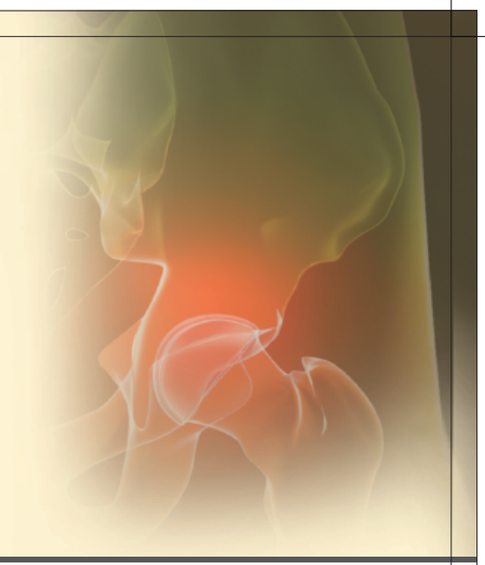

Rheuma Facts

A Quarterly Magazine

6th Issue, March 2015

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Current News

Lower back pain causes more disability globally than any other condition

HOY D. ANN RHEUM DIS 2014

May 13, 2014

Lower back pain is the most common disability globally, with more research needed to better understand the disability, according to results of a recently published study.

Damian Hoy, MD, and colleagues found that of 291 conditions studied in Global Burden of Disease 2010, lower back pain (LBP) had the sixth highest burden.

"Globally, LBP causes more [years lived with disability] YLD than any other condition. Governments, health service and research providers and donors need to pay far greater attention to the burden that LBP causes than what they have done previously," the researchers wrote in the study.

The researchers defined LBP as "pain in the area on the posterior aspect of the body from the lower margin of the twelfth ribs to the lower gluteal folds with or without pain referred into one or both lower limbs that lasts for at least one day." In their systematic reviews on the prevalence, incidence, remission, duration and mortality risk of LBP, Hoy and colleagues identified four levels of severity for LBP with and without leg pain, each with their own disability weights. The global age-standardized point prevalence of LBP in 2010 was estimated to be 9.4%, which was also higher in men.

Hoy and colleagues reported that the disability-adjusted life years increased from 1990 to 2010. Disability-adjusted life year also increased from 58.2 million in 1990 to 83 million in 2010.

"Further research is urgently needed to better understand the predictors and clinical course of LBP across different settings, and the ways in which LBP can be prevented and better managed," Hoy and colleagues wrote. – Robert Linnehan

Rheuma Facts

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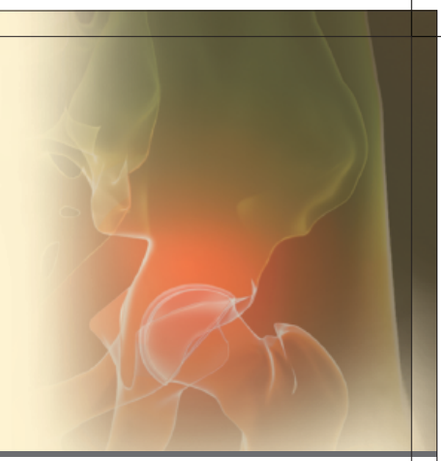

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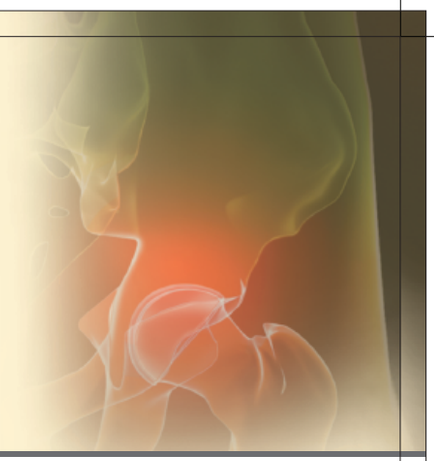
Calcium Pyrophosphate Deposition Disease

Summarized by:
Dr. Ahmed Iqbal Mirza Consultant Rheumatologist
Aga Khan University Hospital, Karachi



Background

Calcium pyrophosphate deposition disease (CPDD) is a metabolic arthropathy caused by the deposition of calcium pyrophosphate dihydrate in and around joints, especially in articular cartilage and fibrocartilage. Although CPDD is often asymptomatic, with only radiographic changes seen (ie, chondrocalcinosis), various clinical manifestations may occur, including acute (pseudogout) and chronic arthritis

Almost any joint may be involved by CPDD, although the knees, wrists, and hips are most often affected. This condition is the most common cause of secondary metabolic osteoarthritis.

