GOUTY ARTHRITIS/CLINICAL AND SURGICAL IMPLICATIONS

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INTRODUCTION

Gout and gouty arthritis represent a clinical syndrome that will frequently present to the podiatric physician. In a general sense, gout results from the tissue deposition of monosodium urate crystals from the extracellular fluid. Although, theoretically any organ can be involved, the disease has a predilection to effect the kidneys, skin, and connective tissue structures. Included within this connective tissue group are synovium, cartilage, bursae, and tendon. Urate crystal deposition reflects the action of one or more of many processes promoting hyperuricemia. Essentially hyperuricemia results from an abnormality in purine metabolism and can be broken up into primary, secondary, and idiopathic types. The biochemistry of purine metabolism and the pathophysiology of hyperuricemia has been well addressed in both the medical and podiatric literature and will not be presented here.

Throughout the remainder of this chapter, we will discuss the arthritic effects the disease has on the lower extremity joints. Special emphasis will be given to the clinical presentation of acute attacks and the implications involved in the perioperative management of the gouty patient.

ACUTE PRESENTATION

The typical acute bout of gouty arthritis involves a violent monarticular inflammatory response. There is a rapid onset of intense pain, edema, and erythema that usually peaks within 24 hours and spontaneously resolves after several days. The disease has a predilection for lower extremity joints with the first metatarsophalangeal joint involved in as many as 75% of cases.[1] Other areas at increased risk include the tarsal, ankle, and knee joints. This preference for lower extremity joints can be due to several factors. First of all, urate deposition is encouraged by a low pH and the pH of joint fluid is typically lower than that of blood.[2] Second, urate precipitation is further increased as the temperature decreases. The temperature of the peripheral joints is normally well below that of the body core temperature.[3] Additionally, it has been proposed that in areas where increased weightbearing is demanded, (the first metatarsophalangeal joint), a traumatic synovial effusion results that when reabsorbed produces a transient increased uric acid concentration.[4,5]

The above factors combined with an overall low solubility of uric acid in body fluids (approximately 7 to 8 mg/dl in plasma) account for the severe inflammatory reaction occurring in select cases of hyperuricemia. At these higher concentrations, a supersaturated state results that has a tendency to precipitate monosodium urate crystals within cartilage, synovium, and other periarticular tissues. Once formed, the crystals are phagocytized by peripheral neutrophils which incorporate them into phagolysosomes. This is followed by rapid degranulation and disintegration with release of cytoplasmic and lysozoman enzymes including collagenase into the surrounding area. These enzymes are responsible for the production of chemotactic factors which result in joint leukocytosis, inflammation, and eventual damage.

The typical patient is a middle-aged or older male (90% of cases are males between 40 and 70 years old), or a post-menopausal woman.[1]

Although the mean age of onset and overall incidence is lower in females, there are some atypical features that they possess which need to be considered. There is a higher incidence of polyarticular onset and subsequent polyarticular attacks in women.[6] It has also been noted that older women on diuretic therapy have a tendency to develop tophaceous deposits in osteoarthritic interphalangeal joints of the hands with only a minimal inflammatory response. Both of these expressions resemble other rheumatic diseases and the correct diagnoses is essential for institution of proper therapy.[7]

DIAGNOSES

Gout can be practically diagnosed and should be highly suspected in all patients presenting with the above history and physical findings. However, due to its similarity to several other diagnoses, including cellulitis, septic arthritis and trauma appropriate work-up is required. Routine lab work can be sensitive but lacks specificity. A leukocytosis and increased sedimentation rate can be expected in both gout and infection. An increased serum uric acid level is a significant finding but is not always consistent with the diagnosis of gout. Serum levels above 7 mg/dl for males and 6 mg/dl for women indicate a supersaturated state and in the right circumstances can precipitate crystals.[8]

