Joint Decisions

WINNIPEG, MAN. — The clinicians wondered whether this patient had rheumatic nodules, gout, or some other disease process.

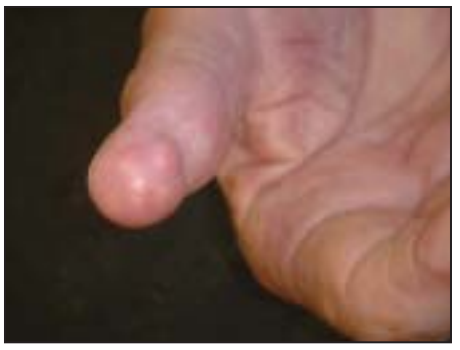
The rheumatologist suggested that blood tests, x-rays, an MRI, and a number of other tests would be needed to determine the cause of the finger nodules. These suggested procedures would require a return visit from the patient several weeks later.

"I was watching this and wondering whether there was an easier way" to make the diagnosis, Dr. Benjamin K. Fisher said at the annual conference of the Canadian Dermatology Association.

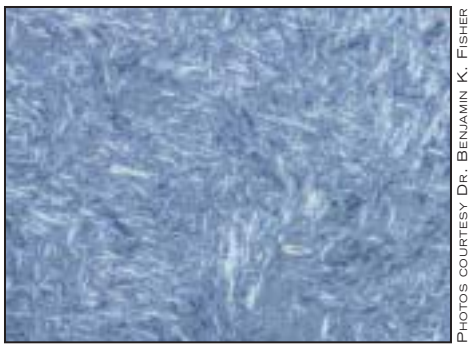
After the clinicians finished discussing the case, with the agreement of his colleagues and the patient, Dr. Fisher incised one of the nodules and pressed against it with the side of the scalpel. Out came yellow toothpaste-like matter that he smeared on a slide and placed under a microscope.

"I'd never done it that way before, and I didn't know if I'd see anything," said Dr. Fisher, professor of dermatology at the University of Tel Aviv and the University of Toronto.

And then, "Lo and behold, there were all these [uric acid] crystals, and obviously this patient had gout," he said.



A pasty exudate gained by incising one of the hand nodules showed crystals.



PHOTOS COURTESY DR. BENJAMIN K. FISHER

Polarized light microscopy showed crystals that were consistent with gout.

This very simple procedure sped up the diagnosis by at least 3 weeks and saved the patient the time, cost, and hassle of undergoing further testing.

"This is a procedure that any one of you can do in your office," Dr. Fisher pointed out.

—Sherry Boschert

EULAR Issues New Guidelines

Gout *from page 1*

2005. The recommendations are graded according to a formula that includes risk/benefit tradeoffs. Expert commentary accompanies recommendations that reflect standard clinical practice but lack significant supporting research (Ann. Rheum. Dis. 2006;65:1312-24).

Treatment in all stages of gout should be tailored to both specific risk factors (serum urate level, radiographic evidence), clinical phase (acute, intercritical, or chronic), and general risk factors (gender, age, obesity, alcohol use, urate elevating drugs, and other comorbidities), according to the guidelines. Nonpharmacologic treatments are also important, since they can potentiate drugs' effectiveness, are inexpensive, and do not cause harmful or distressing side effects.

The EULAR panel's recommendations for managing gout include the following:

► Patients should reduce alcohol and purine-rich food consumption and lose weight if obese.

► Hyperlipidemia, hypertension, hyperglycemia, obesity, and smoking should also be addressed. The link between metabolic syndrome and elevated serum uric acid has been proved in clinical studies. While there is no direct evidence that links smoking to gout, smoking has been associated with increased alcohol consumption, thereby having an association with gout.

► During acute gout, oral colchicine and/or nonsteroidal anti-inflammatory drugs should be the first-line treatment. Patients who cannot tolerate the GI effects of colchicine, however, can be treated with NSAIDs which provide gastroprotection.

► A 1-g loading dose of colchicine followed by 0.5 mg every 2-3 hours relieves symptoms in acute attacks, but even this level may cause serious GI side effects. A lower dose (0.6 mg three times per day) may be appropriate and effective, but there are no studies addressing this.

► Neither intra-articular aspiration nor injection of long-acting steroids has been studied in controlled trials. However, case reports and uncontrolled trials indicate that the treatments are well tolerated and effective in reducing pain. They may be especially important for patients who cannot tolerate pharmacotherapy.

► Only patients with recurrent acute attacks, arthropathy, tophi, or radiographic changes should receive urate-lowering drugs. There is little agreement on whether less severe patients should have long-term urate-lowering therapy. However, all gout patients should make urate-lowering lifestyle changes though diet modification.

