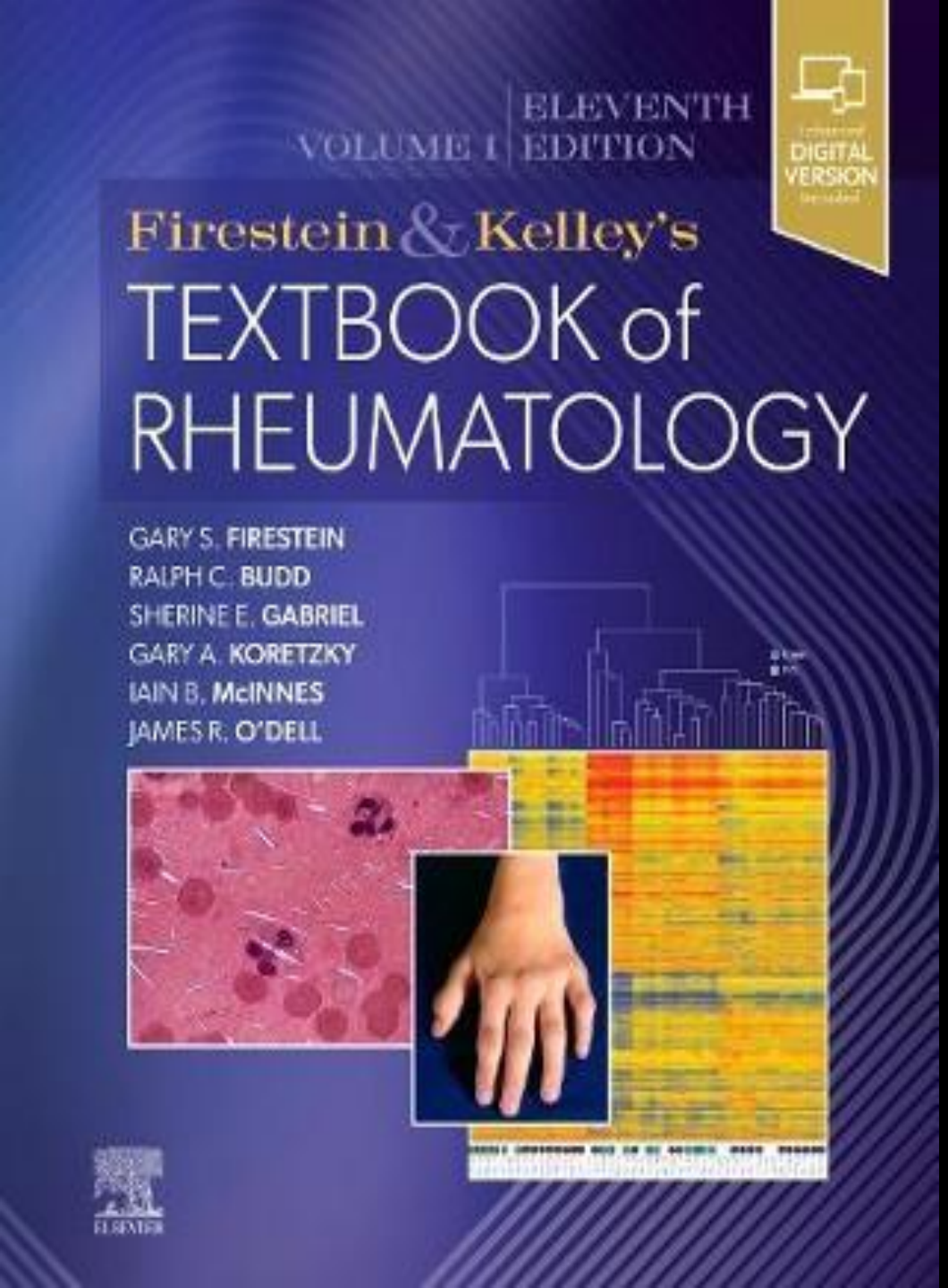

Arthritis Accompanying Endocrine and Metabolic Disorders

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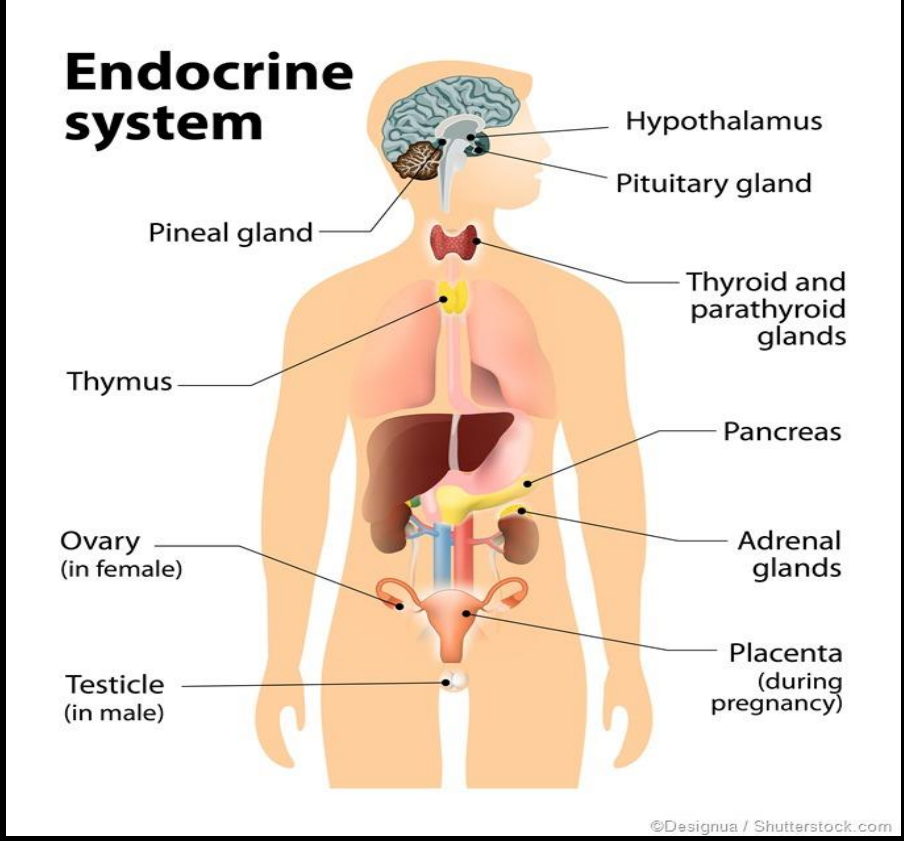
Introduction



- Endocrine disorders can initially present or manifest with a multitude of musculoskeletal signs and symptoms.
- Musculoskeletal symptoms associated with endocrine disorders such as diabetes or acromegaly are a common manifestation associated with morbidity and poor quality of life.
- Each endocrine disorder has its own set of arthritic symptoms that can mimic or present as definitive rheumatic diatheses, requiring a high degree of vigilance and a thorough assessment of patients presenting with such varied symptoms

ADRENAL GLAND DISORDERS

- Cushing described several rheumatic conditions, including osteoporosis, AVN, myopathy, and synovitis.
- Primary CS is usually related to:
- ✓ excessive endogenous production of cortisol
- ✓ and is either ACTH-dependent (pituitary adenomas)
- ✓ or ACTH-independent (adrenal tumor or adrenal hyperplasia),
- whereas exogenous CS is secondary to excessive exposure to exogenous glucocorticoids

ADRENAL GLAND DISORDERS

❑Avascular necrosis:

- ✓ generally occurs after prolonged use of high-dose corticosteroids but can be observed months or even years after the discontinuation of steroids.
- ✓ Less commonly, it can be associated with short courses of therapy or intermittent high-dose IV therapy.
- ✓Although rare, case reports of AVN in patients with endogenous CS do exist

❑Steroid myopathy :

- ✓presents as extreme muscle weakness
- ✓and pain that is most often pronounced around the pelvic girdle.(in 70% of patients with active primary CS)
- ✓ pain may either occur suddenly or be insidious in onset
- ✓Usually a muscle biopsy demonstrates type 2 fiber atrophy,
- ✓Muscle performance such as hand grip strength and performance on the chair rising test is impaired in patients with active CS.
- ✓ the condition gradually improves as corticosteroids are tapered or discontinued completely

ADRENAL GLAND DISORDERS

- ✓ some recent studies using body composition analysis with MRI and DXA in patients with primary CS showed persistence of lower limb skeletal muscle mass decline even up to 13 years after clinical remission.

❑Osteopenia:

- ✓osteopenia occurring in 40% to 78% of patients,osteoporosis in 22% to 57%, and fractures in 11% to 76%.
- ✓The prevalence of osteoporosis is higher in individuals with adrenal CS than in those with pituitary CS.
- ✓ Male patients have a higher prevalence of osteoporosis and vertebral fractures compared with females.
- ✓Several studies show persistently low BMD and altered bone metabolic markers up to several years after achieving clinical remission

ACROMEGALY

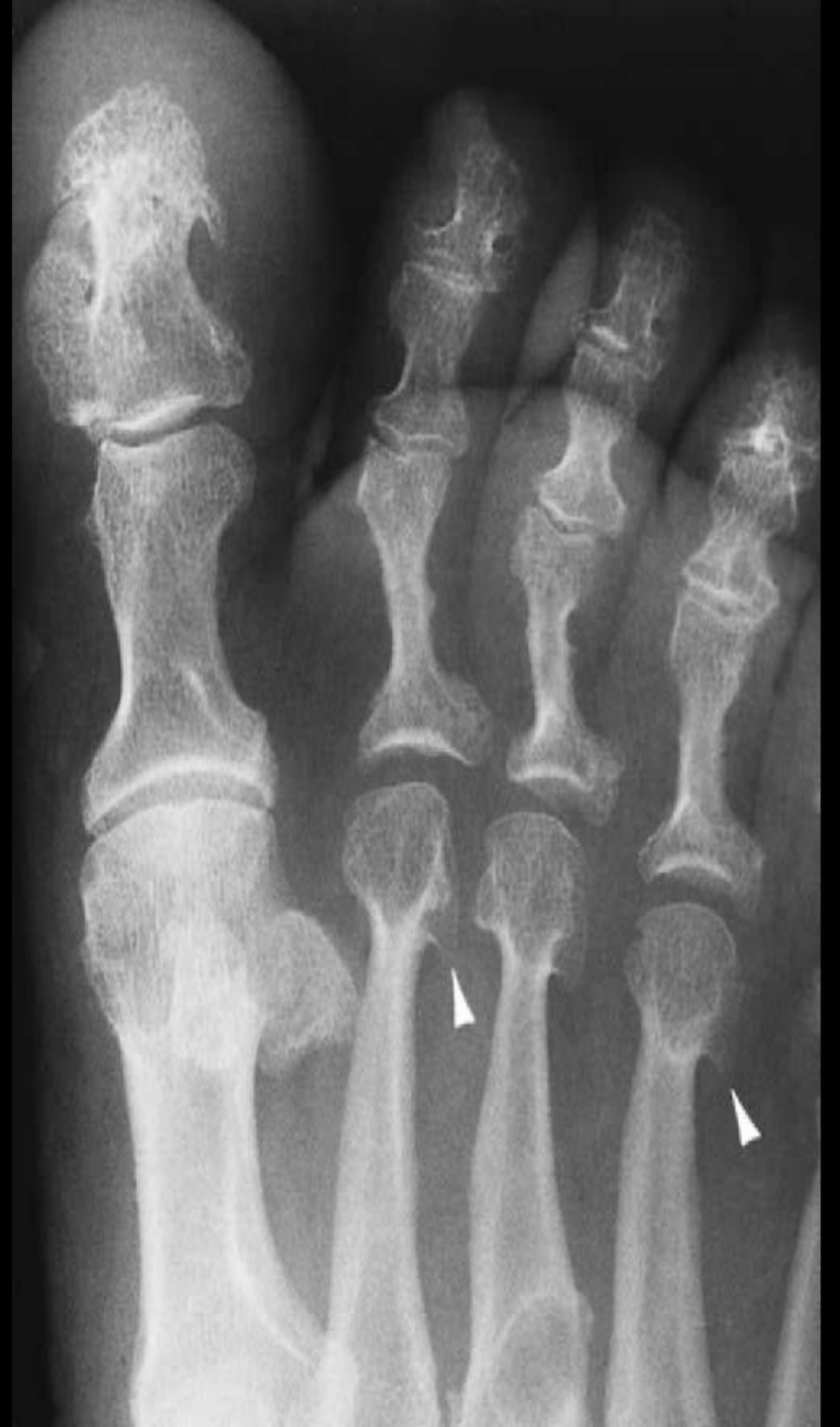
- GH released into circulation stimulates production of IGF-I, and stimulates growth of soft tissue
- and fibroblast proliferation,
- causing an increased thickening of connective tissue.
- GH secreted by the anterior pituitary acts on hepatocytes to secrete somatomedins,which have stimulatory effects on bone and cartilage, including chondrocyte replication, synthesis of proteoglycans,collagen, osteoblastic proliferation, and increased bone collagen(resulting in increased bone mass).
- Somatomedins also have insulin-like activity on muscle and fat.

