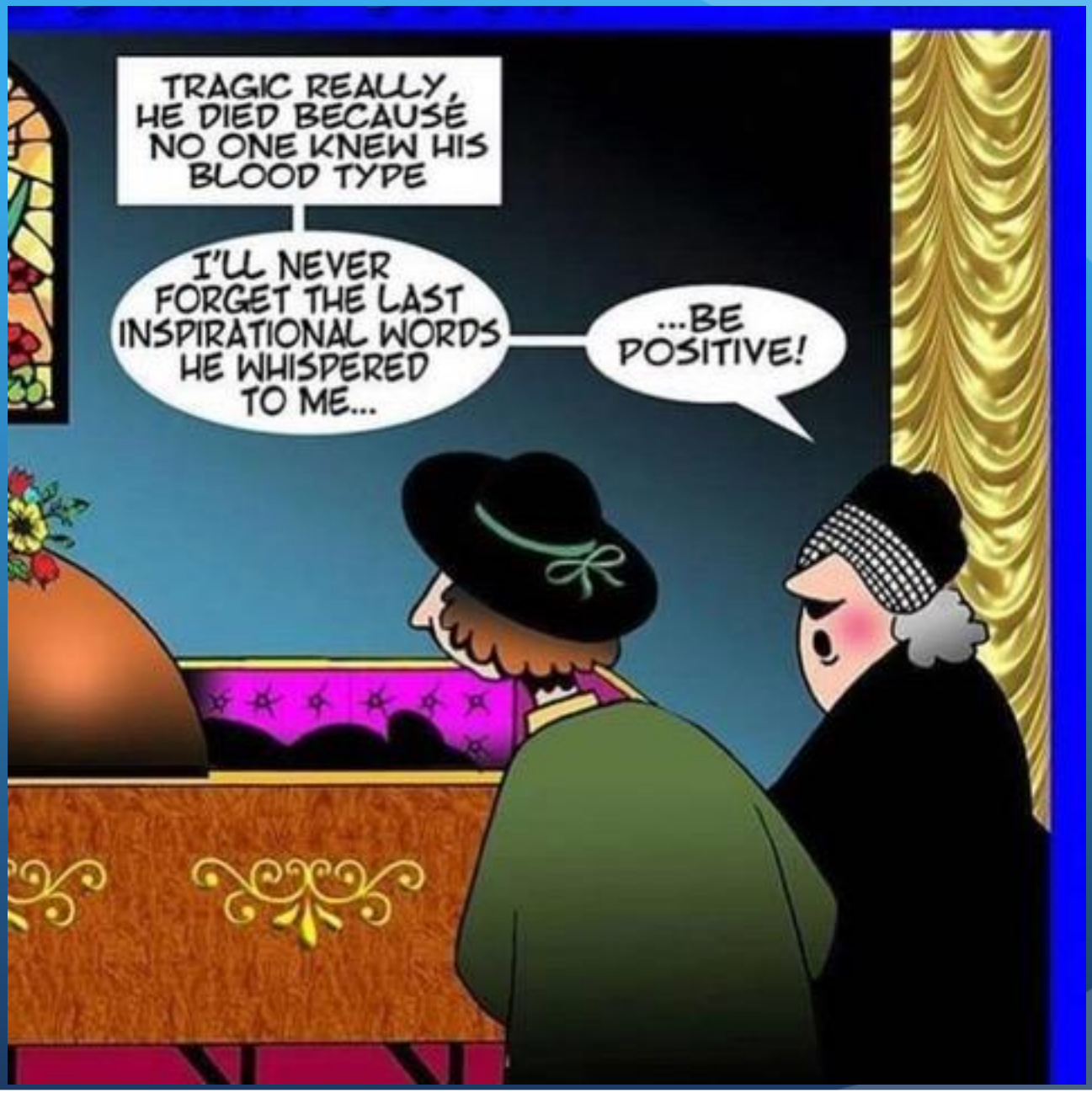
Osteoarthritis

- Evidence exists in the earliest hominids, and it existed in the dinosaurs
- It is the most common form of arthritis. It may account for 30% of physician visits (US data).
- 80% of people have OA by age 80.
- There is only a modest correlation between the severity of anatomic changes/radiological features and symptoms, and these are variable.
- Primary: idiopathic, often genetic factors
- Secondary: due to known disorders or injury

Genetic factors:

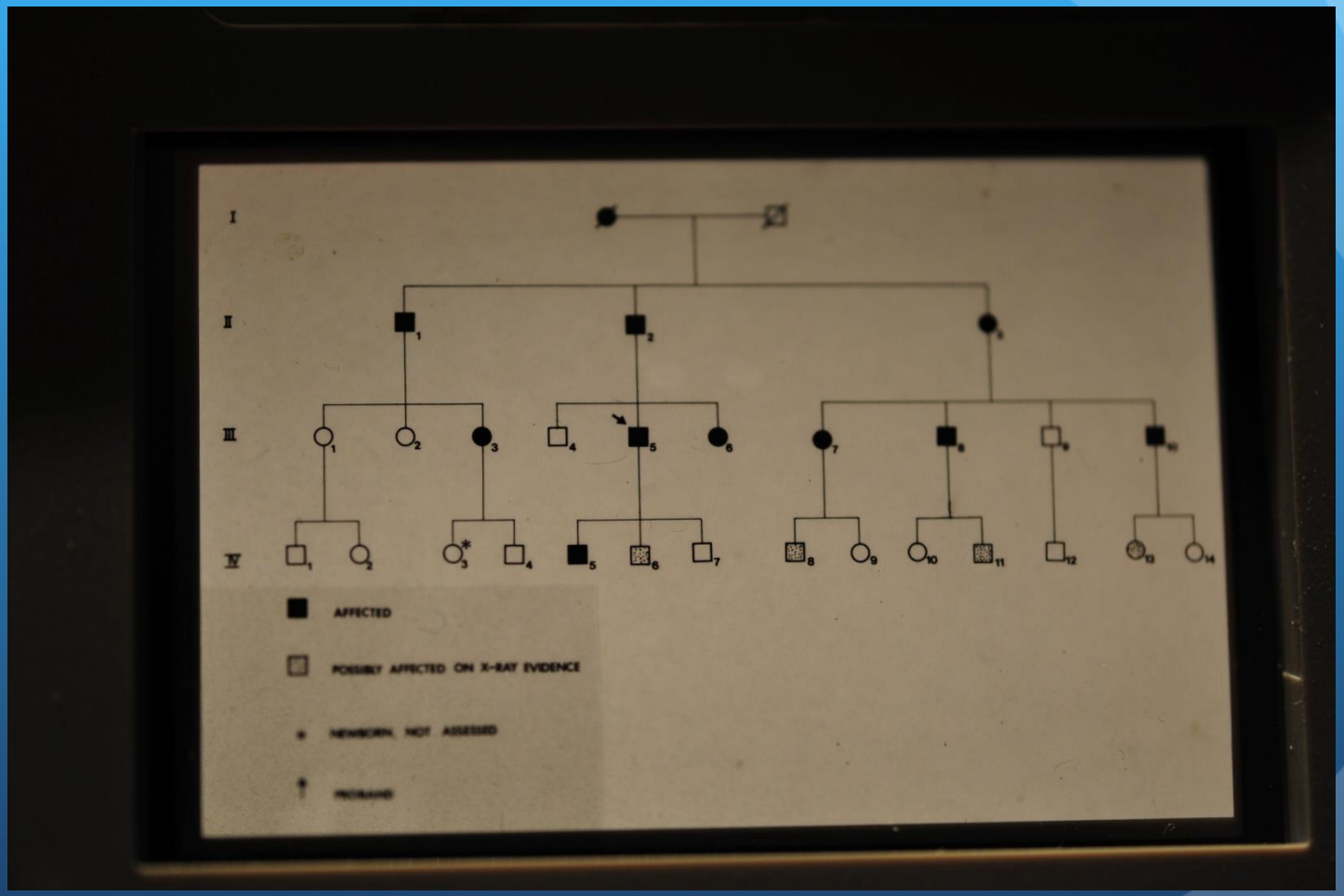
- Originally thought to be the primary tissue failure (hyaline cartilage) and supported by evidence from studies on families with precocious OA caused by SED and other chondro-dysplastic disorders:
  - SED: COL2α1 gene defect on Chromosome 12.
  - SEDTarda: due to a different mutation on Chr Xp22.
- Animal studies mainly done on dogs (cruciate deficient), but also rabbits and guinea pigs.
- More recently, gene manipulation in mice targeting the COL2α gene has been successful


TRAGIC REALLY, HE DIED BECAUSE NO ONE KNEW HIS BLOOD TYPE
I'LL NEVER FORGET THE LAST INSPIRATIONAL WORDS HE WHISPERED TO ME...
...BE POSITIVE!

