# ARTHRITIS

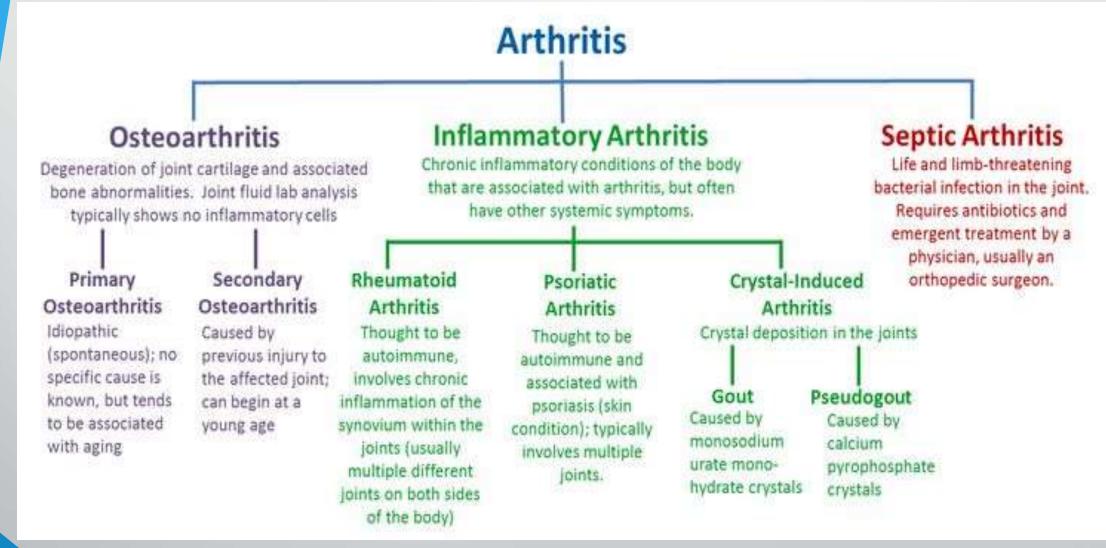
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## **Definition:**

Arthritis is a nonspecific term that means "inflammation of the joints." Arthritic disease encompasses a group of disorders of the rheumatic diseases that affect bones, joints, and muscles.

Some of the more common types include rheumatoid arthritis, osteoarthritis, systemic lupus erythematosus, juvenile arthritis, scleroderma, Sjögren's syndrome, gout, ankylosing spondylitis, Lyme disease, fibromyalgia, and psoriatic arthritis.



## **Types of Arthritis**

Although arthritis comprises a group of more than 60 important diseases, this lecture is limited to a discussion of :

- 1. Rheumatoid arthritis
- 2. Osteoarthritis
- 3. Systemic lupus erythematosus (SLE)
- 4. Sjögren's syndrome

which are among the most common forms encountered and can serve as models for the other forms.

## RHEUMATOID ARTHRITIS

#### Incidence and Prevalence

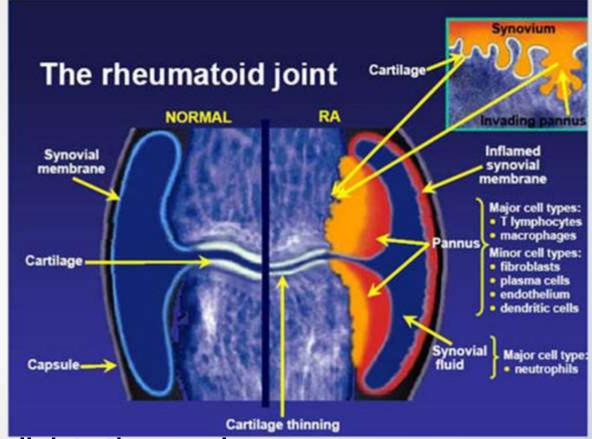
- ✓ Rheumatoid arthritis (RA) is an autoimmune disease of unknown origin that is characterized by symmetric inflammation of joints, especially of the hands, feet, and knees.
- ✓ Severity of the disease *varies widely from patient to patient and from time to time within the same patient.* Determination of prevalence is somewhat difficult to determine because of lack of well-defined markers of the disease.
- ✓ Estimates of prevalence *range from 1% to 2% of the population*.
- ✓ Disease onset usually occurs between ages 35 and 50 years and is more prevalent in women than men by a 3:1 ratio.
- ✓ This gender differentiation indicates involvement of sex hormones in the susceptibility and sensitivity of the disease.
- ✓ Other factors, such as **socioeconomic status, education, and psychosocial stress**, have <sup>5</sup> been suggested to play predisposing roles.

