

MEDICAL AND SURGICAL MANAGEMENT OF PEDAL GOUT

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INTRODUCTION

Gout is a form of arthritis that has been associated with famous individuals throughout history that have had episodic attacks of pain, swelling, and disability of their feet. Hippocrates believed gout was the result of phlegm, one of the body's humors which settled out in the joints.¹ He and others over the ages have attributed gout to the "fine life" and excesses of food, wine, and sex.

Podagra is of Greek derivation, from podus meaning foot and agra meaning attack.² Examples of gout can be found in Roman times with the description of the demon goddess Podagra sent to Earth to cripple all the doctors who have sent her from Earth to overcome her evil. Gout is a derivation of the Latin term gutta meaning drop reflecting the belief that the disease is the result of a drop of poison or malevolent humor dropping into the joint yielding exquisite pain.³

Aretaeus, a contemporary of Galen was the first to suggest a specific "toxic substance" in the blood that was responsible for gout. Aretaeus also provided us with one of the first descriptions of acute gout in the great toe: "The joints begin to be affected in this way: pain seizes the great toe, then the forepart of the heel on which we rest; next it comes into the arch of the foot, but the ankle joint swells last of all. All sufferers at first wish to blame the wrong cause - some friction of a new shoe, others a long walk or others - an accident or being trodden upon ... but the true cause is seldom believed by the patient when he hears it from the physician."¹

It was not until the 19th century that Sir Alfred Garrod identified uric acid as the specific agent that inflames the joints with gout. He also devised a uric acid thread test wherein uric acid could be identified in the serum of patients suffering with gout.⁴ Garrod recognized the relationship between gout and the renal pathology including calculus.

During this time, the use of colchicine also became widespread. Numerous historic figures also suffered with gout including Michelangelo, Sir Isaac Newton, Martin Luther, and Benjamin Franklin. Henry VII had to

postpone his marriage due to an acute attack of gout, which was also a disease found in his descendants. The absence of William Pitt from Parliament due to his suffering with gout led to the passage of the Stamp Act and to the Boston Tea Party.

PATHOLOGY OF GOUTY ARTHRITIS

Gout and the intense inflammatory reaction associated with the disease is due to the deposition of monosodium urate (MSU) crystals within synovial joints and subcutaneous areas of the body. Gout is one of the most well-known but also one of the most treatable of the inflammatory arthritides. Simple chemistries provide for measurement of serum uric acid and allow physicians very rapid assessment of contributing factors leading to acute inflammatory events. Uric acid is a breakdown product of protein metabolism specifically that of purines. Normal serum uric acid values (may vary and are lab-dependent) but are generally less than those expected at the maximum serum monosodium urate solubility concentration (about 7 mg/dl) and the disorder of crystal deposition occurs from precipitation of crystals from supersaturated extracellular fluid.⁵

Hyperuricemia, or an elevated serum uric acid level results from a variety of clinical reasons which leads to the traditional classification of primary versus secondary gout (Table 1). Primary gout refers to an individual with hyperuricemia secondary to an inborn error of metabolism while secondary gout involves individuals with hyperuricemia due to a variety of causes from use of diuretics to myeloproliferative disorders. Primary or idiopathic gout is much more frequent in men versus women (10 to 20:1), with the initial attack occurring in men before their 5th decade of life. Secondary gout is characterized by hyperuricemia often secondary to other causes such as thiazide diuretics, which have been of importance in the treatment of hypertension.