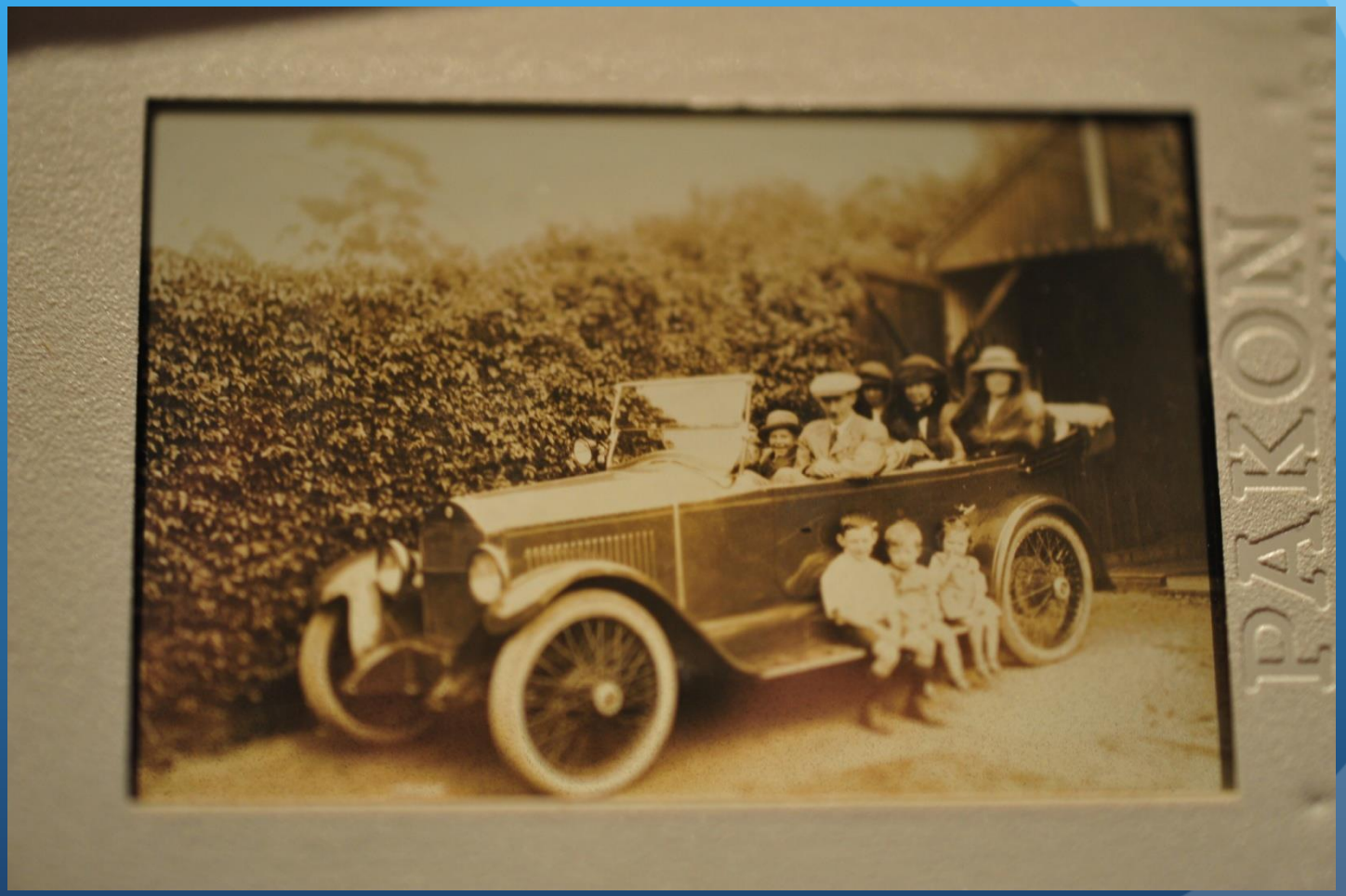
Primary (nodal) osteoarthritis

- Genetically linked to the COL2a1 gene, but polygenic and cartilage is made up of 5 types of collagen (II,XI,IX, lesser amounts of X and VI). When hyaline cartilage is lost in adulthood, some repair occurs with fibrocartilage, which has inferior wear.
- Involves small and large joints: in the hands it affects the distal and proximal interphalangeal joints, and the base of the thumbs. In the feet it is most often the big toe , 1st tarso-metatarsal joint affected.
- There will usually be other affected family members
- Pain and disability are very variable, with poor correlation with Xrays.
- Knees and hips are the commonest large joints affected
- Some genetic disorders lead to precocious OA (SED,SEDT, MED,)

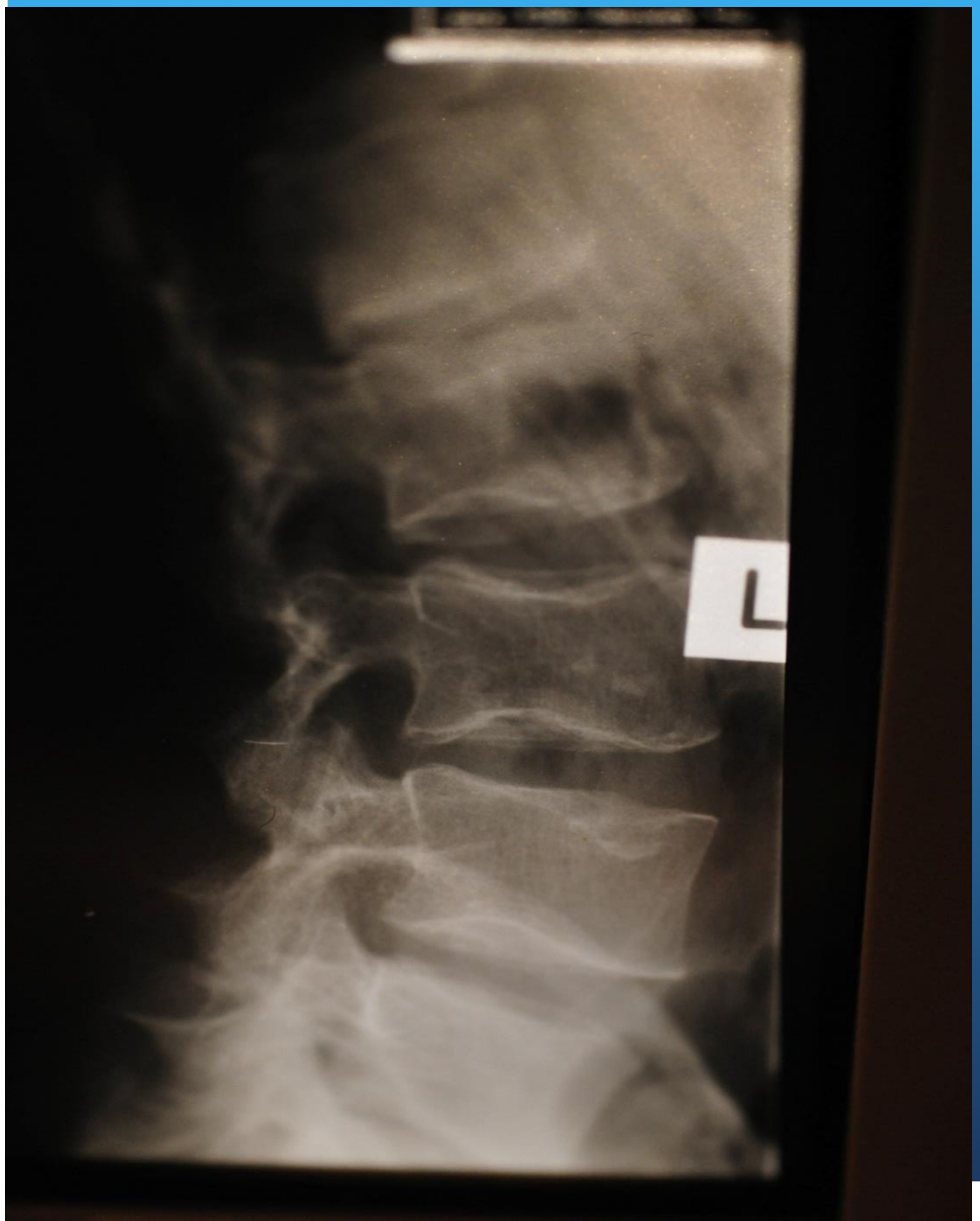
Family tree of 34yo male with SED and precocious OA



The Family 1st two generations!