## **Etiology:**

- > The exact cause of Rh.Ar. Is unknown.
- > Suspected causes are:
  - 1. Bacterial infection.
  - 2. Genetic marker
  - 3. Stress
  - 4. Viral infection
  - 5. Other suspects include female hormones
  - 6. Smoking.

## **PATHOPHYSIOLOGY**

- 1. Edema of the synovium.
- 2. Thickening & folding (pannus).
- 3. Infiltration of lymphocyte and plasma cells into the capsule.
- 4. Gradually granulation tissue covers the articular surfaces and destroys them by enzymatic activity.
- 5. New bone or fibrous tissue is deposited and result in fusion or loss mobility.



## Signs and symptoms

- 1) Painful joint swelling most commonly affected fingers, wrists, feet, ankles, knees, and elbows. Multiple joint changes noted in the hands include a symmetric spindle-shaped swelling of the proximal interphalangeal (PIP) joints, with dorsal swelling and characteristic volar subluxation of the metacarpophalangeal (MCP) joint.
- 2) Deformities like immobility, contractures, subluxation, deviation.
- 3) Generalized joint stiffness after inactivity, and morning stiffness that lasts longer than 1 hour.
- The TMJ is reported to be involved in up to 75% of patients.



metacarpophalangea

Swan-neck deform

The most significant complication of the oral and maxillofacial complex in RA is TMJ involvement, which is found in up to 45% to 75% of patients with RA. This may present as bilateral prequricular pain, tenderness, swelling, stiffness, and decreased mobility of the TMJ, or it may be asymptomatic. Periods of remission and exacerbation may occur, as with other joint involvement. Fibrosis or bony ankylosis can occur. Clinically, patients may present with tenderness over the lateral pole of the condyle, crepitus, limited opening, and radiographic evidence of structural change. Radiographic changes initially may show increased joint space. Later, these changes are primarily erosive and can involve both the condyles and the fossa.

swollen

## Criteria for the Diagnosis of Rheumatoid Arthritis

- Morning stiffness
- Arthritis of three or more joint areas
- Arthritis of hand joints
- Symmetric arthritis
- Rheumatoid nodules
- Serum rheumatoid factor
- Radiographic changes

## **Laboratory Findings**

- No laboratory tests are pathognomonic or diagnostic of RA, although they are used in conjunction with clinical findings to confirm the diagnosis.
- Laboratory findings most commonly seen in RA include
  - 1) an increased erythrocyte sedimentation rate (ESR)
  - 2) the presence of C-reactive protein (C-RP)
  - 3) a positive rheumatoid factor in 85% of affected patients (Rh. F)
  - 4) hypochromic/microcytic anemia.
  - 5) In patients with Felty's syndrome (RA with splenomegaly), a marked neutropenia may be present.

## Physical managements

- Regular exercise is recommended as both safe and useful to maintain muscles strength and overall physical function.
- > It is uncertain if specific dietary measures have an effect example Omega-3
- Physical activity is beneficial for persons with Rheumatoid arthritis complaining of fatigue.
- Occupational therapy <u>Occupational therapy interventions focus on</u> <u>adapting the environment, modifying the task, teaching the skill, and</u> <u>educating the family in order to increase participation in and performance</u> <u>of daily activities</u> has a positive role to play in improving functional ability of persons with rheumatoid arthritis

## **Medical managements**

#### **SALICYLATES**

Aspirin, Ascriptin, Bufferin, Anacin, Ecotrin, Empirin

Prolonged bleeding but not usually clinically significant

#### **NONSTEROIDAL ANTI-INFLAMMATORY DRUGS**

Ibuprofen, Fenoprofen, Indomethacin, Naproxen, Meclofenamate, Piroxicam, Sulindac, Tolmetin, Diclofenac, Flurbiprofen, Diflunisal, Etodolac, Nabumetone, Motrin, Nalfon, Indocin, Feldene, Naprosyn, Meclomen, Clinoril, Tolectin, Voltaren, Ansaid, Dolobid, Lodine, Relafen, Oxaprozin, Ketorolac. *Prolonged bleeding but not usually clinically significant; oral ulceration, stomatitis* 