ACROMEGALY

- Musculoskeletal manifestations :
  - affect both weight-bearing and non–weight-bearing joints.
  - arthropathy is one of the presenting symptoms in up to 50% to 70% of patients. mainly affecting the lumbar area.
  - Between 60% and 70% of patients experience involvement of large peripheral joints.
  - Back pain may be a particularly troubling feature of acromegaly.
  - proximal muscle weakness, although muscle enzymes are generally normal
  - and biopsy specimens may demonstrate variation in fiber size without inflammation.
  - Fibrous synovial thickening and/or bony overgrowth in the PIP and DIP joints is reported in 50% of patients.
  - Morning stiffness and joint swelling are rare,
  - CTS occurs in approximately 60% of patients with acromegaly and is frequently bilateral.

ACROMEGALY

- When compared with patients with primary OA, MRI studies have shown that cartilage hypertrophy and composition differ significantly in patients with acromegalic arthropathy.
- Arthropathy symptoms are significantly incapacitating and lead to a lower quality of life.
- Symptoms are chronic, and current evidence suggests that they can be persistent even after biochemical control of disease.
- with more than 70% of patients having progressive osteophytosis or joint space narrowing despite successful treatment



Bone Mass and Quality in Acromegaly

- GH stimulates, either directly or indirectly through IGF-1, the proliferation of cells of the osteoblastic lineage and the differentiated function of mature osteoblast.
- It also stimulates the carboxylation of osteocalcin and the production of osteoprotegerin.
- GH hypersecretion has different effects on cortical and trabecular bone;
- whereas bone density tends to be increased in the latter because of periosteal ossification.
- These differences, associated with variations in DXA measurements because of bone enlargement in acromegaly patients,
- are a possible explanation as to why patients with acromegaly experience fractures even in the presence of normal BMD.

Bone Mass and Quality in Acromegaly

- The increased risk of vertebral FRX in acromegaly has been confirmed by several studies,
- which describe its association with hypogonadism, higher IGF-1 levels, longer duration of active disease.
- Incident vertebral fractures occurred in 42% of patients followed up for 3 years during the active phase of disease;
- however,progression of FRX was observed in up to 20% of patients with biochemical control of disease.

HYPOTHALAMIC-GONADAL AXIS

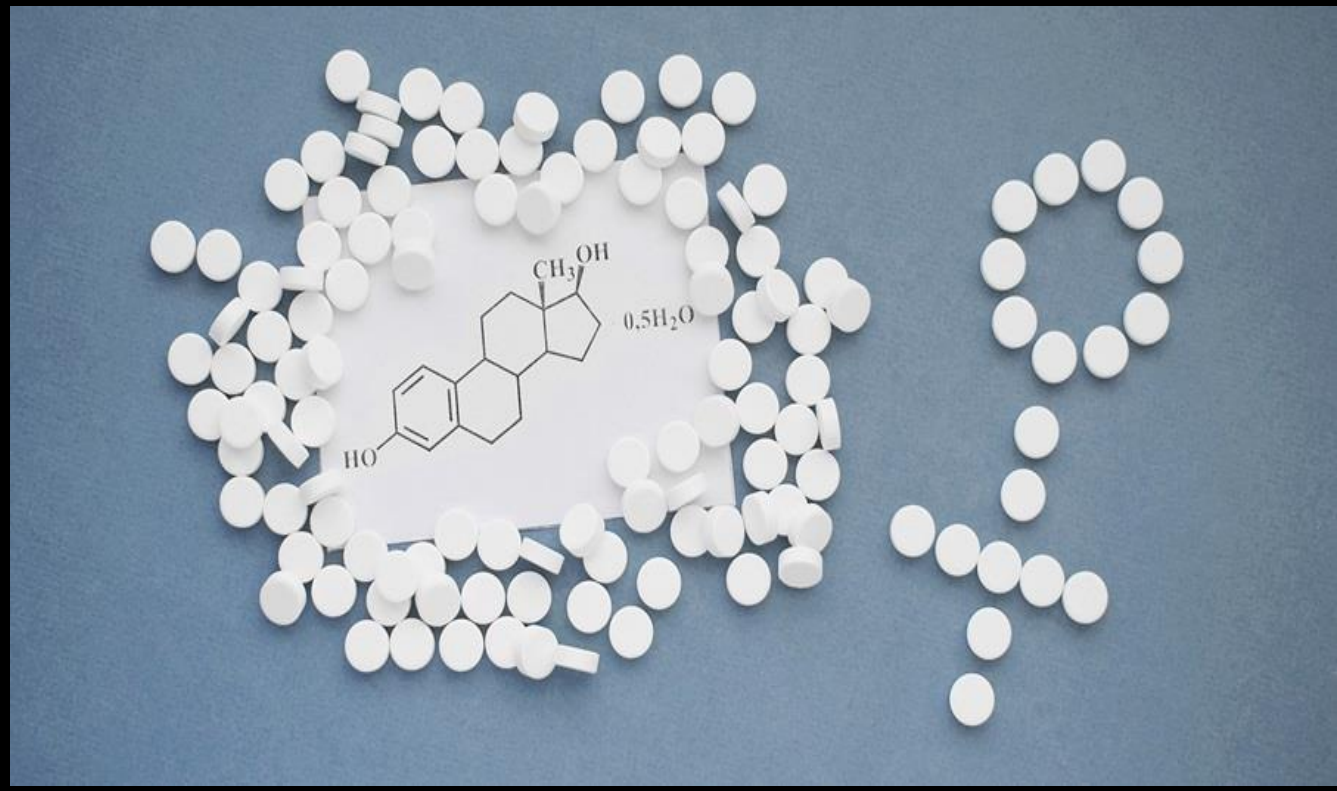
- Many autoimmune disorders are more prevalent in women.
- In RA, the incidence is higher in post-menopausal as opposed to pre-Menopausal women.
- gonadal hormones with protection against RA in younger men (as a result of testosterone) and a predisposition to SLE in women (as a result of estrogens).
- Both sex hormones and genes expressed on sexual chromosomes have been proposed as potential explanations for sex- and age-related effects on musculoskeletal disease presentations.
- This is exemplified in Klinefelter's patients, who have two XX chromosomes (XXY karyotype), reduced levels of androgens, and an increased risk for the Development of autoimmune conditions

Androgens in Rheumatoid Arthritis

- it was only in male patients younger than 50 years with early RA that lower baseline levels of testosterone were found compared with control subjects
- When treated with DMARDs, the males who responded with decreased disease activity also demonstrated significantly increased levels of testosterone.
- The role of androgens in RA susceptibility in women is less well understood,
- Women predisposed to higher androgen concentrations because of a polymorphism in the CYB5A gene are protected from developing seropositive RA.
- Lower androgen levels have occurred in women with RA refractory to treatment.
- Low serum concentrations of androgens have occurred in women with RA,
- but no significant abnormalities have been noted before disease onset.

Ligamentous Injuries and Estrogen

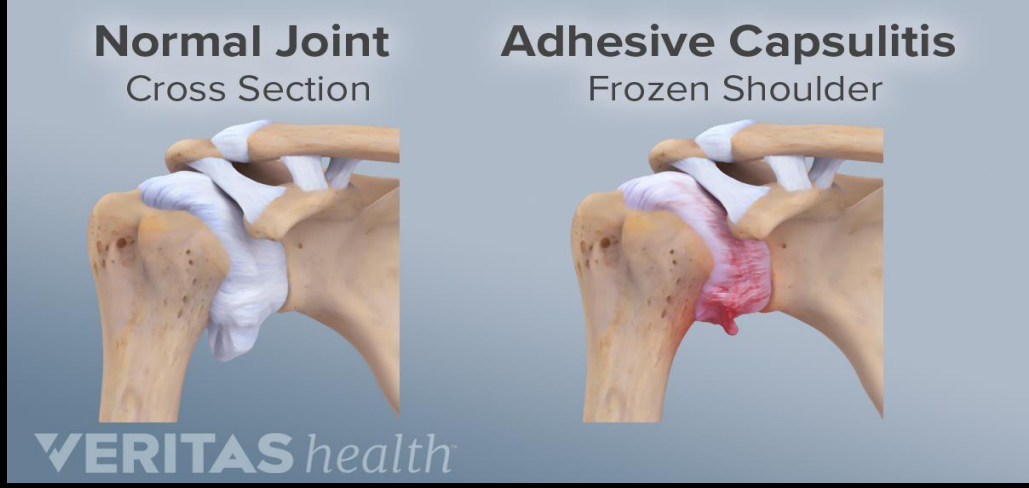
- ❑ Estrogen also fulfills an important role in the development,maturation, aging of bone, muscle, and connective tissue.
- ❑ Increased risk of
  - ✓ musculoskeletal injury,
  - ✓ accelerated bone wasting,
  - ✓ and accelerated muscle wasting
  - • are well characterized in menopause



Ligamentous Injuries and Estrogen

- In general, anterior cruciate ligament (ACL) ruptures occur 2 to 8 times more often in female athletes than their male counterparts.
- One explanation may be the increase in knee laxity during preovulatory and ovulatory phases of the menstrual cycle because knee laxity is directly related to estrogen concentrations.
- Retrospective studies have shown that patients with ACL injuries were less likely to be on oral contraceptives.
- Interestingly, women suffer fewer muscle injuries than men; this is thought to be related to decreased muscle stiffness.

DIABETES MELLITUS



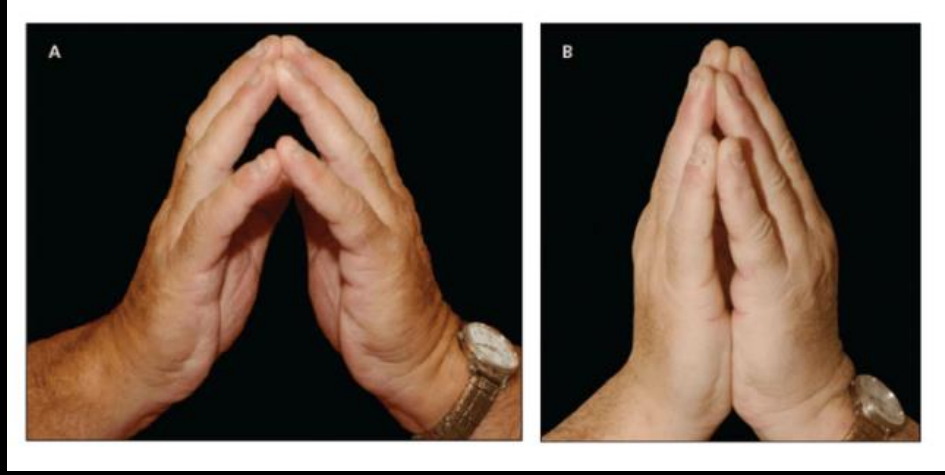
- Shoulder:
- Adhesive capsulitis, or frozen shoulder, represents the most common form of shoulder involvement in diabetes, reportedly occurring in approximately 20% of diabetic patients

➢ Patients generally report shoulder stiffness and impaired external and internal ROM

➢ Another manifestation to consider with diabetics includes calcific shoulder tendonitis, which occurs three times more often in these patients compared with individuals without diabetes but only a third of them experienced any symptoms.

➢ and involves periarticular calcium hydroxyapatite deposition, predominantly in the area of the rotator cuff tendons

DIABETES MELLITUS



- Hands:

❑diabetic “stiff hand” syndrome (or diabetic cheiroarthropathy):

✓ a fibrosing syndrome often resembling scleroderma with features of:

✓thick, tight, and waxy skin—results in contractures at the MCP and PIP joints.

