Osteoarthritis and Rheumatoid Arthritis

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AIMS

• We will all come across osteo- and rheumatoid arthritis whatever our clinical practice
• Overview of pathology of osteoarthritis, its assessment and management options
• Discuss the pathology, assessment and management of rheumatoid arthritis
• Not going to discuss sero -ve arthritides
• Emphasis will be on orthopaedic not medical management (leave that to the clever docs)
Osteoarthritis (OA)

- Non-inflammatory degenerative joint disease characterised by progressive damage and loss of articular cartilage with associated irregular new bone formation and capsular fibrosis.
Hyaline Cartilage

- Few mm thick but withstands tremendous forces during everyday activities (Force acting through hip = 4x body wt during walking)
- Few chondrocytes
- Type II collagen & aggrecan molecules give physical properties
- No blood supply & no innervation
- Relies on synovial fluid for nutrition
Articular Cartilage II

• Load bearing properties are related to its structure
• Transmits force from bone to bone
• Low friction
• Deformability and elasticity distribute loads evenly
Cross Section of Articular Cartilage

Zones

- Superficial tangential (10-20%)
- Middle (40-60%)
- Deep (30%)

Articular surface

- Tide mark
- Subchondral bone
- Cancellous bone

Calcified cartilage
Chondrocytes

- Chondrocytes are important in the control of matrix turnover through production of collagen, proteoglycans, enzymes for cartilage metabolism
Healing of Articular Cartilage

- Hyaline cartilage does not “heal” with hyaline tissue.
- Deep lacerations
  - extend below the tidemark
  - heal with fibrocartilage
- Superficial lacerations
  - above the tidemark
  - chondrocytes proliferate but do not heal
Classification of OA

- Primary
- Secondary
  - previous trauma
  - congenital deformity
  - infection
  - metabolic disorder
Aetiology

- Idiopathic in the majority
- Biomechanical: instability causes eccentric loading
- Biochemical: imbalance between degradation & regeneration (inc. proteases)
Aetiology 2

• Age - exponential rise in prevalence OA
  – NB.: differences in “old” cartilage and OA
• Obesity - increased with BMI
• Occupation - blue collar workers most at risk
• Family Hx:
  – Hereditary disorders (multiple epiphyseal dysplasia)
Aetiology 3

- Injury
  - Intra-articular fractures with step in articular surface
  - Soft tissue injury causing instability
- Menisectomy
  - 3-11 times more likely to develop OA in knee
- Deformity (congenital, developmental and acquired)
  - DDH, SUFE, Perthes are all associated with increased risk
- Calcium deposit diseases, gout, infection,
Presentation

- Pain, pain, pain
- Stiffness
- Functional disability
- Deformity (varus, valgus)
- Instability
- Joint: swollen, tender, crepitus, ROM reduced and painful
Radiology of Knee

Figure 1: Normal knee

Figure 2: Narrow joint space, Bone spur
Radiology of Hip

Figure 1: Normal hip

Figure 2: Bone spurs
Radiology of Hip
Radiology

- Loss of joint space
- Osteophytes
- Subchondral sclerosis
- Subchondral cysts
Investigations

- Consider crystal or inflammatory subsets
  - FBC, ESR, Rh. Screen
- Remember infection, especially if symptoms deteriorate suddenly!!
  - Aspirate & washout
- MRI – assess cartilage thickness, other pathologies
- Arthroscopy – good visualisation & assessment
Pathology

- Disruption of the articular surface -> fibrillation -> eburnation and this causes pain
- Subchondral cysts - due to microfractures that degenerate & accumulate synovial fluid
- Osteophytes caused by proliferation of cartilage adjacent to the weight-bearing which then undergoes endochondral ossification
- Sclerosis - compression of weakened bone with collapse
- Loose bodies - fragmentation of osteochondral surfaces cause mechanical symptoms
Treatment - Conservative

- No cure, therefore treat symptoms
- Education
- Protection of affected joints from overloading
  - weight loss, use of walking stick (opposite hand)
- Exercise of supporting muscles around joints to avoid wasting
- Analgesia: paracetamol or NSAIDs
- Hyaluronic acid injections
- Oral glucosamine & chondroitin
Surgical Treatment

- Indicated for patients with persistent symptoms and pain
- Classically osteotomy, arthroplasty, arthrodesis & excision
- Realignment osteotomies may be done in younger patients to redistribute weight bearing load at the knee to prevent further damage
- Total or partial joint replacement for older patients in advanced cases that are resistant to conservative treatment
Wedge from tibia removed