According to McCarty, the five most common presentations of CPDD are as follows:

- Asymptomatic (lanthanic) CPDD
- Acute pseudogout
- Pseudo-osteoarthritis
- Pseudorheumatoid arthritis
- Pseudoneuropathic joints

Etiology

Although the exact mechanism for the development of CPDD remains unknown, increased adenosine triphosphate breakdown with resultant increased inorganic pyrophosphate in the joints results from aging, genetic factors, or both.

Overactivity of enzymes that break down triphosphates, such as nucleoside triphosphate pyrophosphohydrolase, has been observed in the cartilage of patients with CPDD. Therefore, inorganic pyrophosphate can bind calcium, leading to CPPD deposition in the cartilage and synovium..Hyaline cartilage is affected most commonly, but fibrocartilage, such as the meniscal cartilage of the knee, can also be involved.

Epidemiology

Occurance

CPDD is a common condition that occurs with aging in all races. Nearly 50% of people older than 85 years have radiologic evidence of chondrocalcinosis.

Sex- and age-related demographics

CPDD is slightly more common in women than in men. The exact female-to-male ratio is unknown but is probably 1.4:1.

Types:

Asymptomatic (lanthanic) CPDD

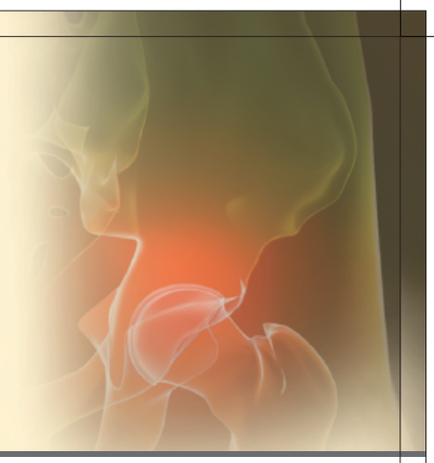
This is usually associated with radiographic findings of chondrocalcinosis in the absence of clinical manifestations and may be the most common form of calcium pyrophosphate deposition disease (CPDD).

The classic radiologic findings include chondrocalcinosis of the hyaline cartilage and fibrocartilage of the knees, the fibrocartilage of the triangular ligament of the wrist, the fibrocartilage of the symphysis pubis, and the acetabulum labrum of the hips.

Acute pseudogout

Acute pseudogout is characterized by acute monoarticular or oligoarticular arthritis. Pseudogout usually involves the knee or the wrist, although almost any joint can be involved, including the first metatarsophalangeal (MTP) joint, as occurs in patients with gout. This form of CPDD accounts for 25% of cases. Glucose levels are usually normal.

Clinical manifestations are similar to those of acute gouty arthritis, typically presenting with an acute monoarthritis with pain and swelling, although generally not as intense. Polyarticular attacks may occur on occasion. Pseudogout may be precipitated by medical illness such as myocardial infarction, congestive heart failure, or



cerebrovascular accident or may occur after surgery. Trauma may also be a precipitating factor. Events that affect serum calcium levels also may precipitate attacks of pseudogout.

Occasionally, pseudogout may present as a pseudoseptic syndrome with acute arthritis, fever, and leukocytosis with a left shift.

Pseudo-osteoarthritis

Pseudo-osteoarthritis often involves the metacarpophalangeal (MCP) joints, wrists, elbows, and shoulders, joints unlikely to be involved in primary osteoarthritis. It affects the knees most commonly and can involve the proximal interphalangeal (PIP) joints and spine, as occurs in patients with primary osteoarthritis. This form of CPDD accounts for 50% of all cases. Approximately half of these patients also have associated pseudogout.

Pseudoneuropathic joints

Neuropathic arthropathy, which is observed in fewer than 5% of patients with CPDD, most commonly involves the knee. This is a severe, destructive arthropathy. Unlike true neuropathic arthropathy, no clear underlying neurologic disorder is present. The presence of chondrocalcinosis can aid in making the diagnosis.

Physical Examination

The physical examination findings vary depending on the form of CPDD in a given patient, who may present with an acute arthritis or different patterns of chronic arthritis.

Acute pseudogout

Physical examination findings show an acutely inflamed joint with swelling, effusion, warmth, tenderness, and pain on range of motion similar to acute gouty arthritis. This typically occurs in the knee but may be present in the wrists, shoulders, ankles, hands, and feet.

