

Rheumatoid Arthritis

Objectives:

- ★ Recognize which patient is likely to have RA
- ★ Know the different modes of presentation of RA
- \bigstar Develop a plan of investigation and management of RA

Color index:

Original text Females slides Males slides Doctor's notes Text book Important Golden notes Extra

Osteoarthritis VS Rheumatoid arthritis (Extra)

• We will take each disease in a separate lecture, this is just an overview to help you differentiate between them.

	Osteoarthritis	Rheumatoid arthritis
Pathogenesis	Mechanical - wear and tear destroys articular cartilage (degenerative joint disorder)	Chronic, systemic autoimmune disease
Predisposing factors	Major risk factor is age (common after 60 years); additional risk factors include obesity and joint trauma.	Associated with HLA-DR4 , classically arises in women of late childbearing age
Presentation	 Pain that gets worse with movement and is relieved by rest Early morning stiffness (lasting LESS than 30min) Involves DIPJs (Heberden nodes), PIPJs (Bouchard nodes) and 1st CMC but NOT MCP Asymmetric joint involvement No systemic symptoms 	 Pain. Early morning stiffness (lasting MORE than 1hr) that gets better with movement Swelling in the small joints of the hands and feet. There's spindling of the fingers caused by swelling of PIPJs and MCP but NOT DIPJs or 1st CMC Symmetric joint involvement Systemic symptoms e.g. fever, fatigue, weight loss Extraarticular manifestations are common*
Joint findings	 Osteophytes (bone spurs) Joint space narrowing 	 Pannus (proliferative granulation tissue) Erosions, cervical subluxation, ulnar finger deviation, swan neck and boutonniere deformities

* Extraarticular manifestations include **rheumatoid nodules** (fibrinoid necrosis with palisading histiocytes) in subcutaneous tissue and lung (+ pneumoconiosis→ Caplan syndrome), interstitial lung disease, pleuritis, pericarditis, anemia of chronic disease, neutropenia + splenomegaly (**Felty syndrome**), **AA amyloidosis**, Sjögren syndrome, scleritis, carpal tunnel syndrome

Rheumatoid Arthritis (RA)

Introduction

- Rheumatoid arthritis is a chronic **Systemic** inflammatory disease mainly affects synovial joints and tendons. It can affect other systems in the body. Early recognition and treatment can prevent joint destruction and disability.
- Variable expression (the severity and limitations caused by RA is variable)
- RA has a worldwide distribution with a prevalence of 3 %. (Female:male ratio 3: 1)
- Peak age of onset: 25-50 years but could occur at any age, even children 'juvenile type'.
- RA remains a significant cause of disability and mortality and carries a high socioeconomic cost.
- Symmetrical polyarthritis ddx: RA, SLE and seronegative arthritis (We will take each in a separate lecture)



Etiology

• The exact etiology is **unknown**, but there are factors like genetics, environmental (**smoking**) & infectious components. These factors may cause modification and production of autoantigens (citrulline) by the process of citrullination, leading to the initiation of an autoimmune reaction

Genetics: associated with **HLA-DR4**¹ and HLA-DRB1

- Autoimmune disorder, associated with IgM autoantibodies against Fc portion of IgG (rheumatoid factor)
- Environmental such as smoking²
- Possible infectious component, most likely viral

Pathogenesis Normal Rheumatoid arthritis Bone and cartilage The pathology of RA: - Joint capsule erosion and synovia **Synovitis** (Joints, Tendon sheaths, Bursae) 0 lining Increased Synovial synovial fluid Vasculitis: widespread cutaneous vasculitis with necrosis of the skin 0 cavity Pannus -Cartilage formation (seen in people with very active, strongly seropositive disease) Subcutaneous Nodules 0 Synovitis with thickening of the Synovium proliferates and grows out over Infiltration by inflammatory cells synovial lining the surface of cartilage a tumour-like mass called 'pannus' is Pannus **destroys** the articular producing bony erosions & joint cartilage and subchondral bone destruction produced Numerous Cellular Interactions Drive the RA Process The interaction between Antigen presenting Cells and HLA-DR4 will activate rial products T cells & B cells. This will lead to the production of Autoantibodies (RF & Anti-CCP) as well as the activation of macrophages leading to the production of inflammatory cytokines (TNF- α , IL-1,6,17). This will lead to the activation of the Chondrocytes and Synoviocytes to produce collagenases and other proteases which will cause bone & cartilage destruction. RA Is Characterised by Synovitis and IL-1 and TNF-a Have a Number of Overlapping Proinflammatory Effects: **Joint Destruction** Proinflammatory effects of IL-1 Proinflammatory effects of TNF-α IL-1 Plays a Pivotal(crucial) Role in Inflammatory and Destructive Processes of RA IL-1 ↑ cyclooxygenase 2 ↑ prostaglandin-E₂ TNF-α ↑ nitric oxide **1**IL-1 duces fibrol ↑ Osteoclast Adhesion molecules ↑cell death activation [†]Chemokines ↑ Angiogenic ↑Collagenases Major cell type in the pannus: factors ↑IL-6 T lymphocytes & macrophages Major cell type in the synovial fluid: Neutrophils

