UNIVERSITY OF LATVIA
FACULTY OF MEDICINE

Diploma work

Differential Diagnosis Of Rheumatoid Arthritis And Osteoarthritis In Different Age Groups

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I dedicate this diploma work to my loving family and to my friends for being a constant source of encouragement and support, my thesis owes much to them.

Mhd Adnan Alsawaf

List of abbreviations

- **OA:** Osteoarthritis
- **RA:** Rheumatoid arthritis
- **WBC:** White blood cell count
- **Hg:** Hemoglobin
- **RF:** Rheumatoid factor
- **CRP:** C-reactive protein
- **ALAT:** Alanine-Amino transferase
- **ASAT**: Aspartate Amino transferase
- **ESR**: Erythrocyte sedimentation rate
- **HLA:** human leukocyte antigen
- **PIP:** Proximal interphalangeal joints
- **MCP:** Metacarpophalangeal joints
- **MTP:** Metatarsophalangeal joints.
- **DIP:** Distal interphalangeal joints
- **SC :** subcutaneous
- **IM :** intramuscular
- **DMARDs:** disease-modifying anti-rheumatic drugs
- **NSAIDs:** Nonsteroidal Anti-Inflammatory Drugs
- **COX:** Cyclooxygenase
- **Yrs:** Years

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Abstract

Arthritis is one of the most prevalent chronic health conditions and the most common cause of physical limitation

The most common forms of arthritis are osteoarthritis (OA) and rheumatoid arthritis (RA). OA, also called degenerative joint disease, is most common in people over 30, and the incidence increases with age. The joints most commonly involved are the hips, knees, hands and spine. The hallmark of OA is the breakdown of the joint cartilage, which cushions the ends of bones. People with OA may experience joint pain and swelling and, frequently, limitation of motion. Risk factors for the development of OA include previous trauma to the joint, obesity and genetic factors.

This common form of arthritis strikes 121 of 100,000 people between the ages of 18 and 79 whereas the next most common form of arthritis, rheumatoid arthritis, affects 9 in 1,000 persons.

RA is a systemic disease that affects 1-2 percent of the world's population and is three times more common in women than men. [6]. Prevalence increases with age, approaching 5% in women over age 55. The average annual incidence is about 70 per 100,000 annually [4]

The peak incidence occurs in the third to sixth decades of life. While the cause of RA is unknown, genetic factors appear to play a role in both susceptibility to and severity of RA in some groups of patients.

One of the earliest symptoms of RA is joint stiffness, which is most pronounced in the morning and usually gets better with movement. Patients experience joint pain, swelling, redness and warmth, which are frequently disabling. In severe cases, internal organs such as the lungs, heart and nerves may be involved. In recent years, research in inflammation has helped scientists begin to unravel the mysteries of RA and has led to the development of targeted therapies. [6]

So as these diseases are chronic and can cause sever pain and disability the early diagnosis is important so we can begin treatment that can help to relieve pain, improve mobility, and minimize disability.

So it is important that the new doctors, general practitioners, family doctors have the essential knowledge to diagnose these diseases early and prevent further complications and for that reason we need to study the simple tests that are essential for diagnosis of such illness and the differences between these two diseases RA & OA.

Aim of the study is searching and analyzing the simple diagnostic tests for OA and RA patients admitted to rheumatology department in PAULA STRADINA Hospital in order to define the differences between these two illnesses in different age groups.

In order to achieve this aim the following tasks were formulated:

- Review the literature about clinical presentation of RA and OA.
- Data collection using and processing the data scheme from the archive for different age groups (under 45 and over 45 years old) using the

relevant statistical software.

- Present the results, to analyze and compare the results with the international guidelines;
- Conclusions and recommendations for the daily practice.

Kopsavilkums

Artrīts ir viens no visizplatītākajiem hroniskajiem veselības stāvokļiem un vissastopamākais fiziskā ierobežojuma cēlonis.

Vissastopamākās artrīta formas ir osteoartritis (OA) un rheumatoid artrīts (RA). AO ir deģeneratīva locītavu slimība, kura biežāk ir novērojama cilvēkiem virs 30 gadiem un kuras sastopamība palielinās ar vecumu. Gurni, ceļi, rokas un mugurkaula stabs ir visbiežāk iesaistītas locītavas. Raksturīga AO iezīme ir locītavu skrimšļa sabrukums, kurš kā spilvens pasargā kaulu galus. Cilvēki, kas slimo ar OA jūt sāpes locītavās un pietūkumu un bieži arī kustību ierobežojumus. OA attīstības riska faktors ir iepriekšējas locītavu traumas, aptaukošanās un ģenētiskie faktori.

Šīs izplatītākās artrīta formas ir novērotas 121 cilvēkam no 100,000, kuri ir no 18 līdz 79 gadiem veci, turpretim cita visizplatītākā artrīta forma, rheumatoid artritis, ietekmē 9 cilvēkus no 1,000.

RA ir sistemātiska slimība, kura ietekmē par 1-2% no visiem pasaules iedzīvotājiem un trīsreiz vairāk sastopama sievietēm nekā vīriešiem. Izplatība pieaug ar vecumu, tuvojoties 5% no sievietēm, kurām ir pāri 55 gadi. Vidēji katru gadu ir apmēram 70 gadījumi no 100,000.

Augstākā pakāpe gadījumu sasniedz trešo līdz sesto dzīves desmitgadi. Tai

laikā kad RA cēlonis ir nezināms, parādās ģenētiskais faktors, kurš spēle lomu gan RA uzņēmībā gan stingrībā dažās pacientu grupās.

Viens no pašiem agrākajiem RA simptomiem – locītavu stīvums, kas skaidri parādās no rīta un parasti atkopjas ar kustību. Pacienti jūt sāpes locītavās, parādās pietūkums, sārtums un siltums, kas bieži bloķē. Smagos gadījumos iespējams, ka tiks iesaistīti arī iekšējie orgāni - plaušas, sirds un nervi. Pēdējos gados pētījums par iekaisumu palīdzēja zinātniekiem sākt atminēt RA mistērijas un aizveda pie mērķtiecīgas terapiju attīstības.

Tā kā šī slimība ir hroniska un var izsaukt pārrautas sāpes un nespēju, agra diagnoze ir svarīga, jo tad mēs varēsim sākt ārstēšanu, kura palīdzēs atvieglināt sāpi, uzlabot kustīgumu un mazināt nespēju.

Līdz ar to ir svarīgi, kad jauniem ārstiem, praktizējošiem ārstiem, ģimenes ārstiem ir būtiskās zināšanas, lai agri diagnosticētu slimības un novērstu tālākus sarežģījumus un tas ar ir cēlonis tam, lai mēs studētu vienkāršu izmeklēšanu, lai diagnosticētu šo saslimstību un atšķirību starp šo divu slimību RA un OA. Studēšanas mērķis ir meklēt un analizēt vienkāršu RA un OA diagnostikas izmeklēšanu pacientiem, kuri ir hospitalizēti reimatoloģijas nodaļā Paula Stradiņa slimnīcā tādēļ, lai noteiktu atšķirības starp divām slimībām atšķirīgās vecuma grupās.
Tādēļ, lai sasniegtu šo mērķi, tika noformulēti sekojoši uzdevumi:

- Aplūkot literatūru par RA un OA klīnisku uzrādīšanu.
- Datu izlase, izmantojot un apstrādājot datu shēmu no arhīva par atšķirīgām vecuma grupām (zem 45 un pāri 45 gadiem), izmantojot saistītu ar ārstēšanu statistisku programmas nodrošinājumu.
- Prezentēt rezultātus, lai analizētu un salīdzinātu rezultātus ar starptautiskajām vadlīnijām

Slēdzieni un rekomendācijas ikdienas praksei..

Chapter I
LITERATURE REVIEW

- **Rheumatoid Arthritis**

Rheumatoid arthritis is a chronic progressive inflammatory polyarthritis often leading to joint destruction, deformity and loss of function, Symmetric swelling of peripheral joints is the hallmark of the disease. Extra-articular features and systemic symptoms can commonly occur and may antedate the onset of joint symptoms. Chronic pain, disability and excess mortality are unfortunate sequelae.

- **Epidemiology**

Rheumatoid arthritis has a worldwide distribution with an estimated prevalence of 1 to 2%. Prevalence increases with age, approaching 5% in women over age 55. The average annual incidence in the United States is about 70 per 100,000 annually. Both incidence and prevalence of rheumatoid arthritis are two to three times greater in women than in men. Although rheumatoid arthritis may present at any age, patients most commonly are first affected in the third decade.

- **Etiology and Pathophysiology**

The etiology of rheumatoid arthritis is not fully understood. Evidence points to a complex interplay between environmental and genetic factors. In monozygotic twins, there is a more than 30 percent concordance rate for rheumatoid arthritis development, and 80 percent of whites with rheumatoid arthritis express the HLA-DR1 or -DR4 subtypes. These and other regions of the Major Histocompatibility Complex may confer susceptibility to more severe disease by causing a specific arthrogenic peptide to be presented to $CD4^+$ T cells.[4]

Joint damage in rheumatoid arthritis begins with the proliferation of synovial macrophages and fibroblasts after a triggering incident, possibly autoimmune or infectious. Lymphocytes infiltrate perivascular regions, and endothelial cells proliferate. Neovascularization then occurs. Blood vessels in the affected joint become occluded with small clots or inflammatory cells. Over time, inflamed synovial tissue begins to grow irregularly, forming invasive pannus tissue. Pannus invades and destroys cartilage and bone. Multiple cytokines, interleukins, proteinases, and growth factors are released, causing further joint destruction and the development of systemic complications.

- **Clinical feature**

The typical case of rheumatoid arthritis begins insidiously, with the slow development of signs and symptoms over weeks to months. Often the patient first notices stiffness .in one or more joints, usually accompanied by pain on movement and by tenderness in the joint. The number of joints involved is

highly variable, but almost always the process is eventually polyarticular, involving five or more joints. Rheumatoid arthritis is an additive polyarthritis, with the sequential addition of involved joints, in contrast to the migratory or evanescent arthritis of systemic lupus erythematosus or the episodic arthritis of gout. Occasionally, patients experience an explosive polyarticular onset occurring over 24 to 48 hours.

The joints involved most often are the proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints of the hands, the wrists (particularly at the ulnar-styloid articulation), shoulders, elbows, knees, ankles, and metatarsophalangeal (MTP) joints. The distal interphalangeal (DIP) joints are generally spared. The spine except the atlanto-axial articulation in late disease is never affected.