Pseudo-osteoarthritis

Physical examination findings show a picture similar to osteoarthritis, sometimes with an unusual joint predilection. If a patient has osteoarthritis involving the MCP joints and wrists, consider CPDD associated with an underlying metabolic disease.

Pseudorheumatoid arthritis

Physical examination findings show a picture similar to rheumatoid arthritis with synovitis in a symmetrical, polyarticular pattern, especially involving the wrists and MCP joints.

Differential Diagnoses

- Gout
- Hemochromatosis
- Hyperparathyroidism
- Hypothyroidism
- Osteoarthritis
- Rheumatoid Arthritis
- Septic Arthritis

Lab Studies

General laboratory studies usually are not helpful in calcium pyrophosphate deposition disease (CPDD). The white blood cell (WBC) count and erythrocyte sedimentation rate (ESR) may be elevated.

Evaluating for an underlying metabolic disease (eg, hemochromatosis, hyperparathyroidism, hypothyroidism) is reasonable, especially in younger patients.

Laboratory tests can include the following:

- Serum calcium, phosphorus, magnesium and alkaline
- phosphatase levels
- Iron levels
- Total iron-binding capacity
- Transferrin saturation and ferritin

- Thyroid-stimulating hormone and free thyroxine levels

Pseudogout

Occasionally, pseudogout may present as a pseudoseptic syndrome with acute arthritis, fever, and leukocytosis with a left shift.

The diagnosis of acute pseudogout is made by performing compensated polarized microscopy after aspiration of fluid from the involved joint. The most commonly involved joint is the knee, followed by the wrist, the MCP joints, the elbows, and the MTP joints.

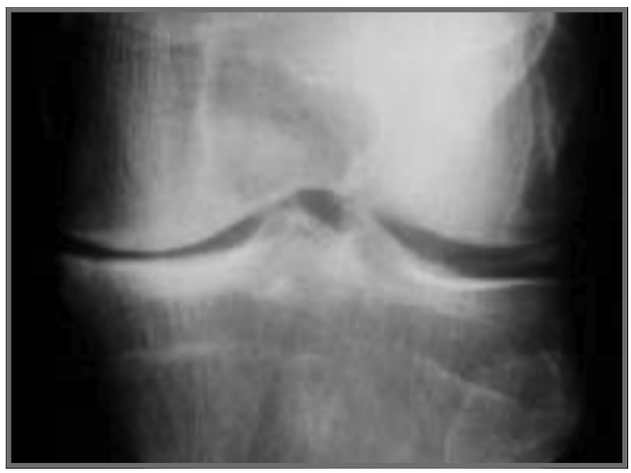
The crystals are rhomboid-shaped, weakly positively birefringent, and difficult to see. If intracellular, an acute attack of pseudogout is strongly suggested. Aspiration of the fluid from affected joints during an acute attack usually yields mildly to moderately inflammatory fluid, with 10,000-50,000 WBCs/ L, more than 90% of which are neutrophils. (See the images below.)

Gout and pseudogout can coexist, even in the same joint; therefore, the presence of gout does not rule out the possibility of pseudogout and vice versa. Ultrasonography may be helpful in diagnosing pseudogout. (See the image below.)

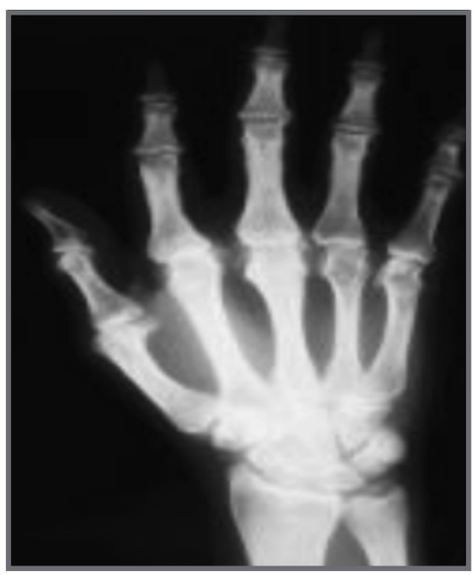
Imaging Studies

Radiography

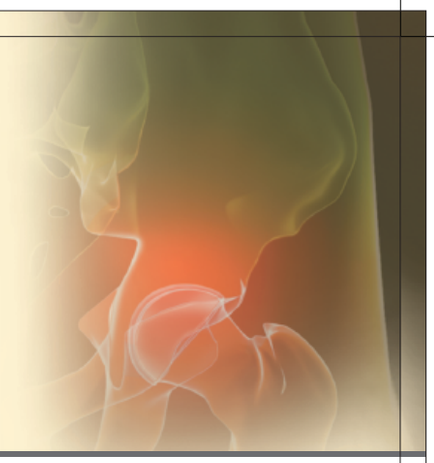
Radiologic studies are important in the diagnosis of CPDD, with radiography being the criterion diagnostic standard (see the images below). Imaging studies usually include the hands, wrists, pelvis, and knees. The pelvic radiograph should include an anteroposterior view that shows the symphysis pubis and hips.



