DISH

- Diffuse Idiopathic Skeletal Hyperostosis
- Calcification and ossification of ligaments, spine and peripheral entheses
- Decreased ROM, Stiffness,dysphagia
- ALL mainly affected , PLL can be affected and can cause spinal stenosis
- Vertebral motion unaffected
- Tx spine most affected (T7-T11) on the right - aortic pulsation

DISH Demographics

- Rare in <50 years old patients
- More males than females
- Usually 3rd and 5th decade of life
- White people affected more than any other race
- **Associated with Diabetes, obesity, gout, hyperlipidemia, HLA-B8 & hypertension**
- No association with HLA-B27

DISH Presentation

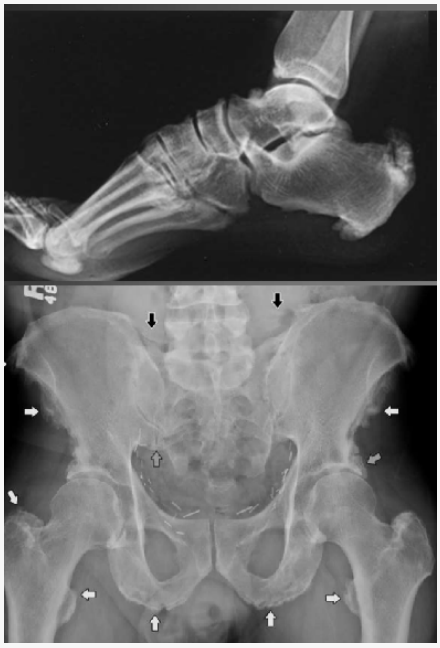
- Pain due to nerve impingement and/or bony growths
- Decreased in ROM
- Dysphagia, hoarseness, sleep apnea if in the cx
- Spinal/extremity pain

DISH Investigations

- CRP, ESR, RF, ANA normal
- AP and lateral X-rays gold standard
- CT and MRI for occult f#

DISH on x-ray

DISH in other areas



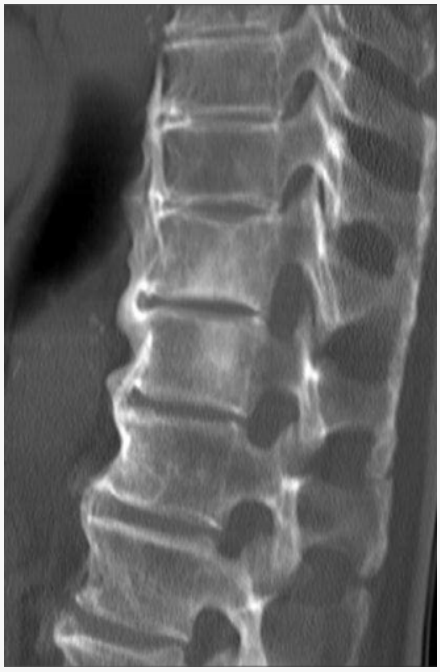
Most commonly in the pelvis, patella, calcaneus, and elbow - can affect any place where there is a ligamentous/tendinous insertion
"Whiskering" of the bone and ossification of ligament/tendon

DISH DDx

- AS
- Spondylosis Deformans (no tx ALL ossification)
- Seronegative spondyloarthropathies
- Charcot Spine
- Acromegaly
- Psoriasis
- Reactive arthritis
- Pseudogout
- Hypoparathyroidism

Management

- Mobilisation and NSAIDs
- Hip and Knee ossification may require surgery if severely affecting ADL
- Exercise (ROM exercises, stretching of muscles, strengthening of muscles)
- Bisphosphonates



Ossification of the Anterior Longitudinal Ligament (ALL)
- Radiolucent horizontal cleft
- Disc height preserved (OA)
- Bony bars
- No sacroiliitis/facet joint involvement (AS)
- Hyperostosis ends from mid-anterior portion of the VB (out and up) on 4 or more contiguous vertebrae
- Looks like flowing candle wax
- Can affect costotransverse, costo-vertebral and other joints

By **Siffi** (Siffi)
cheatography.com/siffi/

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Management (cont)