Morning stiffness, persisting over 45 min to several hours, may be a feature of any inflammatory arthritis but is especially characteristic of rheumatoid arthritis. Its duration is a useful gauge of the inflammatory activity of the disease. In contrast, patients with degenerative arthritis complain of stiffness lasting no more than a few minutes.

Nonspecific systemic symptoms primarily fatigue, malaise, and depression, may occur. Fever occasionally occurs and is almost always low grade (37° to 38°C) .A higher fever suggests another illness, and infectious causes must be considered.

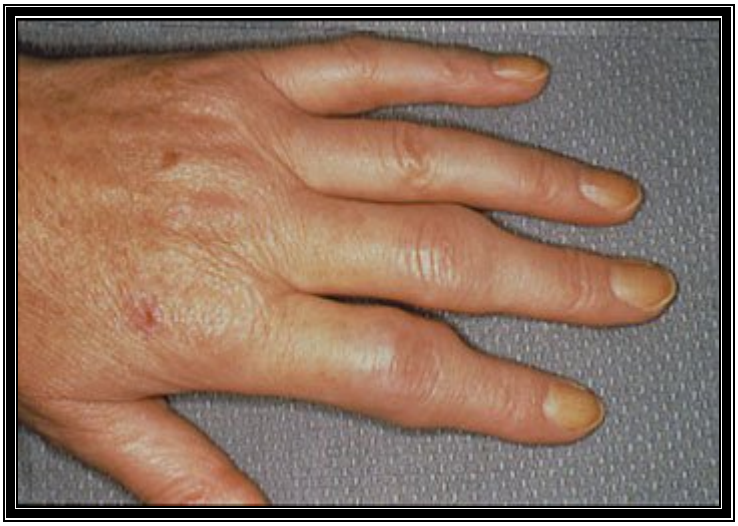
It is typical of patients with rheumatoid arthritis that their symptoms wax and

wane often making diagnosis and treatment decisions difficult. Atypical presentations include intermittent joint inflammation that can be confused with gout or pseudogout, SLE, viral infection, seronegative arthritis and proximal muscle pain and tenderness mimicking polymyalgia rheumatica or diffuse musculoskeletal pain seen in fibromyalgia.

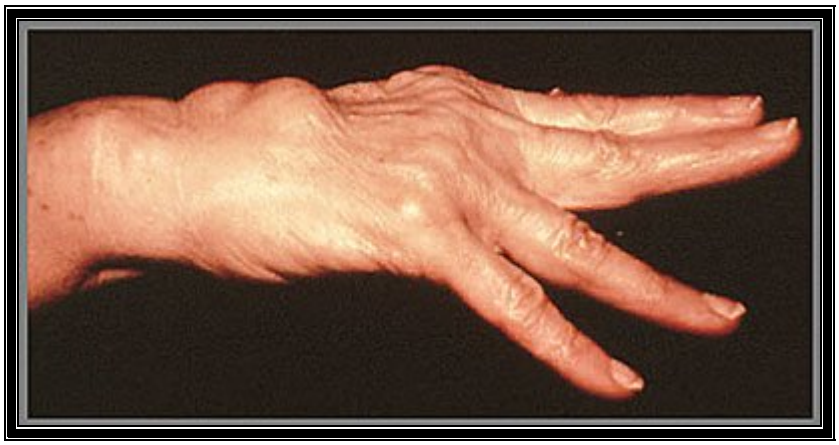
- **Physical Examination**

Symmetric joint swelling, although not invariable, is characteristic of rheumatoid arthritis. Careful palpation of the joints can help to distinguish the swelling of joint inflammation from the bony enlargement seen in osteoarthritis. Fusiform swelling of the PIP joints of the hands is a common early finding. MCP, wrists, elbows, knees, ankles and MTP are other joints commonly affected where swelling is easily detected. In contrast to gout or septic arthritis, redness of affected joints is not a prominent feature of rheumatoid arthritis. Pain on passive motion is the most sensitive test for joint inflammation. Occasionally inflamed joints will feel warm to the touch. Inflammation, structural deformity, or both may limit the range of motion of the joint.

Permanent deformity is an unwanted result of the inflammatory process. Persistent tenosynovitis and synovitis leads to the formation of synovial cysts and to displaced or ruptured tendons. Extensor tendon rupture at the dorsum of the hand is a common and disabling problem.



As evident in the picture below, bony erosions seen at the margins of the joint, at the attachment of the synovium, are the hallmark of rheumatoid arthritis.



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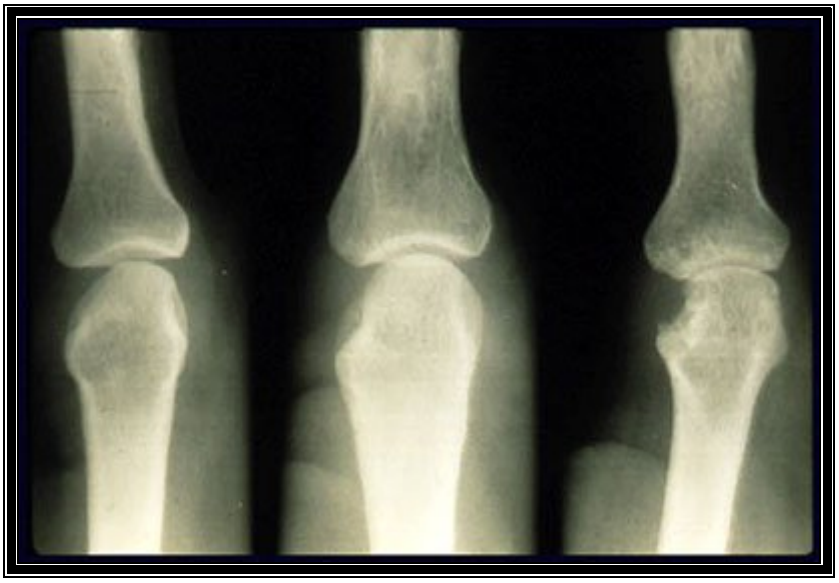
Erosions occur rapidly within the first 2 years of the disease. These anatomic changes result in limitations in range of motion, flexion contractures, and subluxation (incomplete dislocation) of articulating bones.



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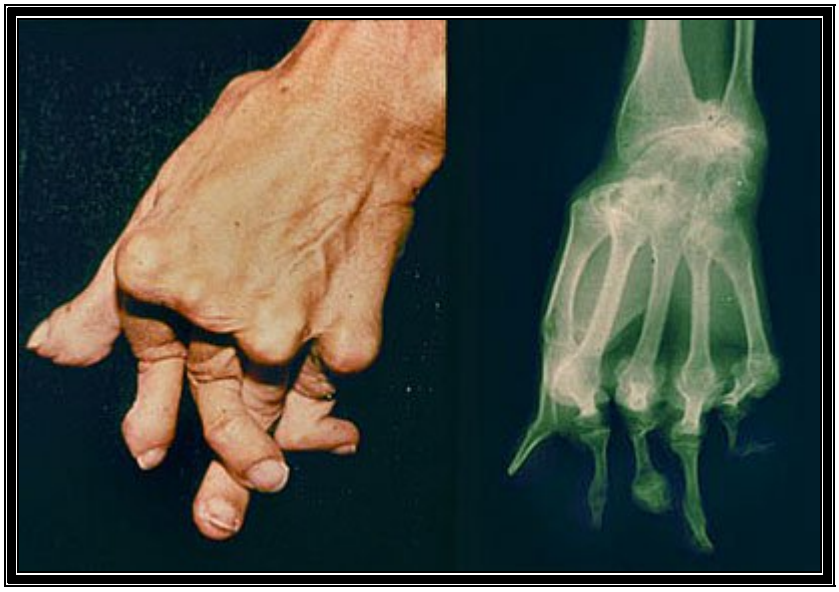
Typical visible changes include ulnar deviation of the fingers at the MCP joints, hyperextension or hyperflexion of the MCP and PIP joints, flexion contractures of the elbows, and subluxation of the carpal bones and toes (cocked —up).



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- **Laboratory Tests**
  - Chemistries
  - Hematology
  - Serology
  - Radiology

Initial Laboratory work-up

- Complete Blood Count
- Platelet count
- CRP
- HLA
- Urinalysis
- ESR.
- Rheumatoid factor
- Anti-nuclear antibody

Chemistries

Chemistries are normal in rheumatoid arthritis with the exception of a slight decrease in albumin and increase in total protein reflecting the chronic inflammatory process. Renal and liver function should be checked prior to instituting therapy.

Hematology

A mild anemia with hematocrit values in the range of 30 — 34% occurs in approximately 25 to 35% of patients with rheumatoid arthritis. In most cases, the reduced red cell mass is caused by the anemia of chronic disease, a normocytic- normochromic process characterized by a normal\or low concentration of serum iron, a low serum iron-binding capacity, and normal or increased serum ferritin concentration. However, occasionally true iron deficiency anemia can develop

secondary to intercurrent blood loss often from gastrointestinal (GI) bleeding due to NSAIDS. The inflammation of rheumatoid arthritis inhibits erythropoiesis, making it difficult to differentiate anemia secondary to chronic blood loss, from the anemia of chronic disease, without an iron stain of the bone marrow. Patients should be monitored closely for symptoms of GI bleeding and consideration must also be given to other causes of GI blood loss such as colonic lesions.

The white cell count is usually normal in patients with rheumatoid arthritis, but can be mildly elevated secondary to inflammation. Similarly, the platelet count is usually normal but thrombocytosis occurs in response to inflammation. Drug reactions and Felty's syndrome are rare causes of leukopenia or thrombocytopenia.

The erythrocyte sedimentation rate (ESR) is usually elevated in patients with rheumatoid arthritis and in some patients is a helpful adjunct in following the activity of the disease.

