Rheumatology

Rheumatology is concerned with diseases of joints & C.T., they are diseases of musculoskeletal system.

Rheumatological conditions are characterized by , chronic pain, stiffness &progressive impairment of joints & soft tisssue.

Economically they are costy , the cost comes from the direct spent of treatment (direct cost) & indirect cost from the lost of productivity.(these costs are millions of dollars).

Rheumatolgical conditions are one of the major causes of disabilities in community.

They may lead to various types of disabilities which could be ;

1-Physical disability (walking, bending,lifting & grasping).

2-Social disability (refers to higher tasks , i.e.eating, dressing, shopping& interacting with other people.)

Arthritis first causes of physical dysfunction which lead to social dysfunction.

Social dysfunction includes :

1-Basic personal care tasks such as eating , grooming& toileting.

2-Instrumental activities of daily living(IADL) (house task) like shopping and paying bills.

JOINTS.

Joint is articulation between two or more bones.

The activities of human body depends on effective interaction between normal joints & neuromuscular units that drive them.

Types of joints are:

1. Synarthrosis (skull joints)
2. Amphiarthrosis(Cartilaginous joints) have modest motion (Intervertebral disc joints).
3. Diarthrosis are mobile joints have synovial membrane (synovial joints).

Anatomy of synovial joints.

Diarthroidal joint consist of :

1. Opposing bony surfaces.
2. Hyaline cartilage covering the articulating surfaces of bones.It is firmly attached to the underlying bone. It is aneural & avascular. It gets the nourishment from synovial fluid , s. membrane &underlying bone. The cartilage is shock absorber.
3. Capsule which is surrounding the articular tissue.

4. Synovial membrane which lines the capsule. Has A cells are macrophage like cells have phagocytic function & B cells are fibroblast like cells (produce hyaluronate part of synovial fluid.

5.Joint cavity.

6. Synovial fluid. Is viscous , pale yellow & clear fluid , present in small amount ranging from (0.1 _ 4). Hyaluronic acid making the fluid viscous.The fluid lubricates the joint.

Rheumtology ; is concerned with the diseases of joints & C.Ts which could be damaged by avariety of pathological processes (infection ,inflammation , degeneration, metabolic disturbances &systemic diseases like leukaemia or Haemochromatosis.

Rheumatological Terminology.

Arthritis: is inflammation of the joint.

Polyarthritis : is inflammation of five or more joints simultaneously.

Oligoarthritis (Pauciarticular) : 2_ 4 joint are involved.

Monoarthritis : is inflammation of one joint.

Arthralgia : is a joint pain without swelling .

Inflammation of joint is suggested by :

- Pain , Swelling, Warmth & reddening of overlying skin.
- Joint stiffness which occur after a period of immobility & lasting for over a period (hour) is more reliable indication of inflammation.
- Reduction of joint motion & loss of function may occur in noninflammatory condition also.

Classification

Inflammatory arthritis.

1. R.A.
2. Seronegative arthropathies.
3. C.T diseases : SLE.
   Scleoderma.
   Dermatomyositis.
4. Crystal induced arthropathies.
5. Degenerative joint disease : primary or secondary.

6. Arthritis associated with infection :
   (septic &nonseptic arthritis).
7. Arthritis associated with systemic diseases
   (bacterial endocarditis, acromegaly, thyroid
   diseases,respiratory diseases & miscellaneous-
   sarcoidosis,amyloidosis etc.
8. Nonarticular rheumatism& localized pain
   (tenosynovitis, bursitis,fibrositis&enthesopathies)

CLINICAL FEATURES

Features help in the diagnosis of arthritis in general are :

Age: as in OA.

Gender: RA & SLE are more common in females.

Race: As arthritis associated with sickle cell anemia.

Occupation: As in soft tissue rheumatism.

Joint pain: the following points are of some value in the diagnosis of arthritic problems regarding pain :

Duration of pain (in Gout is acute while in RA is usually chronic).

Onset: (abrupt in gout).

Precipitating factors: as trauma, use of diuretics etc.

Characterisitics

- Site of pain : This usually indicates the site of the pathology.
- Radiation.
- Severety: i.e. Gout.
- Aggravating & relieving factors.
- Diurnal variation.(i.e.Inflammation).

Episodic arthritis

Morning Stiffness. Duration is important.

Pattern of joint involvement.

Reccurent attack, small joints involvement.

Disability. (depends on joints affected).

Objective signs of arthritis.

Swelling , Tenderness, Limitation of joint movement , deformity, Crepitas & Heat & redness.