Gout may be classified by several stages including asymptomatic hyperuricemia, acute gouty arthritis, intercritical gout, chronic tophaceous gout. Serum uric

Table 1

CAUSES OF HYPERURICEMIA

GENETIC

- excessive production of urate due to defective control mechanisms

ACQUIRED

- high dietary purine intake
- alcohol consumption
- obesity and hyperlipidemia
- rapid turnover of bone marrow cells in leukemia, lymphoma and tumors

GENETIC

- reduced excretion of urate by an otherwise healthy kidney

ACQUIRED

- long-standing or reversible kidney disease
- hypertension
- drugs - diuretics
- inadequate urine volume
- increased body acidity due to lactic acid or ketone acids

** redrawn from: Emmerson, B.: Getting rid of gout. (1996) p.43*

acid levels are age and sex dependent with elevations occurring around the time of puberty with this more pronounced in males. Urate levels remain lower in females till menopause when values increase although still somewhat less than age-matched males. Individuals with hyperuricemia probably will remain asymptomatic for years and only a small proportion go on to have clinical gout.

Acute gouty arthritis is generally the most recognized stage and may recur with variable frequency. Gouty arthritis is generally known to be monoarticular although oligoarticular or even polyarticular involvement occurs. Initially, urate deposition simply may cause an acute inflammatory event with great pain. The period between attacks may be variable and is termed the intercritical phase of gout. Repeated gout attacks and formation of tophi lead to significant erosive bone and joint changes as well as osteopenia and softening of bone. Tophaceous gout may lead to severe joint damage as well as draining sinuses from the areas of urate deposits. Our discussion as foot and ankle surgeons will mainly relate to periods of clinical symptoms most notably acute gout attacks and dealing with the complications of chronic tophaceous gout.

Even today, with gout as well-known as it is, it is often misdiagnosed. Gouty arthritis is the result of lymphocytic ingestion of urate crystals, lysis of the PMN white cell and subsequent localized inflammatory reaction.

Gout is most common in obese males and is quite typical in its clinical presentation although it may be misdiagnosed as cellulitis. Acute gouty arthritis is characterized by the presence of an acute, hot swollen joint with intense pain often with middle of the night onset, crippling the patient, often making weight bearing or wearing a shoe difficult. Gout is frequently overdiagnosed with almost any painful swelling of the foot being labeled gout. This is particularly true in obese type II diabetics,

those typical of the metabolic syndrome that often possess a hyperuricemia, but the differential diagnosis must also include acute Charcot, cellulitis or local infection, bursitis over a bunion, as well as hallux rigidus. Diabetic patients do have a higher incidence of gout than the nondiabetic population. This may be due to the presence of renal impairment, hyperlipidemia and metabolic syndrome, or treatment of comorbidities such as hypertension with diuretics, or simply an acid-base imbalance in the tissues.

Hyperuricemia may generally be attributed to one or both of the following: the kidneys cannot eliminate the circulating uric acid fast enough (underexcretors) or the body simply makes too much uric acid (overproducers). A total of 90% of the circulating uric acid is reabsorbed following clearance by the glomerulus; and 60% of the body's miscible pool of uric acid, 1.2g, is turned over daily. Uric acid is derived from three sources: 1) catabolism of ingested nucleoproteins, 2) catabolism of endogenous nucleoproteins, and 3) direct transformation of endogenous purine nucleotides. Numerous disorders are associated with overproduction of uric acid including myeloproliferative disorders, chemotherapy and high dietary purine consumption. Table 1 summarizes the causes of hyperuricemia.

PROVOCATIVE FACTORS

Several situations deserve mention due to likely influence in development or triggering an acute attack, including local injury, alcohol, drugs, illnesses, or surgery. The first MTP joint and elbow are among the areas subjected to minor strains or injury with subsequent development of gout.

Ethanol consumption may involve several mechanisms leading to hyperuricemia including 1) hyperlactacidemia suppressing renal excretion of uric acid, 2) increased

catabolism of purine nucleotides, and 3) increased purine biosynthesis.⁴ Thiazide diuretics are among the most well-known drugs that contribute to the development of gout. Impairment of tubular secretion of uric acid is notable but volume contraction may be a more important factor. Furosemide has also been associated with hyperlactacidemia, which suppresses the tubular secretion of urate.⁴ Salicylates are well known for their dose-dependent renal effect on uric acid excretion. At high doses, greater than 2-3 grams daily, aspirin is a uricosuric while in low doses it diminishes renal tubular secretion although very small doses of 81-325 mg daily are not believed to be problematic.