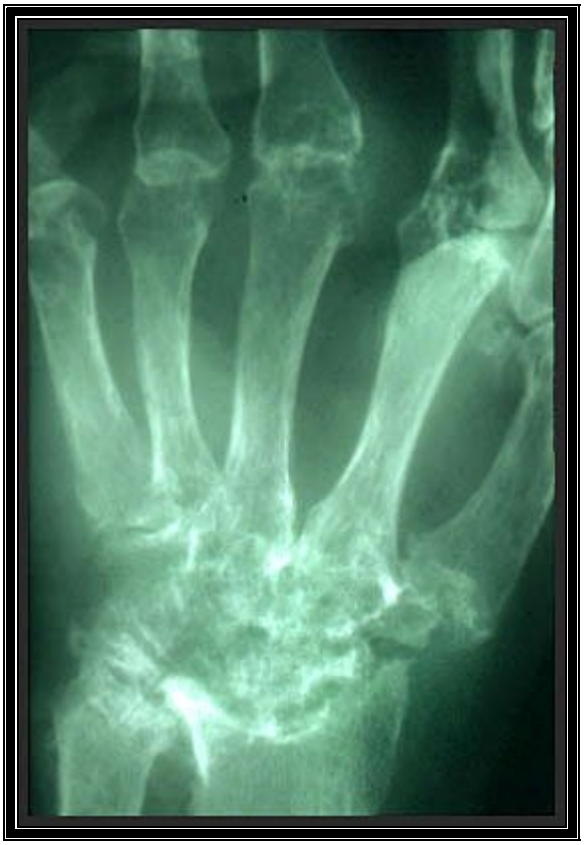
Serology

Rheumatoid factors are antibodies directed against the Fc portion of immunoglobulin G (Ig G). A positive test for rheumatoid factor is by no means pathognomonic of rheumatoid arthritis, but is present in 70 to 90% of patients with the disease. The titer does not correlate with the activity of disease, but patients with a high titer rheumatoid factor are

more likely to have erosive joint disease, extra-articular manifestations, and greater functional disability. In contrast, generally, rheumatoid factor negative patients exhibit a milder disease course. Rheumatoid factors are also detectable in non-rheumatoid patients who have chronic antigenic stimulation, such as prolonged infection (bacterial endocarditis, tuberculosis, cytomegalovirus, human immunodeficiency virus (HIV), collagen vascular disease, or dysproteinemia). Low titers of rheumatoid factors may be detected in the serum of apparently normal people, especially over the age of 70, where its prevalence is anywhere from 10 - 25%. The anti-nuclear antibody (ANA) is positive in 10-20% of patients with rheumatoid arthritis and is more common in patients with extra-articular manifestations.

Radiology

Radiological findings early in the disease may show nothing other than soft tissue swelling. Thereafter, periarticular osteoporosis may develop. With progression of their disease, narrowing of the joint space is caused by loss of cartilage, and juxta-articular marginal erosions appear, generally at the point of attachment of the synovium. In end-stage disease, large cystic erosions of bone may be seen. Bony proliferation may occur because of degenerative changes that follow inflammation. In the picture below, erosive changes at the carpal bones can be seen.



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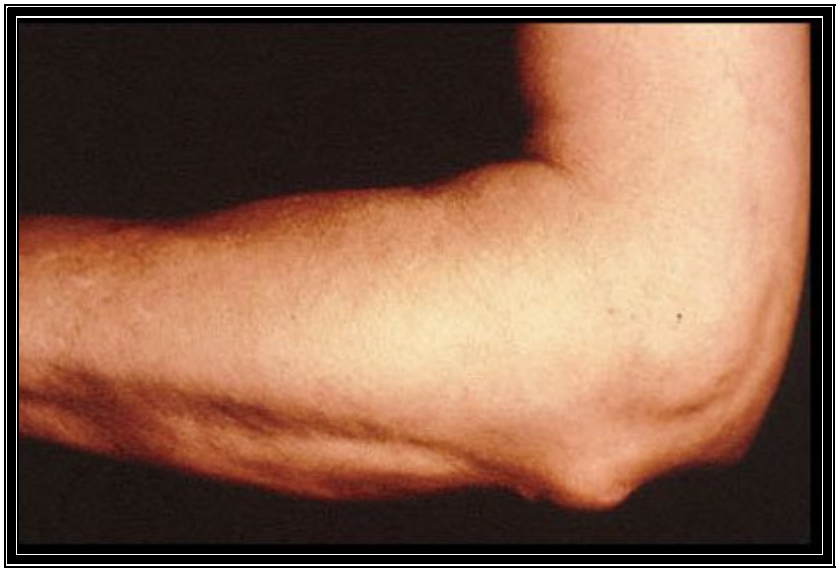
- **Extra-Articular Diseases**
  - Rheumatoid Nodules
  - Cardiopulmonary Diseases
  - Ocular Diseases
  - Neurological Diseases
  - Felty's Syndrome

- Rheumatoid Vasculitis
- Sjogren's Syndrome

Although the joints are almost always the principal focus of the rheumatoid arthritis, other organ systems may also be involved. Extra-articular manifestations of rheumatoid arthritis occur most often in seropositive patients with more severe joint disease. Interestingly, extra-articular manifestations can occur in later stages of the disease when there is little active synovitis ("burnt-out" disease). In contrast to the predilection of rheumatoid arthritis for women, extra-articular manifestations of the disease are more common in men.

Rheumatoid Nodules:

The subcutaneous nodule is the most characteristic extra-articular lesion of the disease. Nodules occur in 20 to 30% of cases, almost exclusively in seropositive patients. They are located most commonly on the extensor surfaces of the arms and elbows, but are also prone to develop at pressure points on the feet and knees.



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Rarely, nodules may arise in visceral organs, such as the lungs, the heart, or the sclera of the eye.

Cardiopulmonary Disease:

There are several pulmonary manifestations of rheumatoid arthritis, including pleurisy with or without effusion, intrapulmonary nodules, rheumatoid pneumoconiosis (Caplan's syndrome), diffuse interstitial fibrosis, and rarely, bronchiolitis obliterans pneumothorax. On pulmonary function testing, there commonly is a restrictive ventilatory defect with reduced lung volumes and a decreased diffusing capacity for carbon monoxide. Although mostly asymptomatic, of greatest concern is

distinguishing these manifestations from infection and tumor. Pericarditis is the most common cardiac manifestation

Ocular Diseases:

Keratoconjunctivitis of Sjogren's syndrome is the most common ocular manifestation of rheumatoid arthritis. Sicca (dry eyes) is a common complaint. Episcleritis occurs occasionally and is manifested by mild pain and intense redness of the affected eye. Cataract Scleritis and scleromalacia, scleromalacia perforans are rare but more serious problems.

Neurological Disease

The most common neurological manifestation of rheumatoid arthritis is mononeuritis and mononeuritis multiplex and mild primarily sensory peripheral neuropathy, usually more marked in the lower extremities. Entrapment neuropathies (e.g., carpal tunnel syndrome and tarsal tunnel syndrome) sometimes occur in patients with rheumatoid arthritis because of compression of a peripheral nerve by inflamed edematous tissue. Cervical myelopathy secondary to atlantoaxial subluxation is an uncommon but particularly worrisome complication potentially causing permanent, even fatal neurologic damage.

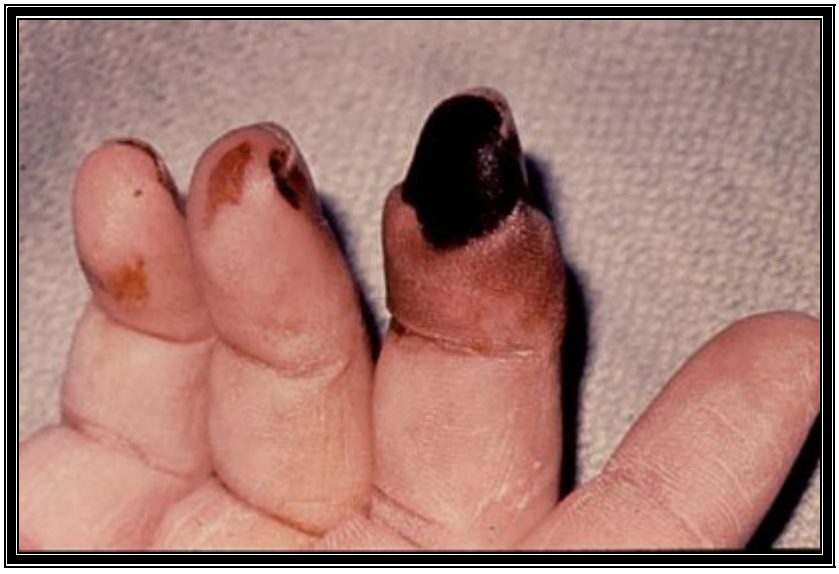
Felty's Syndrome

Felty's syndrome is nowadays a rare complication of rheumatoid arthritis and is characterized by splenomegaly, and leukopenia — predominantly granulocytopenia and lymphoadenopathy. Recurrent

bacterial infections and chronic refractory leg ulcers are the major complications.

Rheumatoid Vasculitis

The most common clinical manifestations of vasculitis are small digital infarcts along the nailbeds. The abrupt onset of an ischemic mononeuropathy (mononeuritis multiplex) or progressive scleritis is typical of rheumatoid vasculitis. The syndrome ordinarily emerges after years of seropositive, persistently active rheumatoid arthritis; however, vasculitis may occur when joints are inactive.



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Sjogren's syndrome:

Approximately 10 to 15% of patients with rheumatoid arthritis, mostly women develop Sjogren's syndrome, a chronic inflammatory disorder characterized by lymphocytic infiltration of lacrimal and salivary glands. This leads to impaired secretion of saliva and tears and results in the sicca complex: dry mouth (xerostomia) and dry eyes (keratoconjunctivitis sicca).

Patients with Sjogren's syndrome have a variable expression of disease in other exocrine glands. This is manifested clinically as dry skin, decreased perspiration, dry vaginal membranes, or a nonproductive cough. Commonly, there is also a polyclonal lymphoproliferative reaction characterized by lymphoadenopathy and splenomegaly. This can mimic and rarely transform into a malignant lymphoma.