Having HLA-DR4 increases the susceptibility for the development of RA and is associated with a poor prognosis.
 Environmental stress, such as smoking, is thought to promote post-translational modifications.

Joint inflammation:

- → Warm swollen and tender joints usually in a symmetrical pattern
- → Early morning stiffness (lasting more than 1hr) that improves with activity
- → Swelling in the small joints of the hands and feet.
- → There's **spindling of the fingers** caused by swelling of PIPJs and MCP but NOT DIPJs or 1st CMC
- → RA does not only affect the hands, once you suspect RA you should examine all the joints such as the temporomandibular joint, sternoclavicular joint, etc..
- → Baker cysts (synovial cysts in the popliteal fossa) may be present

Joint Involvement On Presentation Of RA:

- → 75% of cases presented with **Polvarticular**:
 - 60% of them have only small joints of hand & feet involvement
 - 30% have only large joints involvement,
 - 10% have BOTH small & large joint involvement.
- \rightarrow 25% of cases presented with monoarticular:
 - 50% of them have Knee involvement only.
 - 50% have: shoulder, wrist, hip, ankle, or elbow involvement.

If a patient presents with sudden monoarthritis, ALWAYS consider septic arthritis or infection like TB (in case of chronic), these must be excluded before the symptoms are attributed to a disease flare-up.

Wrist

- Synovitis 0
- **Radial deviation** 0
- Subluxation and collapse of carpus² 0
- Prominent ulnar styloid² 0



Prominent ulnar styloid



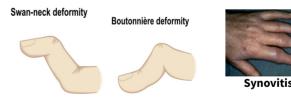
Z deformity

Thumbs

- Synovitis
- "Z" deformity

Proximal Interphalangeal Joints • Boutonniere/buttonhole deformity: Fixed

- flexion³, flexion of PIPJ and extenison of DIPJ.
- Synovitis
- **Swan neck:** Fixed extension³, hyperextension of PIPJ and flexion of DIPJ



Metacarpophalangeal Joints

- Synovitis
- Ulnar deviation⁴
- Subluxation

Important: So, radial deviation at the wrist and ulnar deviation at the MCPJ

Ulnar deviation at MCPJ

- Muscle wasting
- Radial deviation at wrist
- 1. This appearance is called spindling of the digits (swelling of PIPJ). In case an entire finger or toe is swollen, with joint and tendon sheath involvement (Sausage digits/Dactylitis) you must consider psoriatic arthritis (seronegative arthritis) in your differential.
- 2. Swelling and dorsal subluxation of the ulnar styloid, which causes wrist pain and may cause rupture of the finger extensor tendons, leading in turn to a sudden onset of finger drop of the little and ring fingers predominantly; this needs urgent surgical repair.
- 3. These deformities cause impairment of hand function.
- 4. This change leads to unsightly deformity, but function may be preserved once the patient has learned to adapt and pain is controlled.





Joint involvement & Destruction:

Cervical spine

- Painful stiffness of the neck in RA is often muscular, but it may be due to rheumatoid synovitis
- This synovitis leads to bone **destruction**, damages the ligaments and causes atlantoaxial or upper cervical vertebral instability. Particularly at C1 and C2, which can lead to subaxial subluxation
- In late RA, difficulty walking that cannot be explained by articular disease, weakness of the legs or loss of control of bowel or bladder may be due to spinal cord compression and is a neurosurgical emergency.
- MRI is the imaging of choice, but lateral flexed and extended neck X-rays can demonstrate instability.
- **X-RAY Imaging** of the cervical spine in flexion and extension is **recommended** in patients with RA before surgery or upper GI endoscopy to check for instability (Cervical subluxation) and reduce the risk of cord injury during intubation.