Calcium pyrophosphate deposition disease. Radiograph of the knee showing chondrocalcinosis involving the meniscal cartilage, as well as evidence of osteoarthritis.



Calcium pyrophosphate deposition disease. Radiograph of the wrist and hand showing chondrocalcinosis of the articular disc of the wrist and atypical osteoarthritis involving the metacarpophalangeal joints in a patient with underlying hemochromatosis.



Chondrocalcinosis is usually found in the articular cartilage or meniscal cartilage of the knee, the triangular ligament of the wrist, the symphysis pubis, or the glenoid or acetabulum labra.

Chondrocalcinosis has also been noticed in other areas of the wrist (aside from the fibrocartilage), such as the distal radioulnar joint and the midcarpal joint, as well as in the pisotriquetral joint. In addition, it has been reported in the spine as calcification of the ligamentum flavum.

In some situations, hemochromatosis can produce specific radiographic findings, such as large, hooklike osteophytes, especially around the second to fifth MCP joints. However, these findings also can occur in patients with CPDD alone.

Hooklike osteophytes are a common radiologic finding in patients with a pseudo-osteoarthritis condition and are usually present along the second and third metacarpal heads.

Radiologically, erosions can be observed in pseudorheumatoid arthritis but are usually associated with chondrocalcinosis.

MRI and ultrasonography

Routine magnetic resonance imaging (MRI) has not been shown to be as sensitive as radiography in detecting the presence of CPPD deposits. However, 4T MRI holds better promise in detecting these crystals.

Ultrasonography has been significantly beneficial in the visualization of CPDD crystals. (See the image)

In addition, Gutierrez et al reported ultrasonography is accurate and reliable for detecting articular cartilage calcification at the knee level in patients with CPDD. In their study, ultrasonography detected hyaline cartilage spots in at least one knee in 44 of 74 patients with CPDD (59.5%), whereas radiography detected hyaline cartilage spots in 34 patients (45.9%) ($P < 0.001$).

Approach Considerations

Management of calcium pyrophosphate deposition disease (CPDD) depends on the clinical manifestations.

Asymptomatic (lanthanic) CPDD should not be treated unless it is a possible manifestation of other syndromes, such as hyperparathyroidism or hemochromatosis (treatment of which is important to prevent further end-organ damage but cannot reverse the joint disease).

Acute pseudogout may be treated by joint aspiration and intra-articular corticosteroid injection, systemic corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs), or occasionally, high-dose colchicine.

Treatment for pseudo-osteoarthritis is similar to that for typical osteoarthritis. Patients with a pseudorheumatoid arthritis can be treated with small doses of corticosteroids, such as prednisone at 5mg daily.

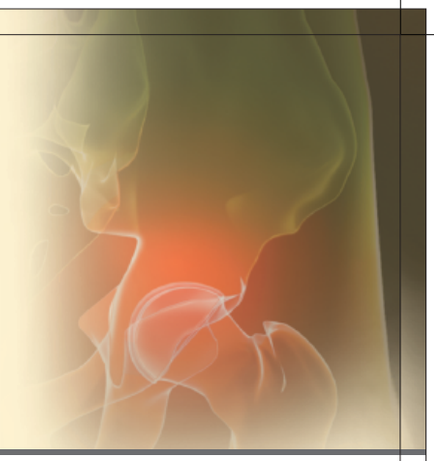
Methotrexate was effective in isolated observations in patients who had severe disease with particular emphasis on joint destruction. However, this treatment was attempted only in patients with the pseudorheumatoid presentation.

Surgical care

Theoretically, surgically removing calcifications from an affected joint could be beneficial, but this is currently considered an experimental procedure.

What Is Gout?

Summarized by:
Dr. Terrence Gibson Consultant Physician and Rheumatologist
Guy's St. Thomas Hospital London



What Is Gout?

Gout is a painful condition that occurs when the bodily waste product uric acid is deposited as needle-like crystals in the joints and/or soft tissues. In the joints, these uric acid crystals cause inflammatory arthritis, which in turn leads to intermittent swelling, redness, heat, pain, and stiffness in the joints.