Diagnostic criteria:

Sign or symptom	*Definition*
Morning stiffness	Stiffness in or around the affected joints for at least one hour
Arthritis of three or more joint areas	Three or more of the following joints noted to be fluid-filled or have soft tissue swelling: wrist, PIP, MCP, elbow, knee, ankle, MTP
Hand joint involvement	Wrist, MCP, or PIP joints among the symptomatic joints observed
Symmetric arthritis	Right and left joints involved for one or more of following: wrist, PIP, MCP, elbow, knee, ankle, MTP
Rheumatoid nodules	Subcutaneous nodules in regions surrounding joints, extensor surfaces, or bony prominences
Serum rheumatoid factor positive	Positive result using any laboratory test that has a positive predictive value of 95 percent or more (i.e., is positive in no more than 5 percent of patients without rheumatoid arthritis)
Radiographic changes	Hand and wrist films show typical changes of erosions or osteoporosis adjacent to affected joints

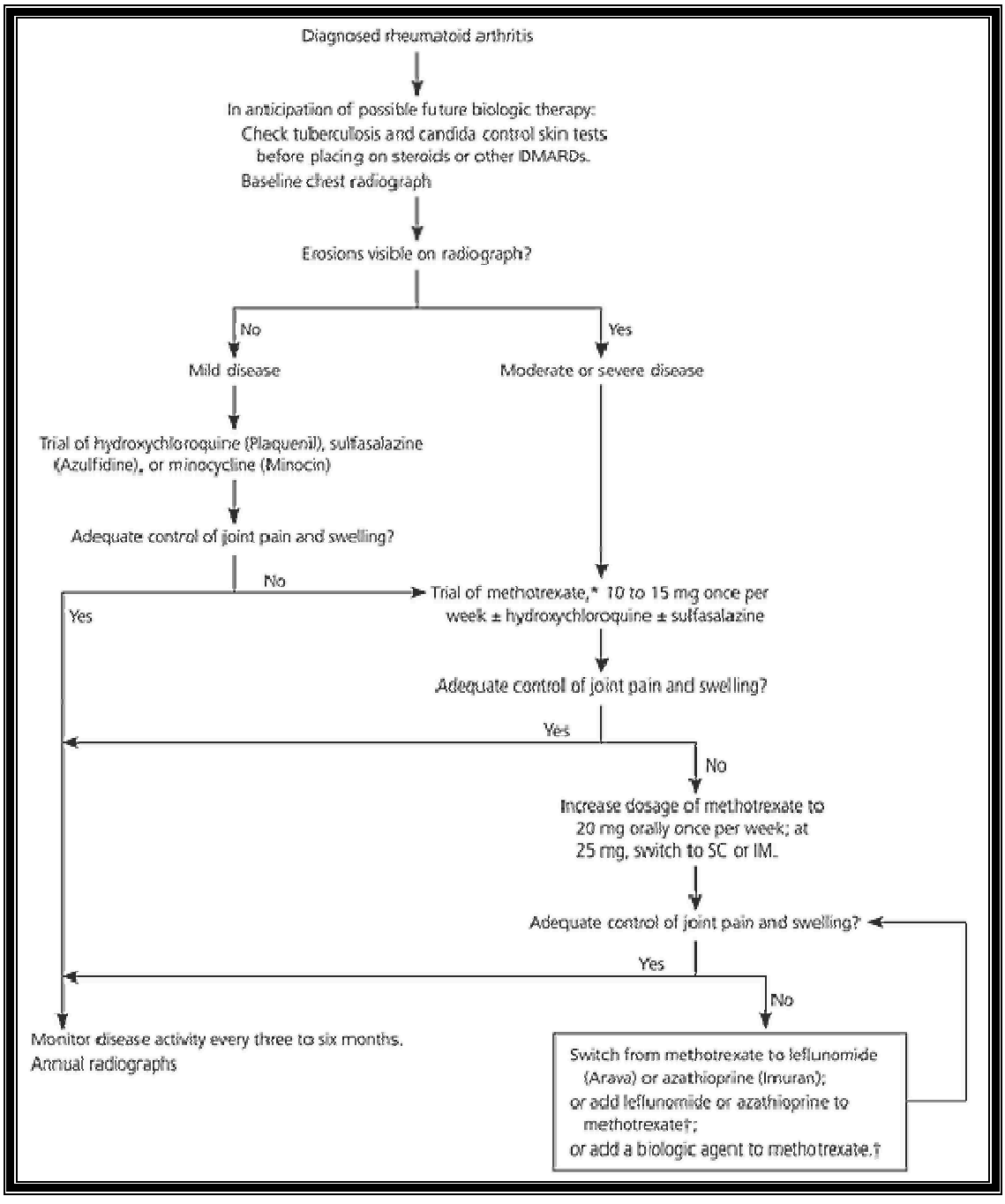
Treatment of RA:

Joint destruction in rheumatoid arthritis begins within the first year of disease onset; early treatment decreases the rate of disease progression.

Therefore, it is imperative to diagnose the disease and initiate treatment as soon as possible.

Therapeutic goals include preservation of function and quality of life, minimization of pain and inflammation, joint protection, and control of systemic complications. And here is an approach to the treatment of a patient with rheumatoid arthritis.

Treatment Approach for a Patient with Rheumatoid Arthritis



Algorithm for treatment of a patient with rheumatoid arthritis.

NSAIDs: Cyclooxygenase-2 inhibitors are used for initial treatment of rheumatoid arthritis to reduce joint pain and swelling. However, because they do not alter the disease course, they should not be used alone.[2] Patients with rheumatoid arthritis are almost two times more likely to have serious complications from NSAID use than patients with osteoarthritis, and they should be observed closely for symptoms of gastrointestinal side effects. Cyclooxygenase-2 inhibitors must be used with caution, given recent findings regarding potential adverse effects.

DMARDs should be considered for all patients with rheumatoid arthritis; DMARDs have a good evidence of beneficial effect.

Patients with mild disease and normal radiographic findings can begin treatment with hydroxychloroquine (Plaquenil), sulfasalazine (Azulfidine), or minocycline (Minocin), although methotrexate also is an option. Patients with more severe disease or radiographic changes should begin treatment with methotrexate. If symptoms are not adequately controlled, leflunomide (Arava) azathioprine (Imuran), or combination therapy (methotrexate plus one of the newer agents) may be considered.

TNF antagonists block the activity of TNF-alpha, which is present in increased concentrations in the synovial fluid in patients with rheumatoid arthritis. Etanercept is a soluble TNF-receptor fusion protein. Its long-term effects are comparable with methotrexate but it

elicits improvement in symptoms much more rapidly, often within two weeks but TNF antagonists are associated with an increased risk of infection, especially tuberculosis reactivation

RITUXAN® (Rituximab) in combination with methotrexate is indicated to reduce signs and symptoms in adult patients with moderately- to severely- active rheumatoid arthritis who have had an inadequate response to one or more TNF antagonist therapies.

Infleximap : given intravenously within 2 to 3 hours 3 mg/ kg of body weight .in the following program zero dose D0 then after 2 weeks , after 6 weeks then 8 weeks thereafter

It should be given with methotrexate.

Anakinra is a recombinant interleukin-1 receptor antagonist. And it has been found to be effective when administered alone or in combination with methotrexate.

Adverse effects include skin irritation at the site of injection, increased risk of infection, and leukopenia.[19]

Glucocorticoids: Steroids at dosages equivalent to less than 7.5 -10 mg of prednisone daily are highly effective for relieving symptoms of rheumatoid arthritis[2] and can slow joint damage. Steroid dosages should be kept at a minimum because of the high risk of side effects, which include osteoporosis, cataracts, cushingoid symptoms, and abnormalities

in blood glucose levels and glucose intolerance

Intra-articular injection of glucocorticoids is a safe and effective intervention; however, the effects are temporary. Infectious arthritis should be ruled out before injections are performed.

Symptoms may recur with steroid discontinuation, especially when high dosages were used, and most rheumatologists withdraw steroids slowly, over a month or more, to avoid rebound effects. Systemic steroids often are used as "bridging therapy" during the period when DMARDs have been initiated but have not yet taken effect

Osteoarthritis

Osteoarthritis is a type of arthritis that is caused by the breakdown and eventual loss of the cartilage of one or more joints. Over time the cartilage may wear away entirely, and the bones will rub together. [8]. Osteoarthritis is also known as degenerative arthritis. Among the over 100 different types of arthritis conditions, osteoarthritis is the most common, affecting over 20 million people in the United States. Osteoarthritis occurs more frequently as we age. Before age 45, osteoarthritis occurs more frequently in males. After age 55 years, it occurs more frequently in females.

Most adults older than 45 years show radiographic evidence of osteoarthritis.

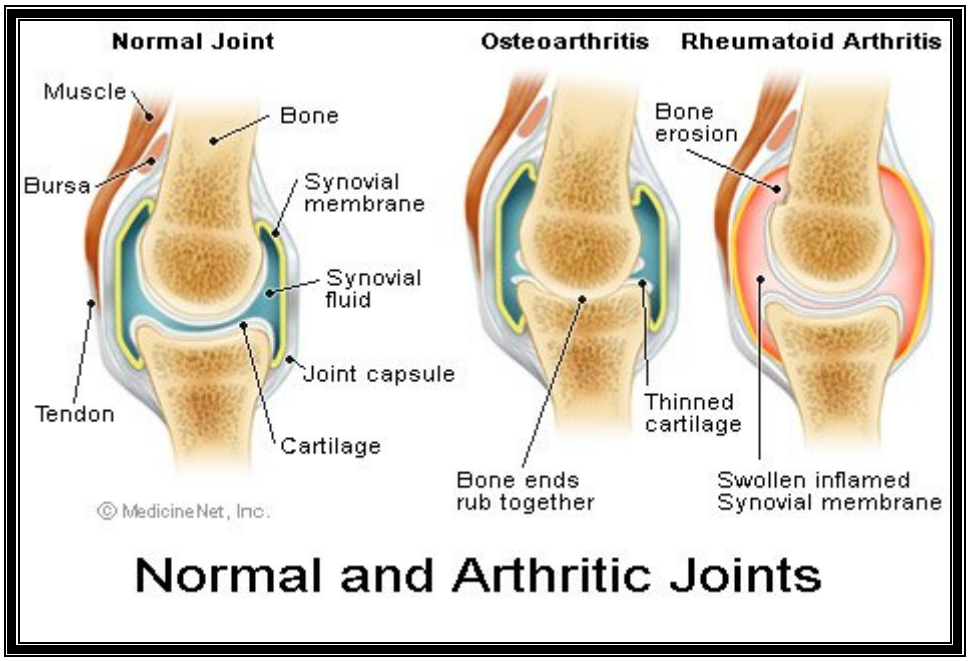
Males develop OA before age 45 years, possibly because of higher incidence of posttraumatic OA. After age 45 years, women are affected more frequently from OA and tend to have more severe disease than men. [7]

In the United States, all races appear equally affected. A higher incidence of osteoarthritis exists in the Japanese population, while South African blacks, East Indians and Southern Chinese have lower rates.

Osteoarthritis commonly affects the large weight-bearing joints, such as the hips and knees and hands, feet, spine,. Most cases of osteoarthritis have no known cause and are referred to as primary osteoarthritis. When the cause of the osteoarthritis is known, the condition is referred to as secondary osteoarthritis.

Causes of osteoarthritis

Primary osteoarthritis is mostly related to aging. With aging, the water content of the cartilage increases and the protein makeup of cartilage degenerates. Repetitive use of the joints over the years irritates and inflames the cartilage, causing joint pain and swelling. Eventually, cartilage begins to degenerate by flaking or forming tiny crevasses. In advanced cases, there is a total loss of the cartilage cushion between the bones of the joints. Loss of cartilage cushion causes friction between the bones, leading to pain and limitation of joint mobility. Inflammation of the cartilage can also stimulate new bone outgrowths (spurs) to form around the joints. Osteoarthritis occasionally can be found in multiple members of the same family, implying an heredity (genetic) basis for this condition.