Joint pathology is indicated by pain & one or of other signs.

Five cardinal sings of inflammation are : swelling, warmth, erythema, tenderness & loss of function.

Joint examination:

Includes three stages.(Look at it , feel it & move it)

Lab. Tests:

1.ESR

2.CRP(c-reactive protein)

3.Hb

4.WBCs count

Rheumatoid factors. **Are autoantibodies directed against antigenic determinants on the FC fragment of immunoglbuline G. RF may be of any isotype IgG, IgM, IgA or IgE.IgM is the one routinely measured by clinical laboratories. RF is detected by agglutination of either latex particles or sheep red cells.** Also by ELISA & Nephelometry.

Anti-CCP(anticyclic citriullinated peptide antibodies) are autoantibodies directed against the amino acids , it's believed that have a role in the pathogenisis of RA.

Antinuclear antibodies(ANA). Are antibodies which react to constituents of nucleous (SLE , Scleroderma, Juvenile RA. Etc.).

Serum uric acid ; (GOUT).

Imagings :

Plain x-ray is important in OA, RA & other rheumatological conditions. i.e (osteophyte , erosion….).

C.T scan & MRI are now more valuable than x-ray in diagnosis.

Synovial fluid anlysis:

Is helpful in distinguishing between inflammatory & noninflammatory arthritis. It is diagnostic in crystal induced arthropathies (gout) & septic arthritis.

Synovial biopsy:

done through needle biopsy or by arthroscopy (which by itself is a valuable investigation).

Ultrasound of joint & soft tissues.

Radioisotope bone scan: (tumors, infection, trauma, lead to increased uptake.).

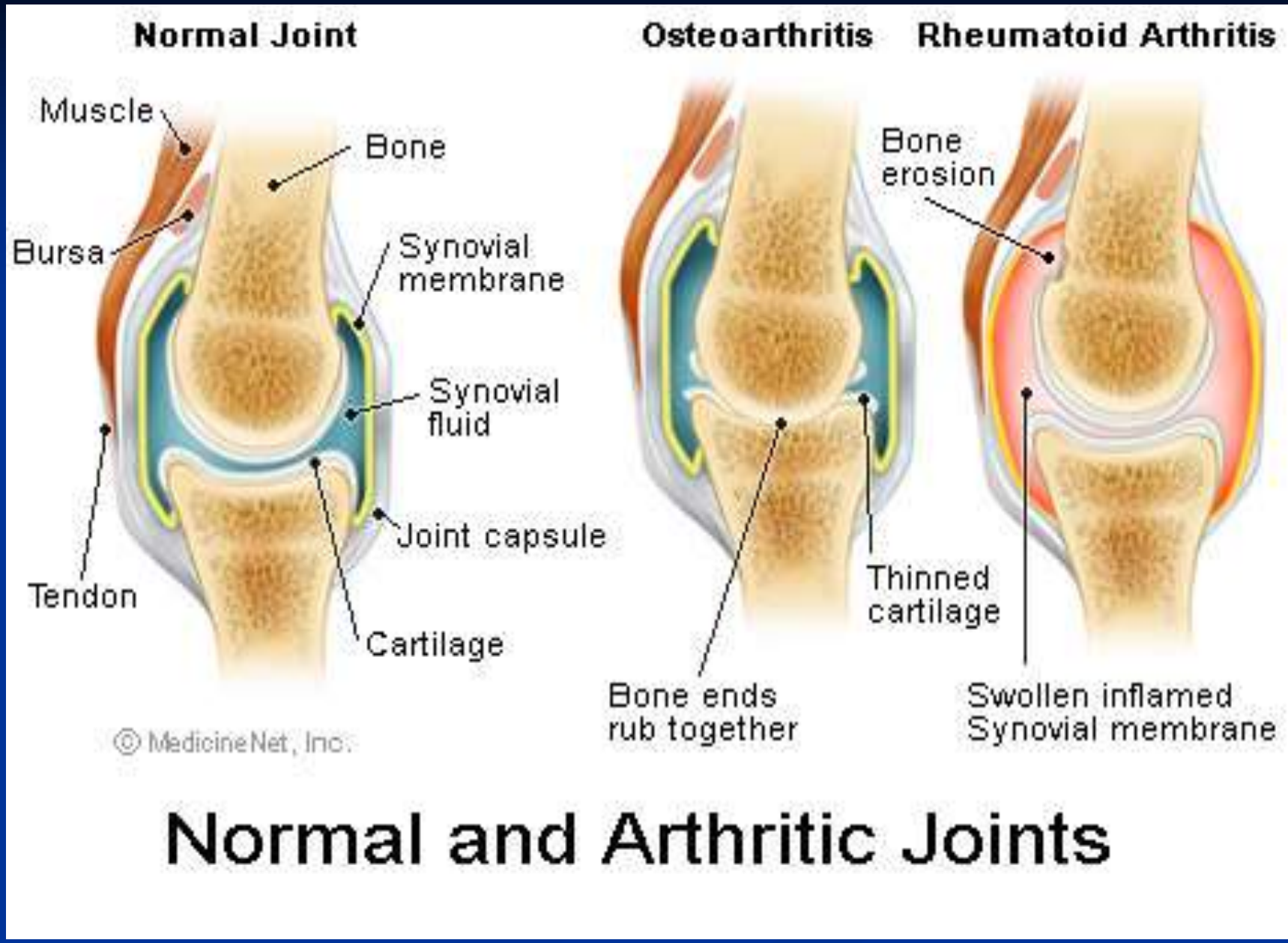
Histocompatability antigens: i.e HLAB27 in Ankylosing Spondylitis 95% , Reiter's disease 60% positive.