✓ This symptom is classically manifested in the advanced stages of the process by the patient’s inability to press his or her palms together completely without a gap remaining between

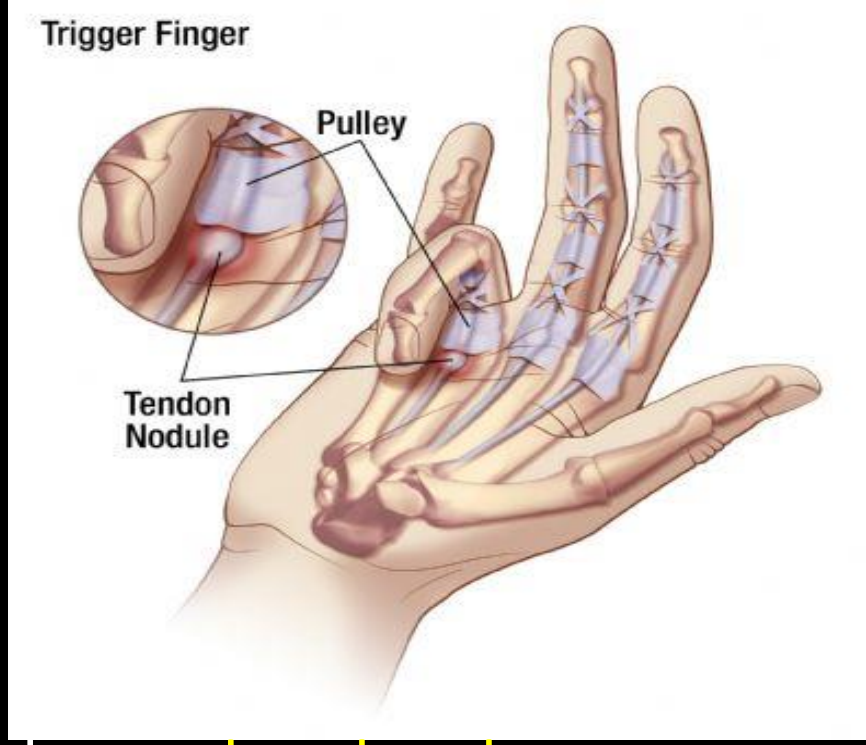
✓opposed palms and fingers and is known as the “prayer sign.”

✓ Larger joints, such as wrists, elbows, knees, and ankles, can be involved.

✓ an increase in frequency with age, disease duration, and poor glycemic control

✓NSAIDs and steroid injections are not effective in management, and glycemic control, as well as physical therapy, is encouraged.

DIABETES MELLITUS



- Hands:

❑Flexor tenosynovitis, or “trigger finger,”

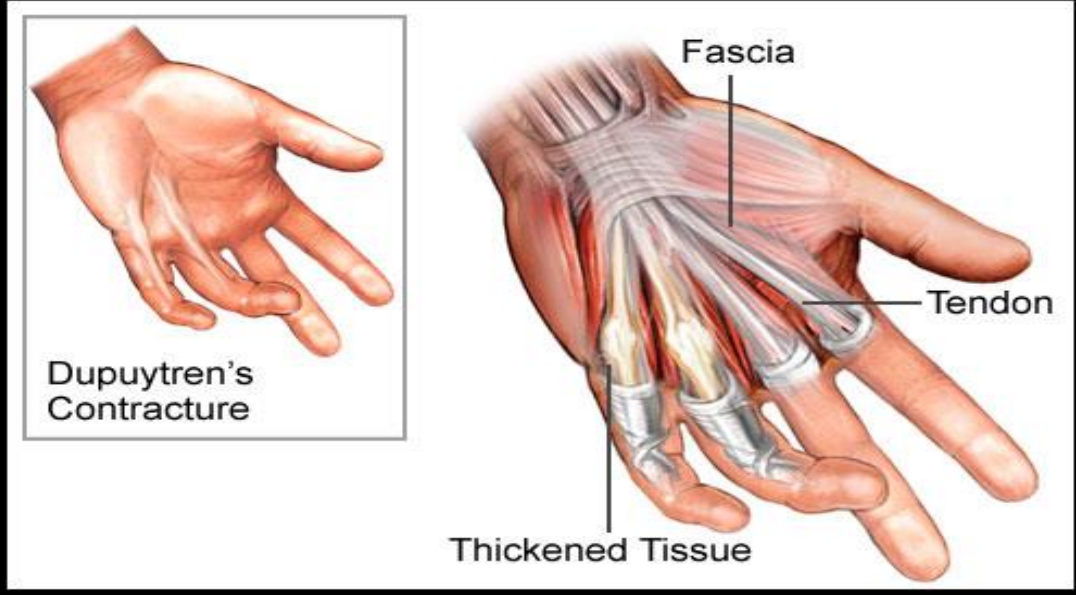
✓ results from the occurrence of fibrosis and thickening of the tendon sheath.

✓which causes pain and ultimately the locking of the involved fingers.

✓ Patients with DM are more likely to have multiple fingers involved simultaneously than non DM patients

✓Management with steroid injections might not be as effective, and patients with multiple digit involvement more often require surgical management.

DIABETES MELLITUS



- Hands:

❑Dupuytren's disease (DD)

✓ is classically described as the thickening of the palmar fascia and flexor tendons, as well as pretendinous bands, palmar or digital nodules, and flexion contractures of fingers, involving the ring and little finger.

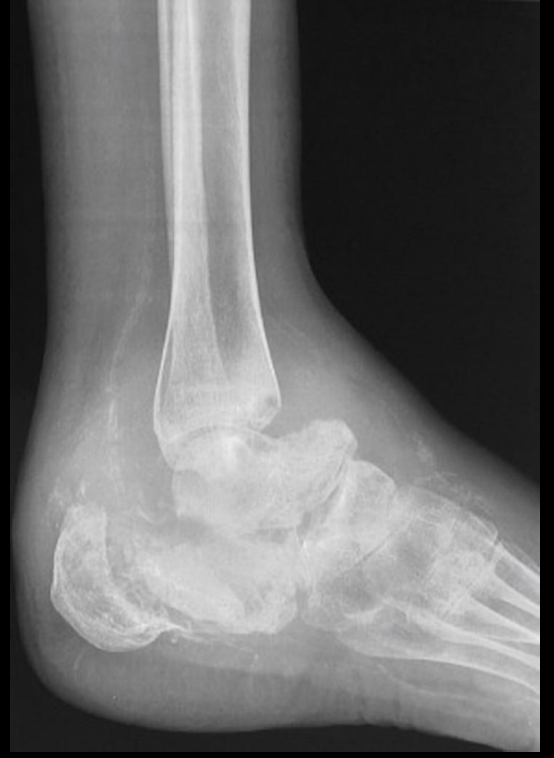
✓ In patients with DM,DD is not only more prevalent, affecting 16% to 42% of individuals,but it also more often involves the middle and ring fingers.

❑Carpal tunnel syndrome (CTS)

✓ is an entrapment neuropathy caused by compression of the median nerve.

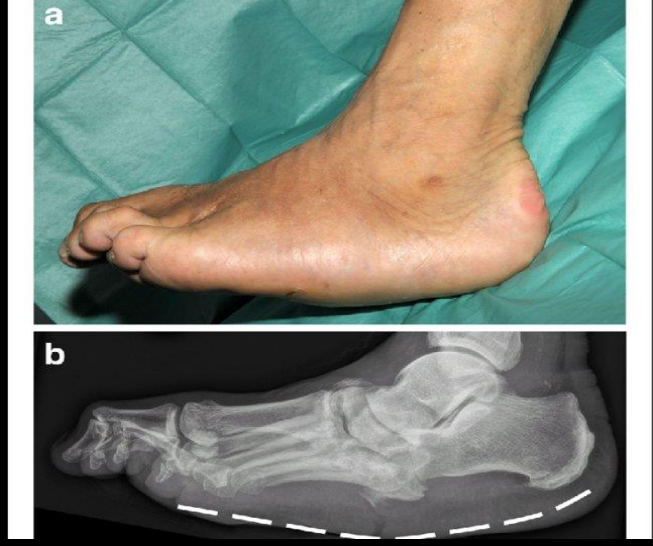
✓ Surgical management outcomes seem to be similar to non-DM patients.

DIABETES MELLITUS Feet



- ❑Charcot's osteoarthropathy (COA), or diabetic osteoarthropathy,
- ✓COA incidence increases with duration of diabetes and diabetic neuropathy,
- ✓ and although it is more common unilaterally, it can involve both extremities in up to 39%
- ❑There are two theories regarding the pathophysiology of COA:
- ✓the neurotraumatic and neurovascular hypotheses.
- ✓The first refers to cumulative trauma as a causative factor, leading to the release of proinflammatory cytokines;which leads to osteoclastogenesis and excessive bone turnover.
- ✓The latter hypothesis proposes that because of autonomic dysfunction, patients with diabetes have increased blood flow to subchondral bone, resulting in increased osteoclastic activity and bone resorption.
- ✓ This phenomenon occurs even in the absence of peripheral vascular disease and creates bone fatigue and disorganization.

DIABETES MELLITUS



- Feet:
- Initially, there is swelling, warmth, and erythema of the foot.
- Involvement of ankle, tarsal, and tarsometatarsal are most common,
- but rarely knees, wrists, shoulder, and intervertebral joints can be affected.

❑"rocker-bottom"

✓Progressive bone resorption, fractures, and dislocation lead to deformity and destabilization of the foot, giving the classic "rocker-bottom" appearance.

✓ In addition to plain radiography, MRI and bone scintigraphy can be useful studies for evaluating the extent of the disease.

✓ The initial approach involves joint stabilization and offloading.

✓ Other measures include antiresorptive therapy and surgery.

DIABETES MELLITUS

- Muscles:
- Diabetic muscle infarction, a rare condition marked by spontaneous infarction of muscle tissue with no preceding injury, occurs largely in patients who are considered "brittle diabetics."

➢ these patients will have accumulated numerous microvascular sequelae from their uncontrolled disease.

➢ Clinically, patients will present with abrupt onset of pain and swelling, usually involving the thigh or calf muscles, and CPK levels may be normal or elevated.

➢ MRI using T2-weighted imaging may be useful in arriving at the diagnosis.

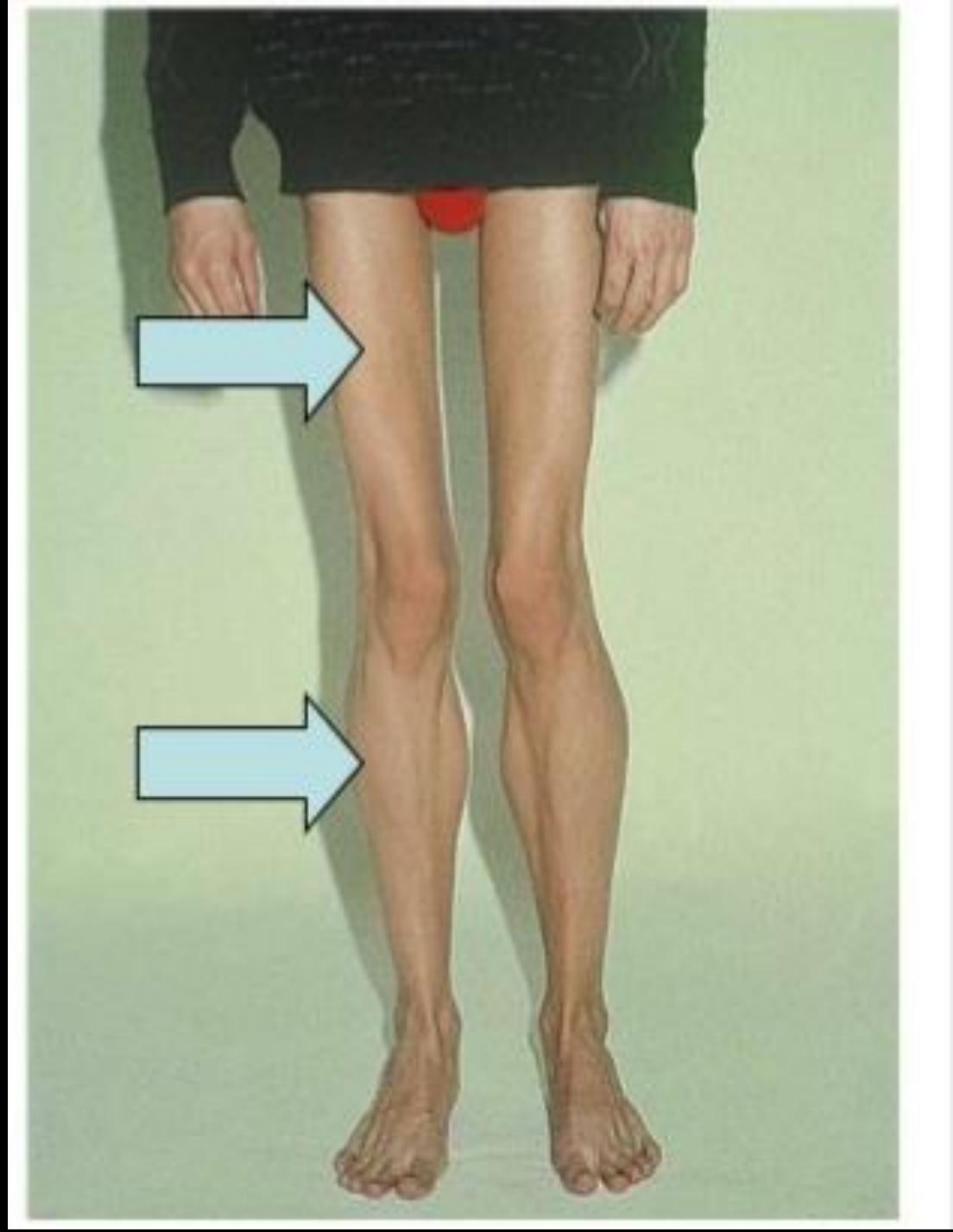
➢ With confirmatory testing involving an incisional muscle biopsy and demonstration of muscle edema and necrosis.