- Activity modification
- Surgery if: F#, Cx myelopathy, lx stenosis, neurological deficits, infection, painful deformity

Complications

Myelopathy

Cx Radiculopathy

Dysphagia

VB f#

Instability

Heterotopic ossification

Gout

- Most Common cause of chronic inflammatory arthritis
- Build up of uric acid - after breaking down purines
- Sodium urate builds up in joints

Gout Causes/Risk Factors

- Hyperurcemia
- Male (>40yo)
- Obesity/ Hyperlipidaemia
- Purine diet (fish, meats)
- Alcohol/soft drinks
- Medication (diuretics, low dose aspirin, ethambutol, pyrazinamide, cyclosporine)
- Genetics (SLC2A9, ABCG2, SLC22A12, GCKR, PDZK1)
- Kidney disease
- Heart failure
- Metabolic syndromes
- Stress (surgery, trauma, starvation), diet, drugs can trigger a flare up

Gout Presentation

- Usually 1st MTP joint, talar, subtalar, ankle and knee can be affected
- Check tendons and bursas
- Acute onset of joint pain

Gout Presentation (cont)

- Wakes patient up/develops gradually over few hours (reaches max intensity within 24 hours)
- Severe pain - sometimes tender to touch
- Swollen, red, warm joint
- Can also have systemic signs- fever, malaise, fatigue
- Tophi on joints, ears, finger pads, tendons, bursae

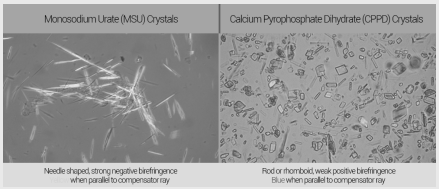
Gout DDx

- CPPD
- Septic Arthritis
- OA
- RA
- Psoriatic arthritis
- Cellulitis

Gout Investigations

- Synovial fluid (yellow and cloudy, crystals and white blood cells)
- Synovial fluid in septic arthritis will be more opaque with yellow-green appearance, higher WBC count and positive gram stain
- Polarising microscopy (needle-shaped, negative birefringent crystals)
- Arthrocentesis (confirms diagnosis and rules out septic arthritis, lyme disease or pseudogout)
- ESR, CRP, serum urate can be elevated
- Urine uric acid
- US (hyperechoic enhancement on the cartilage), DECT

Gout vs CPPD crystals



Above is Gout and CPPD crystals under polarising light microscopy

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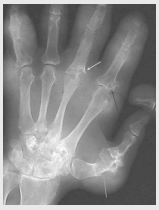
Gout on imaging



Yellow Arrow = Over hanging margin sign
Red Arrow = Marginal Erosions
Green Arrow = Peri-articular Erosion
- Tophi around joints
- Paraarticular erosion + sclerosis and overhanging margin sign
- Feet, hands, elbow mainly affected
- Can destroy the joint if chronic
- "lumpy and bumpy"

Gout Management

- Reducing inflammation + suppresion of serum urate levels

- **Acute**

- Rx should be started within 24 hours of first flare up

- Ice packs, NSAIDs, colchicine, systemic glucocorticoids for 7-10 days

Non-acute:

Urate-lowering therapy (ULT)

Guidelines for ULT:
- Frequent Flares (>2/year)
- CKD stage 2 or more
- Tophus diagnosis on physical exam or imaging
- Past urolithiasis

- Medications including Xanthine Oxidase Inhibitors (XOI) - stops synthesis of uric acid (Allopurinol, febuxostat)
Uricosuric: increases renal urate clearance (Probenecid, Lesinurad)
Interleukin-1 inhibitor: Blocks interleukin-1 (anakinra, canakinumab)

- Modification of lifestyle: limiting alcohol, meats,seafood, sugar, weight loss, drinking more water

Colchicine Contraindications: Older populations, patients with chronic kidney and liver problems, taking other medications that affect cytochrome P450 and P-glycoprotein should stop/modify medication.
Glucocorticoids can be offered instead to the above patients
Colchine side effects: Vomiting, nausea, diarrheoa, myotoxicity, myelosuppression