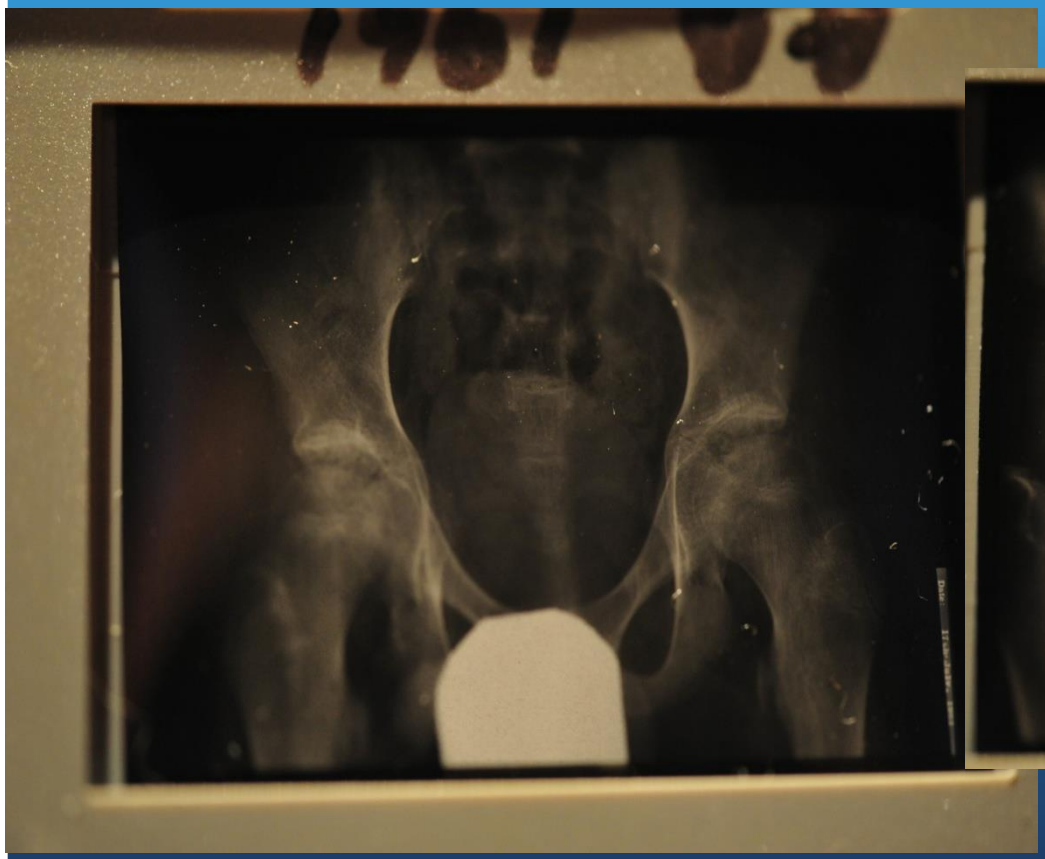


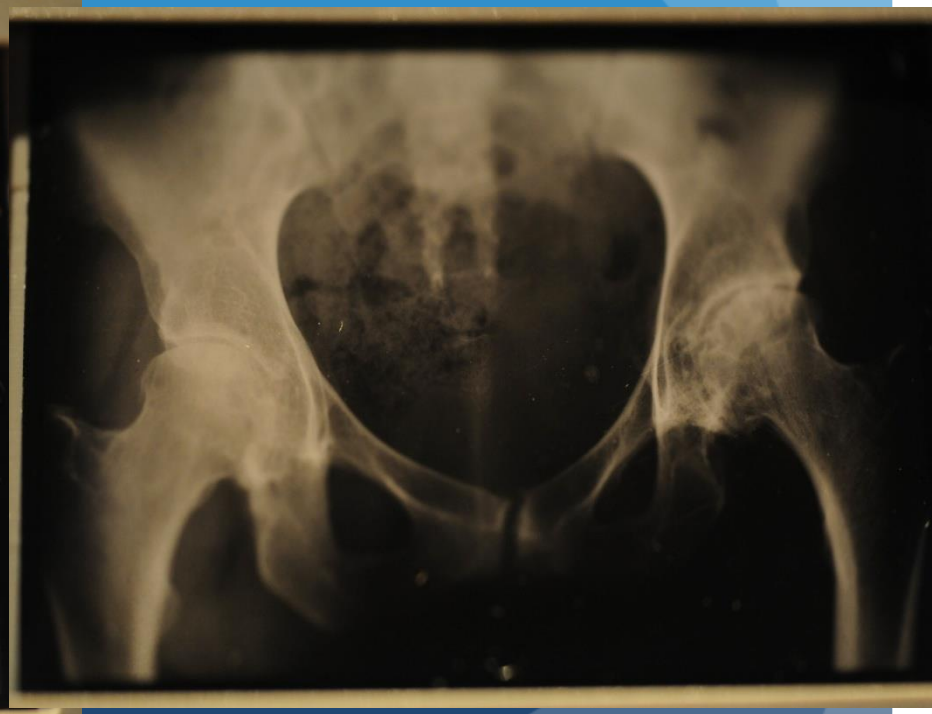
Platyspondyly in thoracic and lumbar spine vertebrae