#### **CYCLOOXYGENASE (COX)-2 INHIBITORS**

Celecoxib ,Rofecoxib

**None** 

#### **TUMOR NECROSIS FACTOR-INHIBITORS**

Etanercept ,Infliximab

**None** 

## **Medical managements**

#### INJECTABLE GLUCOCORTICOIDS

Triamcinolone hexacetonide, Triamcinolone acetonide Prednisolone tebutate, Methylprednisolone acetate, Dexamethasone acetate, Hydrocortisone acetate, Triamcinolone diacetate, Betamethasone sodium phosphate and acetate, Dexamethasone sodium phosphate, Prednisolone sodium phosphate.

\*\*Adrenal suppression, masking of oral infection, impaired healing.\*\*

#### SYSTEMIC GLUCOCORTICOIDS

Hydrocortisone, Cortisone, Prednisone, Prednisolone, Dexamethasone, Methylprednisolone (Deltasone, Meticorten, Orasone, Articulose-50, Delta-Cortef, Medrol).

Adrenal suppression, masking of oral infection, impaired healing.

## **Medical managements**

#### **DISEASE-MODIFYING ANTIRHEUMATIC DRUGS**

Antimalarial agents, Hydroxychloroquine, Quinine, Chloroquine (Plaquenil), Penicillamine (Cuprimine, Depen), Gold compounds. Gold sodium thiomalate (Auranofin, Aurothioglucose, Myochrysine Ridaura, Solganal), Sulfasalazine, Azulfidine.

Increased infections, delayed healing, prolonged bleeding, oral ulcerations, glossitis, stomatitis, Other side effects of gold salts include kidney damage, itching rash, and ulcerations of the mouth, tongue, and pharynx. Approximately 35% of patients discontinue the use of gold salts because of these side effects. Kidney function must be monitored continuously while taking gold salts.

#### **Immunosuppressive**

Azathioprine, Cyclophosphamide, Methotrexate, Cyclosporine, Chlorambucil (Imuran, Cytoxan, Rheumatrex).

Increased infections, delayed healing, prolonged bleeding, stomatitis.

## **Dental Management of the Patient With Rheumatoid Arthritis**

- 1. Short appointments
- 2. Insurance of physical comfort
  - a. Frequent position changes
  - b. Comfortable chair position
  - c. Physical supports as needed (pillows, towels, etc.)
- 3. Drug considerations
  - a. Aspirin and NSAIDs—bleeding may be increased but usually is not clinically significant
  - b. Gold salts, penicillamine, antimalarials, immunosuppressives— get complete blood cell count with differential, bleeding time; treat stomatitis symptomatically.
  - c. Corticosteroids— adrenal suppression possible.
  - d. Joint prosthesis: prophylactic antibiotics are suggested by some authors (cephalosporin or clindamycin)

#### High-Risk Patients With Prosthetic Joints IMMUNOCOMPROMISED/IMMUNOSUPPRESSED PATIENTS

 Inflammatory arthropathies: rheumatoid arthritis; systemic lupus erythematosus; disease-, drug-, or radiation-induced immunosuppression.

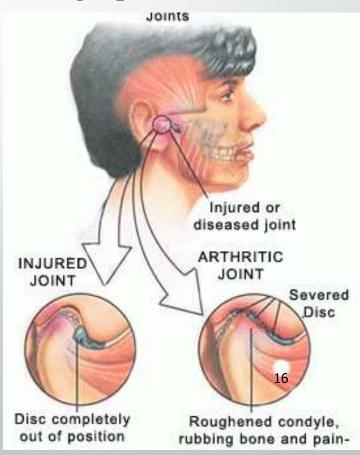
#### OTHER PATIENTS

- Insulin-dependent (type 1) diabetes
- First 2 years after joint replacement
- Previous prosthetic joint infections
- Malnourishment
- Hemophilia

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## **Dental Management of the Patient With Rheumatoid Arthritis**

- 4. Technical treatment modification dictated by patient's disabilities.
- 5. Temporomandibular joint pain/ dysfunction— sudden occlusal changes possible
- a. Decrease jaw function
- b. Soft, non challenging diet
- c. Moist heat or ice to face/jaw
- d. Medication as directed by physician
- e. Occlusal appliance to decrease joint loading
- f. Consideration of surgery for persistent pain or dysfunction