Some other investigations are helpful:

serum alkaline phophatase

ASO

low serum complements (in SLE).



Normal and Arthritic Joints

Osteoarthritis.

OA.Is a disease of diarthroidal joints , is a commonest musculoskeletal disease.

Charecterised by

1. pain & limitation of function,
2. osteophyte & joint space narrowing.
3. Alteration in cartilage integrity.
4. NO systemic manifestations.

The disease occurs through the world & through the history, it is more common in females.

Pathogenesis & pathology.

It is a disease of articular cartilage.

Chondrocytes are maintaining homeostasis of cartilage , it synthesize collagens, proteoglycans & proteinases.

OA results from failure of chondrocytes to synthesize a good quality of matrix in terms of elasticity & resistance & to maintain the balance between synthesis & degredation.

The degeneration process might be initiated by some stimuli (Mechanical insult or Biochemical abnormalities of cartilage).

The chodrocyte is believed to start releasing enzymes which degrade collagen & proteoglycan, breaks in the cartilage allow the uptake of water, cartilage swells & splits leads to breaking of the cartilage(fibrillation of the surface) , clefts down toward the bone surface, gradual lyses of cartilage by synovial enzymes & inflammation of the synovial membrane (might be effusion).

- The underneath bone become smooth & hard called (eburnation) & osteophytes form from boney edges & subchondral bone cyst might form.
- Joint insability may develop as a result of capsulare laxity , collapse of subchondral bone cyst & muscular atrophy.

