Treatment of Acute Gout in Hospitalized Patients

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ABSTRACT. Objective. To record practice patterns of treatment of acute gout in hospitalized patients.

Methods. We performed a retrospective chart review of hospitalized patients diagnosed with gout. Results. Seventy-nine (43%) patients were diagnosed with acute gout during their hospitalization. Fiftyeight (73%) patients with acute gout were found to have a reduction in their glomerular filtration rate. Twenty patients (25%) underwent arthrocentesis. The most widely used drugs for acute gout were colchicine, n = 42 (53%), and nonsteroidal antiinflammatory drugs (NSAID), n = 40 (51%). Combination therapy was used in 52% of patients with acute gout. Thiry-six (86%) patients treated with colchicine and 32 (80%) patients treated with NSAID had renal failure.

Discussion. Crystal analysis, the gold standard for diagnosing gout, was performed in only 25% of patients suspected of acute gout. Combination antiinflammatory agents are used in over 50% of patients despite the absence of evidence to support use of such combinations. Renal failure was present in 73% of patients with acute gout. Colchicine and NSAID should therefore be used with caution in these patients. Practice patterns vary widely and often appear to be in conflict with recommended diagnostic and treatment measures for acute gout. (First Release June 15 2007; J Rheumatol 2007;34:1566-8)

Key Indexing Terms: **GOUT**

TREATMENT

HOSPITAL

Despite centuries of study of gout and the availability of effective treatment, for most patients there are still questions about optimal approaches to the diagnosis and treatment of gout¹. We conducted a study to investigate how hospitalized patients are currently treated for acute and chronic gout.

MATERIALS AND METHODS

We performed a retrospective chart review of patients hospitalized in a 400bed hospital, between October 1, 2002, and October 31, 2004, who were diagnosed with gout and documented the treatment they received. The patients were selected based on a primary or secondary diagnosis of gouty arthropathy or gout not otherwise specified, identified by diagnosis codes 274.0 or 274.9. We recorded patients' history, examination, laboratory values, how the diagnosis of gout was reached, use of consultants, and treatment of acute and chronic gout.

RESULTS

1566

We identified a total of 184 patients diagnosed with gout based upon their diagnosis codes. Of these, 79 (43%) were diagnosed with acute gout during their hospitalization. The average age of the patients was 71 (range 40-96) years. All patients were male.

The leading causes of hospitalization among patients diag-

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nosed as having acute gout while in the hospital were acute gout, n = 15 (19%), and congestive heart failure, n = 15 (19%). Other causes included: chest pain, n = 7 (9%); cellulitis, n = 7(9%); dysrhythmias, n = 4 (5%); infections, n = 4 (5%); deep vein thrombosis, n = 4 (5%); strokes, n = 3 (4%); and gastrointestinal bleeds, n = 3 (4%). Less common diagnoses included hepatitis, cholangitis, headaches, rash, diverticulitis, abdominal pain, proteinuria, syncope, and hypokalemia.

A total of 52 (66%) consultations were performed in patients with suspected acute gout [rheumatology, n = 30(58%), orthopedics, n = 21 (40%), and podiatry, n = 1 (2%)]. Twenty patients (25%) underwent arthrocentesis [14 (47%) patients having been consulted by rheumatology and 6 (29%) by orthopedics]. The joints aspirated were knee, n = 12 (60%); and foot, ankle, olecranon bursa, and hand, n = 2 (10%) each.

The drugs most commonly taken for acute gout were colchicine, n = 42 (53%), and nonsteroidal antiinflammatory drugs (NSAID), n = 40 (51%). Combination therapy was used in 52% of patients with acute gout [prednisone with colchicine, n = 18 (23%); NSAID with colchicine, n = 13(16%); and steroid with NSAID, n = 10 (13%)]. Indomethacin, n = 20 (50%), was the most commonly used NSAID.

Only 51 (27%) of all patients with gouty arthritis had been receiving allopurinol prior to admission. Serum urate (SU) levels were checked in 49 (27%) of all patients. SU level was greater than 6 mg/dl in 9 (60%) patients treated with allopurinol. Allopurinol was stopped during the acute attack in 2 patients (3%) taking it chronically.