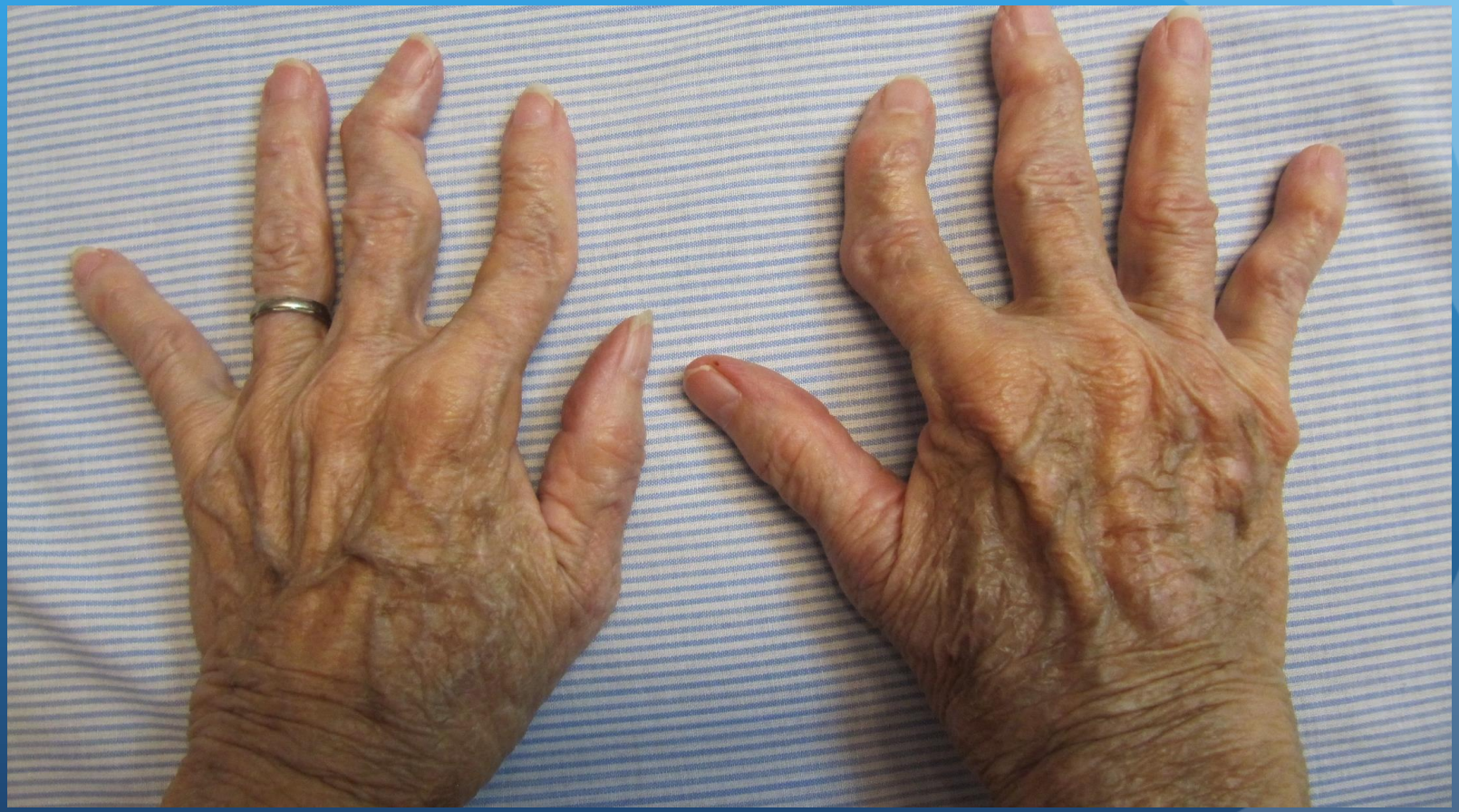
This is a hallmark of the epiphyseal dysplasias

