GOUT & PSEUDOGOUT

HOWARD L. FEINBERG, D.O., F.A.C.O.I., F.A.C.R. ACOI BOARD REVIEW 2019

GOUT

- Hyperuricemia is not gout
- Gout typically follows years of asymptomatic hyperuricemia
- Serum urate increased by alcohol, height, body weight, age, blood pressure, BUN, creatnine

 13.6/1000 in men
 6.4/1000 in women
 estrogen causes increased uric acid excretion

CLASSIFICATION CRITERIA

Step 1: Entry Criteria – swelling pain and redness in a peripheral joint or bursa
 Step 2: Sufficient criterion (if met does not require other criteria) – MSU crystals in a symptomatic joint or bursa
 Step 3: Apply criteria if step 2 is not met

CLASSIFICATION CRITERIA (Requires 9 points)

- Characteristics (1-3 points)
 - Erythema over joint
 - Can't bear touch
 - Inability to walk or use joint
- Time Course (one episode
 - 1 point, recurrent 2 points)
 - <24 hours</p>
 - Resolves in <14 days
 - Complete resolution between episodes
- Evidence of Tophus (4 points)

Serum Urate

- <4 (-4 points)
- 6-8 (2 points)
- 8-10 (3 points)
- > 10 (4 points)
- MSU negative (-2 points)
- Imaging
 - Urate deposit evidence (4 points)
 - Typical damage evidence (4 points)

ASSOCIATED CONDITIONS

Obesity
Ethanol
Diabetes Mellitus
Hypertriglyceridemia
Hypertension
Hypothyroidisim

Atherosclerosis
Metabolic Syndrome
Pregnancy
Acute Illness
Dehydration
Psoriasis

NEGATIVE ASSOCIATIONS

Rheumatoid Arthritis
 SLE
 Ankylosing Spondylitis

CLINICAL

Asymptomatic Hyperuricemia
Acute Gout
Intercurrent Period
Acute Gout
Chronic Gout

PRESENTATION



PATHOLOGY

Monosodium Urate Crystals are formed when the bodies capacity to store uric acid is surpassed Uric acid is a byproduct of purine metabolism

Serum saturation 6.7mg/dl



TOPHI



RADIOGRAPHIC FINDINGS





Hyperuricemia

- Primary Hyperuricemia Hyperuricemia which is not caused by or secondary to another disorder.
- Idiopathic
 underexcretion 90%
 overproduction 10%

<u>Secondary</u>
 <u>Hyperuricemia</u>
 Hyperuricemia which occurs as a result of a drug effect or is secondary to another disease

OVERPRODUCTION PRIMARY HYPERURICEMIA

 HGPRT Deficiency (Hypoxanthine Guanine Phosphoribosyltransferase Deficiency)
 PRPP Synthetase Superactivity (Phosphoribosylpyrophosphate synthetase superactivity)
 G-6-P-D Deficiency
 Fructose-1-Phosphate Aldolase Deficiency

OVERPRODUCTION SECONDARY HYPERURICEMIA

- Diet
- Myeloproliferative Disorders
- Lymphoproliferative Disorders
- Accelerated ATP Degradation
- Glycogen Storage
 Disease (type I, III,
 V, VII)

Severe Muscle Exertion Hemolytic Disease Psoriasis G-6-PD Deficiency Fructose-1-**Phosphate Aldolase** Deficiency HGPRT Deficiency

Under Excretion

- 1º Hyperuricemia
 - Idiopathic

2º Hyperuricemia

- inhibition of tubular urate secretion (DKA, lactic acidosis, Maple Syrup Urine Disease, Alcoholic Ketosis)
- enhanced tubular reabsorbtion (dehydration, diuretics)

Unknown Mechanism

- Hypertension
- Lead
- Hyperparathyroid
- DrugsCyclosporine
 - ASA
 - Ethambutol
 - Pyrazidamide
 - Ethanol
 - Nicotinic Acid

Combined Overproduction & Underexcretion

Glucose - 6- Phosphatase Deficiency
 Fructose -1-phosphate aldolase deficiency

INDICATIONS FOR TREATMENT

Acute Gout
Tophi
Uric Acid Stones
Uric Acid Nephropathy
Interstitial Nephritis

TREATMENT GOALS

Stop acute attacks
Resolve Tophi
Prevent joint damage
Decrease uric acid below 6.0

TREATMENT

Acute

- colchicine
- Indomethacin
- Other NSAID
- Steroid
- Pain Medication
- ACTH
- Joint Injection
- Anakinra (Kineret)
 Interleukin-1 receptor antagonist

Chronic

- Allopurinol
- Febuxostat
- Probenecid
- NSAID
- Colchicine
- Sulfinpyrazone
- Pegloticase (Krystexxa)
- Anakinra ? (Kineret)

CALCIUM PYROPHOSPHATE

Common name Pseudogout
 Occurs exclusively in and around joints
 May be asymptomatic or cause disease

CLINICAL PRESENTATIONS Acute

- similar to gout
- may have fever, leukocytosis, elevated ESR
- Chronic
 - similar to OA
 - symmetrical
 - mainly in knees, wrists, hips
 - isolated patellofemoral disease

CLINICAL PRESENTATIONS

- Polyarticular-may mimic Rheumatoid Arthritis
- Oligoarticular-usually elderly
 Pyrophosphate Arthropathy

 Early-mimics Osteoarthritis
 Late-Charcot Joint
- Precocious Osteoarthritis

CHONDROCALCINOSIS

Rheumatoid 5%

10% RF positive

Gout 25%
OA 50%
Asymptomatic 20%

Present in

4% of adult population

- 50% over age 90

EPIDEMIOLOGY

Hereditary autosomal dominant Post Traumatic Sporadic-rare under age 40 Osteochondrodysplasia

2° To Metabolic Disease

- hemachromatosis
- hyperparathyroid
- hypothyroid
- amyloid
- hypomagnesemia
- hypophosphatemia
- Rickets
- Familial hypocalcuric hypocalcemia

RADIOGRAPHIC FINDINGS

Chondrocalcinosis Crowned Dens - neck pain due to crystal deposits surrounding dens Cord compression Wrap Around Patella Erosive OA



DIAGNOSIS

Definite

- crystals in joint
- Probable
 - other calcium crystals in joint
- Possible
 - X-Ray findings
 - Typical joint distribution
 - History

CALCIUM PYROPHOSPHATE



PATHOLOGY Normal serum phosphate Normal phosphate excretion Elevated levels of inorganic phosphate in synovial fluid NTPPPHase = Cause (Nucleoside triphosphate pyrophosphohydrolase)

TREATMENT

NSAID Colchicine Steroids Physical Therapy Surgery Joint Injections

APATITE -LIKE CRYSTALS

Carbonate substituted apatite
 Octacalcium Phosphate (OCP)
 Tricalcium phosphate (TCP)
 Dicalcium phosphate dihydrate (brushite)

APATITE - LIKE DISEASE Bursitis Crystals not visible on microscopy Tendonitis Alizaren Red-stain Arthritis red Renal Failure von Kossa-stain epiphyseal black dysplasia precise ID requires destructive OA x-ray diffraction

Contact Information

Howard Feinberg, D.O., F.A.C.O.I., F.A.C.R. 1310 Club Drive Vallejo, CA 94592

Howard.Feinberg@TU.edu