Secondary osteoarthritis is caused by another disease or condition. Conditions that can lead to secondary osteoarthritis include obesity, repeated trauma or surgery to the joint structures, abnormal joints at birth (congenital abnormalities), gout, diabetes, and other hormone disorders.

Obesity causes osteoarthritis by increasing the mechanical stress on the cartilage. In fact, next to aging, obesity is the most powerful risk factor for osteoarthritis of the knees. The early development of osteoarthritis of the knees among weight lifters is believed to be in part due to their high body weight. Repeated trauma to joint tissues (ligaments, bones and cartilage) is believed to lead to early osteoarthritis of the knees in soccer players. Interestingly, recent studies have not found an increased risk of osteoarthritis in long-distance runners.

Crystal deposits in the cartilage can cause cartilage degeneration, and osteoarthritis. Uric acid crystals cause arthritis in gout, while calcium pyrophosphate crystals cause arthritis in pseudogout.

Some people are born with abnormally formed joints (congenital abnormalities) that are vulnerable to mechanical wear, causing early degeneration and loss of joint cartilage. Osteoarthritis of the hip joints is commonly related to design abnormalities of these joints that had been present since birth.

Hormone disturbances, such as diabetes and growth hormone disorders, are also associated with early cartilage wear and secondary osteoarthritis.

Symptoms of osteoarthritis

Osteoarthritis is a disease of the joints. Unlike many other forms of arthritis that are systemic illnesses, such as rheumatoid arthritis and systemic lupus, osteoarthritis does not affect other organs of the body. The most common symptom of osteoarthritis is pain in the affected joint(s) after repetitive use. Joint pain is usually worse later in the day. There can be swelling, warmth, and creaking of the affected joints. Pain and stiffness of the joints can also occur after long periods of inactivity, for example, sitting in a theater. In severe osteoarthritis, complete loss of cartilage cushion causes friction between bones, causing pain at rest or pain with limited motion.

Symptoms of osteoarthritis vary greatly from patient to patient. Some patients can be debilitated by their symptoms. On the other hand, others may have remarkably few symptoms in spite of dramatic degeneration of the joints apparent on x-rays. Symptoms also can be intermittent. It is not unusual for patients with osteoarthritis of the hands and knees to have years of pain-free intervals between symptoms.[9]

Osteoarthritis of the knees is often associated with obesity or a history of repeated injury and /or joint surgery. Progressive cartilage degeneration of the knee joints can lead to deformity and outward curvature of the knees referred to as "bow legged." Patients with osteoarthritis of the weight bearing joints (like the knees) can develop a limp. The limping can worsen as more cartilage degenerates. In some patients, the pain, limping, and joint dysfunction may not respond to medications or other conservative measures. Therefore, severe osteoarthritis of the knees is one of the most common reasons for total knee replacement surgical procedures in the United States.

Osteoarthritis of the spine causes pain in the neck or low back. Bony spurs that form along the arthritic spine can irritate spinal nerves, causing severe pain, numbness, and tingling of the affected parts of the body.

Osteoarthritis causes the formation of hard bony enlargements of the small joints of the fingers. Classic bony enlargement of the small joint at the end of the fingers is called a Heber den's node, named after a very famous British doctor. The bony deformity is a result of the bone spurs from the osteoarthritis in that joint. Another common bony knob (node) occurs at the middle joint of

the fingers in many patients with osteoarthritis and is called a Bouchard's node. Dr. Bouchard was a famous French doctor who also studied arthritis patients in the late 1800s. The Heberden's and Bouchard's nodes may not be painful, but they are often associated with limitation of motion of the joint. The characteristic appearances of these finger nodes can be helpful in diagnosing osteoarthritis. Osteoarthritis of the joint at the base of the big toes leads to the formation of a bunion. Osteoarthritis of the fingers and the toes may have a genetic basis, and can be found in numerous women members of some families

Diagnosis of osteoarthritis

Here is no blood test for the diagnosis of osteoarthritis. Blood tests are performed to exclude diseases that can cause secondary osteoarthritis, as well as to exclude other arthritis conditions that can mimic osteoarthritis.

X-rays of the affected joints can suggest osteoarthritis. The common x-ray findings of osteoarthritis include loss of joint cartilage, narrowing of the joint space between adjacent bones, and bone spur formation. Simple x-ray testing can be very helpful to exclude other causes of pain in a particular joint as well as assist the decision-making as to when surgical intervention should be considered.

Arthrocentesis is often performed in the doctor's office. During arthrocentesis, a sterile needle is used to remove joint fluid for analysis. Joint fluid analysis is useful in excluding gout, infection, and other causes of arthritis. Removal of joint fluid and injection of corticosteroids into the joints during arthrocentesis

can help relieve pain, swelling, and inflammation.

Arthroscopy is a surgical technique whereby a doctor inserts a viewing tube into the joint space. Abnormalities of and damage to the cartilage and ligaments can be detected and sometimes repaired through the arthroscope. If successful, patients can recover from the arthroscopic surgery much more quickly than from open joint surgery.

Finally, a careful analysis of the location, duration, and character of the joint symptoms and the appearance of the joints helps the doctor in diagnosing osteoarthritis. Bony enlargement of the joints from spur formations is characteristic of osteoarthritis. Therefore, Heberden's nodes, Bouchard's nodes, and bunions of the feet can help the doctor make a diagnosis of osteoarthritis.

Management of osteoarthritis

Aside from weight reduction and avoiding activities that exert excessive stress on the joint cartilage, there is no specific treatment to halt cartilage degeneration or to repair damaged cartilage in osteoarthritis. The goal of treatment in osteoarthritis is to reduce joint pain and inflammation while improving and maintaining joint function. Some patients with osteoarthritis have minimal or no pain, and may not need treatment. Others may benefit from conservative measures such as rest, exercise, weight reduction, physical and occupational therapy, and mechanical support devices. These measures are

particularly important when large, weight-bearing joints are involved, such as the hips or knees. In fact, even modest weight reduction can help to decrease symptoms of osteoarthritis of the large joints, such as the knees and hips. Medications are used to complement the physical measures described above. Medication may be used topically, taken orally, or injected into the joints to decrease joint inflammation and pain. When conservative measures fail to control pain and improve joint function, surgery can be considered.

Resting sore joints decreases stress on the joints, and relieves pain and swelling. Patients are asked to simply decrease the intensity and/or frequency of the activities that consistently cause joint pain.

Exercise usually does not aggravate osteoarthritis when performed at levels that do not cause joint pain. Exercise is helpful in osteoarthritis in several ways. First, it strengthens the muscular support around the joints. It also prevents the joints from "freezing up" and improves and maintains joint mobility. Finally, it helps with weight reduction and promotes endurance. Applying local heat before and cold packs after exercise can help relieve pain and inflammation. Swimming is particularly suited for patients with osteoarthritis because it allows patients to exercise with minimal impact stress to the joints. Other popular exercises include walking, stationary cycling, and light weight training.

Physical therapists can provide support devices, such as splints, canes, walkers, and braces. These devices can be helpful in reducing stress on the joints. Occupational therapists can assess daily activities and determine additional devices that may help patients at work or home. Finger splints can support

individual joints of the fingers. Paraffin wax dips, warm water soaks, and nighttime cotton gloves can help ease hand symptoms. Spine symptoms can improve with a neck collar, lumbar corset, or a firm mattress, depending on what areas are involved.

In many patients with osteoarthritis, mild pain relievers such as aspirin and acetaminophen (Tylenol) may be sufficient treatment. Studies have shown that acetaminophen given in adequate doses can often be equally as effective as prescription anti-inflammatory medications in relieving pain in osteoarthritis of the knees. Since acetaminophen has fewer gastrointestinal side effects than NSAIDS, especially among the elderly patients, acetaminophen is generally the preferred initial drug given to patients with osteoarthritis. Medicine to relax muscles in spasm might also be given temporarily. Pain-relieving creams applied to the skin over the joints can provide relief of minor arthritis pain. Examples include capsaicin (Arthricare, Zostrix), salycin (Aspercreme), methyl salicylate (Bengay, Icy Hot), and menthol (Flexall).

Nonsteroidal anti-inflammatory drugs (NSAIDs) are medications that are used to reduce pain and inflammation in the joints. Examples of NSAIDs include aspirin (Ecotrin), ibuprofen (Motrin), nabumetone (Relafen), and naproxen (Naprosyn). It is sometimes possible to use NSAIDs for a while and then discontinue them for periods of time without recurrent symptoms, thereby decreasing side effect risks.

The most common side effects of NSAIDs involve gastrointestinal distress, such as stomach upset, cramping diarrhea, ulcer and even bleeding. The risk of

these and other side effects increases in the elderly. Newer NSAIDs called Cox-2 Inhibitors have been designed that have less toxicity to the stomach and bowels. Because osteoarthritis symptoms vary and can be intermittent, these medicines might be given only when joint pains occur or prior to activities that have traditionally brought on symptoms.

Recently, the food supplements glucosamine and chondroitin have been shown to relieve symptoms of pain and stiffness for some persons with osteoarthritis. Fish oil supplements have been shown to have some anti-inflammation properties and increasing the dietary fish intake and/or fish oil capsules (omega 3 capsules) can sometimes reduce inflammation of arthritis.

While oral cortisone is generally not used in treating osteoarthritis, when injected directly into the inflamed joints, it can rapidly decrease pain and restore function. Since repetitive cortisone injections can be harmful to the tissue and bones, they are reserved for patients with more pronounced symptoms.

For persisting pain of osteoarthritis of the knee that does not respond to weight reduction, exercise or medications, a series of injections of hyaluronic acid (Synvisc, Hyalgan) into the joint can sometimes be helpful, especially if surgery is not being considered. These products seem to work by temporarily restoring the thickness of the joint fluid, allowing better joint lubrication and impact capability, and perhaps by directly affecting pain receptors.

Surgery is generally reserved for those patients with osteoarthritis that is

particularly severe and unresponsive to the conservative treatments. Arthroscopy, discussed above, can be helpful when cartilage tears are suspected. Osteotomy is a bone removal procedure that can help realign some of the deformity in selected patients, usually those with knee disease. In some cases, severely degenerated joints are best treated by replacement with an artificial joint (arthroplasty). Total hip and total knee replacements are now commonly performed in community hospitals throughout the world. These can bring dramatic pain relief and improved function. For further information on joint surgeries,

An extensive literature review has been performed to summarize existing knowledge concerning OA and RA as basis and guide for this survey

CHAPTER II
MATERIALS AND METHODS

In this study we look for the differences in clinical and Para clinical features between RA and OA in patients less than 45 years old and over 45 year.