In a study conducted by Hall et al. 5% of the general adult population was found to have or at one point to have had hyperuricemia. Yet, only a small number went on to develop clinical symptomatology.[9] It is equally wrong to dismiss the diagnosis of gout in light of normal serum uric acid levels. The use of anti-inflammatory medication, i.e. aspirin, NSAIDs, or corticosteroids, may reduce a naturally elevated level. The only definitive means of establishing a diagnosis of gout is the demonstration of intracellular, monosodium urate crystals in leukocytes aspirated from the synovial fluid of the involved joints. A compensated polarized light microscope with a first order red compensator is used to visualize the crystals which appear strongly birefringent. The synovial fluid will also reveal an increased white count with a predominance of polymorphonuclear leukocytes in addition to the crystals.

Radiographic evaluation is of questionable value in the initial acute attack and is often without signs of degenerative changes. In a prospective study conducted by Barthelemy et al. on 60 gouty patients, some with symptoms, others asymptomatic, a large number of positive radiographs were found.[10] Of the 60 patients, 36 (60%) were considered to have radiographic changes diagnostic of gout in one or more anatomic areas. Twenty-four percent of these patients with positive x-ray findings denied symptoms in the affected joints.

Barthelemy et al. strongly recommend the use of x-ray evaluation in the management of gout. The specific findings which they considered diagnostic for gout were not identified in the paper. However, some of the more common changes seen early include an increase in the soft tissue density about the joint and possibly an increase in the joint space secondary to effusion. As the disease progresses, more profound changes can be seen including extensive erosions on the peri-articular bones and possibly the presence of tophi within the bone or surrounding soft tissues. When tophi are found in the chondral or subchondral areas the bone will often take on the characteristic *punched-out* appearance. Joint space narrowing is usually not seen until late in the disease.

TREATMENT

Once the diagnosis of gout has been established, either definitively through crystal identification or presumptively by the presenting history and physical findings, a treatment regimen should be promptly started. The management of a patient with gout involves two independent considerations: 1) treatment of the acute attack and 2) long term control of hyperuricemia and prevention of future attacks.

The acute arthritic episode is generally easy to treat and can respond dramatically to a course of colchicine or one of the non-steroidal anti-inflammatory medications. For this reason, it is often tempting for the podiatrist to assume primary control of these attacks. However, it is important for the podiatrist, who is often the diagnosing physician, to work in conjunction with a medical internist or rheumatologist for both acute and long term management of hyperuricemia.

When using colchicine, once considered the standard of treatment for the acute flare in many institutions, one must be aware of the complications and potential toxicities associated with it. Normal renal and hepatic function are mandatory with both oral and intravenous use of colchicine. It has been shown that the majority of systemic toxicity seen with colchicine was a result of inappropriate use of the drug.[11] Either the cumulative dose was too high or the patient had renal or hepatic disease that was not considered.

The use of non-steroidal anti-inflammatory medications, mainly indomethacin and phenylbutazone, has largely replaced colchicine as the treatment of choice for the acute gouty attack. Though much less than with colchicine, significant side effects and toxicities can also result from use of these drugs. The systems most commonly affected are the kidneys and gastrointestinal tract.

For a complete description of these medications including their dosage schedules and toxicities, see the article published by Wallace and Singer.[12] The current thought on the pharmacologic therapy of both acute gouty arthritis and long term management of hyperuricemia are presented in excellent fashion.

COURSE

An acute attack of gouty arthritis may persist for several days to weeks depending on the severity of the bout and on the timing of therapy. In the earlier stages of the disease, upon resolution of the acute symptoms the joint normally returns to its previous state without permanent damage. In between acute attacks, the patient enters an intercritical period where they are normally asymptomatic. The length of this quiescent period may last for many years, though as many as 60% of the patients experience a second attack within one year.[13] In time if the disease goes untreated, the bouts become increasingly frequent with an increased number of areas being affected.