Acute gouty attacks can be precipitated by various factors, including acute illness or surgery. Acute gouty arthritis in patients undergoing surgery is documented and well-known, most recently in patients undergoing bariatric surgery.⁶ Following surgery, attacks typically occur by the eighth postoperative day, with an average onset at postoperative day.³ Known risks factors for acute flares include an elevated presurgical serum urate, a history of cancer surgery, and failure to administer gouty prophylaxis.⁷ The lower extremity joints, particularly the first metatarsophalangeal joint, are affected with the highest frequency and the number of affected joints tends to correlate with previously involved joints. Although dosage and administration varies widely throughout the literature, the most commonly used medication for gouty surgical prophylaxis is colchicine. Administration of colchicine prior to and even following surgical procedures in patients with a known history of gout can prevent this undesirable surgical complication.

MANAGEMENT OF THE PATIENT WITH GOUT

The inflammatory event of acute gouty arthritis is generally the most recognized event with nonsteroidal anti-inflammatory drugs (NSAIDs) being the most utilized (Table 2.) Colchicine, which dates back to the sixth century as hermodactyl and believed to be an extract of the colchicum plant was used as a purgative and treatment of podagra. Colchicine, the active ingredient of the colchicum plant was first isolated in 1820 and has been utilized for almost 200 years for the acute treatment of gout.⁸ Colchicine may be derived from the colchicum plant or commercially synthesized and is available for oral or intravenous use. Oral therapy with 0.5 or 0.6 mg tablets taken every other hour until the patient gets better or until gastrointestinal side effects of diarrhea or abdominal pain limits subsequent ingestion has been described. Alternatively due to at times severe GI intolerance, administration of 1 tablet every t.i.d. is more patient friendly yet patients may still experience GI difficulties. Patients obviously need to be advised of the side effects and to stop the drug at least temporarily until GI symptoms subside.

Colchicine may be given intravenously and although known to be quite irritating to veins it must be given slowly intravenously to avoid the gastrointestinal side effects. However, the intravenous route has been recently associated with death due to toxicity and no longer recommended.⁹

Colchicine toxicity is mainly associated with its GI effects including abdominal cramping pain, diarrhea, nausea and vomiting and profound dehydration,

Table 2

DRUG THERAPY FOR AN ACUTE GOUT ATTACK

NSAIDS

- Indomethacin (Indocin)
50mg tid - qid x 2 days, then decrease
- Celecoxib (Celebrex)
400mg stat then 200 bid
- Ibuprofen (Motrin)
800mg tid - qid
- Ketorolac (Toradol)
30mg IV/IM then 10mg tid (10day limit)
or give parenteral dose and follow with another NSAID

CORTICOSTEROIDS

- Corticosteroid Dose Packs*
Medrol Dosepak
Decadron Dose Pack

**Probably best alternative in anticoagulated patient*

Notes: Allopurinol should be withheld for 7-10 days during an acute attack or acute joint flare. Proton-pump inhibitors (PPI) should be administered concomitantly with NSAIDs other than celecoxib.

hypokalemia, hyponatremia, metabolic acidosis, and renal shutdown. Caution must be used in patients with chronic liver and kidney disease.⁵

Oral NSAIDs have also been used for the acute gout attack with indomethacin being the most widely recognized although many of the newer NSAIDs including ibuprofen and celecoxib are also recommended.^{10,11} Treatment of the acute gout attack with colchicine or NSAIDs is recommended prior to the initiation of allopurinol therapy to reduce serum uric acid.^{12,13} Corticosteroids, with dosing regimens such as found in “dose packs” are a good alternative particularly in the anticoagulated patient. A methylprednisolone or dexamethasone dose pack may be prescribed, but should be avoided in diabetics.