DIABETES MELLITUS

- Muscles:

❑DIABETES amyotrophy

- usually present as weakness involving proximal muscles of the lower extremities and is also associated with pain.
- Treatment usually involves glycemic control and physical therapy.



DIABETES MELLITUS

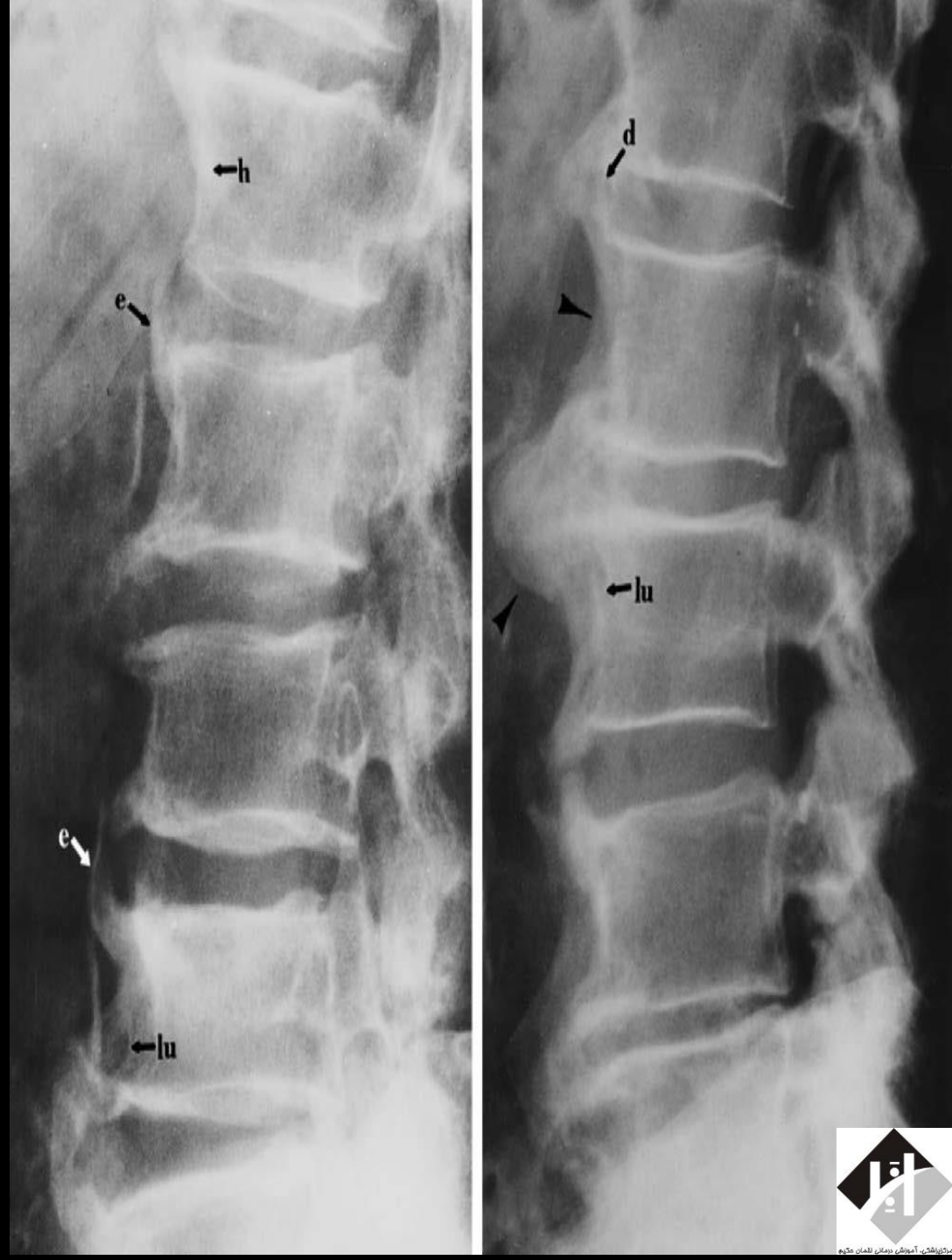
- Diffuse Idiopathic Skeletal Hyperostosis:
- DISH is characterized by ossification of the anterior longitudinal ligament of the spine, can involve various other extraspinal ligaments.

➢ is generally accompanied by osteophyte formation

➢ intervertebral disk spaces and SIJ are commonly spared.

➢ A high incidence of DISH has been demonstrated in a cohort of patients affected by both clinically active and subclinical DM (both type 1 and type 2), as well as in obese patients.

➢ Elevation IGF-I and insulin leads to calcification and ossification in ligaments.



DIABETES MELLITUS

- Diffuse Idiopathic Skeletal Hyperostosis

➢A more recent study found that individuals in :

➢their fifth decade of life are more likely to be affected by DISH

➢if they were obese,

➢had a first-degree relative with either DM or hypertension,

➢had lumbar or thoracic spinal pain,

➢and were affected by enthesopathies or tendonitis

✓The likelihood of relatively young patients with three or more of these clinical parameters being affected by DISH

✓ was six times higher compared with age- and sex-matched control subjects

Summary of Musculoskeletal Complications of D M

❑Shoulder

- Adhesive capsulitis (frozen shoulder)
- Calcific shoulder tendinitis

❑Hand

- diabetic cheiroarthropathy
- trigger finger
- Dupuytren’s disease
- Carpal tunnel syndrome

❑Feet

- Charcot’s osteoarthropathy

❑Muscle Diabetic

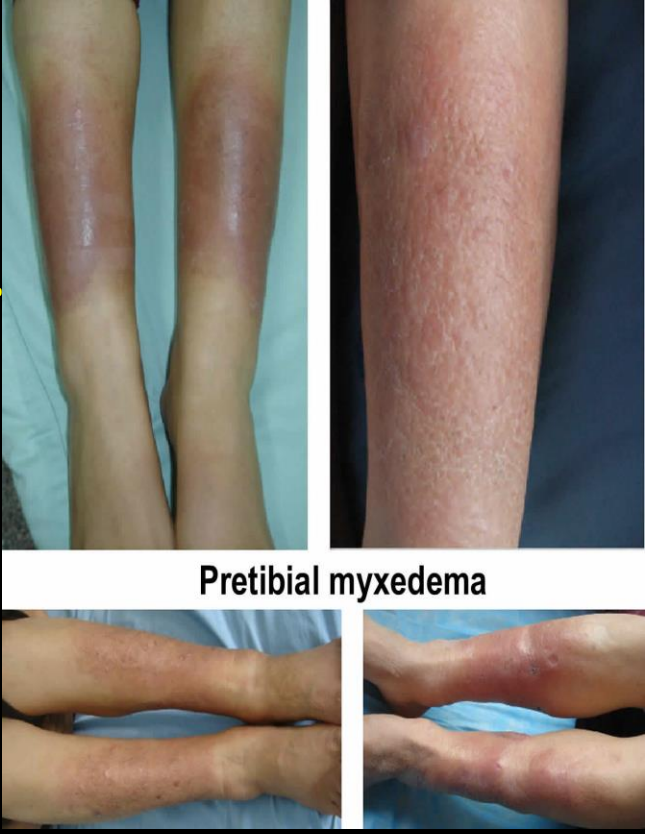
- muscle infarction
- Diabetic amyotrophy

❑Skeletal

- Diffuse idiopathic skeletal hyperostosis

Hyperthyroidism

- Patients with hyperthyroidism because of Graves' disease
- may present with pretibial myxedema and ophthalmopathy.



➢The myxedema appears as:

❖ nodules over the pretibial surface (usually 1 cm or larger)

❖They are composed of hyaluronic acid,

❖are pink to purple in color, and are painless.

➢Patients with Graves' disease can also have :

❖proximal muscle weakness, associated shoulder adhesive capsulitis,

❖ loss of muscle mass, and weight loss.

❖Nail changes, onycholysis,

❖clubbing (thyroid acropachy) may be associated with periostitis around the metacarpal joints, as well as soft tissue swelling of the digits.

❖osteopenia and osteoporosis.

HYPOTHYROIDISM

- Hypothyroidism often presents with a characteristic symmetrical arthropathy involving stiffness of the joints of the hands and knees.

➢Upon palpation, the joints feel “gelatinous,” and aspiration of fluid is usually non-inflammatory and viscous, with high levels of hyaluronic acid.

➢CPPD crystals are often found In patients with hypothyroidism,

➢a myopathy has been reported that presents with pain, cramps, stiffness, proximal weakness, fatigue, elevated CPK levels,

❖abnormal muscle disease on biopsy (atrophy of type II fibers and an increase in type I fibers), and hypercholesterolemia.

HYPOTHYROIDISM

- Hoffman's syndrome is a rare disorder of hypothyroidism
- manifesting with increased muscle mass.

➢Explanations for the effect of thyroid hormone on muscle include

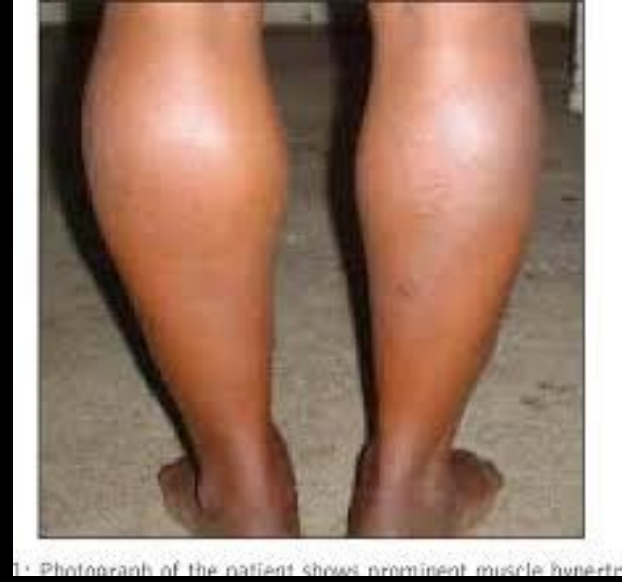
✓delayed contraction and relaxation

✓alteration in myosin heavy chain gene forms

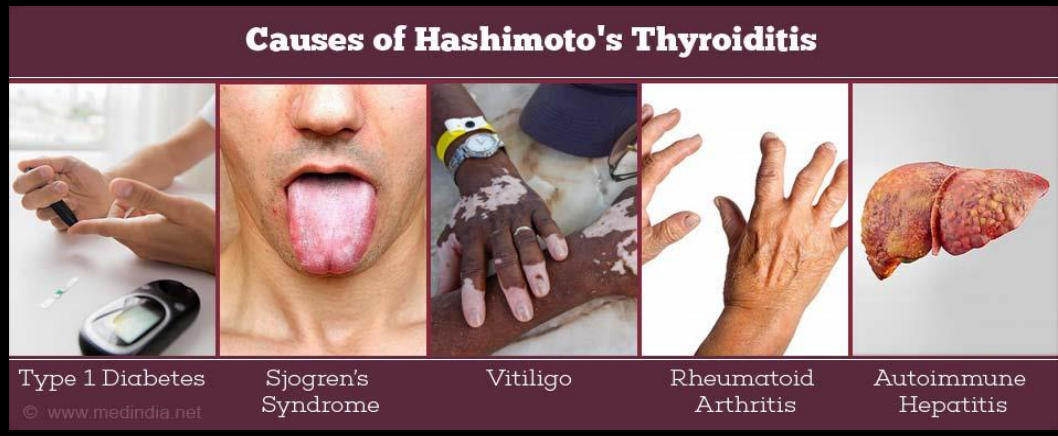
✓and impaired glycogenosis.