If recurrent attacks are not prevented, the swelling and disability that result will persist for longer and longer periods of time and eventually become permanent. Joint destruction including erosion of cartilage and subchondral bone are readily apparent in this stage of chronic gouty arthritis.

It is also in these later, uncontrolled stages of hyperuricemia, that gouty tophi become clinically evident. The tophi are rarely noted early in the disease and usually do not appear until an average of 10 years after onset.[1] Tophus formation is related to serum levels of uric acid and generally the higher the level, the earlier and more extensive the deposits appear. Tophi can appear anywhere in the body but are most commonly seen in subchondral bone, synovium, olecranon bursa, Achilles tendon, the ears, and in subcutaneous tissue on the extensor surface of the forearm and overlying joints. It is the subchondral deposits that initiate the inflammatory response which leads to the erosive changes described earlier, often producing a *punched-out* appearance. In more severe cases, the skin overlying the large and more superficial tophi may ulcerate and produce a chronic draining sinus tract.

Fortunately, due to the effectiveness of acute and long term therapy, only a minority of patients go on to this debilitating stage of tophaceous gout. A portion of those who do go on to develop these deposits may respond to a course of a uricosuric drug or allopurinol. While others may require surgical removal of the tophi and reconstructive procedures to repair the normal periarticular structures that may have been damaged.

SURGICAL INDICATIONS

In 1961, Straub et al.,[14] reclassified the earlier indications of Linton and Talbot,[15] and of Larmon and Kurt[16] into four main categories: functional, metabolic, symptomatic, and cosmetic.

The functional indications for surgical correction include destructive gouty arthritic changes especially in the first metatarsophalangeal joints. Though not contraindicated, a word of caution is given to the use of implants in these patients. The only reported case of a postoperative gouty attack following foot surgery occurred after implant arthroplasty of the first metatarsophalangeal joint.[17] In retrospect, the authors felt that an adjunctive synovectomy should be performed to prevent the development of postoperative crystal-induced synovitis. Other functional considerations include procedures designed to relieve the encroachment of gouty tophi on vital structures such as nerves, vessels, and tendons. Gout infiltration of upper extremity tendons which

simulated rupture has been documented in literature.[18] Also the inability to wear shoes is an indication for surgical management.

Surgical procedures performed for metabolic considerations are aimed at decreasing the total amount of urates in the body by removing large tophi. As was mentioned previously, there is only a small percent of tophi that will not resolve with antihyperuricemic therapy and require surgical ablation. Other metabolic indications include surgical procedures designed to minimize the danger of skin breakdown and subsequent necrosis with ulcer formation over larger gouty deposits.

The symptomatic and cosmetic considerations are many times the only complaints the patient has. In these instances it can be quite pleasing and comforting to the patient to see the removal of large unsightly tophi.

Prior to performing surgery on these areas of tophaceous deposits, consideration must be given to the surrounding skin and underlying vital structures. The skin overlying the tophi becomes increasingly thin as the deposits enlarge. It will remain viable until trauma (including surgery) or distention causes the tophus to rupture. When this occurs, urate crystals will be deposited on the skin and the chance of slough and ulcer formation is very real. In this situation, it would seem obvious to remove all the underlying tophi and alleviate this problem. However, many times the tophi have incarcerated underlying vital nerves, vessels, tendons, and joint structures preventing complete excision. In light of the above findings, the degree and nature of the reconstructive procedure will be affected by the extent of tophi involvement and potential for complete removal.

PERIOPERATIVE MANAGEMENT

Patients with chronic tophaceous gout usually represent that portion of the disease population that is under poor therapeutic control. Because gout has systemic manifestations itself as well as an association with several fellow travelers including hypertension, heart disease, and peripheral vascular disease, a thorough medical examination should be performed prior to surgical consideration.[19] It is also well known that the stress and trauma associated with surgery can result in an acute postoperative gouty attack.[16,17] This not only applies to local procedures, but may also occur following major surgery performed elsewhere in the body. So, for the above reasons it is important for the surgeon to identify the patients who are at risk for the above sequelae and apply appropriate prophylactic management preoperatively.