B: As the diseases progress, we can see some destruction. **C:** The hypertrophied synovium (Pannus) will invade the

в CACR

bone & cartilage showing Erosion on x-ray

intact



- osteopenia
- Small erosive changes

Different presentations of RA: (Obj.)

Palindromic	Palindromic monoarticular attacks last 24-48 h; 50% progress to other types of RA.
Transient	Disease is self-limiting, lasting <12 months and leaving no permanent joint damage. It is usually seronegative for lgM RF and ACPA. Some of these patients may have undetected postviral arthritis.
Remitting	There is a period of several years during which the arthritis is active but then remits, leaving minimal damage.
Chronic, persistent	This is the most typical form; it may be seropositive or seronegative for lgM RF. The disease follows a relapsing and remitting course over many years. Seropositive (plus ACPA) patients tend to develop greater joint damage and long-term disability. They warrant earlier and more aggressive treatment with disease-modifying agents.
Rapidly progressive	The disease progresses remorselessly over a few years and leads rapidly to severe joint damage and disability. It is usually seropositive (plus ACPA), has a high incidence of systemic complications and is difficult to treat.





Extra-Articular features

Hematological

- Felty's syndrome: A triad of Rheumatoid Arthritis + Neutropenia + Splenomegaly
 - Associated with increased risk of developing non-Hodgkin lymphoma
 Treatment: DMARD (Methotrexate)
- Anaemia of chronic disease is almost universal and is usually normochromic and normocytic. It may be iron deficient owing to gastrointestinal blood loss from NSAID ingestion, or rarely haemolytic (Coombs positive).
- There may be a **pancytopenia** due to hypersplenism in Felty's syndrome or as a complication of DMARD treatment.
- A high platelet count indicate an active disease. it just indicate active inflammation, not specific to Rheumatoid arthritis
- Large granular lymphocyte syndrome, Lymphomas (RA patients are at high risk)

Cardiac & Pulmonary

- **Cardiac¹:** Pericarditis, myocarditis, coronary vasculitis², nodules on valves or the conducting system of the heart.
- Pulmonary:
 - Pleuritis, interstitial lung disease³, bronchiolitis obliterans, arteritis, effusions.
 - **Peripheral, intrapulmonary nodules:** asymptomatic but may cavitate, especially with pneumoconiosis (Caplan's syndrome⁴)

Ocular (rare)

- Sjogren's syndrome (Sicca syndrome): Dry eyes, Dry mouth
- Episcleritis/scleritis⁵
- Scleromalacia perforans (Thinning and perforation of the eye)
- Choroid and retinal nodules
- Peripheral ulcerative keratitis

Neuromuscular

- Entrapment neuropathy
 - Median nerve compression is the most common and bilateral carpal tunnel syndrome can occur as a presenting feature of RA
- Peripheral neuropathy
- Mononeuritis multiplex: Involvement of the nerve trunk In case of rheumatoid vasculitis

Others

- Dermatological: Palmar erythema, Subcutaneous rheumatoid nodules over extensor surfaces (pressure areas), vasculitis
- Occasional fever, weight loss, fatigue, malaise
- Lymphadenopathy



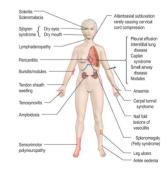


Small vessel vasculitis



Episcleritis

Scleritis, Episcleritis, with Scleromalacia



- **1.** Poorly controlled RA with a persistently raised CRP and high cholesterol is a cardiovascular **risk factor (for IHD and CAD)**, independent of traditional risk factors (i.e. high cholesterol and hypertension). it is like diabetes and hypertension.
- **2.** The most common cause of death in RA is coronary artery disease.
- 3. There is some evidence that the risk of pulmonary fibrosis is increased by anti-TNF therapy
- 4. Caplan syndrome: Rheumatoid Arthritis + Pneumoconiosis + Lung nodules
- 5. The eye involvement in rheumatoid is usually Episcleritis/scleritis. While in seronegative arthritis usually uveitis.