Gout Prognosis

- Depends on comorbidity
- Mortality higher in people with cardiovascular problems
- Most patients live normal life
- Younger patients have gout more severelyr

Gout Complications

- Tophi
- Joint deformity
- OA
- Bone loss
- Urate nephropathy and nephrolithiasis
- Conjunctivitis
- Uveitis
- Scleritis

HADD

- Hydroxyapatite Deposition Disease
- AKA calcific tendinitis
- Common in shoulder, elbow, wrist, hip, knee, ankle, spine
- Metaplastic transformation of tenocytes to chrondrocytes
- Women 4th and 5th decade of life
- Some can be bilateral
- Critical area of suprapinatus tendon is most commonly affected then lower side of infraspinatous and preinsertional area of the subscapularis tendon

HADD Stages

- **Precalcific stage:** Tendon transforms into fibrocartilaginous tissue

- **Calcific stage:** Calcium deposits
Consists of formative and resorptive phase
Formative: Calcium crystals deposit into the tendon by chondrocytes
Resorptive: Vascular weaving + Macrophages phagocytose calcium, oedema and increased pressure in the tendon, calcium crystals may move into the bursa - **Most painful phase**

- **Postcalcific:** Tendon remodelled by fibroblasts - lasts several months , complete healing occurs

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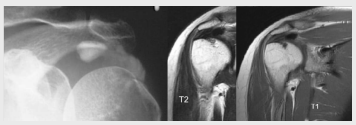
HADD Presentation

- Low grade pain
- Acute/gradual restricted ROM
- Can resolve spontaneously
- +ve shoulder impingement signs
- Can be severe and wake patient up at night

HADD on imaging



Findings

- Toothpaste- Like
- Low signal on both T1 & T2
- Outside articulating surfaces

HADD radiograph appearance

- Type A: Sharply defined, homogenous, dense calcification
- Type B: Sharply defined, dense in appearance, multiple fragments
- Type C: Heterogenous calcification in appearance with dawny deposit
- Type D: Dystrophic calcification in the tendon insertion
- C and D = resorptive phase

HADD Management

- Rest
- Physical therapy (Shoulder ROM, scapular strengthening)
- SMT
- NSAIDs
- Corticosteroid injections
- Shockwave therapy
- US therapy
- Surgery if no better within 6 months (last resort)

Charcot Joint

- Damage of the nerves, injured extremity due to lack of sensation
- Decreased Pain sensation and proprioception
- Joint is swollen + unstable
- Thought to be

Neurotraumatic: Neuropathy + repeated microtrauma = joint destruction

Neurovascular: Increased peripheral blood flow= osteolysis + demineralisation

- Dysfunction between Calcatonin gene related peptide (CGRP), nuclear factor-kB ligand (RANKL) and osteoprotegerin (OPG)

Charcot Stages

Eichenholtz:

- **Stage 0:** Red, hot, swollen foot with no deformity with normal radiographs

Stage I: Erythema, foot oedema, elevated temperature, no pain
Boney debris, fragmenation of subchrondral bone, joint subluxation/-f#/dislocation on X-ray

Stage II: Decreased signs of inflammation
Absorption of boney debris + new bone formation, merging of large fragments with sclerosis of bone ends, stability slowly increases, however x-ray looks worse than stage I

Stage III: Inflammation resolves, changes in foot architecture due to bone remodelling - risk of ulceration due to new pressure points

Charcot Causes

- Diabetic Neuropathy
- Spinal Cord injuries
- Poliomyelitis
- Leprosy
- Syphilis
- Syringomyelia
- Chronic alcoholism
- Charcot Marie Tooth Disease
- Steroids

Knee Involvement: Tabes Dorsalis
Talonavicular/ Tarsometatarsal: Diabetes

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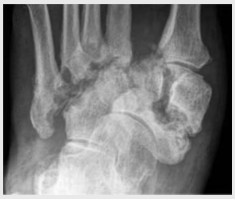
Charcot Presentation