## **Osteoarthritis**

#### Incidence and Prevalence

- > Osteoarthritis (OA, <u>degenerative joint disease</u>), another of the rheumatic diseases, is the most common form of arthritis.
- Almost everyone older than 60 years of age develops OA to some degree.
- Most people are minimally symptomatic; however, approximately 17 million people in the United States have OA to the extent that it results in pain.
- OA is the leading cause of disability among the elderly.
- OA, which is considered <u>a regional disease, usually affects often used joints such as hips, knees, feet, spine, and hands.</u>
- The TMJ also is affected.
- Women are afflicted twice as often as men; however, men are afflicted at an earlier age.
- It is generally a disease of middle to older age, first appearing after the age of 40.
- Racial differences have been noted in the prevalence of OA and in the pattern of joint involvement.

## **Etiology**

Although the exact cause of OA is **not known**, it has been thought to **result from normal wear and tear on joints over a long period**.

However, other factors are now believed to be of significance: Preexisting structural joint abnormalities

Intrinsic aging

Metabolic factors

Genetic predisposition

Obesity leading to overloaded joints

Macrotrauma or microtrauma are considered causative or contributory factors in the origin of the disease.

## Pathophysiology and Complications

In early stages of the disease, the <u>articular cartilage actually becomes thicker than normal</u>, and wate content and the synthesis of proteoglycans are increased.

This reflects a repair effort by the chondrocytes and may last for several years.

The joint surface thins and proteoglycan concentration decreases, leading to softening of the cartilage.

Progressive splitting and abrasion of cartilage down to the subchondral bone occur.

The exposed bone becomes polished and sclerotic

Some resurfacing with cartilage may occur if the disorder is arrested or stabilized.

New bone forms at the margin of the articular cartilage in the non-weight-bearing part of the joint creating osteophytes (or spurs), often covered by cartilage, that augment the degree of deformity.

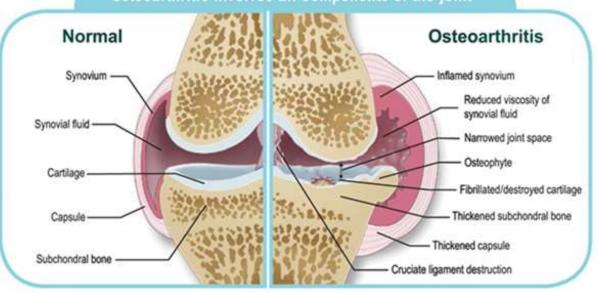
In contrast to RA, OA has a more <u>favorable prognosis and less serious complications</u>, depending on the joint or joints involved.

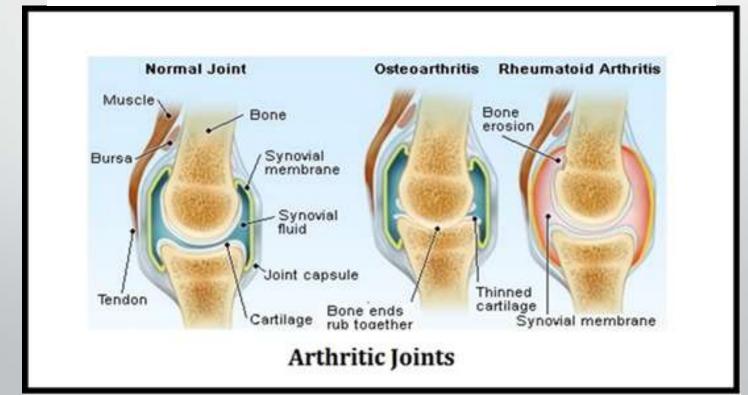
The two most important complications associated with OA are pain and disability.