In 1977, Wallace et al. presented a classification scheme to identify patients with a history of acute gout. If one or more of the following has occurred, a positive history is assumed:

1) a positive crystal examination of synovial fluid
2) positive examination for monosodium urate crystals in a sampled tophi diagnosed by chemical means or by polarized light microscopy, or
3) the presence of six of the following twelve clinical, laboratory, and radiographic findings:
    1. More than one attack of acute arthritis.
    2. Maximal inflammation development within one day.
    3. Attack of monarticular arthritis.
    4. Joint redness observed.
    5. First metatarsophalangeal joint painful or swollen.
    6. Unilateral attack involving the first metatarsophalangeal joint.
    7. Unilateral attack involving a tarsal joint.
    8. Suspected tophus.
    9. Hyperuricemia.
    10. Asymmetric swelling within a joint on radiographs.
    11. Negative culture of joint fluid for microorganisms during attack of joint inflammation.
    12. Subcortical cysts without erosions seen on radiograph.

In 1984, Roper et al. developed a scheme that identified patients who are at risk for developing postoperative gouty arthritis and put them into one of three major classes.[21] 1) high level risk, 2) moderate-level risk, and 3) low-level risk patients.

High-level Risk Group

High level risk patients include those who have had one or more of the following:

1. One or more attacks of acute gout during the past year, at which time the joint aspirate revealed uric acid crystals.

2. A dramatic symptomatic relief of joint pain and inflammation following a test course of colchicine.
3. The documented need for antihyperuricemic medication (e.g., allopurinal, sulfinpyrazone, probenecid, and others).

Moderate-level Risk Group

Moderate-level risk patients include those who have had one or more of the following:

1. Prior episodes of an acute monarthritis clinically resembling gout.
2. A diagnosis of gout made by history, clinical observation, and the presence of hyperuricemia during the acute episode, but not documented by a joint aspiration.
3. Prior symptomatic relief during a bout of monarthritis from use of a nonsteroidal anti-inflammatory medication such as indomethacin.

Low-level Risk Group

Low-level risk group patients include those who have had one or more of the following:

1. Asymptomatic primary hyperuricemia.
2. Asymptomatic hyperuricemia secondary to some other medical condition or drug reaction.

Once the patient is determined a high or moderate risk for the development of an acute postoperative attack, considerations must be given for preoperative prophylaxis. The classic prophylactic agent, and still used quite extensively today, is colchicine. When used the dosage should be approximately 1.5-2.0 mg daily, divided into dosages of .5 mg every 6 to 8 hours. The drug can be administered either intravenously or by mouth. The regimen is normally started 2 to 3 days preoperatively and continued for 3 to 5 days postoperatively. An alternate strategy involves use of NSAIDS, namely Indocin 50 mg po, 2 to 3 times daily with the same perioperative schedule as colchicine. The disadvantage lies in the fact that the patient would have to discontinue the medication when NPO before surgery and may have difficulty tolerating it postoperatively.

If the patient has been on antihyperuricemic therapy coming into surgery, this medication should be continued until the day of surgery, when NPO status begins and started immediately postoperatively. The use of adjunctive colchicine or Indocin in the peri-operative period as described above, will add additional protection against an acute postoperative gouty attack.

CONCLUSION

Gout is one of the more common and easy to treat arthritic disorders that will present to the podiatric physician. However, because of the multifaceted nature of the disease and systemic implications, co-management with other medical specialist is recommended. We have outlined both the acute presentation and chronic gouty arthritic patient. Considerations for therapy, both pharmacologic and surgical have been presented. Though not a contraindication to surgery, these patients carry certain perioperative risks that have to be recognized. When indicated, appropriate preoperative prophylaxis will help assure the patient of an uneventful postoperative course and optimal result.

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