I collected the data from the archive of Paula Stradina hospital for patients with diagnosis RA and OA in 2005, the study included 40 patients (28 females and 12 males) divided into two groups 20 of them with RA and 20 with OA.

Each group was divided into two sub-groups according to the age; ten patients were over 45 and ten under 45 years old.
The following data were looked at:

- WBC
- Hg
- ALAT
- ASAT
- Creatinine
- Glucose
- Uric acid
- CRP
- ESR

- RF
- Arthritis (Mono-\ Oligo-\ Poly).
- Morning stiffness
- Weight loss
- X-Ray results.
- Medication

Then I gave numeric code of the data collected which ranged from (0-2), e.g. 0 means the patient has WBC count below 4 while 1 means he has WBC count between 4-11 and 2 means over 11.

Data analysis was performed with statistical software package SPSS 14.0

Chapter III
RESULTS

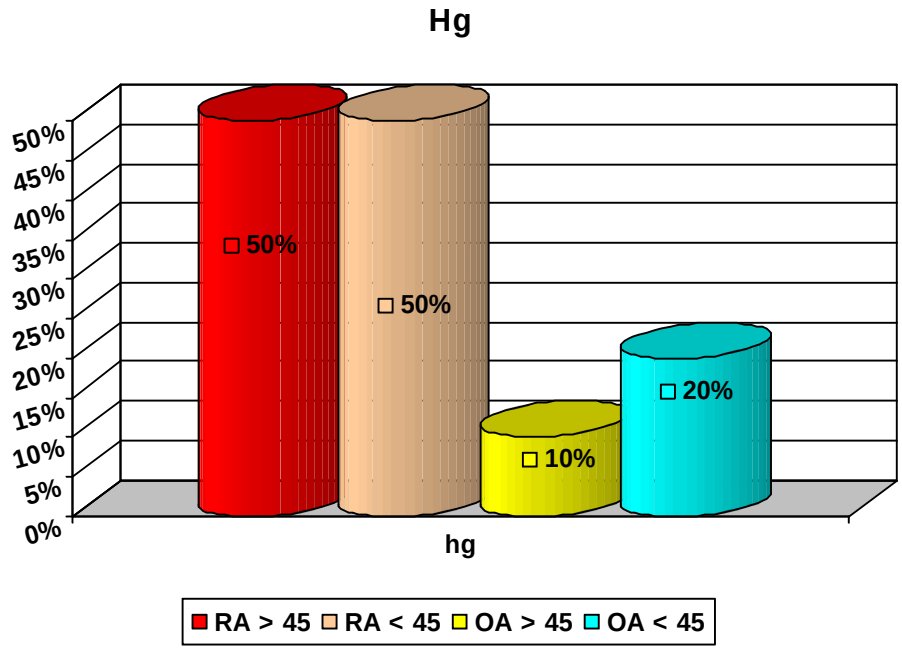
Laboratory results:

WBC

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
WBC	Decreased	0%	0%	0%	20%	5%
	within normal range	80%	100%	100%	80%	90%
	Elevated	20%	0%	0%	0%	5%
Total		100%	100%	100%	100%	100%

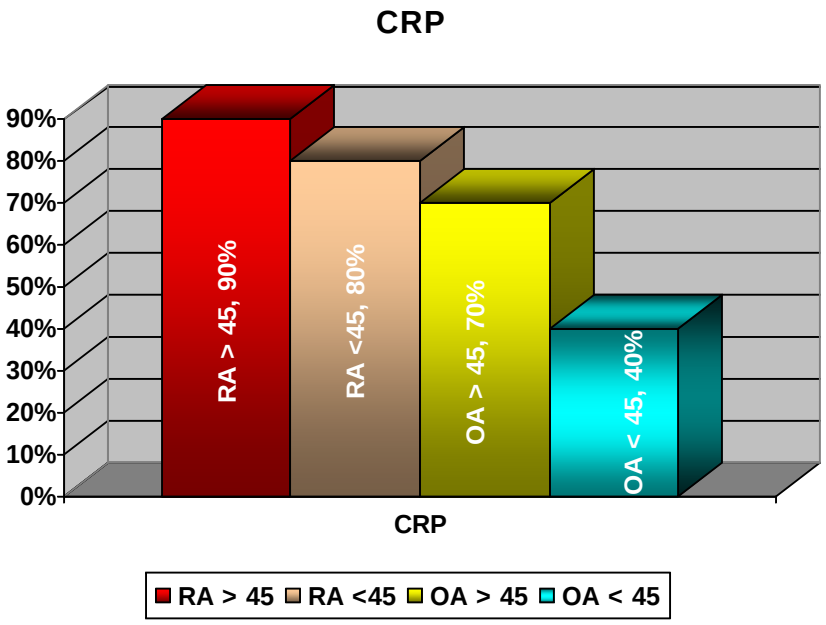
Hg

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
Hg	Decreased	50%	50%	10%	20%	32.5%
	within normal range	50%	50%	90%	80%	67.5%
Total		100%	100%	100%	100%	100%



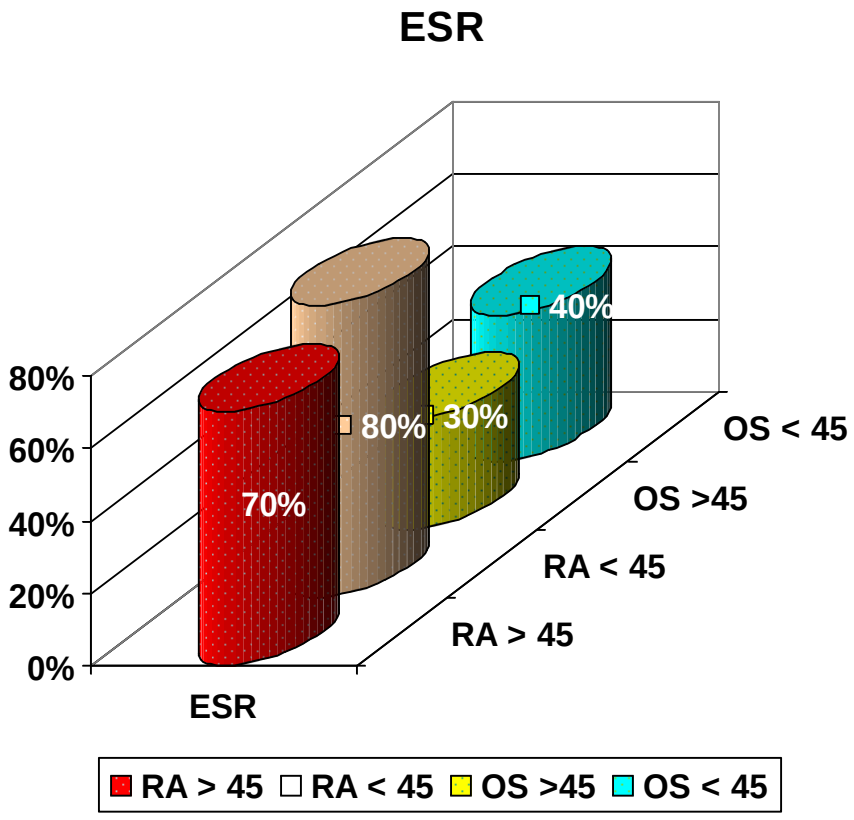
CRP

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
CRP	within normal range	10%	20%	30%	60%	30%
	elevated	90%	80%	70%	40%	70%
Total		100%	100%	100%	100%	100%



ESR

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
ESR	within normal range	30%	20%	70%	60%	45%
	Elevated	70%	80%	30%	40%	55%
Total		100%	100%	100%	100%	100%



ALAT

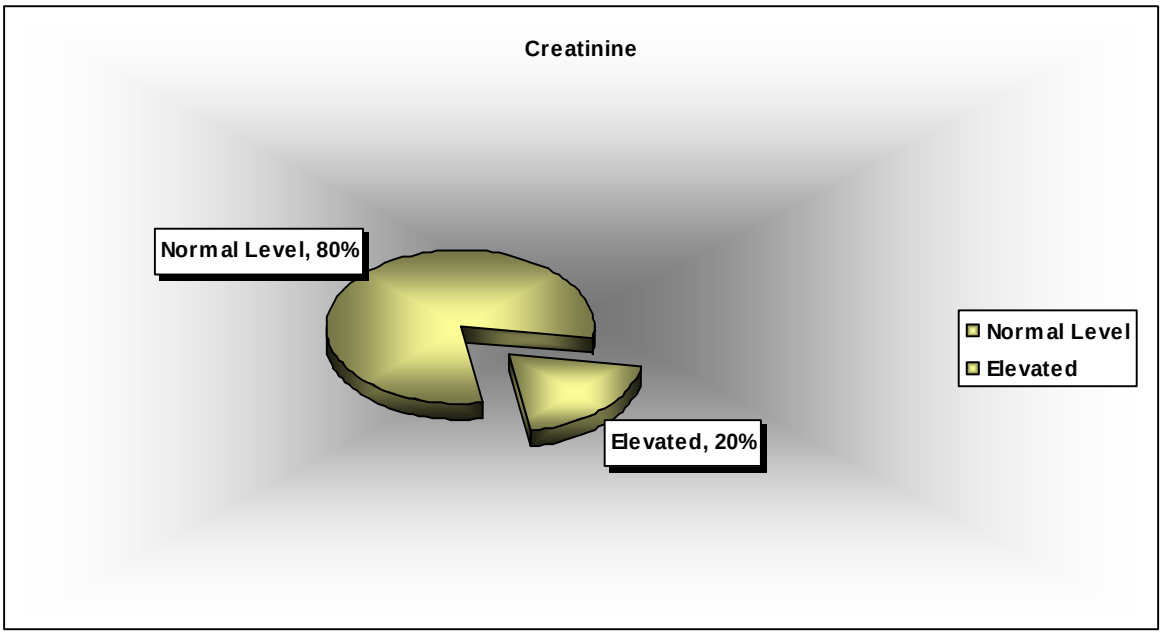
		Patients				Total
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	
ALAT	within normal range	90%	100%	90%	100%	95%
	elevated	10%	0%	10%	0%	5%
Total		100%	100%	100%	100%	100%

ASAT

		Patients				Total
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	
ASAT	within normal range	90%	100%	80%	100%	92.5%
	elevated	10%	0%	20%	0%	7.5%
Total		100%	100%	100%	100%	100%

Creatinine

		Patients				Total
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	
Creatinine	within normal range	80%	80%	100%	100%	90%
	Elevated	20%	20%	0%	0%	10%
Total		100%	100%	100%	100%	100%