CLASSIFICATION OF OA. S

A. Primary OA.

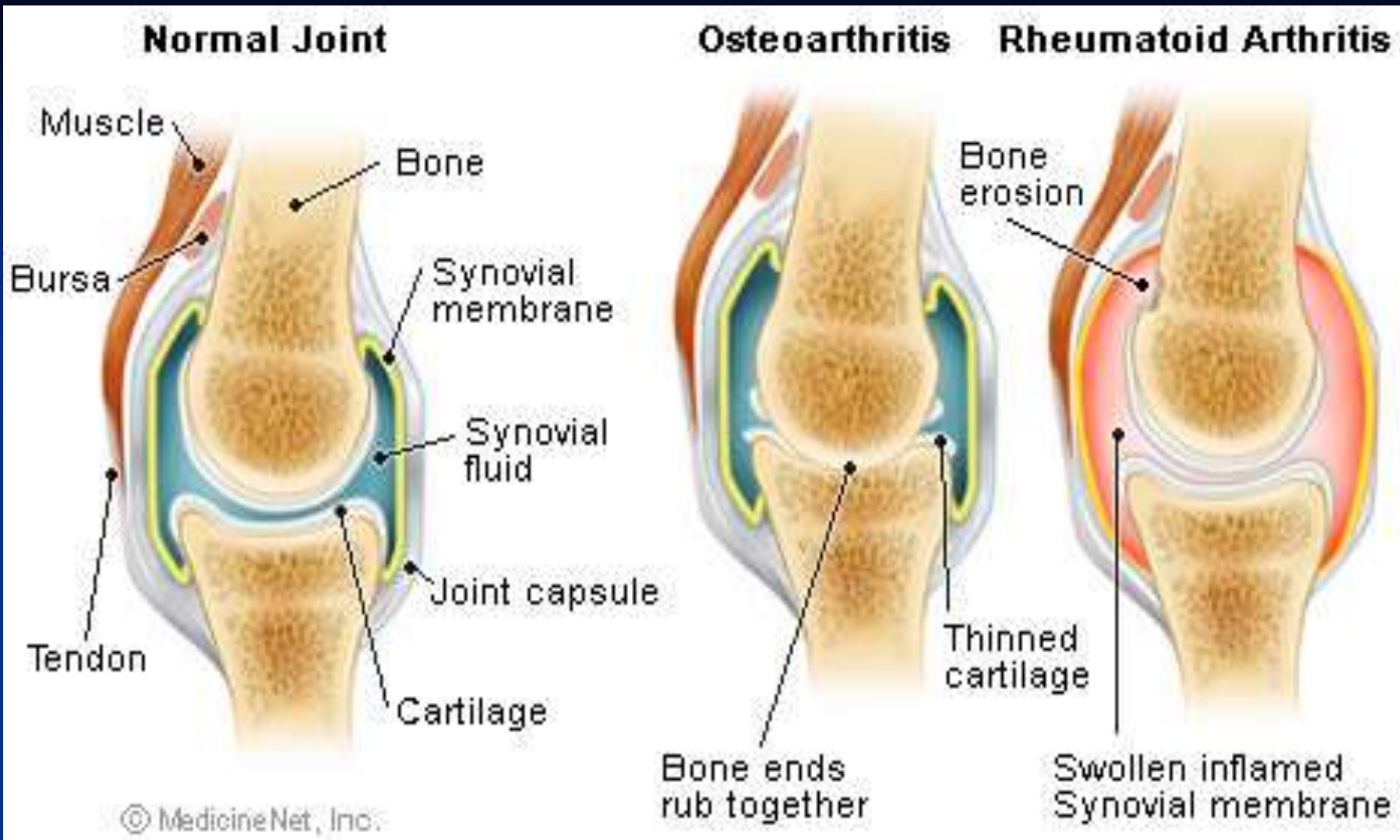
1. Localised (Heberden s & Bouchard s nodes)
2. Primary generlised OA.(involvement of 3 or mor joints or joint groups).

B. Secondary OA.

Any malalignment , damage or alteration to the constituents of the joint (the bone, cartilage, capsule, ligamemts or synovium may result in accelerated wear& the development of OA.

1. Trauma.(Intraarticular fracture, maalignment of fratcture, unequal leg length, occupation).
2. Genetic, congenital & developmental abnormalities: Hypermobility, DDH, Perth's disease etc.

3. Post inflammatory (RA, Septic arthritis etc).
4. Haemorrhage to the joint(Haemophilia).
5. Metabolic & endocrine diseases.
6. Bone disorder(Paget s disease)
7. Neuropathic joints.



Normal and Arthritic Joints

Risk factors for OA are:

- Obesity,
- Heridity(in DIP joints),
- Age,
- Previous joint disease (trauma),
- Abnormal joint mechanics(varus or valgus) ,
- Smoking(in disc disease).

Aetiology

OA is probably is multifactorial .

1. **Age & gender. OA is more in aged & females.**
2. **Trauma & obesity.**
3. **Hormonal & metabolic causes.**
4. **Genetic factor.**
5. **Diatery causes.**

Clinical Features.

Commonly affected joints are spine , knees, DIPs, PIPs, thumb joint, & first MTP joints

Symptpms

Pain.

Is deep dull aching pain, worse by use (in evening) may be present at rest. Trauma precipitate it , related to weather (more in cold & damp weather), it may radiate to the surrounding structures.It may be persistent interfering with normal function & sleep.

Pain arises from structures possessing nerve endings & may result from microfructures in subchondral bone.

Stiffness.

It is less sever than in inflammatory arthritis & lasts for a shorter period (few minutes).

Disability.

It depends on a number & severity of joint affection.

Signs of OA

1. Swelling. May be due to soft tissue, fluid or boney overgrowth.

   Heberdens & Bouchards nodes. Are bony articular nodes. (H.N are seen on DIPs & B.N on PIPs).

2. Crepius.
3. Signs of inflammation.
4. Limitation of joint movement.
5. Joint deformity.(valgus, varus , flexion deformities or joint instability).
6. Loss of function.

Investigations.

There are no diagnostic makers in OA.

X- Ray changes.

1. Narrowing of the joint space
2. Oteophyte.
3. Sclerosis of subchondral bone (eburnation).
4. Subchondral bone cyst.
5. Deformity resulting from sublaxation.
6. Presence of loose bodies.
7. Irregularity of bone surfaces.