Investigations & Classification

Investigations



Hematological

- CBC: Anemia of chronic disease (Normochromic normocytic anemia), thrombocytosis. You may see leukopenia if the patient is on methotrexate or iron deficiency anemia if the pt is using NSAIDs
- ESR and CRP: Usually high in active disease, useful in monitoring treatment

Biochemistry

- Liver function test (LFT)
- **Renal profile:** Because the medications can cause side effects, so you should have a baseline before you start.
- Aspiration of the joint may be needed if an effusion is present. The aspirate looks cloudy owing to white cells. In a suddenly painful joint, septic arthritis should be suspected.



Radiological (X-ray)¹

- Joints: Do x-ray to look for any subluxations, Swelling in early disease and later joint narrowing and erosions.
- Spines: Cervical spine x-ray
- Chest: For lung involvement or effusion



Serology

- Rheumatoid factor (**RF**): IgM²
- Anti-citrullinated peptide antibodies (ACPA/Anti-CCP)³

Anti-CCP is more specific but still not diagnostic for RA.

High titer of **RF** or **Anti-CCP** is associated with more susceptibility to **extra-articular** manifestations (like lung involvement or vasculitis)

The 2010 ACR/EULAR classification criteria for rheumatoid arthritis:

- In the old criteria (1987) a score of 4/7 is diagnostic, however this classification has been disregarded now. why? because it contains erosions as a criterion, but you should NEVER wait for erosion to happen, but rather try to prevent it at all costs by medications
- Target population Patients (who should be tested?):
 - Who have at least 1 joint with definite clinical synovitis (Swelling) Not explained by other disease..
 - With the synovitis not better explained by another disease.
- The cut-off point for RA is at ≥6 points. Patients can also be classified as having RA if they have both typical erosions and longstanding disease previously satisfying the classification criteria.

ACR 1987 Classification Crite for Rheumatoid Arthritis

Patients Must Have Four of Seven Criteria: Morning Stiffness Lasting at Least 1 Hour* Swelling in 3 or More Joints* Symmetric Joint Swelling* Erosions or Decalcification on X-ray of Hand Rheumatoid Nodules Abnormal Serum Rheumatoid Factor _____* Must Be Present at Least 6 Weeks.

2010 criteria:

Criteria	Points
1. Joint involvement 1 medium to large joint	0–5 0
2-10 medium to large joints	1
1-3 small joints (large joints not counted)	2
4-10 small joints (large joints not counted)	3
>10 joints; at least one small joint	5
2. Serology At least 1 result is needed for classification Negative RF and negative ACPA	0–3 0
Low positive RF or low positive ACPA	2
High positive RF or high positive ACPA	3
3. Acute-phase reactants Normal CRP and normal ESR	0–1 0
Abnormal CRP or abnormal ESR	1
4. Duration of symptoms <6weeks	0–1 0
≥6weeks	1

1. In severe disease, extensive imaging of joints may be required. MRI is the technique of choice, especially for the knee and cervical spine

- 2. **IgM RF** is not diagnostic of RA and its **absence does not rule the disease out**; however, it is a useful predictor of prognosis. A persistently high titre in early disease implies more persistently active synovitis, more joint damage and greater disability eventually, and justifies earlier use of DMARDs. The term 'seronegative RA' is used when the standard tests for IgM RF are persistently negative. These patients tend to have a more limited pattern of synovitis.
- 3. ACPAs are usually present with RF in RA. They are better predictors of a transition from early transient inflammatory arthritis to persistent synovitis and early RA.

Complications and Management

Complications

Amyloidosis

- RA is the most common cause of secondary AA amyloidosis.
- AL amyloidosis causes a polyarthritis that resembles RA in distribution and is also often associated with carpal tunnel syndrome and subcutaneous nodules.
- Amyloidosis causing proteinuria, nephrotic syndrome and chronic kidney disease.
 So, if a rheumatoid patient presented with proteinuria, edema, etc. consider amyloidosis.

Septic arthritis (Monoarticular)

- In immunosuppressed patients, the affected joints may not display the typical signs of **inflammation** with accompanying **fever** found in patients with an intact immune system
- It has significant morbidity and mortality (medical emergency).
- Risk factors: Prosthetic joints, pre-existing joint disease
- Treatment is with systemic **antibiotics**¹ and **drainage**
- The most common organism is **S. aureus**
 - **MSSA:** Flucloxacillin + drainage
 - **MRSA:** Vancomycin + drainage

Cigarette **smoking**

C-reactive protein >20 g/dl

Positive rheumatoid factor (RF) and

Comorbidity

7.