Creatinine level in all RA patients

Glucose

		Patients				Total
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	
Glucose	Decreased	0%	20%	0%	0%	5%
	within normal range	70%	80%	80%	90%	80%
	Elevated	30%	0%	20%	10%	15%
Total		100%	100%	100%	100%	100%

Uric Acid

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
Uric Acid	decreased	0%	0%	0%	10%	2.5%
	within normal range	90%	100%	70%	70%	82.5%
	elevated	10%	0%	30%	20%	15%
Total		100%	100%	100%	100%	100%

Polyarthritis

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
Polyarthritis	Oligoarthritis	0%	0%	40%	50%	22.5%
	Polyarthritis	100%	100%	60%	50%	77.5%
Total		100%	100%	100%	100%	100%

Stiffness

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
Stiffness	No stiffness	0%	30%	60%	30%	30%
	15 - 30 min	10%	0%	40%	40%	22.5%
	more than 1 h	90%	70%	0%	30%	47.5%
Total		100%	100%	100%	100%	100%

RF

		Patients				Total
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	
RF	within normal ranges	10%	40%	80%	70%	50%
	positive	90%	60%	20%	30%	50%
Total		100%	100%	100%	100%	100%

Weight loss

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
Wieght loss	stable	40%	20%	90%	60%	52.5%
	weight gain	30%	30%	0%	0%	15%
	weight loss	30%	50%	10%	40%	32.5%
Total		100%	100%	100%	100%	100%

Erosion

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
Erosion	No erosions	50%	40%	90%	100%	70%
	with erosions	50%	60%	10%	0%	30%
Total		100%	100%	100%	100%	100%

Subluxation

		Patients				Total
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	
Subluxation	Without subluxation	80%	80%	80%	80%	80%
	Subluxation	20%	20%	20%	20%	20%
Total		100%	100%	100%	100%	100%

Osteophytes

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
Osteophytes	No Osteophytes	70%	80%	10%	20%	45%
	With osteophytes	30%	20%	90%	80%	55%
Total		100%	100%	100%	100%	100%

Subchondrial Cysts

		Patients				Total
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	
Subchondrial Cysts	No subchondrial cysts	50%	80%	80%	80%	72.5%
	with subchondrial cysts	50%	20%	20%	20%	27.5%
Total		100%	100%	100%	100%	100%

Subchondrial Sclerosis

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
Subchondrial Sclerosis	without subchondrial sclerosis	60%	90%	0%	50%	50%
	with subchondrial sclerosis	40%	10%	100%	50%	50%
Total		100%	100%	100%	100%	100%

PAIN KILLER

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
PAIN KILLER	PAIN KILLER WAS PRESCRIBED	100%	100%	100%	100%	100%
Total		100%	100%	100%	100%	100%

NSAID

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
NSAID	NO NSAIDS WAS USED	10%	0%	30%	20%	15%
	COX 1 NSAID	80%	80%	60%	70%	72.5%
	COX 2 NSAID	10%	20%	10%	10%	12.5%
Total		100%	100%	100%	100%	100%

S.STEROIDS

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
S.STEROIDS	Didn't use	40%	80%	100%	100%	80%
	used	60%	20%	0%	0%	20%
Total		100%	100%	100%	100%	100%

L.STEROIDS

		Patients				
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	Total
L.STEROIDS	NO INTRA-ARTICULAR STEROID INJECTION	80%	70%	70%	70%	72.5%
	WITH INTRA-ARTICULAR STEROID INJECTION	20%	30%	30%	30%	27.5%
Total		100%	100%	100%	100%	100%

DMARD

		Patients				Total
		Rheumatoid arthritis over 45	Rheumatoid arthritis under 45	Osteoarthritis over 45	Osteoarthritis under 45	
DMARD	NO DMARD WAS USED	30%	50%	100%	100%	70%
	MTX alone	10%	10%	0%	0%	5%
	SULPHASALAZINE alone	30%	20%	0%	0%	12.5%
	MTX AND SULPHASALAZINE	30%	20%	0%	0%	12.5%
Total		100%	100%	100%	100%	100%

Descriptive Statistics for all RA patients

	N	Minimum	Maximum	Mean	Std. Deviation
WBC	20	4.20	16.90	8.6055	2.79113
Hg	20	98.00	154.00	127.1500	16.42295
ALAT	20	8.00	79.00	21.0000	15.02979
ASAT	20	13.00	51.00	18.9000	8.58334
Creatinine	20	.70	1.90	.9900	.30070
CRP	20	3.20	81.20	24.0300	21.34383
ESR	20	5.00	60.00	25.2500	16.39279
Glucose	20	4.40	7.70	5.2350	.91667
RF	20	8.80	560.00	176.6400	205.82836
Uric Acid	20	201.00	512.00	265.5000	69.19500
Valid N (listwise)	20				

Descriptive Statistics for RA over 45

	N	Minimum	Maximum	Mean	Std. Deviation
WBC	10	4.50	16.90	9.6000	3.21524
Hg	10	98.00	134.00	121.1000	12.48510
ALAT	10	14.00	79.00	27.2000	19.30343
ASAT	10	13.00	51.00	20.7000	11.50893
Creatinine	10	.70	1.90	1.0600	.33066
CRP	10	3.20	81.20	34.0700	24.31255
ESR	10	13.00	60.00	34.3000	17.12081
Glucose	10	4.50	7.70	5.6100	1.18082
RF	10	13.00	560.00	206.0300	195.35089
Uric Acid	10	201.00	512.00	266.8000	89.47104
Valid N (listwise)	10				

Descriptive Statistics for RA under 45

	N	Minimum	Maximum	Mean	Std. Deviation
WBC	10	4.20	10.40	7.6110	1.97757
Hg	10	100.00	154.00	133.2000	18.22574
ALAT	10	8.00	18.00	14.8000	4.34102
ASAT	10	13.00	26.00	17.1000	3.98469
Creatinine	10	.70	1.40	.9200	.26583
CRP	10	3.20	36.10	13.9900	12.10917
ESR	10	5.00	33.00	16.2000	9.60093
Glucose	10	4.40	5.10	4.8600	.25906
RF	10	8.80	560.00	147.2500	222.16305
Uric Acid	10	212.00	330.00	264.2000	45.81557
Valid N (listwise)	10				

Descriptive Statistics for all OA patients

	N	Minimum	Maximum	Mean	Std. Deviation
WBC	20	3.40	10.70	6.3600	1.81989
HG	20	81.00	168.00	136.3000	17.55023
ALAT	20	5.00	82.00	22.0500	16.96893
ASAT	20	7.00	50.00	21.4000	9.68124
Creatinine	20	.50	1.20	.8550	.21145
CRP	20	1.00	50.50	11.2435	12.97209
ESR	20	2.00	67.00	18.4500	16.41718
GLUCOSE	20	4.00	9.50	5.4450	1.13855
RF	20	8.30	330.00	32.4900	72.83904
Uric Acid	20	203.00	679.00	337.9000	134.81641
Valid N (listwise)	20				

Descriptive Statistics for patients with OA over 45

	N	Minimum	Maximum	Mean	Std. Deviation
WBC	10	4.60	10.70	6.6900	1.89177
HG	10	127.00	168.00	142.1000	12.60026
ALAT	10	11.00	82.00	27.0000	21.68973
ASAT	10	12.00	50.00	25.1000	11.77049
Creatinine	10	.50	1.20	.9100	.21318
CRP	10	3.20	50.50	13.7400	15.88096
ESR	10	6.00	49.00	18.0000	12.56981
GLUCOSE	10	4.00	6.50	5.3500	.78493
RF	10	8.50	330.00	42.0700	101.20142
Uric Acid	10	221.00	548.00	344.5000	101.01403
Valid N (listwise)	10				

Descriptive Statistics for patients with OA less than 45

	N	Minimum	Maximum	Mean	Std. Deviation
WBC	10	3.40	8.30	6.0300	1.78079
HG	10	81.00	163.00	130.5000	20.41378
ALAT	10	5.00	33.00	17.1000	9.10982
ASAT	10	7.00	24.00	17.7000	5.37587
Creatinine	10	.50	1.10	.8000	.20548
CRP	10	1.00	32.40	8.7470	9.44419
ESR	10	2.00	67.00	18.9000	20.26190
GLUCOSE	10	4.30	9.50	5.5400	1.44929
RF	10	8.30	85.00	22.9100	27.47510
Uric Acid	10	203.00	679.00	331.3000	167.54041
Valid N (listwise)	10				

Paired Samples Test

	Paired Differences			t	df	Sig (p value)
	Mean	Std. Deviation	Std. Error Mean			
WBC	.15000	.36635	.08192	1.831	19	.083
Hg	-.35000	.67082	.15000	-2.333	19	.031
CRP	.35000	.58714	.13129	2.666	19	.015
ESR	.15000	.74516	.16662	.900	19	.379
ALAT	.00000	.32444	.07255	.000	19	1.000
ASAT	-.05000	.39403	.08811	-.567	19	.577
Creatinine	.20000	.41039	.09177	2.179	19	.042
Glucose	-.10000	.55251	.12354	-.809	19	.428
Uric acid	-.15000	.48936	.10942	-1.371	19	.186
Polyarthritis	.40000	.50262	.11239	3.559	19	.002
Stiffness	1.00000	1.16980	.26157	3.823	19	.001
RF	.50000	.76089	.17014	2.939	19	.008
Weight loss	.50000	1.31789	.29469	1.697	19	.106
Erosion	.50000	.51299	.11471	4.359	19	.000
Subluxation	.05000	.51042	.11413	.438	19	.666
osteophytes	-.55000	.51042	.11413	-4.819	19	.000
Subchondrial cyst	.15000	.58714	.13129	1.143	19	.267
.Subchondrial sclerosis	-.50000	.60698	.13572	-3.684	19	.002

Discussion

20% of patients with RA have elevated WBC count and all of them are over 45 years old while we see that in OA 20 % of patients had decreased WBC count and all of them are under 45 years old. But in OA the WBC is usually normal.

Regarding the Hg level it is more decreased in RA and in equal proportion 50% in both age groups while in OA only 15 % of patients have decreased Hg level and it is twice decreased in patients under 45 years old. World statistics for RA shows 35-50% has low Hg level, 50% of them due to chronic disease, 29% due to vitamin B12 deficiency, and 21% due to folic acid deficiency.

The CRP level was elevated in both diseases but with greater percentage in patients with RA (85%) in patients over 45 yrs and 80% in patients under 45 yrs. While in OA 65% of all patients have elevated CRP level and there is a big difference between the different age group as 70% of OA patients over 45 have elevated CRP but only 30 % of OA patients under 45 have elevated CRP level. But the world statistics shows 30% of OA patients of both age group has elevated CRP level.