Joint not aligned due to arthritis
New & Experimental Treatments:

- Mirco-fracture/drilling
- Chondrocyte transplantation (collagen gels etc.)
- Fresh osteochondral grafts – auto or allograft
- Soft tissue interposition grafts - periosteal / perichondral
- Doxycycline - In animal studies, doxycycline inhibits cartilage collagenase activity and reduces the severity of osteoarthritis
- Transforming growth factor beta (TGF beta) - can repair partial thickness lesions in animal models
- Metalloproteinase inhibitors - inhibit enzymes responsible for cartilage matrix destruction and block cytokines
- Gene therapy - anti-arthritic or synthetic properties can be delivered into the joint via non-replicating viral vectors
The potential long-term treatment or cure of osteoarthritis lies in the development of agents that reverse the balance between degradative and synthetic processes of the chondrocyte.
Rheumatoid Arthritis

- 3% of women & 1% of men
- **Diagnosis: ARA Criteria** (revised 2000): need 4 of the 7 below for 6/52

  - **Morning stiffness**
  - **Arthritis of 3 or more joint areas must have simultaneous soft tissue swelling**
  - **Arthritis of hand joints**
  - **Symmetric arthritis**
  - **Rheumatoid nodules**
  - **Serum rheumatoid factor**
  - **Radiographic changes**
    Radiographic changes typical of RA on posteroanterior hand and wrist radiographs, which must include erosions or unequivocal bony decalcification localized to or most marked adjacent to the involved joints (osteoarthritis changes alone do not qualify).
Aetiology & Pathology

• Genetic susceptibility (HLA-DR4 & DW4) &/or environmental trigger factors -> Auto-antibodies (IgG & IgM) to the body's own IgG -> T-cell infiltration & B-cell reactivity in the synovium.

• **Stage 1: Synovitis**
  - vascular congestion
  - synoviocyte proliferation
  - infiltration of subsynovial layers by PMNs, lymphocytes & plasma cells

• **Stage 2: Destruction**
  - a pannus of granulation tissue creeps over the articular surface eroding cartilage & bone
  - direct invasion occurs at the margins of the joint
  - similar changes occur in tendon sheaths

• **Stage 3: Deformity**
  - From:
    - articular destruction
    - capsular stretching
    - tendon ruptures
Clinical Findings

- **Extra-articular Manifestations:**
  - Nodules - in 20% of RA - skin, synovium, tendons, sclera, viscera
  - Lymphadenopathy
  - Splenomegaly
  - Vasculitis
  - Myopathy
  - Sensory changes - neuropathy, or direct compression from synovitis
  - Visceral – pericarditis, pulmonary fibrosis, nodules, pleurisy

- **Syndromes:**
  - *Felty's Syndrome* - splenomegaly + leukopaenia
  - *Still's Disease* - fever, rash + splenomegaly
  - *Sjorgen Syndrome* - decreased salivary & lacrimal gland secretion & lymphoid proliferation

- **Laboratory Findings:**
  - Incr. ESR
  - Incr. CRP
  - RF +ve in 80%
Management Principles

• **Stop the Synovitis**
  – Rest
  – Drugs - Pyramid Approach = NSAIDs - antimalarials - disease modifying agents (MTX, sulphasalazine, gold, penicillamine) - steroids - cytotoxic drugs - experimental drugs.
  – Synovectomy - chemical, irradiation, surgical

• **Prevent Deformity**
  – Splintage
  – Physiotherapy
  – Tendon repairs & joint stabilisation

• **Reconstruct**
  – Arthroplasty
  – Arthrodesis
  – Osteotomy

• **Rehabilitate**
  – Occupational therapists - aids, support
  – Physiotherapy
Complications:

- Fixed Deformities
- Joint Rupture
- Infection
- Spinal cord compression
- Peripheral nerve compression
- Vasculitis
- Amyloidosis - proteinuria & progressive renal failure

Prognosis:
- 10% improve
- 60% have remissions & exacerbations
- 20% have severe joint erosions requiring multiple operations
- 10% become completely disabled

Poor prognostic signs:
- Very high RF
- Peri-articular erosions
- Nodules
- Muscle wasting
- Joint contractures
- Vasculitis
Summary

- Brief overview of pathology, assessment and treatment options in OA and RhA suitable for FY1 and FY2 level