MRI is superior to X-Ray (showing cartilage changes).

MANAGEMENT

Aims are.

1. Relieving pain.
2. Maintaining & improving joint function.
3. Preventing or minimizing disability improving functioning.

General measures.

1. Education of the patient.
2. Maintain & improve joint function.
3. Short lower limb should be correted.
4. Reduce weight .
5. Avoid trauma.
6. Use aid .
7. Change job.

Phamcological therapy.

1. Simple analgesia(paracetamol).
2. NSAIDs.
3. Intraarticular injections(steroid , Hyaluronic acid).
4. New agents (might reduce cartilage degredation & stimulate cartilage synthesis)(Glucosamininglycan & chondroitoin).

Physiotherapy.

To maintain joint mobility &function & improve muscular condition.

Surgery.

(i.e joint replacement).

Rheumatoid Arthritis.

Is a chronic systemic inflammatory disease of unknown aetiology characterized by symmetrical arthritis of hands , feet & other joints (PIP & MCPs) with bone erosion.

RA is a commonest inflammatory arthritis.

RA is characterized by:

1. A symmetrical inflammatory polyarthritis.
2. It has extraarticular features.
3. Progressive joint damage causing sever disability in young people.

Epidemiology.

RA is a world wide disease affects all racial & ethinic groups.It affects 1—3% of population , female/ male is 3/1.

Age range is 10 – 70y. (starts 30& 40ys.).

5—10% having family history & 70% have HLADR4.

Aetiology & Pathogenesis.

The cause of RA is unknown. Several factors may possibly operate to produce the disease (i.e. Genetic, environment..). RA is said to be an autoimmune disease.(there are many immunological disturbances in RA. As autoantibodies).(Cytokines , growth factors , tumor necrosis factors& metalloproteases have role in the production of the disease.).

Pathology.

RA is a disease of synovial membrane.

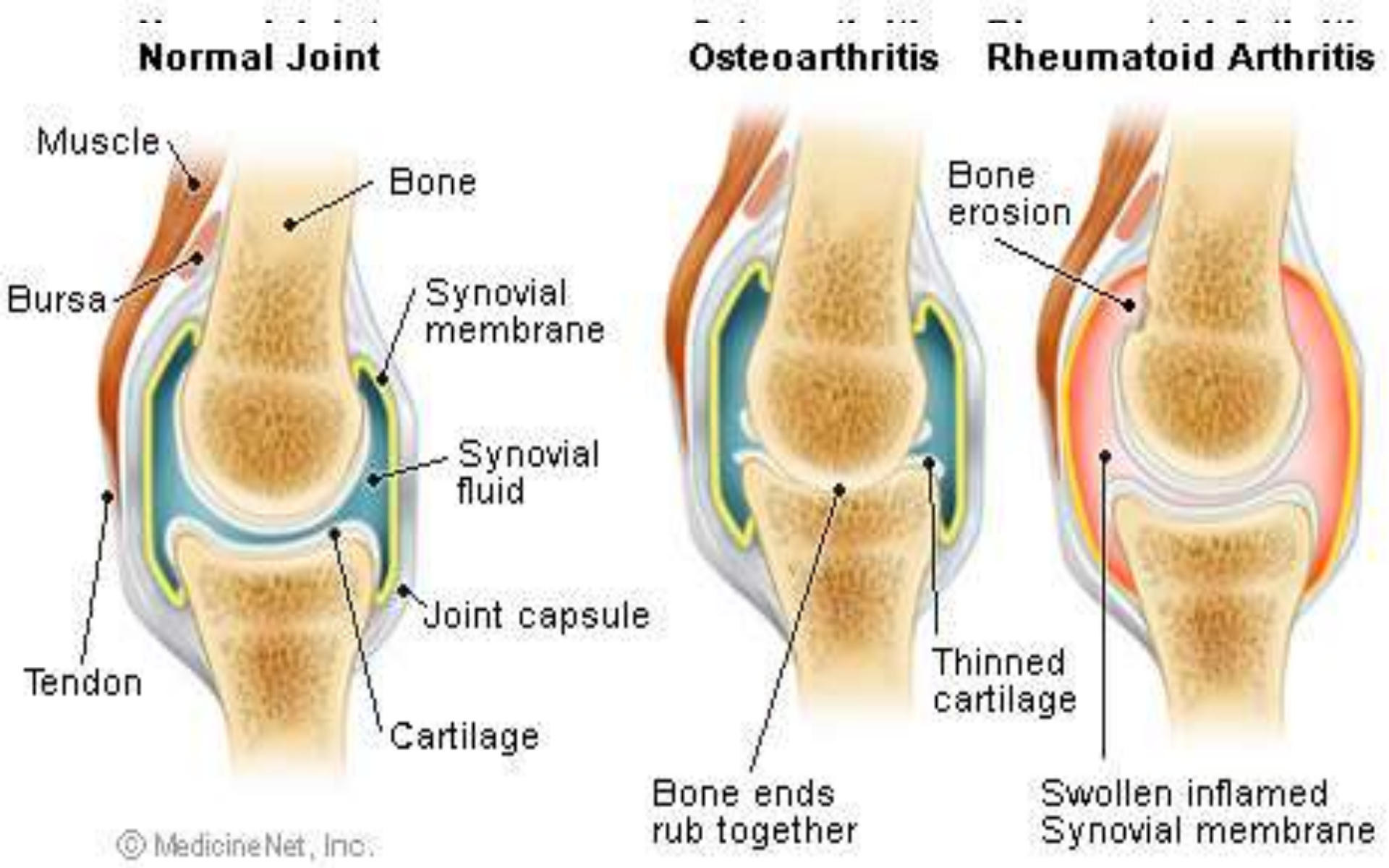
There is Inflammation & proliferation.(synovitis occur with chronic inflammatory cells infiltration, lymphocyte, plasma cells & macrophages followed by gaint cells infiltration, fibrinoid degeneration& hyperplasia of lining cells followed by granulation tissue (Pannus) formation which is a tumor like mass. This leads to destruction of the cartilage & bone & might be effusion.