Although RA is a more serious disease, <u>OA has a 30-fold greater economic impact, resulting in 68 million lost workdays per year compared with 2 million for RA.</u>

Conservative treatment often can retard the progress of the disease; however, surgery may be required to restore function and reduce pain

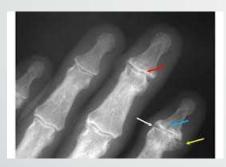
#### Osteoarthritis involves all components of the joint

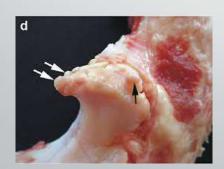




## Signs and symptoms







#### Rheumatoid arthritis and Osteoarthritis differences

Rheumatoid arthritis	Osteoarthritis
Usually begin b/w ages 25-50 years	Usually begins after age 40 years
Autoimmune response affecting the synovial membrane leads to joint destruction	Biomechanical: Leads to loss of cartilage matrix
Develops within weeks or months	Develops slowly, over many years
Usually symmetrical, primarily affects small joints, may involve large joints like elbow	Usually affects weight bearing joints such as knee, hip, lower spine, may be uni or bilateral.
Signs of inflammation present	Pain begins with the use of joints, inflammatory signs are less common
Morning stiffness often >1 hour	Morning stiffness usually lasts <20 minutes
Generalized symptoms, such as fatigue, weight loss and anemia may be present	Does not cause a general feeling of unwellness
More common in females	Commonly found in both male and females
Osteophytes absent	Osteophytes may be present
Rheumatoid factor (RF) frequently present	Rheumatoid factor (RF) absent



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## **MEDICAL MANAGEMENT**

- > The management of **OA** is palliative.
- drug therapy is limited to <u>analgesics</u>. Acetaminophen frequently is effective in the management of OA and is recommended as a <u>first-line drug</u>.
- > <u>Aspirin or NSAIDs</u> also are commonly employed when acetaminophen is not effective.
- Narcotic analgesics are generally used only for acute flares for short periods.
- Intra-articular steroid injections also may be used for acute flares for short periods.
  Intra-articular steroid injections may be used intermittently to reduce acute pain and inflammation.
- Patient education, physical therapy, mild exercise, weight reduction, and joint protection are all important aspects of management.
- Surgery, including joint replacement, may be required to improve function or relieve pain.

## **Dental Management of the Patient With osteoarthritis**

#### 1. Short appointments

#### 2. Insurance of physical comfort

- a. Frequent position changes
- b. Comfortable chair position
- c. Physical supports as needed (pillows, towels, etc.)

#### 3. Drug considerations

- a. Aspirin and NSAIDs—bleeding may be increased but usually is not clinically significant
- b. Joint prosthesis: prophylactic antibiotics are suggested by some authors (cephalosporin or clindamycin)
- 4. Technical treatment modification dictated by patient's disabilities.

#### 5. Temporomandibular joint pain/ dysfunction— sudden occlusal changes possible

- a. Decrease jaw function
- b. Soft, non challenging diet
- c. Moist heat or ice to face/jaw
- d. Medication as directed by physician
- e. Occlusal appliance to decrease joint loading
- f. Consideration of surgery for persistent pain or dysfunction

### SYSTEMIC LUPUS ERYTHEMATOSUS

#### **DEFINITION**

- Lupus erythematosus has two forms: one that predominantly affects the skin (discoid, DLE) and a more generalized one that affects multiple organ systems (systemic, SLE).
- DLE is characterized by chronic, erythematous, scaly plaques on the face, scalp, or ears. Most patients with DLE do not have systemic manifestations, and the course tends to be benign.
- > SLE involves the skin and many other organ systems and is the more serious form.

#### **Incidence and Prevalence**

- > SLE is a prototypical autoimmune disease that predominantly affects women of childbearing age,
- With a <u>female/male ratio of 5:1</u>; it is more common and severe among African Americans and Hispanics than whites.
- A defining feature of SLE is the almost invariable <u>presence in the blood of antibodies</u> directed against one or more components of cell nuclei; certain manifestations of the disease are associated with the <u>presence of one or more of these different antinuclear antibodies.</u>

#### **Etiology**

- The etiology of SLE is unknown, although it is clearly an autoimmune disease.
- A <u>strong familial aggregation exists</u>, with a much higher frequency noted among first-degree relatives of patients.
- immune abnormalities that can be triggered by exogenous and endogenous factors. Among these triggering factors are infectious agents, stress, diet, toxins, drugs, and sunlight