Uricosuric drugs were often a consideration in the treatment of chronic gout. Probenecid is probably the most well-known of the uricosuric agents and was initially utilized as a blocking agent to help limit the renal excretion of penicillin. Phenylbutazone, a potent NSAID type anti-inflammatory was a popular agent for the treatment of gout due to its uricosuric properties. Phenylbutazone fell into disrepute due to the incidence of aplastic anemia but a sulfoxide derivative, sulfinpyrazone was synthesized as an exceedingly potent uricosuric but possessed no anti-inflammatory properties.

Allopurinol, a structural analog of hypoxanthine, inhibits the enzyme, xanthine oxidase and thus reduces the formation of uric acid. Allopurinol helps to regulate the excretion of uric acid and is frequently used in patients with chronic gout, patients with renal calculi and hyperuricemia secondary to myeloproliferative diseases. In patients with tophaceous gout, treatment with allopurinol may be combined with uricosuric agents in efforts to reduce tophaceous deposits.

The initial drug of choice for the treatment of acute gout in most patients would include an NSAID.¹² However, in the anticoagulated patient, careful consideration must be given due to the increased incidence of gastrointestinal complications, most notably gastrointestinal bleeding. Multiple studies have noted comparable efficacy in patient reported pain control with the use of celecoxib, when compared with nonselective NSAIDs, during acute gouty attacks. Recent literature has also demonstrated a comparable cardiovascular risk with an improved gastrointestinal risk profile, even with co-administration of aspirin. Although allopurinol has known interactions with anticoagulants, primarily warfarin, there has been little evidence to indicate an adverse drug interaction with the use of colchicine in these patients. Most agree that limited colchicine use with patient monitoring is a safe and effective treatment in this patient population.

CASE PRESENTATIONS

Case 1

A 42-year-old obese man presented with a inflamed first MTP joint on one foot with the onset occurring in the early morning hours (Figure 1). The patient describes awakening in sudden pain and being unable to place weight on the foot due to the severe pain. This type of patient often presents to the emergency room due to acute nature of the joint inflammation or may show up on your office door step awaiting your arrival.

The patient will be afebrile with a tender swollen foot localized to the first MTP joint. Localized skin temperatures will be elevated and overlying skin erythematous. Range of motion will be impaired and guarded due to the significant associated pain. Deformity may or may not be present and has no association with the arthritis itself.

The diagnosis of gout is often a clinical one with the typical presentation as described. Laboratory examination with serum uric acid and sedimentation rates are useful adjuncts and help confirm the diagnosis. Diagnostic gold standard is synovial aspiration with demonstration of birefringent urate crystals identified utilizing polarized microscopy. Radiographs are striking for soft tissue swelling but joint spaces are generally well-maintained without erosive changes unless the patient has had multiple gouty episodes with tophaceous deposits. Gouty tophi are simply collections of urate crystals that accumulate in the tissues and generally result in both acute inflammation as well as chronic erosive precipitates.

NSAIDs have probably supplanted the use of colchicine for the treatment of acute gout although the latter is still quite effective. Alternatively, a periarticular injection or a field block of a corticosteroid/local anesthetic solution has been very effective in providing immediate relief of acute joint symptoms. Oral corticosteroids, particularly as dose packs have been advocated. Adjunctive treatment of acute attacks include immobilization and limitation of activities until subsidence of the acute symptoms.

Case 2

Case 2 is a 59-year-old obese man with type II diabetes and a positive cardiovascular history. He possesses a mild bunion deformity with limited abduction of the great toe but medial bony prominence (Figure 2). Radiographically, he showed an opaque soft tissue area along the medial eminence of the first metatarsal. Clinically, he presented more as a symptomatic hallux rigidus with limited first MTP joint motion as well as heel complaints typical of plantar fasciitis.

CASE 1



Figure 1A. Acute gouty arthritis is well-known for its predilection for the first MPJ. This patient presented with the typical pain, swelling and local inflammation.