- Erythematous Foot with oedema and calor
- Unilateral, sudden onset after trauma/repetitive microtrauma
- Inflammation
- Gait and biomechanical altercations

Charcot DDx

- Osteomyelitis (can trigger Charcot disease)
- Cellulitis
- Septic Arthritis
- Gout/pseudogout
- Foot/ankle sprain
- F#
- DVT

Charcot in Imaging



- Dislocation + displacement of the joint rules out infection.
- Can present similar to DJD (ghost chrondrocytes, subchondral cysts, sclerotic bone, fragmented and irregular cartilage thinning) and Osteomyelitis
- Bone biopsy and histology to determine charcot joint vs osteomyelitis (OM has plasma cells, lymphocytes, neutrophills + reactive new bone formation + necrosis + capillary fibrosis and proliferation
- CT can also be prescribed - labeled white blood cell nuclear imaging

Findings

The 6 Ds

Distended Joint	Density Increase
Debris	Dislocation
Disorganisation	Destruction

Charcot Management

- In early stages, immbolise foot and restrict weight bearing (crutches, wheelchairs)
- Fractures may heal on their own in a stable position if not stressed
- Bisphosphonates (to inhibit osteoclastic reabsorption)
- Calcitonin supplements
- Pamidronate, Zoledronic acid
- Surgery (although controversial in acute stages)
- Stopping smoking if relevant

Charcot Prognosis

- 8 months recovery time
- Majority develop ulcerations

Charcot Complications

- Foot deformities (flatfoot, rocker bottom foot, hammer toes, ankle equinus contracture)
- Boney prominences - ulcerations, infection, possible amputation
- Condition can reoccur again
- 5 year mortality = 13%

CPPD

- Calcium Pyrophosphate Dihydrate Disease
- Involves synovial and periarticular tissues
- Can cause acute inflammatory reaction (pseudogout)
- Chondrocalcinosis = deposition of crystals in cartilage
- Can also be mistaken for RA
- Affects large, weight -bearing joints

CPPD Causes

- Imbalance between production of pyrophosphate and pyrophosphatases in the cartilage
- Hyperparathyroidism
- Gout
- OA
- RA
- Haemochromatosis
- Osteoporosis

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CPPD Causes (cont)

- Hypomagnesaemia
- Chronic kidney disease
- Calcium supplements
- >65 years old, male

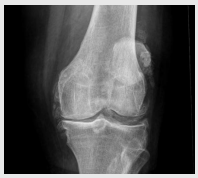
CPPD Presentation

- Symptoms caused by the crystals causing an inflammatory response from the immune system
- Joint Oedema
- Erythma
- Tenderness
- Some can have a low grade fever
- Waxing and waning of non-synchronous inflammatory arthritis in the non-weight bearing joints

CPPD DDx

- Gout
- RA
- AS
- Erosive OA
- Haemochromatosis
- Hyperparathyroidism
- Wilson's disease

CPPD Imaging



CPPD of the Knee
- Calcification in joint compartments
DJD presentation in an unusual location = CPPD
- In scapho - lunate ligament , causes a wide scapho-lunate joint + collapse of wrist
- Arthrocentesis for synovial fluid analysis (rhomboid crystals) + radiography
- US for cartilage abnormalities
- MRI

CPPD Management

- Decrease inflammation and stabilising the underlying disease
- Joint aspiration and intraarticular glucocorticoid if acute and 1-2 joints affected
- >3 joints affected - NSAIDs if contraindicated, colchicine/systemic glucocorticoids
- **Rule out septic arthritis** (synovial fluid cultures)
- Ice packs and joint rest
- If younger patient, screen for metabolic conditions (Hyperparath-yroidism, haemochromatosis and family hx)

CPPD Prognosis

- Self limiting usually involves within days-weeks
- **Complications:**
- Degradation of menisci and synovial tissue
- Gout Tophi
- Spinal involvement (Mistaken for AS, DISH)

Refer if

- Unclear aetiology with hyperuricemia
- Unclear aetiology with normal serum urate levels
- Patients with renal impairment
- Failed trial of XOI
- Multiple side effects from medications
- Refractory gout (Level 1)

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