Proband was a 34yr male, said to have Perthes as a 10 yr old, Hips on R at 24y



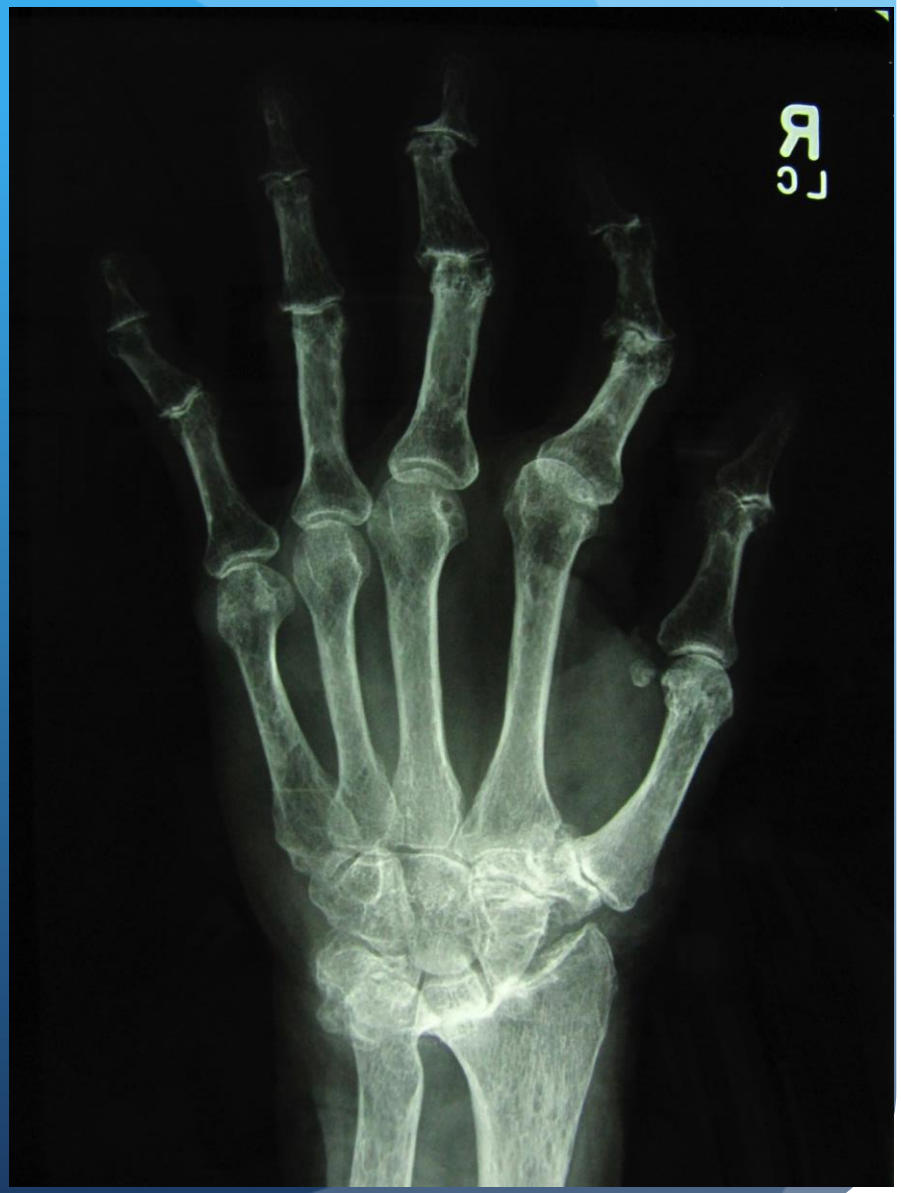


Heberden's and Bouchard's nodes

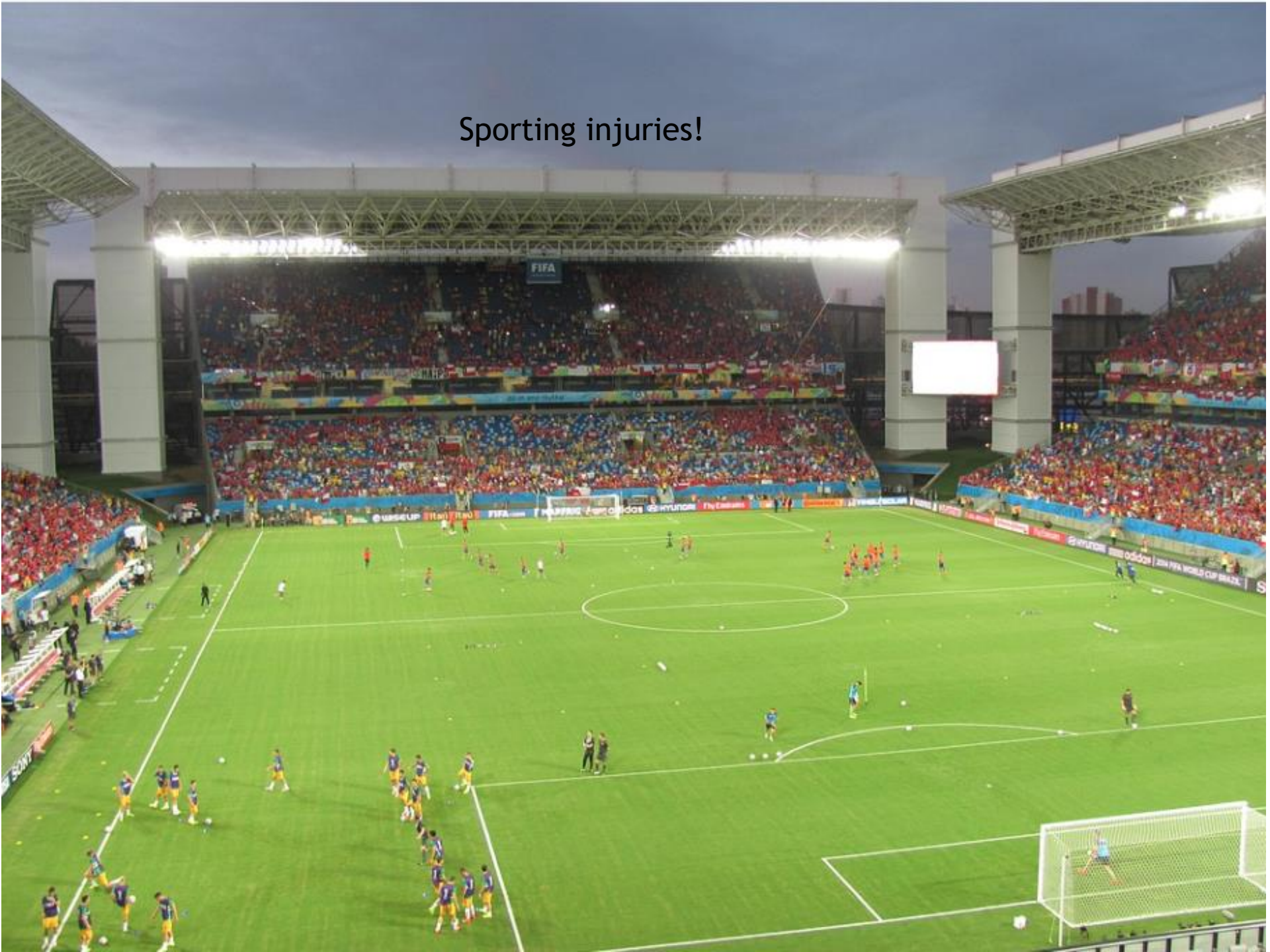


Xray images:

This shows advanced, erosive OA of the DIP, PIP joints sparing of the MCPs, and pan-trapezial OA in the thumb base. The wrist may also be badly affected, often due to calcium pyrophosphate deposition in the triangular cartilage as in this patient.
Caution: sometimes psoriatic arthritis may affect similar joints with indistinguishable xray changes in DIP/PIP joints.