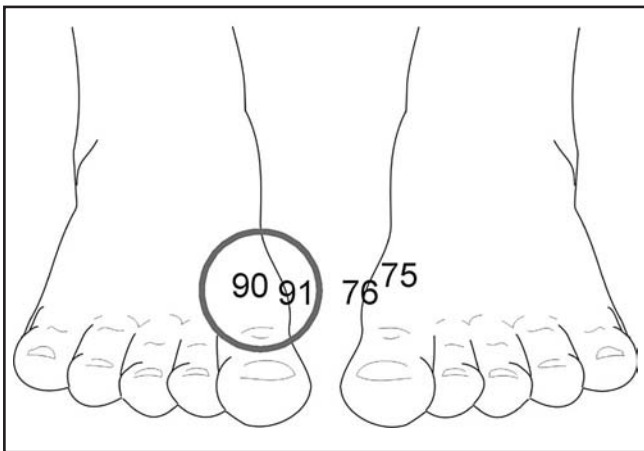


Figure 1C. Skin temperatures will be locally elevated particularly when compared to the contralateral foot.

The patient had been treated with anti-inflammatory drugs, maintenance doses of colchicines, as well as allopurinol and orthotics. Due to continued symptoms, he eventually went to surgery with performance of plantar fasciotomy as well as surgical management of the hallux rigidus. Intraoperatively, chalky capsule or deposits were quite evident particularly in the medial aspect of the first metatarsal head. Due to combination of degenerative change with gouty infiltrates, joint destructive arthroplasty was performed with a hemi metallic implant. He was maintained on colchicine throughout the perioperative period.

Case 3

A 47-year-old man with an unremarkable medical history presented with a painful bunion on the left foot of 10 days duration (Figure 3). He described prior injections from an orthopedic surgeon, but no prior diagnosis of gout.



Figure 1B. Radiographs may show soft tissue swelling and associated deformity.

Clinically, his first MTP joint range of motion was somewhat limited and tender with elevation in local skin temperatures. Radiographically, the deformity was mild to moderate in severity with good maintenance of joint space and no erosions. His serum uric acid level was 7.9 mg/dl with a laboratory normal range of 3.4-7.0. Bunion deformity was present with a degree of osteopenia. He was initially treated with NSAIDs, Celebrex, and subsequently allopurinol.

He continued to have low-grade symptoms, and colchicine 0.6 mg twice daily was added. Three months after his initial presentation, he was brought to surgery for hallux valgus repair during a period of intercritical gout. Intraoperatively, extensive urate infiltrations of the medial joint capsule were apparent as well as areas of inflammatory pannus. The articular surfaces were in good condition and

CASE 2



Figure 2A. This 59-year-old man presented with a painful first MTP joint, low-grade in nature with an opaque soft tissue area along the medial eminence of the first metatarsal head.



Figure 2B. He was later brought to surgery with extensive gouty infiltrates observed.



Figure 2C.



Figure 2D. Implant arthroplasty was performed with a metallic hemi great toe implant.

CASE 3



Figure 3A. This patient presented with localized joint pain and low grade inflammation with concomitant bunion formation.



Figure 3B. He underwent bunion surgery with debridement of the joint capsule. Postoperative radiographs at 6 weeks show Austin/Chevron type osteotomy.

typical hallux valgus correction was performed with an Austin-type osteotomy although the joint capsule required extensive debridement. Postoperatively, he experienced little difficulty and felt immensely improved almost immediately postoperatively. He was continued on allopurinol as well as colchicine for more than 3 months postoperatively. Healing and his postoperative course were eventful. His serum uric acid level only diminished to 6.1 mg/dl and the allopurinol was continued.

Case 4

This case involves an elderly patient again with a long history of gout. He presented with a rather large bullous-like lesion involving the dorsal medial aspect of the first MTP joint (Figure 4). The area was chronically swollen and almost tumor-like. He was unable to wear a regular shoe and cut-out the forefoot area. Bunion deformity was present with significant osteopenia medial erosion along the sagittal groove with a great deal of soft tissue swelling medially. The patient had a history of hypertension and his serum uric acid level was elevated.