## Table 1

## **Clinical Symptoms of SLE**

Organ System	Symptoms
Musculoskeletal	Arthritis, arthralgia
Constitutional	Fever (absence of infection), fatigue, weight loss
Skin	Malar (butterfly) rash, alopecia, photosensitivity, purpura, Raynaud's phenomenon, urticaria, vasculitis
Gastrointestinal	Nausea, vomiting, abdominal pain
Renal	Proteinuria, hematuria, nephrotic syndrome
Hematologic	Anemia, thrombocytopenia, leukopenia
Cardiac	Pericarditis, endocarditis, myocarditis
Neurologic	Seizures, psychosis, peripheral and cranial neuropathies
Pulmonary	Pulmonary hypertension, pleurisy, parenchymal disease





THE CLASSIC MALAR 'BUTTERFLY' RASH OF S.L.E. AND TYPICAL RASHES ON THE HANDS



## **Laboratory Findings**

- The <u>antinuclear antibody test is the best screening test for SLE because it is positive in 95% of patients</u>. This positivity also occurs in patients with other rheumatic diseases.
- Anti-DNA assays—double helix and single helix—also are elevated in 65% to 80% of patients with active untreated SLE.
- Hematologic abnormalities include hemolytic anemia, leukopenia, lymphopenia, and thrombocytopenia.
- Leukopenia in SLE usually is not associated with recurrent infection.
- Autoimmune thrombocytopenia occurs in as many as 25% of patients with SLE and may be severe in 5% of these. Patients with severe thrombocytopenia are at risk for bleeding spontaneously or after trauma.
- **Elevated partial thromboplastin time (PTT).**
- ESR erythrocyte sedimentation rate often is elevated, but this does not reflect disease activity.
- With active nephritis, proteinuria is present, as are hematuria and cellular or granular casts.
- Other abnormalities include false-positive serologic tests for syphilis.

## **Oral Complications and Manifestations**

- Oral lesions of the lips and mucous membranes have been reported to occur in up to 5% to 25% of patients with SLE.
- These lesions are <u>rather nonspecific</u> and may be <u>erythematous</u> with white spots or radiating peripheral lines; they also may occur as <u>painful ulcerations</u>
- Lesions frequently <u>resemble lichen planus or leukoplakia</u>. When they occur on the lip, a <u>silvery, scaly margin</u>, similar to that seen on the skin, may develop. <u>Skin and lip lesions</u> <u>frequently are noted after exposure to the sun.</u>
- Treatment of these lesions is symptomatic, and future sun exposure is avoided
- Other oral manifestations of SLE may include <u>xerostomia and hyposalivation</u>, <u>dysgeusia (metallic taste sensation)</u>, <u>and glossodynia (burning tongue)</u>.

#### **Dental Management of Patient With Systemic Lupus Erythematosus**

#### 1. Consultation with physician

- A. Patient status and stability
- B. Extent of systemic manifestations (i.e., kidney, heart)
- C. Hematologic profile (complete blood cell count [CBC] with differential, prothrombin time [PT], partial thromboplastin time [PTT], bleeding time [BT])
- D. Drug profile

#### 2. Drug considerations

- Aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs)—bleeding may be increased but is not usually clinically significant; if patient is concurrently taking corticosteroids, bleeding is more likely—suggest obtaining pretreatment bleeding time (<20 minutes)</li>
- b. Gold salts, antimalarials, penicillamine, and cytotoxic drugs may <u>cause leukopenia and</u> <u>thrombocytopenia</u>; <u>also, severe stomatitis—treat symptomatically</u>
- c. Corticosteroids may cause adrenal suppression

#### **Dental Management of Patient With Systemic Lupus Erythematosus**

#### 3. Hematologic considerations

- a. Leukopenia with corticosteroids or cytotoxic drugs may predispose patient to infection; <u>use of</u> <u>postoperative antibiotics can be considered with surgical procedures</u>
- b. Platelet count <50,000/mm may result in severe bleeding—consultation with physician
- c. Elevated PTT associated with lupus anticoagulant usually does not cause increased <a href="bleeding-surgery">bleeding-surgery can be performed</a>
- **4. Infective endocarditis is a potential problem**—antibiotic prophylaxis is not recommended by the American Heart Association.