➢CTS can also be the initial manifestation of hypothyroidism.

➢The mechanism for compression is thought to be a result of the accumulation of glycosaminoglycans within the surrounding tissues



Hypothyroidism



- Hashimoto's Thyroiditis

➢The most common cause of primary hypothyroidism

in the United States is Hashimoto's thyroiditis

- Patients with HT had a significantly higher prevalence of ANAs than did control subjects
- ANA+ patients were younger than ANA− patients and had significantly higher anti-TG values
- The prevalence of HT in people with RA and SLE was significantly higher than in the control subjects

Chronic Autoimmune Thyroiditis

- ATDis an inflammatory thyroiditis that is sometimes characterized by lymphocytic infiltration of the thyroid gland.
- The presence of arthritis in patients with ATD (even those with normal thyroid function tests) is being increasingly noted.
- ATD encompasses a group of illnesses that include Graves' disease, chronic lymphocytic thyroiditis (CLT),and Hashimoto's disease.
- ✓Synovial effusions can occur and are non-inflammatory.
- Affected joints are knees, MCP joints, PIP joints, and MTP joints.
- Most patients are hypothyroid, joint complaints resolve when treated with thyroid hormone suppression and decrease in TSH

Chronic Autoimmune Thyroiditis

- complaints in patients with ATD include:

- ✓ Carpal tunnel syndrome
- ✓ Chondrocalcinosis
- ✓ Shoulder capsulitis
- ✓ Neck pain
- ✓ Generalized stiffness
- ✓ Myopathy
- ✓ Fibromyalgia

Chronic Autoimmune Thyroiditis

- ✓CLT can present with either hyperthyroidism or, more often, hypothyroidism.Both can have positive ANA autoantibody expression
- ✓In a group of euthyroid patients with CTL and no well-defined connective tissue disease,
- ✓ 98% presented with arthralgia and 59% with fibromyalgia, 28% had Raynaud's disease, and 26% had sicca symptoms.
- ✓Arthritis was radiographically present in 88% of these patients (arthritis of the spine accounted for 45%).
- ✓The authors concluded that rheumatic manifestations frequently occur in patients with CTL(chronic lymphocytic thyroiditis)in the absence of overt thyroid dysfunction.

Medications that affect thyroid function

1. glucocorticoids inhibit TSH secretion, thereby slightly lowering serum thyroid hormone concentrations.

2. Aspirin and NSAIDs may lower serum thyroid hormone by interfering with the binding to their carrier proteins

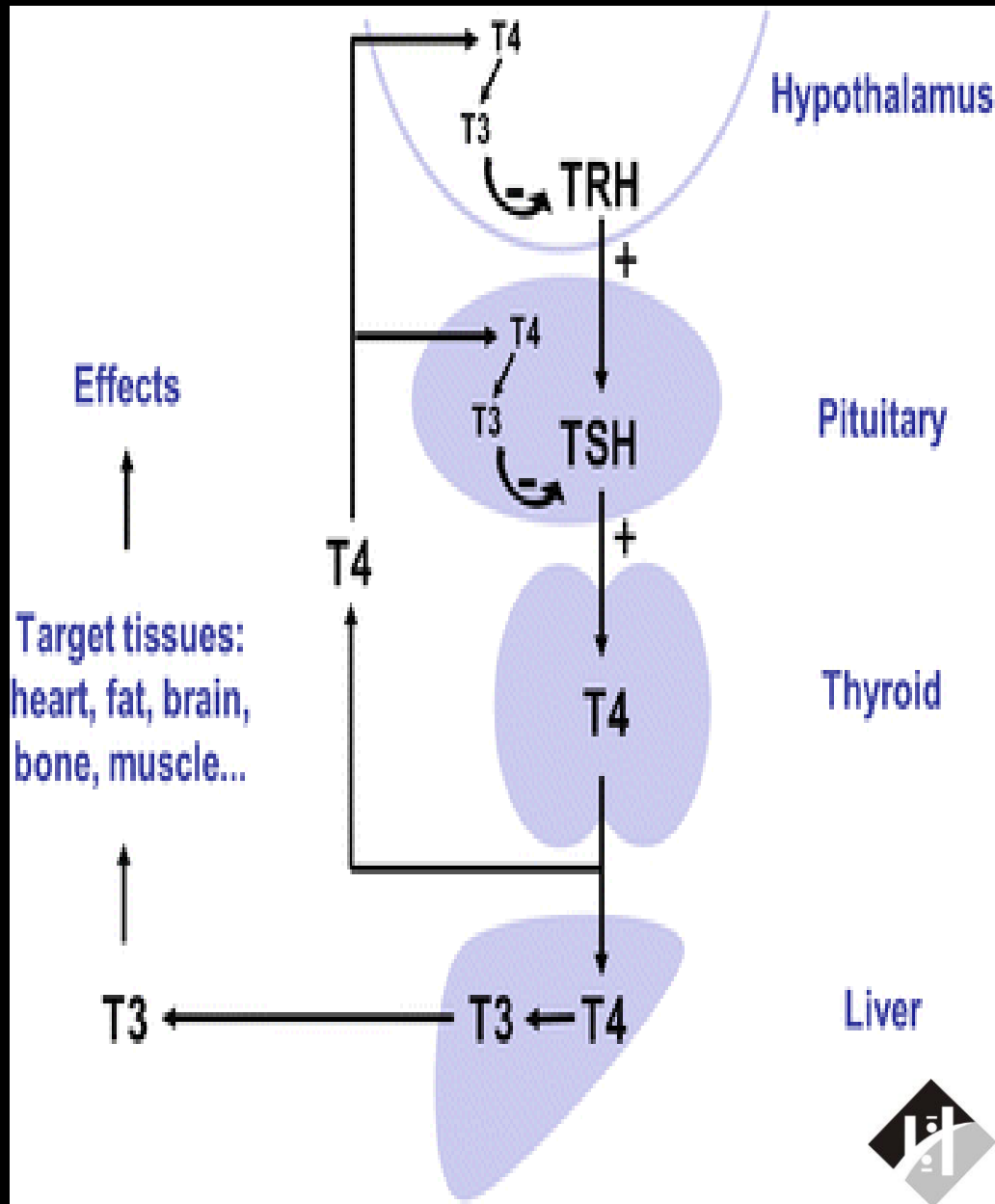
Thyroid Disorders in Patients with Rheumatic Disease

- Systemic Lupus Erythematosus:

➢ Anti-thyroid antibodies are present in 15% to 20% of patients with SLE.

- Concomitant hypothyroidism is five times greater than expected, as opposed to hyperthyroidism, similar to the normal population.

➢ a susceptibility gene shared by patients with SLE and autoimmune thyroid disease.



Thyroid Disorders in Patients with Rheumatic Disease

Systemic Lupus Erythematosus:

- ❑ HT

✓ patients with SLE, there was a twofold increase in HT.

✓ Anti-SM antibodies and antibody to dsDNA favors this association but disease activity are not related to HT or the presence of autoantibodies.

- ❑ Graves' disease

✓ Studies of patients with Graves' disease and chronic autoimmune thyroiditis have demonstrated upward of 26% ANA+ and 34% for anti–single-stranded DNA antibodies.

✓ No patients in these studies had antibodies to ds DNA, anti-Ro-La,anti-SM, or anti-RNP.

Thyroid Disorders in Patients with Rheumatic Disease

- polymyalgia rheumatica or giant cell arthritis:
  - may be more common in patients with autoimmune thyroid disease.
- fibromyalgia:
  - In people with fibromyalgia, an attenuated response of TSH to TRH,
  - as well as an increased prevalence of anti-thyroid Ab compared with control subjects, has been reported.
- Sjögren's Syndrome
  - Symptoms of Sjögren's syndrome, such as conjunctivitis sicca and xerostomia, have been reported in up to 32% of patients with autoimmune thyroiditis.
  - The frequency of HT was found to be higher than in the normal population to however,
  - this increased frequency was not seen for Graves' disease.

Thyroid Disorders in Patients with Rheumatic Disease

❑Rheumatoid Arthritis and Psoriatic Arthritis:

✓an increase in the frequency of anti-thyroid antibodies has been demonstrated compared with control subjects

✓Even though this increase in anti-TPO and anti-TG antibodies

✓ and abnormal variation in TSH level was demonstrated,

✓no significant alteration of hormone levels was found,

✓RA patients, 11.8% with thyroid disease and other comorbid diseases had significantly poorer initial response to RA treatment compared with patients with isolated RA.

Thyroid Disorders in Patients with Rheumatic Disease

❑Scleroderma and Overlap Syndromes

✓fibrosis of the gland can lead to hypothyroidism,

✓ and in this group only 50% of patients had positive tests for anti-thyroid antibodies.

✓Scarce data is available for overlap syndromes.

✓ Anti-thyroid antibodies were found in a quarter of the patients,

✓ and overt hypothyroidism was found in fewer than 20% of patients.

Thyroid Disorders in Patients with Rheumatic Disease

❑Juvenile Idiopathic Arthritis

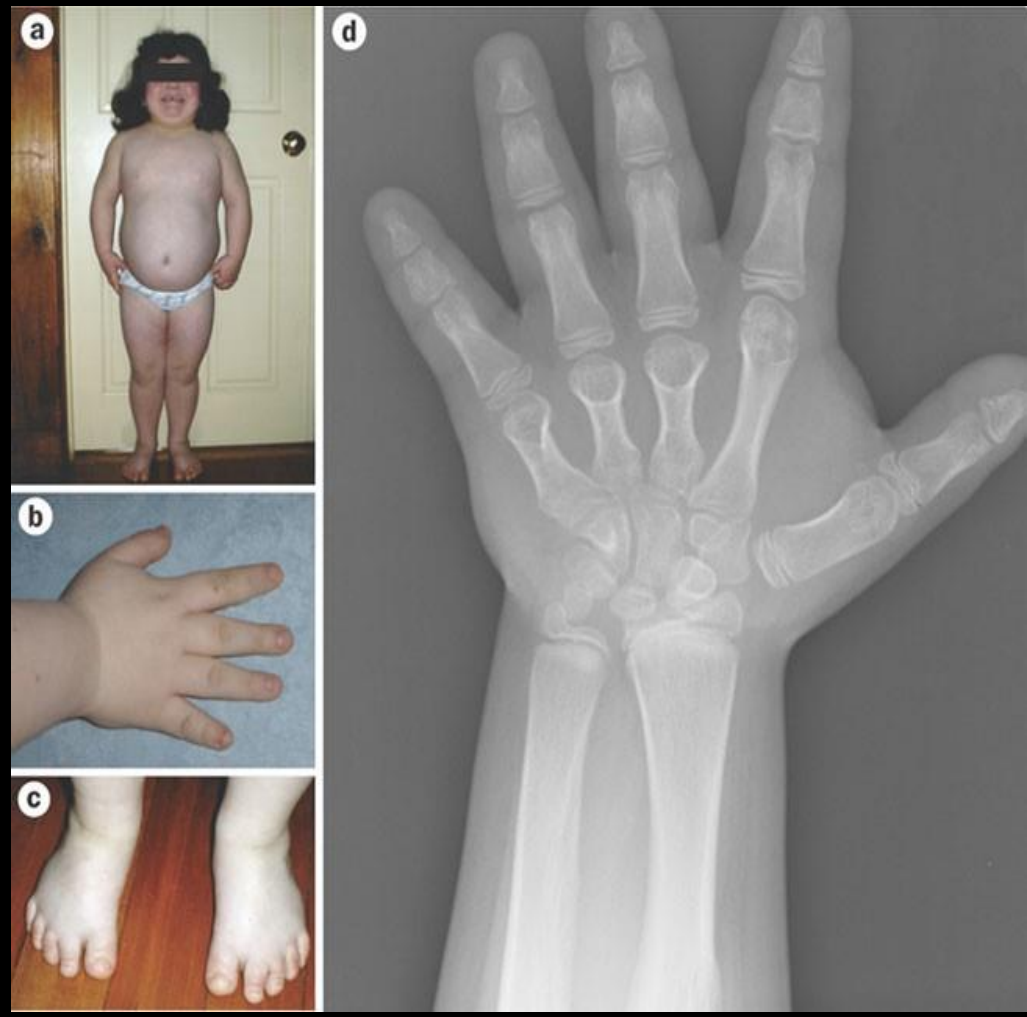
- ✓The investigators concluded that children with JIA have a higher incidence of anti-thyroid antibodies and subclinical hypothyroidism than do normal children.
- ✓ The children who were positive for Ab all had oligoarticular JIA.
- ✓In another study looking at the prevalence of other autoimmune diseases in patients with JIA and their families (more common in first-degree relatives).
- ✓The most common was autoimmune thyroid disease.