In many people, gout initially affects the joints of the big toe (a condition called podagra). But many other joints and areas around the joints can be affected in addition to or instead of the big toe. These include the insteps, ankles, heels, knees, wrists, fingers, and elbows. Chalky deposits of uric acid, also known as tophi, can appear as lumps under the skin that surrounds the joints and covers the rim of the ear. Uric acid crystals can also collect in the kidneys and cause kidney stones.

What Is Uric Acid?

Uric acid is a substance that results from the breakdown of purines. A normal part of all human tissue, purines are found in many foods. Normally, uric acid is dissolved in the blood and passed through the kidneys into the urine, where it is eliminated.

If there is an increase in the production of uric acid or if the kidneys do not eliminate enough uric acid from the body, levels of it build up in the blood (a condition called hyperuricemia). Hyperuricemia also may result when a person eats too many high-purine foods, such as liver, dried beans and peas, anchovies, and gravies. Hyperuricemia is not a disease, and by itself it is not dangerous. However, if excess uric acid crystals form as a result of hyperuricemia, gout can develop. The crystals form and accumulate in the joint, causing inflammation.

What Are the Four Stages of Gout?

Gout can progress through four stages:

1. **Asymptomatic (without symptoms) hyperuricemia.** In this stage, a person has elevated levels of uric acid in the blood (hyperuricemia), but no other symptoms. Treatment is usually not required.

2. **Acute gout, or acute gouty arthritis.** In this stage, hyperuricemia has caused the deposit of uric acid crystals in joint spaces. This leads to a sudden onset of intense pain and swelling in the joints, which also may be warm and very tender. An acute attack commonly occurs at night and can be triggered by stressful events, alcohol or drugs, or the presence of another illness. Attacks usually subside within 3 to 10 days, even without treatment, and the next attack may not occur for months or even years. Over time, however, attacks can last longer and occur more frequently.

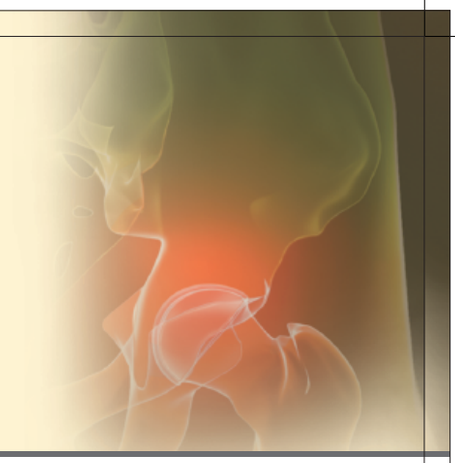
3. **Interval or intercritical gout.** This is the period between acute attacks. In this stage, a person does not have any symptoms.

4. **Chronic tophaceous gout.** This is the most disabling stage of gout. It usually develops over a long period, such as 10 years. In this stage, the disease may have caused permanent damage to the affected joints and sometimes to the kidneys. With proper treatment, most people with gout do not progress to this advanced stage.

What Causes Gout?

A number of risk factors are associated with hyperuricemia and gout. They include:

- **Genetics.** Many people with gout have a family history of the disease. Estimates range from 20 to 80 percent.
- **Gender and age.** It is more common in men than in women and more common in adults than in children.
- **Weight.** Being overweight increases the risk of developing hyperuricemia and gout because



there is more tissue available for turnover or breakdown, which leads to excess uric acid production.

- **Alcohol consumption.** Drinking too much alcohol can lead to hyperuricemia, because alcohol interferes with the removal of uric acid from the body.
- **Diet.** Eating too many foods that are rich in purines can cause or aggravate gout in some people.
- **Lead exposure.** In some cases, exposure to lead in the environment can cause gout.
- **Other health problems.** Renal insufficiency, or the inability of the kidneys to eliminate waste products, is a common cause of gout in older people. Other medical problems that contribute to high blood levels of uric acid include:
  - high blood pressure
  - hypothyroidism (underactive thyroid gland)
  - conditions that cause an excessively rapid turnover of cells, such as psoriasis, hemolytic anemia, or some cancers
  - Kelley-Seegmiller syndrome or Lesch-Nyhan syndrome, two rare conditions in which the enzyme that helps control uric acid levels either is not present or is found in insufficient quantities.
- **Medications.** A number of medications may put people at risk for developing hyperuricemia and gout. They include:
  - **Diuretics,** which are taken to eliminate excess fluid from the body in conditions like hypertension, edema, and heart disease, and which decrease the amount of uric acid passed in the urine
  - **Salicylate-containing drugs,** such as aspirin
  - **Niacin,** a vitamin also known as nicotinic acid
  - **Cyclosporine,** a medication that suppresses the body's immune system (the system that protects the body from infection and disease). This medication is used in the treatment of some autoimmune diseases, and to prevent the body's rejection of transplanted organs.
  - **Levodopa,** a medicine used to support communication along nerve pathways in the treatment of Parkinson's disease.