Renal failure (creatinine > 1.5 mg/dl) was present in 98 (53%) patients with gout and in 51 (65%) patients with acute gout. Mean creatinine in patients with acute gout was 1.8 mg/dl (range 0.5–9.9). In adults the best equation for estimat-

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ing glomerular filtration rate (GFR) from serum creatinine is the Modification of Diet in Renal Disease (MDRD) Study equation. When estimating the GFR by using the MDRD formula an even higher number of patients with acute gout were found to have renal failure. Fifty-eight (73%) patients with acute gout were found to have a reduction in their GFR. A moderate reduction (GFR 30–59 ml/min) was seen in 37 (47%) patients, a severe reduction (GFR 15–29 ml/min) was seen in 16 (20%), and renal failure (GFR < 15 ml/min) was seen in 4 (5%).

Thirty-six (86%) patients treated with colchicine and 32 (80%) patients treated with NSAID had renal failure. Thirty (83%) received colchicine 0.6 mg daily to 3 times a day. Six patients received hourly colchicine 0.6 mg despite their renal failure (5 of whom had GFR < 29 ml/min). A 92-year-old man with GFR of 55 ml/min received hourly colchicine 0.6 mg. He developed diarrhea followed by dehydration and hypotension requiring transfer to the intensive care unit. Thirty-six (46%) patients with acute gout were taking diuretics.

DISCUSSION

Although crystal analysis is the gold standard for the diagnosis of gout, only 25% of the patients had arthrocentesis for crystal analysis. The diagnostician was likely to be the admitting physician for these patients, with only 38% receiving a rheumatology consultation. Crystal identification in patients with gout is commonly recommended; however, the extent of its use is not clear, as was seen in our study.

The leading causes of hospitalization among patients diagnosed as having acute gout while in the hospital were acute gout and congestive heart failure (19% each). This was a retrospective study. Lifestyle details of the patients such as drinking habits would be helpful, but were not known to us due to the retrospective design of the study.

Combination antiinflammatory agents are taken by over 50% of patients despite the absence of evidence for use of combinations. Indomethacin was the most commonly used NSAID. The use of combination antiinflammatory drugs to treat acute gouty arthritis is not what is described in most textbooks and reviews^{1,2}; there is little or no literature to support such a practice. Combination therapy potentially puts the patient at risk of increased morbidity/mortality due to the combined effects upon the kidney.

Allopurinol was the only urate-lowering drug used. Only 27% of our gout cohort were prescribed allopurinol and even when it was taken SU was not lowered to a SU goal of 6 mg/dl. In our study, SU level was > 6 mg/dl in 60% of patients treated with allopurinol. Maintaining a SU level at < 6 mg/dl and not just within the "normal range" has been proposed to help assure resolution of tophi and eventual cessation of acute gouty attacks. Lowering serum urate levels to < 6 mg/dl leads to dissolution of urate crystals and regression of tophi³. In addition there are fewer acute flares⁴ as well as fewer monosodium urate crystals in the joints⁵.

Renal failure was present in 65% of patients with acute gout. This is more common than previously reported⁶. When estimating the GFR by using the MDRD formula an even higher number of patients with acute gout were found to have renal failure: 73%. This may be due in part to diuretics used in approximately half of these patients. Over 80% of patients given colchicine or NSAID had renal failure.

Colchicine and its metabolites are excreted through the urinary and biliary tracts, and the presence of renal or liver disease increases the time needed for clearance of colchicine 2-to 3-fold⁷. This suggests that patients with either liver or renal disease should be closely monitored while taking even low doses of colchicine⁸. The potential toxicity of colchicine is often underappreciated. Colchicine has the smallest risk-to-benefit ratio of all drugs used for the treatment of patients with acute gouty arthritis and should be used with caution, especially in patients with renal disease. NSAID too should be used with caution in patients with renal disease to avoid renal toxicity.

The association of uncontrolled hyperuricemia causing diminished renal function has recently been studied. Kang, *et al* hypothesized the association of elevated SU causing deterioration of renal function. The elevation of SU was shown to increase blood pressure, proteinuria, vascular disease, and renal scarring in rats. All these conditions are associated with worsening renal function⁹. In yet another study, the association of an elevated SU was found to be a risk factor for developing renal failure¹⁰. This information establishes a need for tighter control of SU and appropriate urate-lowering therapy, as well as possible screening for impending renal disease in patients with hyperuricemia and gout.

Practice patterns vary widely and support the need for education of healthcare professionals taking care of patients with gout. In our study all patients were male, which may suggest that the cohort was somewhat biased. Longterm prospective, placebo-controlled studies are needed to establish guidelines for the diagnosis and treatment of gout.

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