He was brought to surgery and more than 25 ml of a milky fluid was aspirated. Surgery was continued with a first MTP joint fusion attempted due to extensive infiltrates and poor osseous tissue. Soft tissue debridement of the joint capsule and chalky gouty infiltrates was also

accomplished. Such significant osteopenia was present that fixation was performed with multiple Kirschner wires in effort to stabilize the fusion site. Rather poor purchase of the wires was noted although the patient did go on to full radiographic union of the arthrodesis.

Case 5

This case involves a long-standing diabetic patient with a history of renal impairment who has been treated numerous times for gouty arthritis and maintained on colchicine and allopurinol. Her radiographs were remarkable for cystic-like erosions involving the first metatarsal head as well as the proximal phalangeal base.

She was brought to surgery and showed typical chalky-like tophaceous gout with very soft osteopenic bone. First MTP joint fusion was performed with resection of articular surfaces and use of a platelet rich plasma gel combined with ground-up resected bone. Fixation was accomplished with a dorsally based plate. She was placed in a below-knee fracture brace or CAM-walker for 6 weeks postoperative. Clinically, the patient did well, but radiographic union was never achieved. This patient has been followed for more than 3 years postoperatively and has remained asymptomatic and overall pleased with her foot despite lack of complete arthrodesis.

CASE 4



Figure 4A. This elderly patient presented with a very large fluctuant soft tissue mass along his bunion.



Figure 4B. Aspiration of more than 25 ml of a white fluid containing urate crystals was performed.



Figure 4C. Preoperative radiograph revealed the large soft tissue swelling as well as erosive changes in the first metatarsal.



Figure 4D. A first MPJ fusion was performed with multiple Kirschner wire fixation.



Figure 4E. Complete bony consolidation did occur despite his poor bone stock.

CASE 5



Figure 5A. Preoperative AP radiograph of a 57-year-old woman who underwent first MPJ fusion with PRP.



Figure 5B. Intraoperative photo.



Figure 5C. Fixation was accomplished with a dorsal plate, postoperative radiographs AP.



Figure 5D. And lateral.

DISCUSSION

Gout is frequently seen in podiatric practices and patients may present with involvement of the first MTP joint as well as concomitant pedal complaints. Gout is most frequently associated with acute inflammatory flares with more than 60% occurring at the first MTP joint. Gout is often thought to be monoarticular and a lower extremity problem but polyarticular and upper extremity involvement at the elbow, hand, and ear may be seen.

Generally, gout is not believed to be etiologic to the development of bunion deformities but concomitant hallux valgus or hallux rigidus deformities may be present. These patients often develop tophaceous deposits within the joint capsule as well as osseous structures; or as subcutaneous excrescences at the interphalangeal joints of the toes. Tophi may also present as draining sinuses and may become secondarily infected. Gout may also present as more of a low-grade problem in other joints such as the subtalar and ankle joints or entheses such as the inferior calcaneal tuberosity, Achilles insertion, and fifth metatarsal base.

Primary gout does occur and may be exacerbated in patients who have a history of alcohol use or ingestion of over-the-counter analgesics containing salicylates. Hypertension, hyperlipidemia, and diabetes are common comorbidities that are associated with renal impairment and may contribute to the frequency of secondary gout in both men and women. Acute symptoms are generally treated with oral anti-inflammatories or colchicines (Table 2.) Surgical management of gout is not frequent but several cases are presented with good success of symptom resolution. There were no postoperative infections and no wound healing complications occurred.

Gout is a frequent presenting pathology to foot and ankle surgeons and may mimic disorders such as infection,

Charcot and a synovitis/capsulitis associated with hallux valgus. Gouty arthritis with its full-blown inflammatory presentation with a red hot, painful and swollen joint is characteristic and is still a common occurrence. Symptoms also may be low-grade and chronic and occur in patients with first MTP joint disorders particularly deformities such as hallux valgus.

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