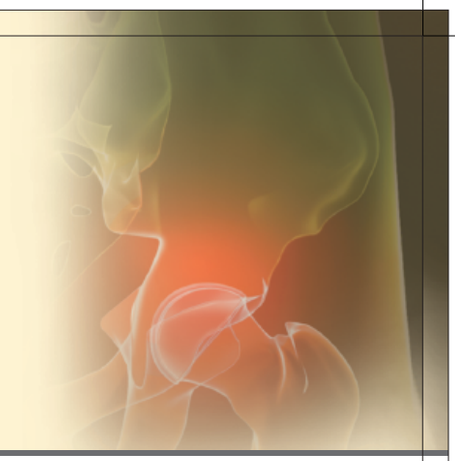
Who Is Likely to Develop Gout?

Scientists estimate that 6 million adults age 20 and older report having had gout at some time in their lives.[1] It is rare in children and young adults. Men, particularly those between the ages of 40 and 50, are more likely to develop gout than women, who rarely develop the disorder before menopause. People who have had an organ transplant are more susceptible to gout.

[1]According to the National Arthritis Data Workgroup, this estimate is based on self-reports, which may produce an overestimation of prevalence, as cited in Helmick CG, Felson DT, Lawrence RC, Gabriel S, ***Hirsch R, Kwoh CK, et al.;*** National Arthritis Data Workgroup. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. ***Part 1. Arthritis and Rheumatism 2008;1:15-25.***

How Is Gout Diagnosed?

Gout may be difficult for doctors to diagnose because the symptoms can be vague, and gout often mimics other conditions. Although most people with gout have hyperuricemia at some time during the course of their disease, it may not be present during an acute attack. In addition,



having hyperuricemia alone does not mean that a person will get gout. In fact, most people with hyperuricemia do not develop the disease.

To confirm a diagnosis of gout, a doctor may insert a needle into an inflamed joint and draw a sample of synovial fluid, the substance that lubricates a joint. The joint fluid is placed on a slide and examined under a microscope for uric acid crystals. Their absence, however, does not completely rule out the diagnosis.

The doctor also may find it helpful to look for uric acid crystals around joints to diagnose gout. Gout attacks may mimic joint infections, and a doctor who suspects a joint infection (rather than gout) may also culture the joint fluid to see whether bacteria are present.

How Is Gout Treated?

With proper treatment, most people who have gout are able to control their symptoms and live productive lives. Gout can be treated with one or a combination of therapies. The goals of treatment are to ease the pain associated with acute attacks, to prevent future attacks, and to avoid the formation of tophi and kidney stones. Successful treatment can reduce discomfort caused by the symptoms of gout, as well as long-term damage to the affected joints. Treatment will help to prevent disability due to gout.

The most common treatments for an acute attack of gout are nonsteroidal anti-inflammatory drugs (NSAIDs) taken orally (by mouth), or corticosteroids, which are taken orally or injected into the affected joint. NSAIDs reduce the inflammation caused by deposits of uric acid crystals, but have no effect on the amount of uric acid in the body.

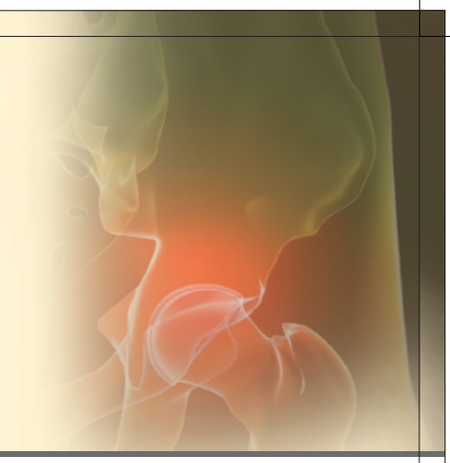
Warning: NSAIDs can cause stomach irritation or, less often, they can affect kidney function. The longer a person uses NSAIDs, the more likely he or she is to have side effects, ranging from mild to serious. Many other drugs cannot be taken when a patient is being treated with NSAIDs because NSAIDs alter the way the body uses or eliminates these other drugs. Check with your health care provider or pharmacist before you take NSAIDs. Also, NSAIDs sometimes are associated with serious gastrointestinal problems, including ulcers, bleeding, and perforation of the stomach or intestine. People age 65 and older and those with any history of ulcers or gastrointestinal bleeding should use NSAIDs with caution.