Synovitis occurs in tendon sheath & bursae. Nodules might occur over pressure areas (more on extensor surfaces).

Vasculitis(panarteritis) may occur.

Chest could be involved.

Eyes might be affected.



Normal and Arthritic Joints

Clinical Features.

The onset is variable (70% insidious 15- 20% is subacute & 5—10% acute while few having episodic symptoms that progress to persistant diseases).

Commonly affected joints are MCPs, PIPs, wrists, MTPs & larger joints.

RA might starts with few joints then progress& become symmetric.

Thoracolumbare , sacroiliac & DIP joints are very rarely involved.

RA is generally symmetrical, destructive, disabling & deforming polyarthritis , affecting small & large joints.

It has systemic manifestations, extraaricular features& circulating antiglobulin antibodies (RA factor).

Symptoms.

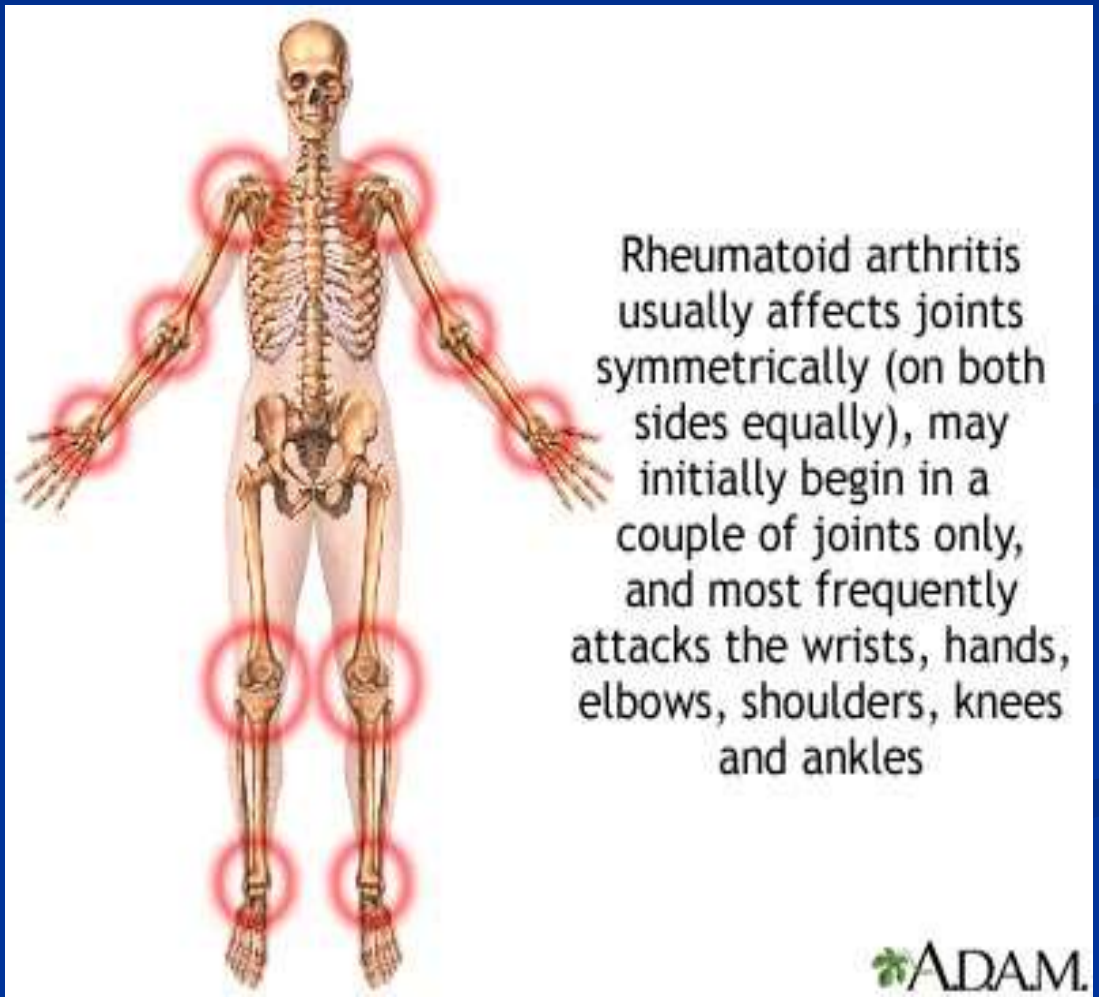
1. Joint pain.(More in the morning & might disturb the sleep).
2. Morning stiffness. Is a prominent feature , present after aperoid of rest often lasts for several hours.
3. General symptpms. Malaise, fever,loss of weight & strength & diffuse muscle wasting .
4. Disability. This depends on joints affected & destructions occurred.
5. Non articular symptoms: e.g CTS.