## **SJÖGREN'S SYNDROME**



#### **DEFINITION**

- Sjögren's syndrome (SS) is an <u>autoimmune disease complex classified among the many</u> <u>rheumatic diseases.</u>
- SS is characterized by a triad of clinical conditions that consists of <u>keratoconjunctivitis sicca</u>, <u>xerostomia</u>, and a connective tissue disease (usually, rheumatoid arthritis).
- SS presents in two different forms: primary SS and secondary SS.
- Primary SS (SS-1) clinically manifests with the primary ocular complication of keratoconjunctivitis sicca; in the oral cavity, it presents as various levels of salivary gland dysfunction (xerostomia).
- Secondary SS (SS-2) manifests as the presence of keratoconjunctivitis sicca or xerostomia in the presence of a diagnosed systemic connective tissue disease. The connective tissue disorder from which SS develops most commonly is rheumatoid arthritis; SLE, primary biliary cirrhosis, fibromyalgia, mixed connective tissue disease, polymyositis, Raynaud's syndrome, and several others are among the associated inflammatory conditions

## **Etiology and Pathophysiology**

- The precise cause of SS, as of many of the autoimmune rheumatic disorders, is unknown, although several contributing factors have been identified.
- > One theory is that the disease results from:
- Complications of <u>viral infection with EBV (Epstein-Barr Virus) [Infectious mononucleosis, also called "mono,"] is a contagious disease.</u> Exposure to or reactivation of EBV elicits expression of the HLA (human lymphocyte antigen) complex; this is recognized by the T-cell (CD4+) lymphocytes and results in
  - 1. The release of cytokines (tumor necrosis factor [TNF], interleukin [IL]-2, interferon [IFN]-y, and others).
  - 2. Chronic inflammation,
  - 3. Infiltration of lymphocytes
  - 4. Ultimate destruction of exocrine gland tissue follow.

## **Signs and Symptoms**

The oral clinical manifestations of SS typically include:

- Hyposalivation
- Glossitis
- Mucositis
- Parotid gland hypertrophy
- Angular cheilosis
- Dysgeusia (taste dysfunction)
- Secondary infection
- A significantly increased caries rate



## Symptoms of Sjögren's Synarome

#### PRIMARY SYMPTOMS

#### Dry eye

Gritty, sandy feeling Stinging feeling

#### Dry mouth

Dry, cracked tongue

Sore throat

Burning throat

Difficulty talking

Difficulty swallowing

Difficulty chewing dry food

Change in sense of taste/smell

Increase in cavities

Mouth sores

Cracked lips

#### OTHER SYMPTOMS

Swollen parotid glands

Nausea

Dry skin

Joint pain

Dry nose

Reflux

Muscle pain

Fatigue

Muscle weakness

Low-grade fever

Vaginal dryness

Neuropathy

Dizziness

## **Laboratory Findings**

# European Study Group Diagnostic Criteria

#### Criteria

- Ocular symptoms
- 2. Oral symptoms
- 3. Ocular signs (Schirmer's or Rose Bengal test)
- Diagnostic histopathologic features in salivary gland biopsy
- Presence of abnormality in at least one salivary gland study: parotid sialography, salivary scintigraphy, or unstimulated salivary flow
- Presence of at least one: Ro/SS-A, La/SS-B antibodies, antinuclear antibodies, rheumatoid factor

# **DENTAL MANAGEMENT**



Gluten

High-sodium

foods



Trans fat & some saturated fats



Alcohol & caffeine



Added sugar

Certain legumes

#### Dr. Axe FOOD ! MEDICINE

## FOODS TO EAT



Organic, unprocessed foods



Raw vegetables



Wild-caught fish



High-antioxidant foods



Bone broth



Avocado



Nuts and seeds



Coconut oil



Olive oil



Raw milk



Cucumbers



Melon

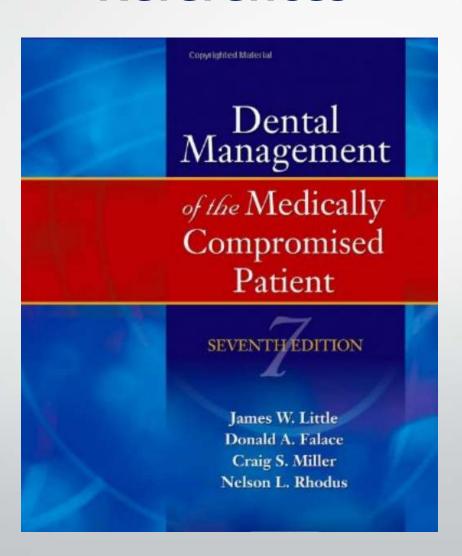


Water



Herbal tea and green tea

## References



# JHANKYOU FOR



YOUR

ATTENTION !