8.

9.

10.

11.

NZ

Factors predicting poor prognosis

- 1. Older age
- 2. Female sex
- 3. Symmetrical small joint involvement
- 4. Morning stiffness >30 min
- 5. >4 swollen joints
- 6. Normochromic normocytic anaemia

Treatment



Treatment Goals:

- Relieve pain.
- Reduce inflammation.
- Prevent/slow joint damage.
- Improve functioning and quality of life.

Rationale for the Early Treatment of RA:

- Erosions develop early in the disease course
- Destruction is **irreversible**.
- Disease activity is strongly associated with joint destruction later in the disease course.
- Early treatment can slow down or prevent radiographic progress
- Disease activity must be suppressed maximally in its early stages to prevent destruction and preserve function.

Early erosive damage (On x-ray, US, MRI)

anti-citrullinated peptide antibodies (ACPA)

- Lifestyle modification: such as weight reduction and smoking cessation
- Rest: <u>JUST</u> During active phase. not prolonged rest as it is may lead to stiffness and muscle wasting.
- Physical and occupational therapy
- Medications & surgery (repair or replacement).

i Box 18.28 Management of rheumatoid arthritis

- Establish the diagnosis clinically.
- Use NSAIDs and analgesics to control symptoms.
 Try to induce remission with i.m. depot methylprednisolone 80–120 mg if synovitis persists beyond 6 weeks.
- If synovitis recurs, refer to a rheumatologist to start DMARDs and consider combinations of sulfasalazine, methotrexate and hydroxychloroquine. Give a second dose of i.m. depot methylprednisolone or oral steroids.
- Refer for physiotherapy and general advice through a specialist team.As improvement occurs, as measured by less pain, less morning
- stiffness and reduced acute-phase response, tail off steroids and possibly reduce drugs.
- If no better, use anti-TNF- α therapy or other biological agent.

DMARDs, disease-modifying anti-rheumatic drugs; TNF- $\!\alpha\!,$ tumour necrosis factor alpha.

Suspected Gram +ve? 1st line is vancomycin, with clindamycin or cephalosporin second line. Suspected Gram- negative? 1st line is 3rd Gen. cephalosporin with IV ciprofloxacin second line. In confirmed (MSSA) primary options include flucloxacillin alone or combined with fusidic acid or gentamicin, while penicillin-allergic patients should receive clindamycin or a 3rd Gen. cephalosporin. In cases of (MRSA), vancomycin is the drug of choice. Antibiotics should be altered according to sensitivities. Drainage of the joint and/or arthroscopic joint washouts are required in all cases.

Management

NSAIDs		
Drug name	Aspirin Ibuprofen - Ketoprofen - Naproxen	Celecoxib - Etoricoxib Selective COX-2 Inhibitors (less GI side effects)
Uses	 1st line to <u>relieve pain</u> and inflammation. NO ROLE in preventing the progression Use in combination with a DMARD 	
Side effect	• If GI side-effects are prominent, or the	e patient is over 65, add a PPI

Corticosteroids	
General info	 Low dose use if NSAIDs do not provide adequate pain relief or during flares Short-term treatment may be appropriate but avoid long-term use. Used as a bridge when waiting for DMARDs to take effect; DMARDs are much slower in onset of action than steroids. Sometimes if 1 or 2 joints are active, we may give local injections, provided that we excluded septic arthritis. Corticosteroids are the most common cause of secondary osteoporosis, Therefore, concomitant vitamin D and bisphosphonates are necessary to reduce fracture risk in patients expected to be on corticosteroid therapy for more than 3 months' duration.