Corticosteroids are strong anti-inflammatory hormones. The most commonly prescribed corticosteroid is prednisone. Patients often begin to improve within a few hours of treatment with a corticosteroid, and the attack usually goes away completely within a week or so.

When NSAIDs or corticosteroids do not control symptoms, the doctor may consider using colchicine. This drug is most effective when taken within the first 12 hours of an acute attack. For some patients, the doctor may prescribe either NSAIDs or oral colchicine in small daily doses to prevent future attacks. The doctor also may consider prescribing other medicines to treat hyperuricemia and reduce the frequency of sudden attacks and the development of tophi.

People who have other medical problems, such as high blood pressure or high blood triglycerides (fats), may find that the drugs they take for those conditions can also be useful for gout.

The doctor may also recommend losing weight, for those who are overweight; limiting alcohol consumption; and avoiding or limiting high-purine foods, which can increase uric acid levels.



What Can People With Gout Do to Stay Healthy?

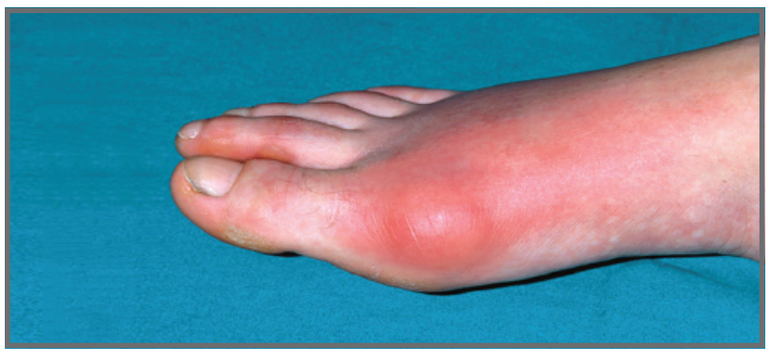
Fortunately, gout can be controlled. People with gout can decrease the severity of attacks and reduce their risk of future attacks by taking their medications as prescribed. Acute gout is best controlled if medications are taken at the first sign of pain or inflammation. Other steps you can take to stay healthy and minimize gout's effect on your life include the following:

- Tell your doctor about all the medicines and vitamins you take. He or she can tell you if any of them increase your risk of hyperuricemia.
- Plan followup visits with your doctor to evaluate your progress.
- Drink plenty of nonalcoholic fluids, especially water. Nonalcoholic fluids help remove uric acid from the body. Alcohol, on the other hand, can raise the levels of uric acid in your blood.
- Exercise regularly and maintain a healthy body weight. Lose weight if you are overweight, but avoid low-carbohydrate diets that are designed for quick weight loss. When carbohydrate intake is insufficient, your body can't completely burn its own fat. As a consequence, substances called ketones form and are released into the bloodstream, resulting in a condition called ketosis. After a short time, ketosis can increase the level of uric acid in your blood.
- Avoid foods that are high in purines.

Because uric acid's role in gout is well understood and medications to ease attacks and reduce the risk or severity of future attacks are widely available, gout is one of the most—if not the most—controllable forms of arthritis. But researchers continue to make advances that help people live with gout. Perhaps someday these advances will prevent this extremely painful disease

Most Hospitalizations for Gout Are Preventable

Summarized by:
Dr. Kamran Hameed Consultant Physician and Rheumatologist
Dean; Ziauddin Medical College, Karachi



Inefficient or inadequate clinical care led to unnecessary hospitalizations in 89% of patients with gout, according to a retrospective review conducted at the Geisinger Health System in Central Pennsylvania.

"Most of these hospitalizations could have been avoided with a consult with a rheumatologist," said Thomas Olenginski, MD, from the Geisinger Medical Center in Danville, Pennsylvania.

In fact, the hospitalizations were often the result of failure to follow the guidelines of the American College of Rheumatology (ACR) and European League Against Rheumatism (EULAR), hospitalizing patients who present to the emergency department for care, and noncompliance with prescribed medications. However, rheumatologists "were rarely asked to see the patients once they were admitted," he explained.

"Because patients presented so often to the emergency department rather than their doctor's office and were in pain with other comorbidities, admission may have seemed to be the correct medical care decision," Dr. Olenginski said during a press conference here at the ACR 2014 Annual Meeting.