Signs.

- Swelling,
- Warmth,
- Limitation of movements,
- Deformities &
- Nodules.

Pettern of joint involvement.

RA is usually starts from small joints of hands & feet but in most patients eventually many joints are involved (wrists, knees, ankls , shoulders etc…).



Hands & feet.

MCPs &PIPs are early involved (spindle shape swelling of PIPs). , Swelling of wrists.

Progression of the disease will lead wasting & atrophy of small muscles , weakening of capsule & other supportive tissue & joint destruction result in limitation of joint motion , instability, sublaxation & deformities.

Hand deformities.

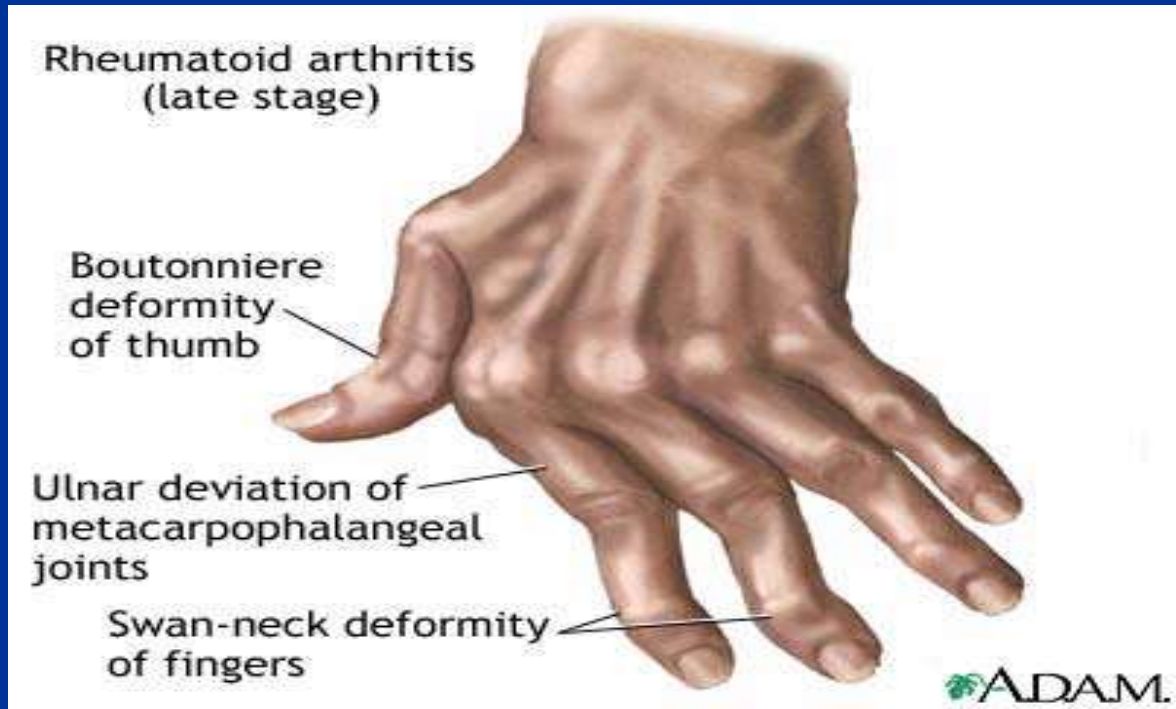
Radial deviation at wrist.