► A uric acid level of less than 6 mg/dL—below the saturation point for monosodium urate—should be the target serum level. Many patients with serum uric acid in the normal range can still have joint damage. Maintaining the lower level keeps crystals from precipitating into the joints.

► "Start low, go slow" with allopurinol for long-term urate-lowering therapy. To avoid the rare but potentially life-threatening allopurinol hypersensitivity sydrome, begin with 100 mg/day and increase by 100 mg/day every 2-4 weeks.

► Patients with normal renal function who cannot tolerate allopurinol may do well with a uricosuric drug such as probenecid or sulphinpyrazone. For renal patients, benzbromarone can be tried, but liver function should be carefully monitored.

► Low-dose colchicine (0.5-1.0 mg daily) and/or NSAIDs may have a place in preventing additional attacks. One placebo-controlled trial suggested that colchicine could prevent half of acute attacks, although all colchicine-treated patients experienced diarrhea. NSAIDs are not as effective; evidence suggests they may prevent one attack in every seven patients.

► If possible, stop diuretic therapy in gout patients. Replace the diuretic with another antihypertensive; losartan and fenofibrate are effective and have modest uricosuric effects.

Physicians may perceive gout as a less-serious disease—a mistake, Dr. Saag said. "It's an area where a lot of medical errors are made, because gout is a condition that affects people with multiple comorbidities. Many patients have alcohol abuse, renal insufficiency, and other challenging problems, including an inability to tolerate the medications used to treat gout."

EULAR's diagnosis and management guidelines are similar to the quality care indicators for gout published by the American College of Rheumatology in 2004 (Arthritis Rheum. 2004;50:937-43), according to Dr. Saag, who added that the indicators are used mostly by those doing research on quality of care issues. ■

EULAR's New Guidelines Outline 10 Recommendations on Gout Diagnosis

The presence of monosodium urate monohydrate crystals in synovial fluid remains the gold standard for gout diagnosis, with a sensitivity of 84% and a specificity of 100%, according to new gout diagnostic guidelines issued by the European League Against Rheumatism.

But the crystals may be present only during an acute attack, and their identification requires a clinician who is both well trained and experienced, the document notes. Additionally, the cost-effectiveness of the test has not been fully demonstrated (Ann. Rheum. Dis. 2006;65:1301-11).

The guidelines put forth 10 diagnostic criteria for gout, ranked not only upon extant research but also on benefit and risk, cost, and the clinical expertise required to effectively use the criteria.

"This seems a better system, reflecting both research evidence and expert opinion, than the traditional semiautomatic estimation based on category of research data alone," wrote Dr. Weiya Zhang of the University of Nottingham (England), who led a 20-person panel in developing the document.

The recommendations were drawn from the same 181 studies, published from 1945 to 2005, from which the EULAR panel drew its 12 gout management recommendations (see accompanying story).

The recommendations address clinical, biochemical, and radiographic diagnostic techniques for the disease:

1. Clinical symptoms of rapidly developing severe joint pain and swelling, especially with overlying erythema, are highly indicative of crystal formation but have low sensitivity (23%) for a gout diagnosis without further evidence.

2. Monosodium urate monohydrate (MSU) crystals in synovial aspirate have both high sensitivity and high specificity, although variability in lab expertise may affect the accuracy of the sample findings.

3. A clinical diagnosis of classic recurrent podagra with hyperuricemia can be reasonably assumed to be gout but requires MSU crystal confirmation.

4. All patients with inflamed joints should have their synovial fluid examined for MSU crystals.

5. MSU crystal testing can also be performed between bouts of inflammation, because urate crystals persist in intercritical periods in up to 70% of gout patients.

6. Because gout and sepsis may occur simultaneously, all possible gout patients should also have Gram staining and culture of synovial fluid. The test should be performed even if the fluid is positive for MSU crystals, because septic arthritis can progress rapidly and carries a significant risk of morbidity and mortality.

7. Serum uric acid can't be used exclusively as a diagnostic tool. Many people with high serum uric acid don't develop gout, and some patients with confirmed MSU crystals have normal serum uric acid.

8. Gout patients under age 25 years with a family history of the disease or who have renal calculi should have a 24-hour urinary uric acid/creatinine ratio done. The 24-hour screen appears to be more accurate and cost effective than an early morning spot sample.

9. Radiographs might be useful for a differential diagnosis, but they can't confirm gout. There are radiographic changes in all stages of gout, but many affected joints can be radiographically normal. Patients with intradermal tophi are more likely to show severe radiographic changes.

10. Male gender, diet, alcohol use, and diuretics increase the risk of gout, but don't ignore other important risk factors. These include hypertension (relative risk of 4, compared with controls), coronary heart disease (odds ratio, 1.75), chronic renal failure (odds ratio, up to 5), and obesity (odds ratio, 3.8).