Thyroid Disorders in Patients with Rheumatic Disease

- Spondyloarthritis
- In the SpA group, a higher prevalence of HT was found in patients with active disease than in those with low to moderate disease levels.
- Also in the SpA group, patients with a disease duration greater than 2 years had a higher prevalence of HT and anti-TPO antibody positivity than did patients with a disease duration less than 2 years.
- Ultrasonography detected a significantly higher frequency of thyroid nodules and hypoechoic pattern in patients with SpA than in control subjects.
- HT and anti-TPO antibodies were significantly more frequent in patients with SpA who had peripheral involvement than in patients with axial involvement

HYPOPARATHYROIDISM

- Albright's osteodystrophy or pseudo-hypoparathyroidism results from end organ resistance (bone and kidney) to PTH
- and presents with elevated levels of PTH, hypocalcemia, hyperphosphatemia.



❑ Type 1a HoPT (autosomal dominant) is inherited maternally characterized:

- ✓ by calcification of the paraspinal ligaments,
- ✓ and mental retardation.
- ✓ have shortened metatarsal and metacarpal bones
- ✓ and a defect in the genes encoding the α-subunit of the cell membrane– associated guanine nucleotide stimulating unit of adenyl (GNSA1)cyclase.

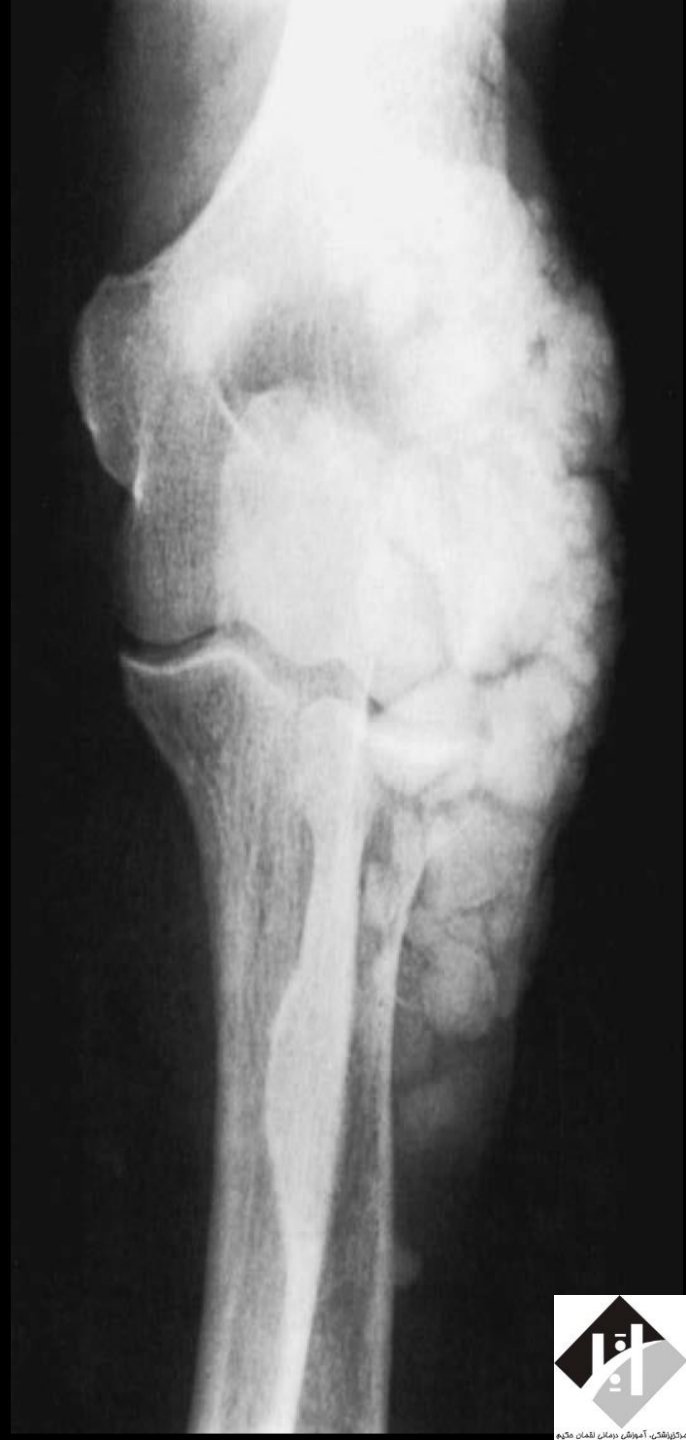
HYPOPARATHYROIDISM

- ❑Type 1b HoPT also has resistance to PTH but has a normal phenotype and is inherited paternally.
- ✓ Soft tissue calcifications not clinically relevant have been reported in HoPT
- ✓ and infrequently in pseudo HoPT.
- ❑Surgically induced HoPT may also be accompanied by muscle weakness that is usually related to the degree of hypocalcemia and is responsive to treatment with vitamin D and calcium

HYPOPARATHYROIDISM

❑crystal deposition

- ✓ Renal disease can result in crystal deposition disease (sodium urate, cppd, basic calcium phosphate hydroxyapatite) as a result of hyperphosphatemia from reduced glomerular filtration and secondary hyperparathyroidism
- ➢ Gout is rare in patients undergoing dialysis but can be seen after renal transplantation with decreased creatinine clearance and use of calcineurin inhibitors(cyclosporine).
- ➢ Hydroxyapatite deposition can cause acute synovitis and periarticular inflammation.
- ➢ Painful subcutaneous nodules or chronic asymptomatic nodules (uremic humoral calcinosis) can occur.
- ➢ Prevention is achieved through phosphate restriction, adequate dialysis, and oral phosphate binding agents.



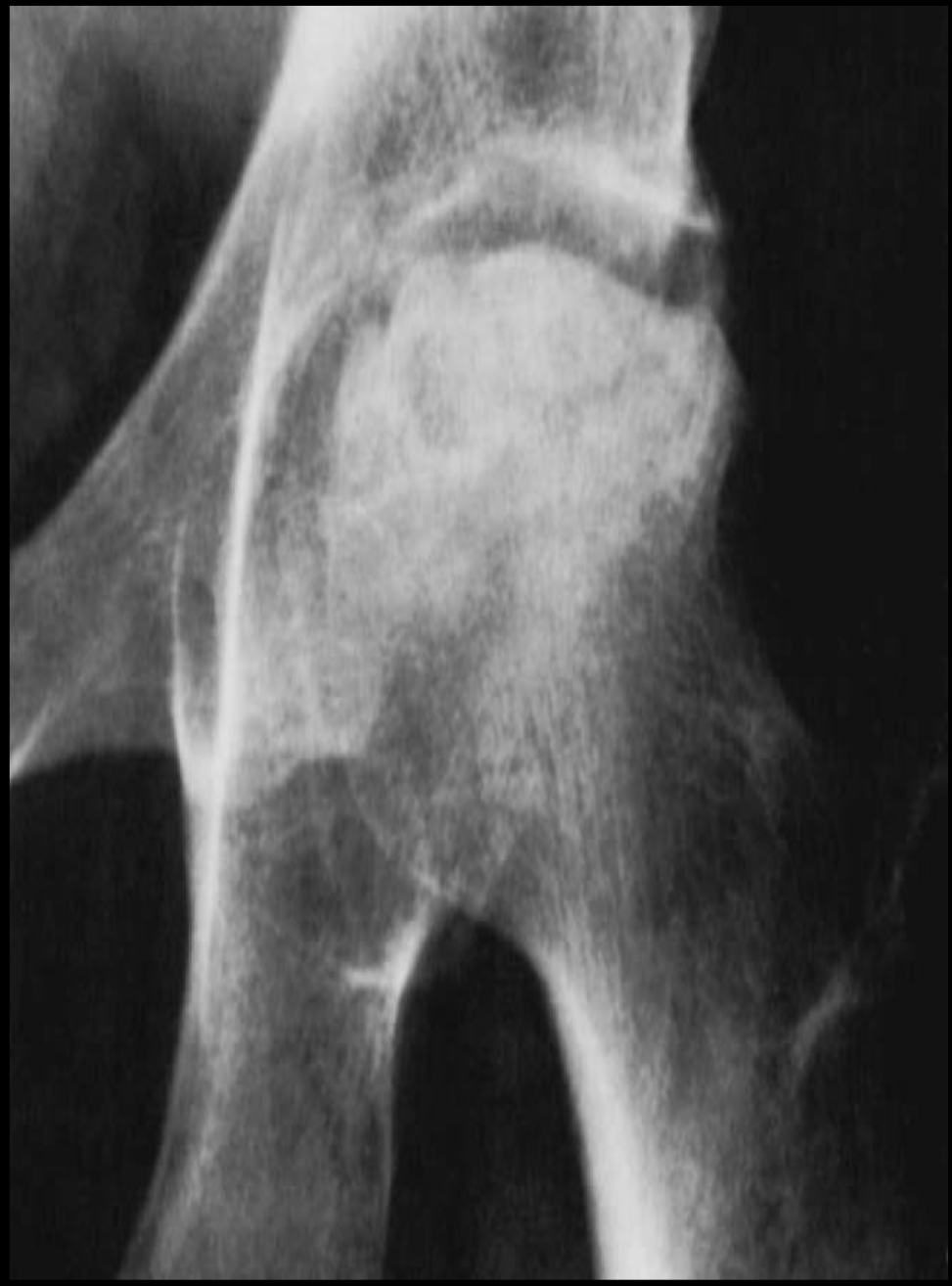
HYPOPARATHYROIDISM

❑Renal osteodystrophy

- is a result of osteomalacia, osteitis fibrosa cystica, osteosclerosis, aluminum toxicity, osteoporosis, and β2 microglobulin amyloid deposition
- presents with bone pain, muscle wasting, and myalgias, as well as bone fractures.