Ulnar deviation at MCP joints.

Swan neck deformity (hperextension at PIPs).

Boutonnier deformity(flexion at PIPS)

Feet deformities are more or less are like those occur in hands.



The knees.

1.Synovial effusion

2.Deformities(valgus., varus &/ or flexion).

3.Bakers cyst in popliteal fossa which may repture.(sudden onset of pain & swelling in calf and ankle) diagnosed by US. & arthrogram.

Presentations.

RA is commonly affecting small joints of hands & feet.

It could be presented as acute polyathritis (15%).

Involvement of large joints.

Monoarticular is not uncommon.

Palindromic(episodic).

Soft tissue involvement.

Prodromal systemic symptoms.

With the progression of the disease get(destructive changes, deformities, symptoms of mechanical effects as pressure on the nerves(CTS)).

Criteria for the diagnosis of RA.

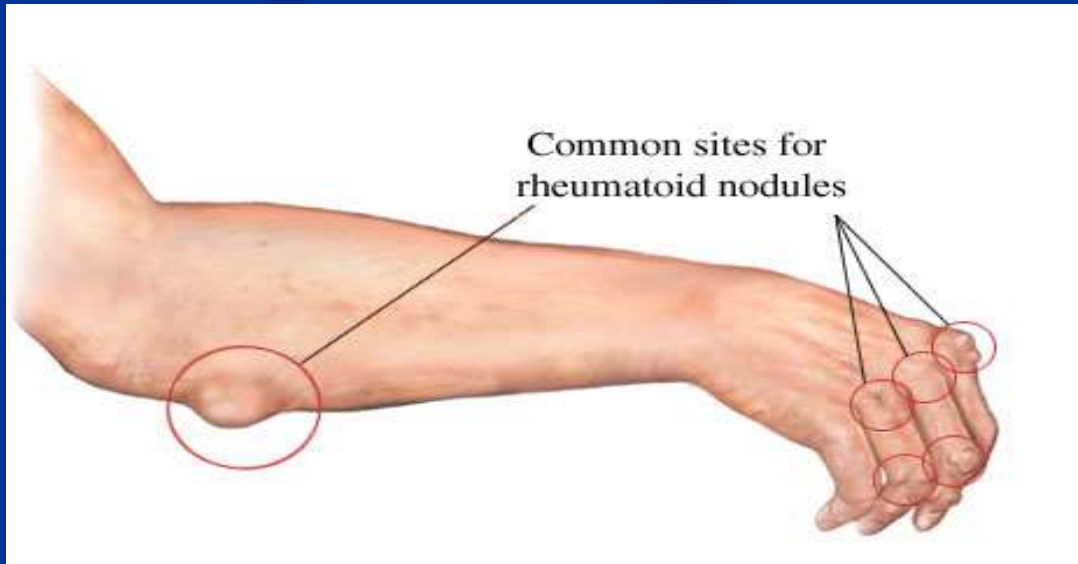
1. Morning stiffness for> one hour.
2. Swelling of 3 or more joints(arthritis).
3. Swelling of hand joints (PIPs , MCPs & wrist).
4. Symmetrical polyarthritis.
5. Subcutaneous nodules.
6. Serum rheumatoid factor.
7. Radiological changes (erosion).

Duration of (1,2,3,4) for 6 weeks or more.

Extra-articular manifestations.

A. In soft tissue surrounding joints.

1. Rheumatoid nodules.(found in 20—30%) , mostly in seropositsve . Are subcutaneous, not attached to skin or underlying tissue, mobile , not tender & located on extensor surface of elbow , forearm, wrist , occiput& achills tendon. Might be found in chest , heart or eye.



2. Bursitis. Olecranon or other bursae.
3. Tenosynovitis. (flexor tendons in palm—Trigger finger).
4. Muscle wasting. As in small muscles of hands.
5. Cyst & repture joints.(baker s cyst).