	Disease-Modifying Antirheumatic Drugs (DMARDs) Click here for a summary of all DMARDs	
Drug name	Methotrexate	
General info	 Methotrexate is the 1st line & Gold standard DMARD MUST be part of the initial therapy with NSAIDS Monitoring requirement (CBC, LFTs) bc it causes liver toxicity and bone marrow suppression MOA: Inhibits DNA synthesis and cell division. 	
Uses	 Should be initiated early (at the time of diagnosis) to control symptoms. No immediate analgesic effects. So, begin treating With NSAID while waiting for DMARDs to take effect (~6 weeks).¹ Once the effect is evident, gradually taper and discontinue NSAIDs and continue DMARDs. Can delay progression of the disease (prevent/slow joint and cartilage damage and destruction) Should not be used in pregnancy and conception should be delayed until women have been off the drug for 3-4 months (in kumar it's 6 weeks) 	
<u>Alternatives</u> to Methotrexate	 Hydroxychloroquine It requires eye examinations every 6 months because of the risk of visual loss due to retinopathy. Used in mild non-erosive disease. Sulfasalazine: used as first line in young females of reproductive age, requires blood monitoring is because of the risk of leukopenia, thrombocytopenia and bone marrow toxicity. (CBC, LFTs) Both hydroxychloroquine and sulfasalazine are safe in pregnancy. Leflunomide, Azathioprine (especially if there are severe extra-articular manifestations, like lung involvement or vasculitis. You may also use other drugs like cyclophosphamide) For both (Leflunomide & Azathioprine) monitor CBC, LFTs. 	

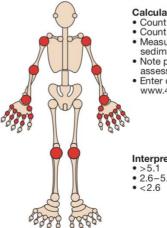
1. In early RA, the combination of at least one swollen joint for more than 6weeks with no prior injury, no associated history or family history of spondyloarthritis or associated conditions such as psoriasis, and a positive ACPA test is the best way to select patients for earlier treatment to avoid joint damage.

If not improving, use: Biologic Response Modifiers		
General info	Before using any drug (especially anti-TNF) we have to exclude infections like latent TB by screening with a purified protein derivative (PPD = Mantoux test)	
Drugs	 TNF Inhibitors: Etanercept, infliximab, Adalimumab. → first line as DMARDs for those not responding to methotrexate or intolerant of methotrexate. They are often used initially in combination with methotrexate to prevent disease progression. Contraindicated in CHF. → TNF inhibitors are safe in pregnancy. IL-6 receptor inhibitor : Tocilizumab. T-Cell costimulation modulator: Abatacept. Anti-CD20: Rituximab 	

Others	
Physiotherapy	 Effective in maintaining the range of motion. Strengthening of muscles, Prevent contractures, Prevent deformities. Maintain activities of daily living.
Occupational therapy	 Education of patients in the use of daily living activities Prevention of joint contractures and deformities.
Surgery	 Surgery has a useful role in the long-term approach to patient management but is less frequently needed as therapeutic disease control becomes more effective. Its main objectives are prophylactic, to prevent joint destruction and deformity, and reconstructive, to restore function.

Calculation of the Disease Activity Score 28 (DAS28)

- DAS28 is widely used to assess disease activity, response to treatment and need for biological therapy.
- It involves counting the number of swollen and tender joints in the upper limbs and knees, and combining this with the ESR and the patient's assessment of the activity of their arthritis on a visual analogue scale, where 0 indicates no symptoms and 100 the worst symptoms possible.
- This data are entered into a calculator to generate a numerical score. The higher the value, the more active the disease



Calculation

- Count swollen joints
 Count tender joints
- Measure erythrocyte sedimentation rate*
- Note patient global health assessment (1–100)
- · Enter data into calculator: www.4s-dawn.com/das28

Interpretation

- >5.1 High activity
 2.6-5.1 Moderate activity
 - Remission

Summary

Rheumatoid Arthritis Unknown could be : Etiology 1. Genetic 2. infectious 3. environmental 4. Autoimmune **Articular features:** Wrist (Radial deviation) • Metacarpophalangeal Joints (Ulnar deviation) • Thumbs ("Z" deformity) • Proximal Interphalangeal Joints (Swan neck, boutonniere deformity)) • Cervical spine (Do X-ray imaging before any procedure) • Features **Extra-articular features:** Felty's syndrome (RA + Neutropenia + Splenomegaly) • Cardiac: RA is a cardiovascular risk factor • Pulmonary: Interstitial Lung Disease • Neuromuscular: Carpal Tunnel syndrome • Dermatological: Subcutaneous nodules mostly in the pressure areas Investigations Anti-CCP is more specific than RF **NSAIDs** 1st line to relieve pain and inflammation. NO ROLE in preventing the progression • Corticosteroids During flares or if NSAIDs do not provide adequate pain relief • Methotrexate Best initial and Gold standard in managing RA. it Inhibits DNA synthesis and cell • division. Treatment initiated early (at the time of diagnosis) to control symptoms. No immediate analgesic effects (~6wks) Can delay progression of the disease Anti-TNF For those **not responding** to methotrexate or **intolerant** of methotrexate. Before using any drug we have to exclude infections like latent TB by screening with a **PPD** 7. Cigarette smoking 1. Older age 8. Comorbidity 2. Female sex 9. C-reactive protein >20 g/dl Factors 3. Symmetrical small joint involvement predicting poor 10. Positive rheumatoid factor (RF) and 4. Morning stiffness >30 min anti-citrullinated peptide antibodies prognosis 5. >4 swollen joints (ACPA) 6. Normochromic normocytic anaemia **11.** Early erosive damage (On x-ray, US, MRI)