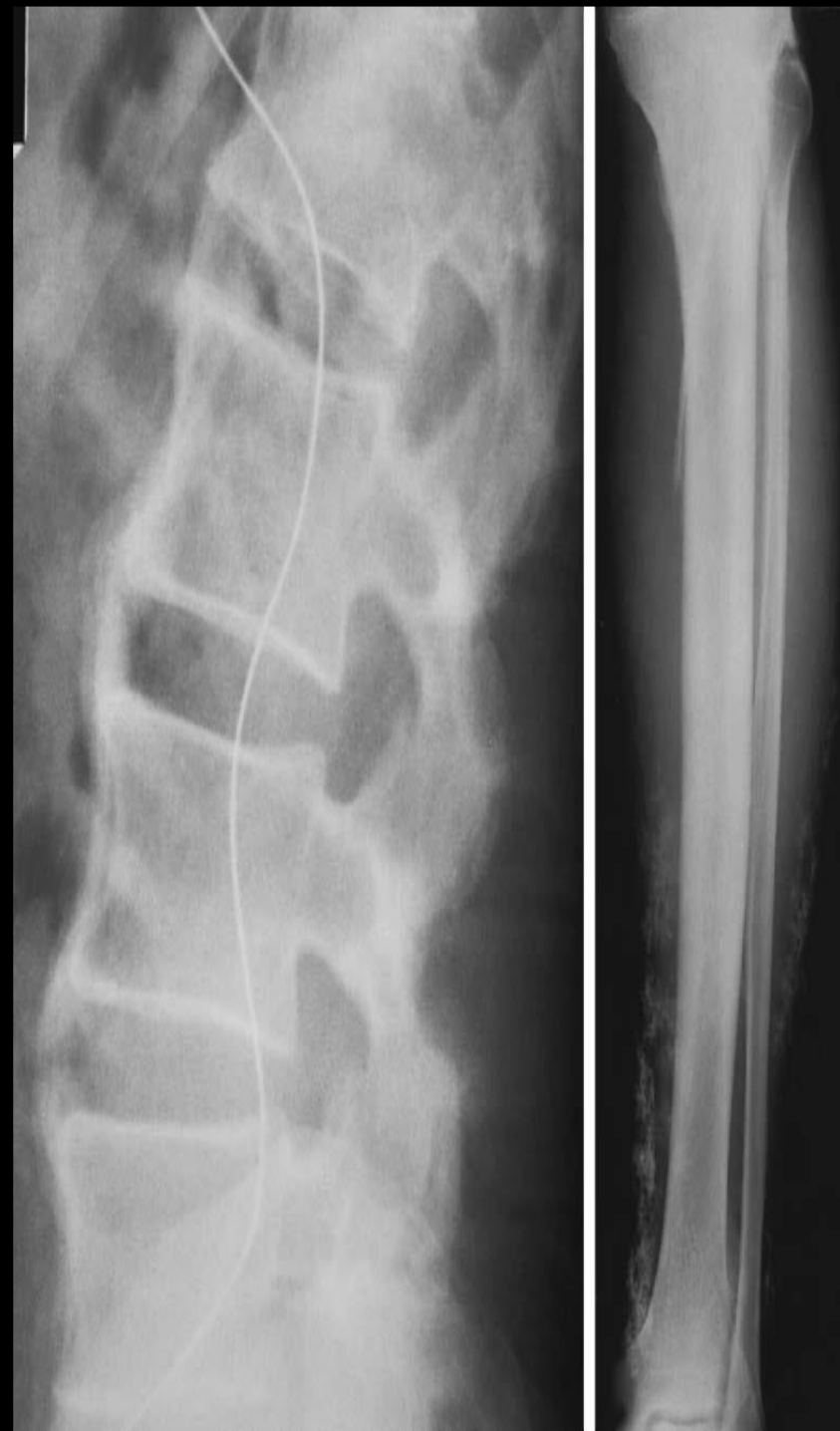
❑ skeletal abnormalities

- The investigators concluded that the abnormal microstructure of the skeleton in hypoparathyroidism reflects the absence of PTH.
- Participants were treated with teriparatide for 2 years and compared with age- and sex-matched control subjects with no abnormalities in parathyroid function.
- Replacement therapy with PTH has the potential to correct these abnormalities and to reduce the requirements for calcium and vitamin D.



HYPOPARATHYROIDISM

- The skeletal abnormalities of hypoparathyroidism are:
- caused by calcification, which can simulate AS with clinical signs, including morning stiffness, gait.
- Sacroiliitis is not expected,
- syndesmophytes in patients with hypoparathyroidism can resemble those of AS with origin from the vertebral margin and preserved disk space, but more often there is also involvement of the posterior paraspinal ligament
- ✓ It is important to differentiate hypoparathyroid-related spondylitis from AS because the management for the two disorders is different.
- ✓ In fact, some of the drugs used for AS(bisphosphonates) may worsen hypocalcemia.



HYPOPARATHYROIDISM

- Spinal changes in hypoparathyroidism have also been described to be similar
- to those in DISH, which is characterized by ossification of the anterior longitudinal ligament of the spine and of various extraspinal ligaments,

✓but is rarely reported before 50 years of age.

✓The pain is not responsive to immunosuppressive agents and NSAIDs but may resolve completely upon treatment with calcitriol.

✓ossifying diathesis of paravertebral ligaments,which is the origin of DISH, might be initiated or aggravated by hypoparathyroidism

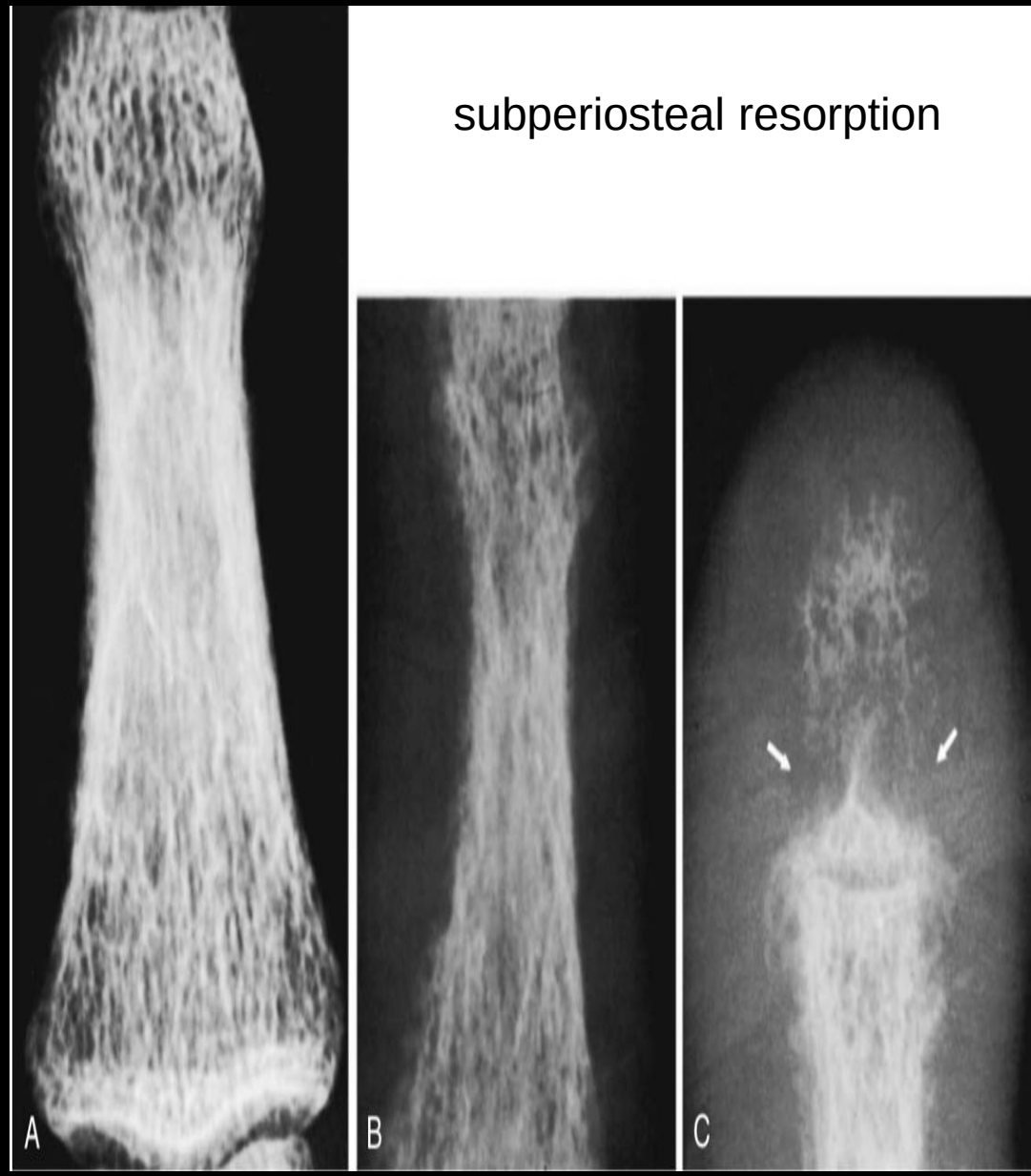
HYPOPARATHYROIDISM

❑SLE and hypoparathyroidism

- ✓hypoparathyroidism has been found to occur in 4.0% to 5.7% of patients with SLE
- ✓acute hypocalcemia typically gives symptoms and signs of musculoskeletal irritability,
- ✓chronic hypocalcemia usually gives mild symptoms and can even be asymptomatic.
- ✓hypocalcemia as a result of QT interval prolongation and the consequent risk of sudden death.
- ✓These cases raise the question of whether periodic calcium and phosphate screening should be routine care for patients with SLE

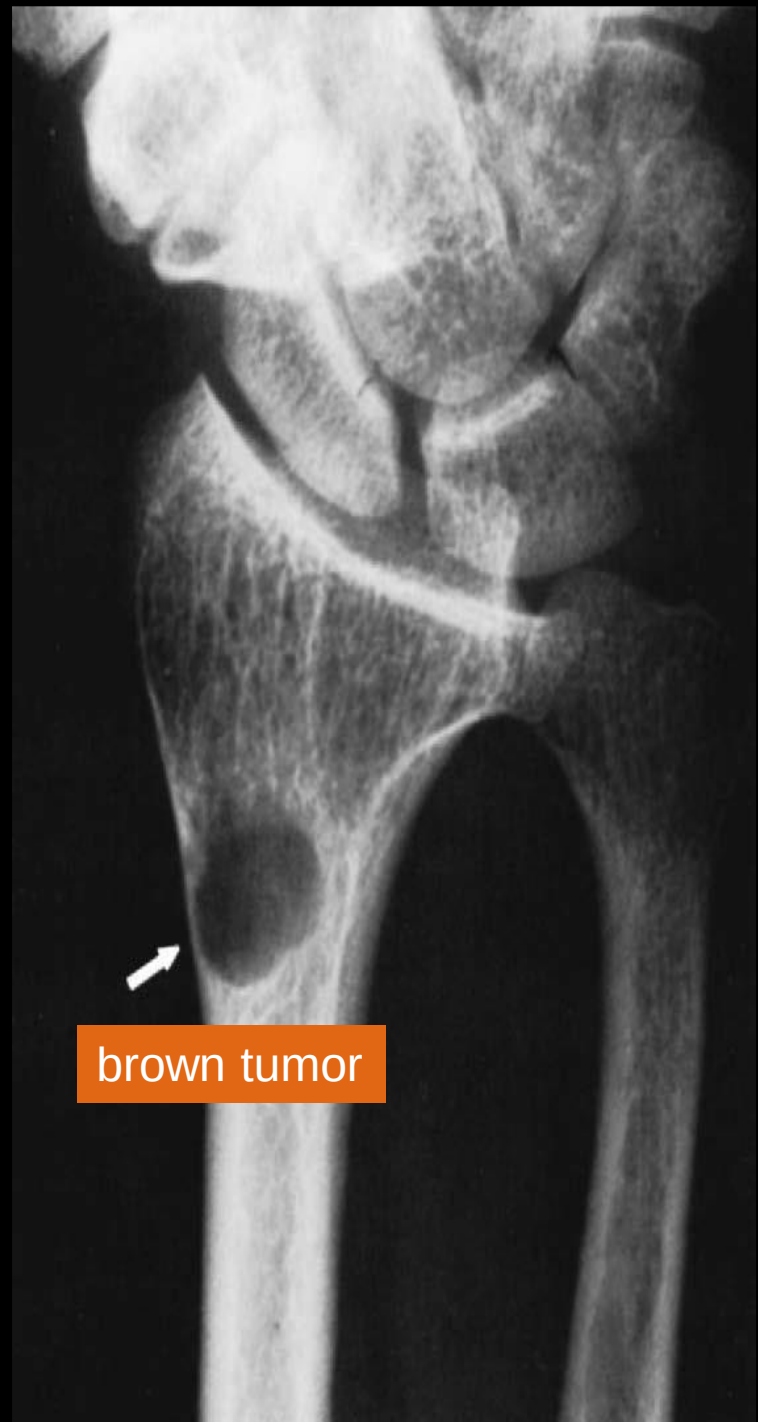
HYPERPARATHYROIDISM

- Secondary HPT is most commonly the result of:
- osteomalacia,vitamin D deficiency, and renal failure.
- Skeletal symptoms include arthralgias,
- as well as radiographic findings such as osteitis fibrosa cystica and subperiosteal resorption along the radial side of the phalanges.
- Arthritis associated with HPT involves the small joints of the hand (possibly becoming erosive) and spares the PIP joints.