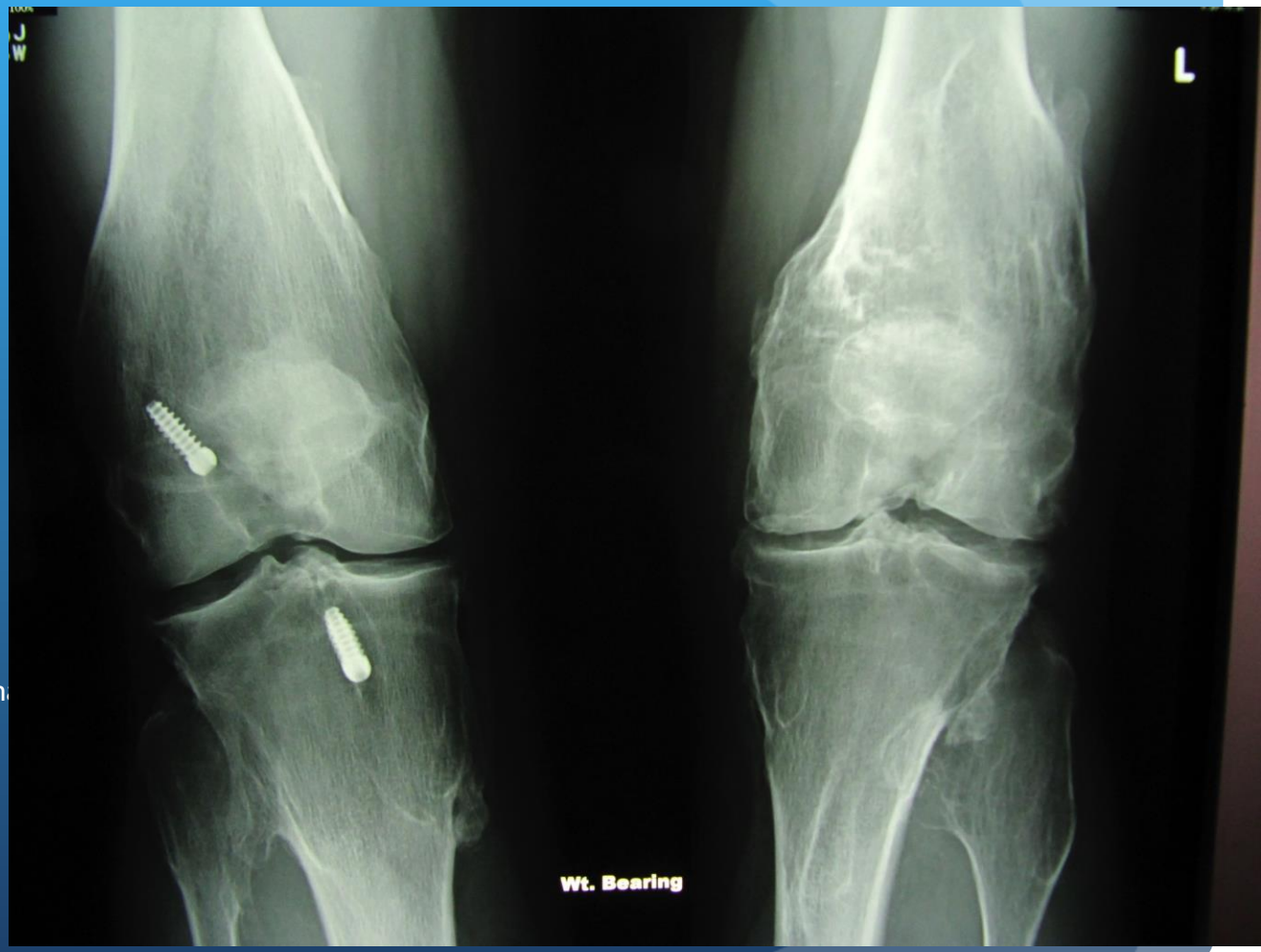



Have a seat Kermit. What I'm about to tell you might come as big shock...


Sporting injuries!

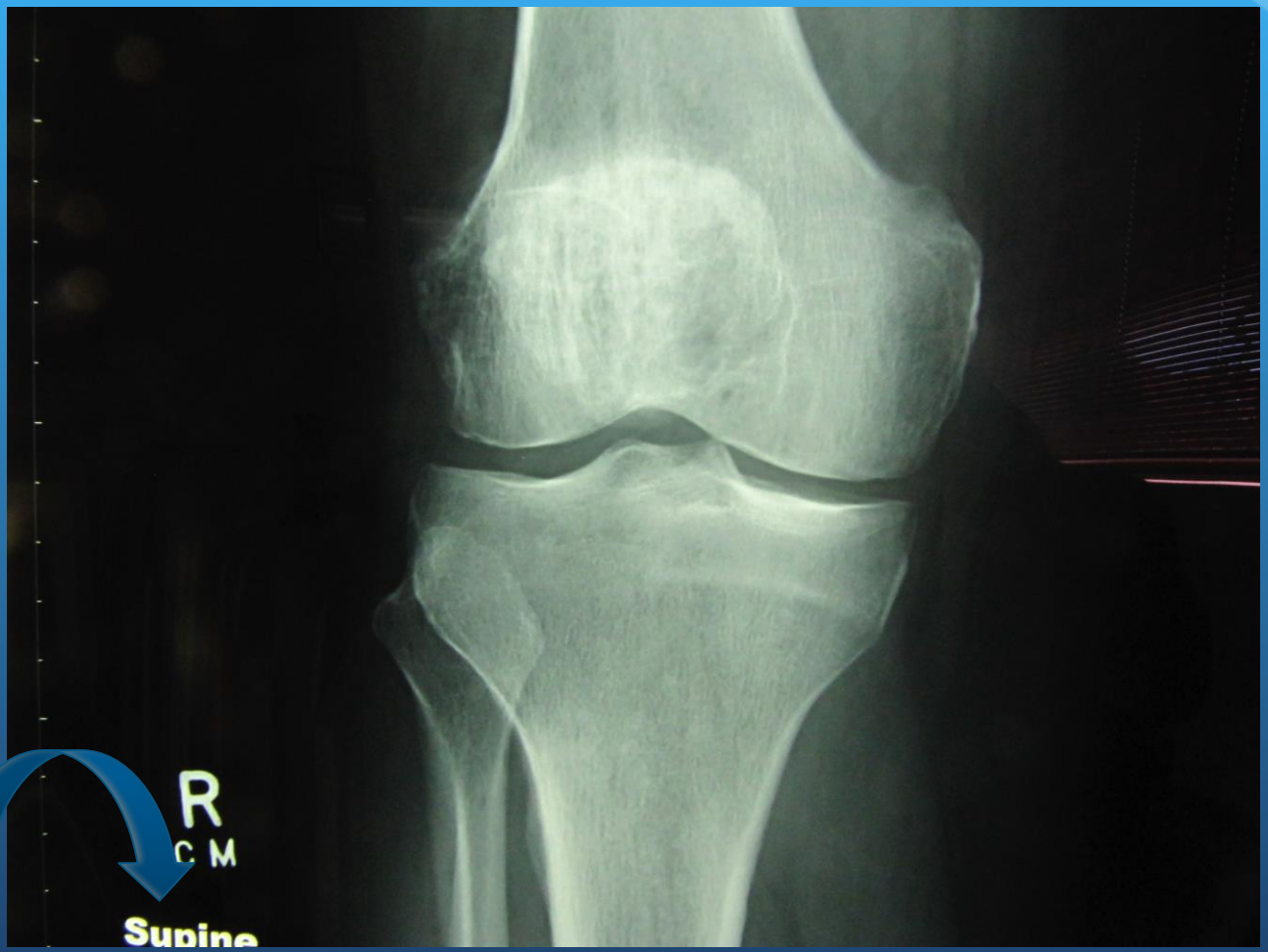
Secondary Osteoarthritis

- Obesity
- Injury
- Malignment
- Shallow acetabula
- Infection
- Excess iron
- Gout
- Pseudogout
- Ischaemia (AVN, barotraum
- Neurological
- malignancy