We see that 70 % to 80% of patients with RA have elevated ESR while in OA patient about 30-40 % have elevated ESR level.

So the percentage is higher in RA patients than patients with OA.

Concerning liver enzymes ALAT & ASAT there is no much difference despite an elevation in 10 % of patients over 45 years old in both diseases and this cloud be due to NSAIDs and properly concomitant liver disease.

The Creatinine level is elevated in 20 % of patients with RA in both age groups while it's absolutely normal in patients with OA

In patients over 45 yrs 30 % of RA patients have elevated glucose level and 20 % of OA and this could be due to concomitant diabetes mellitus illness which is common among elderly

But surprisingly 20 % of patient with RA less than 45 yrs have decreased glucose level!

This may be due to connective tissue disorder in patients with RA and affecting on contra-insular organs such suprarenal gland and alpha island of pancreas which produce contra insular hormones.

Even that the uric acid level is not specific for any of these two diseases but we can see an elevation in 10 % of patients with RA over 45, 30 % in patients with OA over 45 and 20 % in patients with OA under 45 yrs.

The elevation of RF level is more pronounced in Patients with RA especially among elderly, where the study shows about 90% of patients over 45 with RA have elevated level of RF and 60 % in patients under 45 yrs. The world statistics also shows 85-90% positive RF.

While in OA the percentage is only 20 % in patients over 45 and 30 % in patients less than 45 yrs. This is near to the world statistics 15% positive in general population.

From clinical aspect we see that 100% of patients with RA in both age groups have Polyarthritis and this could be because most of the patients are suffering of the disease for a long time, the world statistics shows that 30% of RA patients have oligoarthritis.

While in OA only about 40 -50 % of patients who have Polyarthritis and the rest have mono- or Oligoarthritis and this is near the world statistics.

The duration of morning stiffness is more prolonged in patients with RA than in patients with OA as we see that 70 -90% of RA patients have morning stiffness more than 1 h but in OA 40 % of patients in each age group have morning stiffness only for less than15 min and 30 % of patients under 45 yr have stiffness around 1 h .

And we see that patients with age less than 45 tend to loss weight more than patients over 45 in both diseases and this could be due to encouragement by the doctor to reduce their weight .

In X-Ray examination we see that 50 -60% of patients with RA have erosions and in greater proportion among the patients less than 45 yrs old while in OA patients only few patients over 45 ~10 % have erosion .

The subluxation is equal in proportion 20% in all age groups for both diseases while in world statistics it is 40% for RA patients and only 2% or OA.

The discrepancy is due to small sample use

The osteophytes are seen in greater percentage among OA patients 80-90% and there presence is characteristic for the disease while only 20 -30 % of RA patients have osteophytes.

The subchondrial cysts are mostly seen among patients with RA over 45 yrs and in small proportions among the rest groups

Subchondrial sclerosis is seen in all patients with OA over 45 and that's one of the signs of OA but in patients less than 45 only 50 % have these changes on X-Ray and most properly the rest will have it later in there life as the disease progress. while in RA patients we see a 10 % among patients less than 45 and 40 % among patients over 45 and that's may be due to secondary osteoarthritis.

Concerning the medications all patients with RA & OA receive pain killer,

NSAIDs use is almost the same for both diseases with slightly increased percentage among RA patients, most of the patients are taking COX-1 NSAIDs (~72.5%) and only 12 % used COX-2

While we see that the use of systemic steroid is conserved for RA patients especially those over 45 (~ 60%)and in small proportion for those less than 45 yrs old (~ 20 %)mostly because the disease is well controlled by other

medications and avoiding the adverse affect of long term use of corticosteroids.

Only 27.5 % of all patients' received local injections of corticosteroids and almost in the same percentage for each age group (~ 20-30%), the world statistics shows more percentage for RA 40-50%

The use on DMARDs is conserved for patients with RA only and the use of these agents is more among patients over 45 (70 %) 10% used MTX alone and 30 % used SULFASALAZINE alone ,30% used MTX and SULFASALAZINE

50% of patients under 45 used DMARDs 10% used MTX alone 20% used SULFASALAZINE alone, 20% used MTX and SULFASALAZINE

Conclusion:

- The differences between RA and OA:

– Polyarthritis - 100% in RA

- 45% in OA.

P = 0.002 < 0.05

– Morning stiffness more than 1 hour: 70-90% in RA.

30% in OA

P = 0.001<0.05

- RF elevated: 75% in RA

  25% in OA

  P = 0.008 <0.05

- WBC: leukocytosis in 20% of RA

  Leucopenia in 20% of OA

  P = 0.083 > 0.05

- Hg: 50% decreased in RA

  15 % decreased in OA

  P = 0.031<0.05

- ESR: 70% elevated in RA

  30% elevated in OA

  P = 0.379> 0.05

- CRP: 85% elevated in RA

  40-70% elevated in OA

  P = 0.015 <0.05

- EROSSION 60% in RA

  10% in OA

  P = 0.00 <0.05

- Osteophytes 20 % in RA

  85% in OA

  P = 0 <0.05

- Subchondrial sclerosis 10-40% in RA

  50-100% in OA

  P =0.002< 0.05

- We don't find statistical trustworthy in sign and features between young" till 45" and old "over 45" patients with RA and OA.

Recommendation:

The most important signs and features for diagnosis of RA and OA are the followings:

RA	**OA**
Polyarthritis	Mono-Oligo arthritis
Morning stiffness	osteophytes
anemia	Subchondrial sclerosis
CRP elevation	
ESR elevation	
Erosion of cartilage in X-ray	

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Appendix

Case Summaries of patients with RA over 45

	WBC	HG	ALAT	ASAT	Creatinine	CRP	ESR	Glucose	RF	Uric Acid
1	4.50	98.00	14.00	13.00	.70	3.20	13.00	4.50	13.00	201.00
2	8.00	108.00	15.00	15.00	.80	11.40	18.00	4.50	29.10	210.00
3	8.20	108.00	16.00	15.00	.90	15.40	18.00	4.60	33.80	220.00
4	8.70	120.00	17.00	16.00	.90	24.00	23.00	4.90	42.70	235.00
5	8.90	122.00	22.00	16.00	1.00	26.00	24.00	5.00	102.00	240.00
6	9.00	127.00	22.00	16.00	1.00	30.40	40.00	5.60	197.70	250.00
7	9.20	129.00	26.00	18.00	1.10	39.30	42.00	5.70	276.50	260.00
8	10.40	132.00	26.00	18.00	1.10	44.70	51.00	6.10	401.50	263.00
9	12.20	133.00	35.00	29.00	1.20	65.10	54.00	7.50	404.00	277.00
10	16.90	134.00	79.00	51.00	1.90	81.20	60.00	7.70	560.00	512.00
Total N	10	10	10	10	10	10	10	10	10	10

Case Summaries for patients with RA less than 45 yrs

	WBC	Hg	ALAT	ASAT	Creatinine	CRP	ESR	Glucose	RF	Uric Acid
1	4.20	100.00	8.00	13.00	.70	3.20	5.00	4.40	8.80	212.00
2	6.10	110.00	8.00	13.00	.70	3.20	8.00	4.40	8.80	220.00
3	6.10	116.00	12.00	13.00	.80	7.50	10.00	4.80	8.80	222.00
4	6.30	137.00	12.00	16.00	.80	7.50	11.00	4.90	8.80	222.00
5	7.00	137.00	18.00	16.00	.80	10.50	14.00	4.90	20.50	250.00
6	8.60	140.00	18.00	18.00	.80	11.80	15.00	5.00	49.00	270.00
7	8.60	140.00	18.00	18.00	.80	12.00	16.00	5.00	123.90	286.00
8	8.80	149.00	18.00	18.00	1.00	12.00	17.00	5.00	123.90	300.00
9	10.01	149.00	18.00	20.00	1.40	36.10	33.00	5.10	560.00	330.00
10	10.40	154.00	18.00	26.00	1.40	36.10	33.00	5.10	560.00	330.00
Total N	10	10	10	10	10	10	10	10	10	10

Case Summaries for patients with OA over 45

	WBC	HG	ALAT	ASAT	Creatinine	CRP	ESR	Glucose	RF	Uric Acid
1	4.60	127.00	11.00	12.00	.50	3.20	6.00	4.00	8.50	221.00
2	5.10	128.00	12.00	13.00	.60	3.20	9.00	4.60	8.50	250.00
3	5.40	133.00	13.00	17.00	.90	4.54	9.00	5.00	8.80	252.00
4	5.90	133.00	14.00	19.00	.90	6.26	12.00	5.00	8.80	291.00
5	6.20	140.00	15.00	19.00	.90	6.30	13.00	5.10	8.80	314.00
6	6.20	146.00	22.00	27.00	1.00	6.70	14.00	5.20	8.80	351.00
7	6.30	147.00	30.00	28.00	1.00	8.10	20.00	5.90	10.70	376.00
8	7.40	147.00	31.00	29.00	1.00	14.40	23.00	5.90	10.80	397.00
9	9.10	152.00	40.00	37.00	1.10	34.20	25.00	6.30	17.00	445.00
10	10.70	168.00	82.00	50.00	1.20	50.50	49.00	6.50	330.00	548.00
Total N	10	10	10	10	10	10	10	10	10	10

Case Summaries for patients with OA less than45

	WBC	HG	ALAT	ASAT	Creatinine	CRP	ESR	Glucose	RF	Uric Acid
1	3.40	81.00	5.00	7.00	.50	1.00	2.00	4.30	8.30	203.00
2	3.40	127.00	8.00	15.00	.60	3.08	5.00	4.60	8.50	239.00
3	4.30	127.00	8.00	15.00	.70	3.20	5.00	5.10	8.50	239.00
4	5.90	127.00	12.00	15.00	.70	3.20	5.00	5.10	8.80	240.00
5	6.60	133.00	19.00	15.00	.70	3.80	7.00	5.20	8.80	250.00
6	6.60	133.00	19.00	18.00	.80	4.30	11.00	5.20	9.50	271.00
7	6.80	135.00	19.00	22.00	.80	9.59	27.00	5.30	9.50	292.00
8	6.80	137.00	19.00	22.00	1.00	13.00	30.00	5.30	20.00	292.00
9	8.20	142.00	29.00	24.00	1.10	13.90	30.00	5.80	62.20	608.00
10	8.30	163.00	33.00	24.00	1.10	32.40	67.00	9.50	85.00	679.00
Total N	10	10	10	10	10	10	10	10	10	10