Lecture Quiz

Q1: A 45-year-old woman presents to the rheumatology outpatient clinic with a three month history of stiff hands and wrists. She mentions that the pain is particularly bad first thing in the morning. On examination, the wrists, metacarpophalangeal joints and proximal interphalangeal joints are swollen and warm. A diagnosis of rheumatoid arthritis is suspected. Which of the following investigations is most specific for confirming the diagnosis? A-X-rays

- B- Rheumatoid factor levels
- C- Anti-citrullinated peptide antibody (anti-CCP) levels
- D- C-reactive protein
- E- Erythrocyte sedimentation rate

Q2: A 40-year-old woman presents to the rheumatology outpatient clinic with a three month history of stiff hands and wrists. She mentions that the pain is particularly bad first thing in the morning. On examination, the wrists, metacarpophalangeal joints and proximal interphalangeal joints are swollen and warm. A diagnosis of rheumatoid arthritis is suspected. Blood tests for rheumatoid factor return as positive. What is the most appropriate management? A- Non-steroidal anti-inflammatory drugs (NSAIDs)

B- Intramuscular depot injection of methylprednisolone plus NSAIDs

C- Anti-TNF therapy

D- Intramuscular depot injection of methylprednisolone plus NSAIDs and methotrexate and sulfasalazine

E- Physiotherapy

Q3: A 55-year-old woman presents to her GP with shortness of breath and dry cough. The symptoms began a few months ago and have progressed. She has a past medical history of rheumatoid arthritis, diagnosed ten years earlier. On respiratory examination, there are bibasal fine inspiratory crackles on auscultation. What is the most likely cause of her symptoms?

- A- Pulmonary oedema
- **B-** Consolidation
- C- Pleural effusions
- D- Pulmonary fibrosis
- E- Intrapulmonary nodules

Q4: A 30-year-old woman presents to accident and emergency with worsening stiffness in the hands, wrists and feet. She mentions that the pain has been particularly bad in the mornings. On examination, there is a palpable spleen. Initial blood tests reveal a low neutrophil count and a raised C-reactive protein. The most likely diagnosis is

- A- Felty's syndrome
- **B-** Reactive arthritis
- C- Still's disease
- **D-** Infectious mononucleosis
- E- Serum sickness

Q5: A 40-year-old woman complains of pain and swelling in both wrists and ankles for 7 weeks. She has several months of fatigue. Morning stiffness impairs her activities for approximately 2 hours. OTC naproxen provides temporary relief. On examination, the metacarpophalangeal (MCP) joints and wrists are warm and tender; there is slight tenderness to pressure over the ankles and metatarsophalangeal (MTP) joints as well. All other joints are normal. There is no alopecia, photosensitivity, kidney disease, or rash. Which of the following is correct?

A- The clinical picture suggests early rheumatoid arthritis (RA), and a rheumatoid factor and anticyclic citrullinated peptide (anti-CCP) should be obtained.

- B- The prodrome of lethargy suggests chronic fatigue syndrome (CFS).
- C- Lack of systemic symptoms suggests osteoarthritis.
- D-X-rays of the hand are likely to show joint space narrowing and erosion.
- E- An aggressive search for occult malignancy is indicated.

THANKS!!

This lecture was done by:

Faisal Alqifari Jehad Alorainy



Females co-leaders:

Raghad AlKhashan Amirah Aldakhilallah Males co-leaders: Mashal AbaAlkhail Nawaf Albhijan

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Send us your feedback: We are all ears!