Obesity is the major secondary cause in the western world

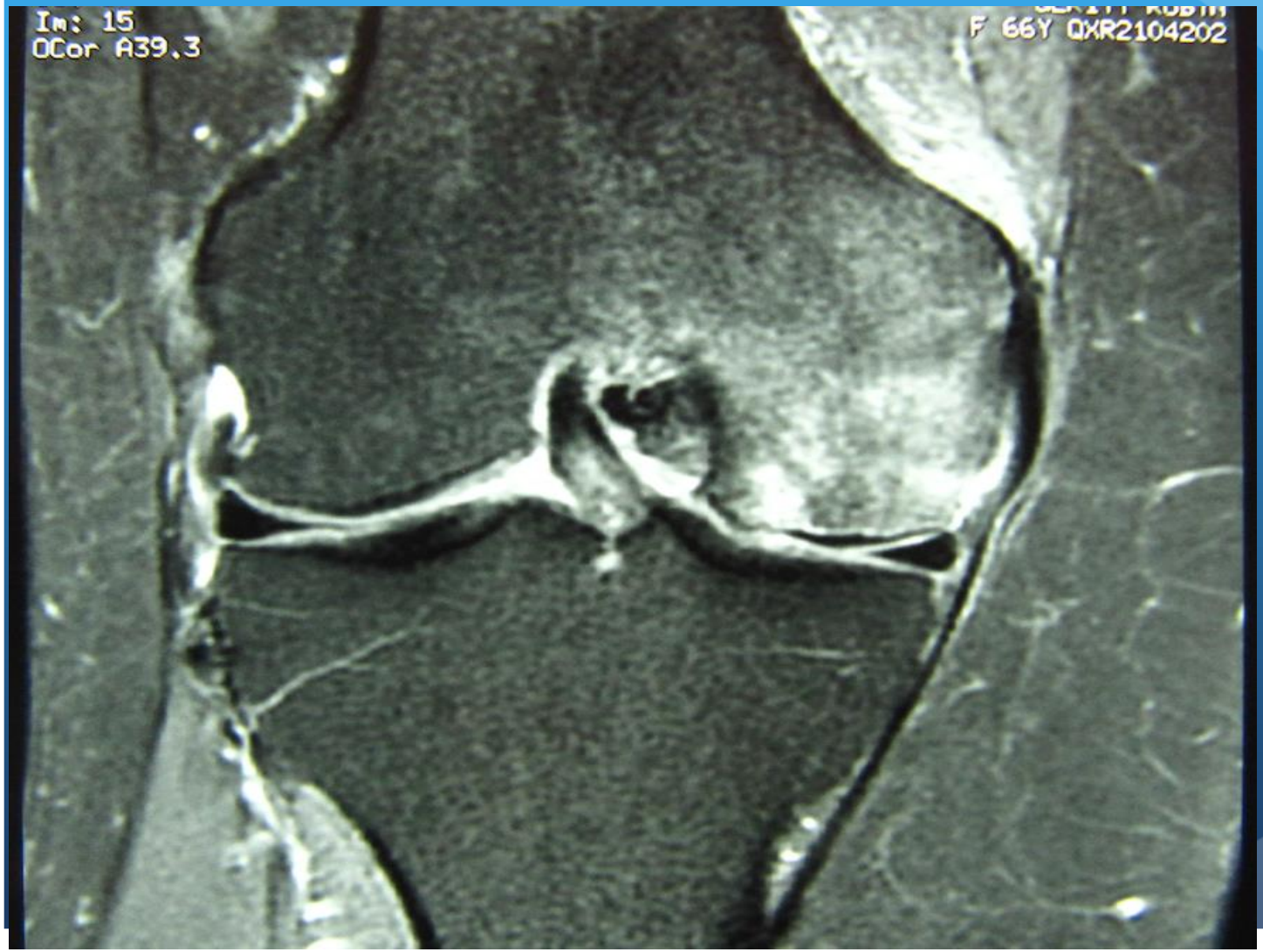
The plain film Xray accompanying a person with medial R knee pain



66 yr old female, recently taken up walking again.

<- Note

The MRI tells a different story!



1.Loss of hyaline cartilage
2. Bone marrow oedema medial femoral condyle
3.Oedema insertion of anterior cruciate
4. Menisci intact

Common symptoms of OA

- Pain
- Swelling
- Stiffness
- Loss of function
- Deformity
- Weakness
- Grinding or clicking
- Instability

Pain

- This is the most common complaint when OA is symptomatic, but less than 50% of people with Grade III or IV (Kellgren and Lawrence severe grades) radiographic OA report pain. Pain turns out to be surprisingly complex. (Mease etal J Rheum Aug 2011).
- Nociceptive pain arises from the affected structures but not cartilage, as there are no nerves in cartilage. Subchondral bone, capsule, synovium, ligaments, bursae, tendons and muscle may all be sources of the pain.
- Inflammation is often present and correlates with pain. The release of cytokines from inflamed tissue may explain the development of some centrally mediated pain. Neurotrophins such as NGF are new targets for treatment.
- More chronic pain may be neuropathic, ie due to central sensitization, and could explain some of the 30% of patients with Total Knee Replacements still experiencing pain afterwards.

Treatment : try to address the cause

- If the pain is arising from a periarticular structure, this may be treated with direct means
- If it is arising from the joint itself, then exclusion of meniscal injury or foreign bodies and where possible, joint aspiration may isolate the cause.
- Sarcopaenia may be very obvious with OA. Treatment is thus directed at restoring muscle where possible. Appropriate exercise is vital.
- Correction of gait and other alignment factors and reduction of joint loading, if possible. Orthotics and braces have mixed reports of benefit.

Treatment:

- Reduction of inflammation where present:
  - Judicious use of anti-inflammatory agents (oral, topical)
  - Intraarticular injections: a)Steroids, b)hyaluronic compounds or c) PRP??.
- Pain control with NSAIDs/COX-2 i, if safe! (CVS and GI guidelines). Topical gels in certain joints. Paracetamol not great. Use of cold or hot packs? Try capsaicin creams. Rarely need opioids, and these have major long term problems. Avoid!(see current US opioid crisis).
- If neuropathic pain, then neuromodulators may help.
- Surgery – fusion in some instances, or replacement.
- Novel agents. (oral steroids, methotrexate poor results in RCT). Anti-IL1 likewise. Zolendronate: failed ZAP2 RCT. Tanezumab?-pain relief but accelerated joint destruction. Lots of interest!