HYPERPARATHYROIDISM

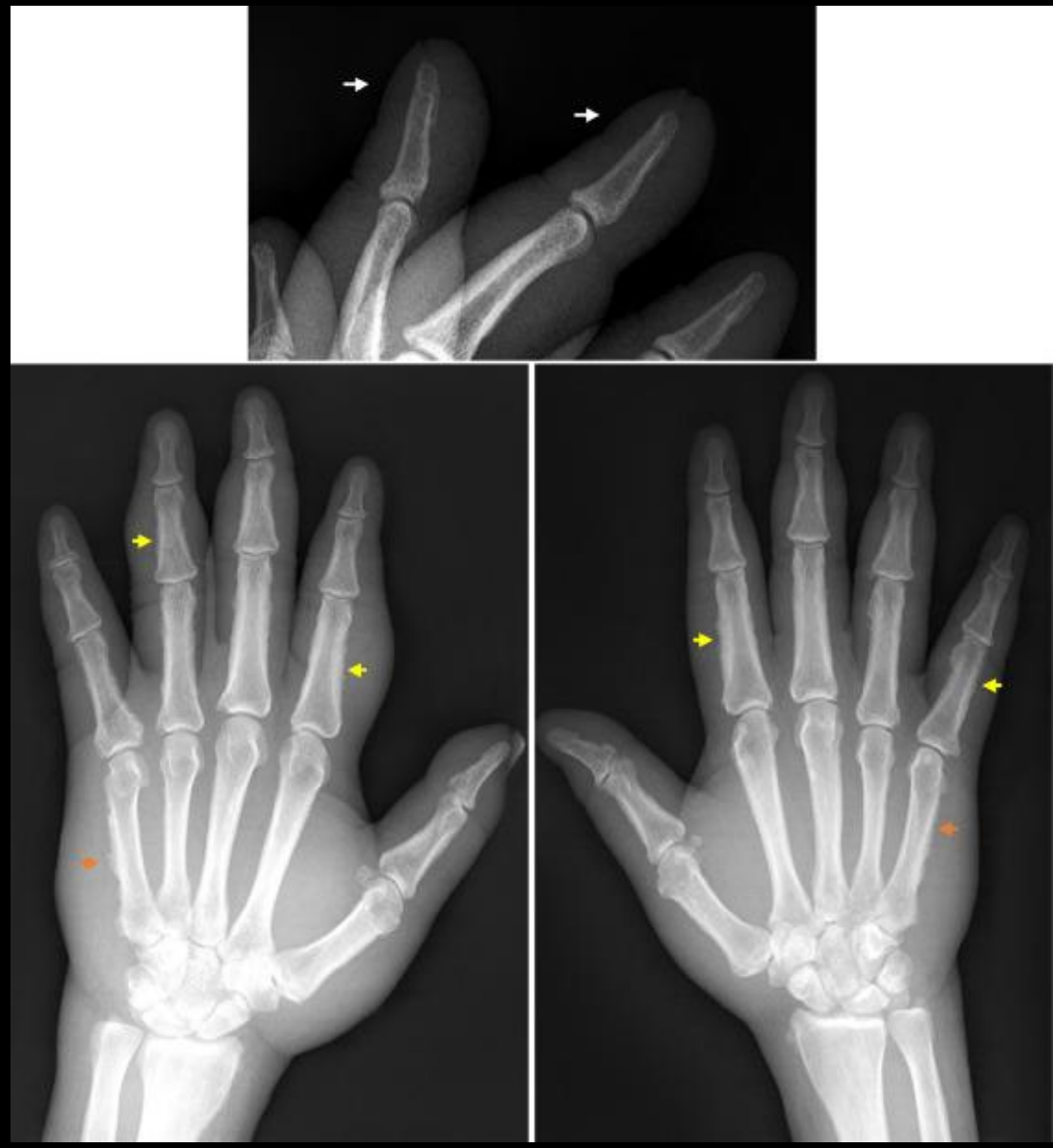
- Lytic lesions resulting from HPT in the skeleton (Brown's tumors) are localized areas of fibrous tissue resulting from increased osteoclastic activity, giant cells, and decomposing blood.
- CPPD (pseudo gout or chondrocalcinosis)is also associated with HPT.
- Muscle involvement can occur in HPT, presenting with proximal muscle weakness. Muscle enzymes are not elevated,
- and both EMG studies and muscle biopsy results demonstrate a picture of denervation.



HYPERPARATHYROIDISM

❑compared with RA.

- In contrast to RA, these patients generally have low ESR rates, negative RF , ACPA , and laboratory values consistent with HPT.
- HPT more commonly involves the radiocarpal,inferior radioulnar, and MCP joints compared with RA.



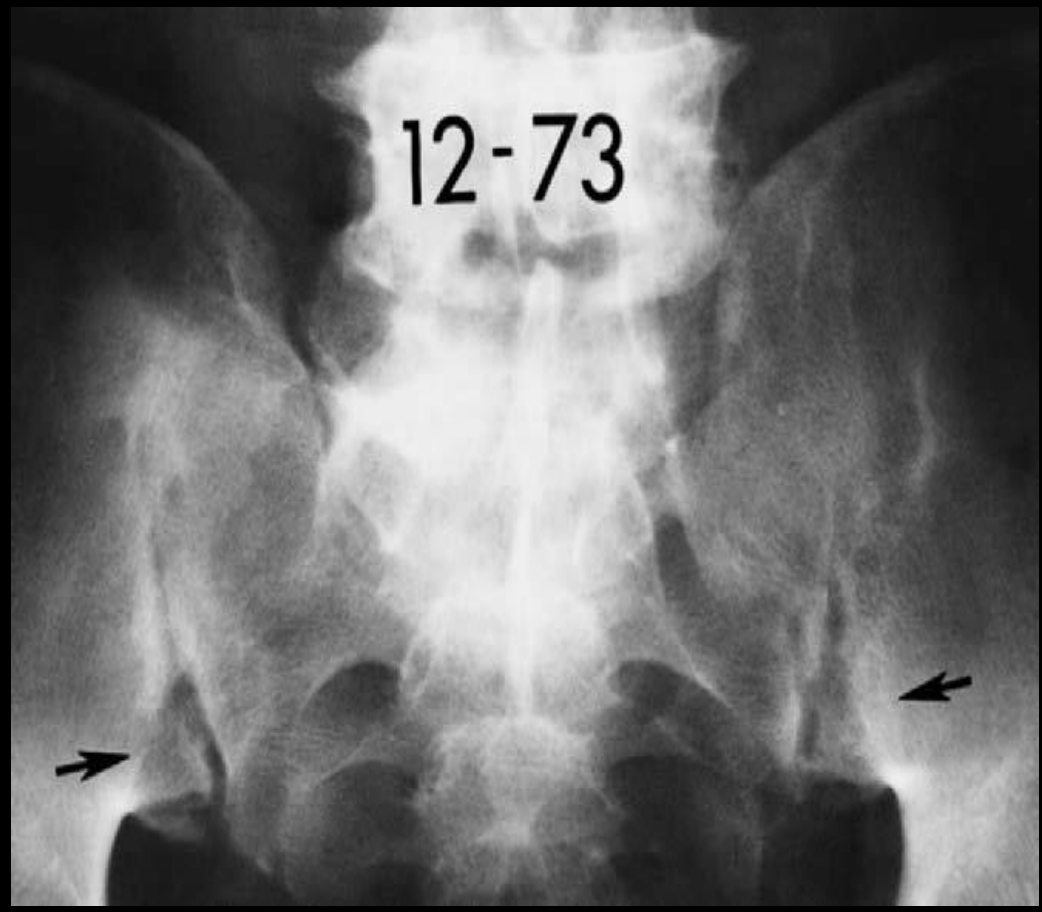
HYPERPARATHYROIDISM

- Early changes in bone such as sacroilitis may occur in HPT.

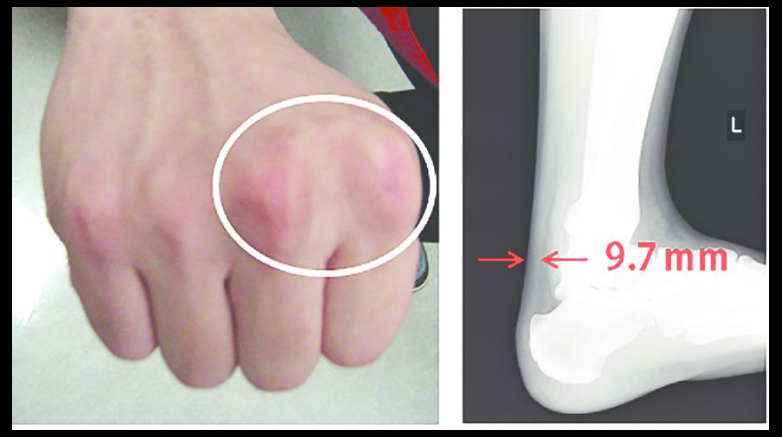
In one study using MRI patients with asymptomatic HPT

- Bone marrow edema was seen in 16.3% of asymptomatic HPT patients and acute inflammatory lesions as well as chronic structural sacroiliac lesions, similar to controls but statistically lower than seen in axial SpA.

- These findings could not be attributed to excessive secretion of PTH hormone.



Fredrickson Hyperlipoproteinemia Classification



- Type 1 → Familial Hyperchylomicronemia
- Type 2A → Familial Hypercholesterolemia
- Type 2B → Familial Combined Hyperlipoproteinemia
- Type 3 → Dysbetalipoproteinemia
- Type 4 → Primary Hypertriglyceridemia
- Type 5 → Mixed Hypertriglyceridemia

Lipid Disorders

- Tendinopathy

➢Tendon xanthomas have been described in dyslipidemia types II and III,

➢type II has been associated with tendinopathy.

➢Tendon xanthomata, which are collections of lipid-laden macrophages typically observed over the Achilles tendons, can be either symptomatic or asymptomatic.

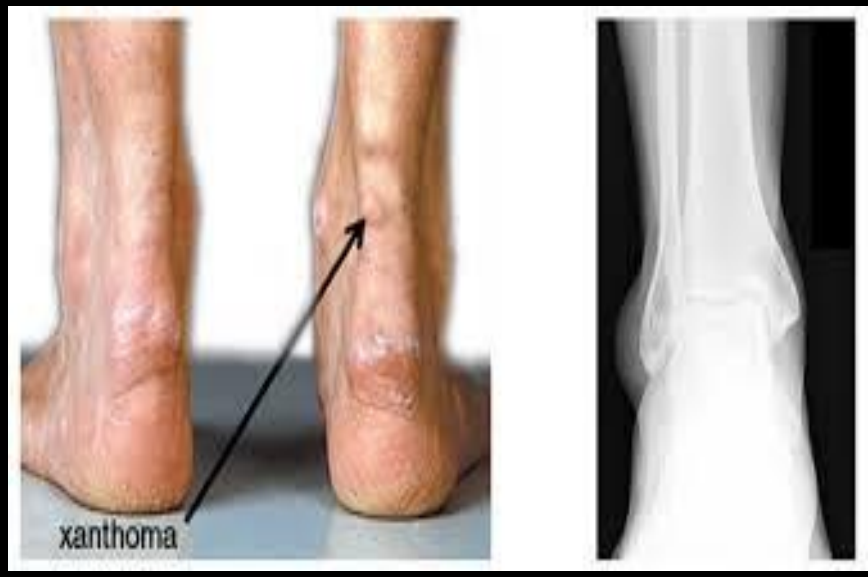
➢ Presence of tendon xanthomata correlate with a 3.2-fold higher risk of cardiovascular disease.

❑ Type III dyslipidemia patients present with:

✓ tuberoeruptive xanthomata

✓and plantar crease xanthomata,

✓ which are typically asymptomatic and do not involve tendons.



Lipid Disorders

- Arthritis

✓several other reports have been published of arthritis in patients with type II dyslipidemia.

❑A case series of 41 patients followed up for 4 years described a transient migratory polyarthritis,which affected both large and small joints in 10 of the patients.

✓ Episodes lasted between 3 to 12 days, with moderate to severe pain.

✓Non-inflammatory synovial fluid was obtained in some cases,

✓supporting the hypothesis that symptoms arise secondary to periarthritis

✓Type IV dyslipidemia patients have also developed episodes of arthritis,

✓ presenting as an acute or subacute pauci-arthritis of large and small joints.

✓Arthritis and musculoskeletal symptoms have responded to lipid-lowering therapy


THANKS FOR YOUR ATTENTION