"Gout is a misunderstood disease, and not considered to be a condition that can lead to complications. Gout attacks are preventable. In 2014, deposition of crystals on the joint should not occur. When the patient comes in, it is critical to make the diagnosis by aspirating fluid and defining crystals under the microscope," he emphasized.

"Gout patients are problematic," he continued. "Even before we undertook this study, we suspected that gout was not being properly managed, and not just by primary care providers. We expected to find preventable hospital admissions and patients who could have had shorter hospital stays."

Most of these hospitalizations could have been avoided with a consult with a rheumatologist.

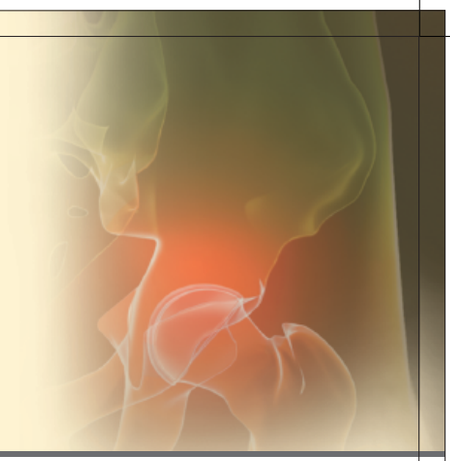
Dr. Olenginski and his team retrospectively reviewed the cases of 56 hospitalized adults with a primary discharge diagnosis of gout from 2009 to 2013. Of these hospital admissions, 50 (89%) were considered to be preventable, defined as a primary diagnosis of gout with no concomitant illness at presentation that required hospitalization.

Of the 50 patients with preventable admissions, 21 (42%) had three or more risk factors for gout, such as diabetes, chronic kidney disease, cardiovascular disease, malignancy, diuretic use, and low-dose aspirin use.

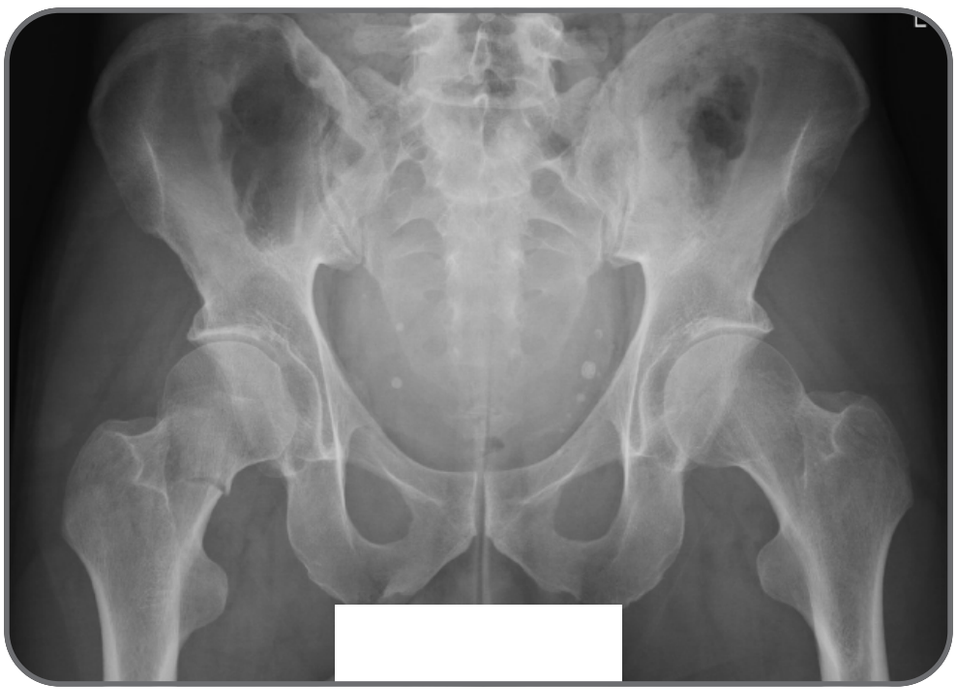
On admission, cellulitis was suspected in 8% of the patients, inflammatory polyarthritis in 14%, and septic arthritis in 76%. Two-thirds of patients underwent arthrocentesis, 73% of which were performed in the emergency department.

Photo Quiz

Pain & Weakness in Lower Limb



25 years old female presented with severe pain & weakness in lower limb since last 3 months. She has difficulty while sitting up from floor



Question

- Based on history, what is the most obvious finding on this X-ray?
- What is your diagnosis?

Answer of last quiz

Erythema Nodosum