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THE ORIGIN OF GOUT AND METHODS OF ITS TREATMENT

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Annotation: This article provides information on gout, one of the currently common biohimic pathologies. In this article, you can get information about gout disease. The article was written using internet resources , articles , tutorials. I hope this article will be of interest to students.

Key words: Gout, hyperuricaemia, arthropathies, kidney, uric acid.

Gout is a true crystal deposition disease in which all clinical manifestations are considered to be directly attributable to the presence of monosodium urate (MSU) crystals. It is one of the most prevalent inflammatory arthropathies with a prevalence of approximately 1.4%, and is the most common inflammatory arthropathy in men . Both the prevalence and incidence of gout appear to be rising . The primary risk factor for the development of gout is elevation of serum uric acid (urate) levels, or hyperuricaemia. As uric acid levels rise and exceed the physiological saturation threshold of uric acid in body tissues, formation and deposition of MSU crystals occurs in and around joints.

Gout is a form of inflammatory arthritis that results from an excess of uric acid in the blood. Among people with gout, 90% have kidneys that don't adequately remove uric acid from their urine, while 10% have high uric acid levels because they produce too much uric acid.

Uric acid is a chemical that is created in the body when it digests and breaks down proteins. Among the breakdown products of protein are compounds called purines, and these, in turn, get broken down to uric acid.

Gout statistics and facts

- 8.3 million people in the United States have gout (6.1 million men and 2.2 million women).[1]
- 90% of people with gout have kidneys that don't remove enough uric acid, while 10% have an overproduction of uric acid.
- 90% of gout attacks start in a single joint. Most often, it is the "bunion joint" of the big toe.
- 90% of gout patients have one or more of the following conditions (comorbidities), which make it more difficult to manage gout: kidney dysfunction, coronary heart disease, obesity, high cholesterol and/or triglycerides, diabetes mellitus.

Gout is caused by an elevated amount of uric acid, either because a person's kidneys do not adequately remove uric acid from their body, or because their body simply makes too much of it. The overabundant uric acid can then crystallize in the joints, causing inflammation and pain. A condition known as pseudogout has similar symptoms but is caused by a very different type of crystals. In both conditions, white blood cells surround the crystals, which leads to inflammation.

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In pseudogout, the associated crystals are formed from calcium pyrophosphate dihydrate, rather than from uric acid, as in gout. The inflammation looks similar in a joint with pseudogout as in gout. However, there are some differences regarding which joints are commonly affected. For example, the big toe, very common in gout, is not so common with pseudogout. The treatment of a flare of gout and pseudogout are treated similarly, but the prevention regimens are very different, since the crystals that cause the problems are different.

The propensity of gout for the foot was recognised by the ancient Greeks who referred to it as podagra, literally "foot-grabber" . The name "gout" derives from humoral theory and the Latin word *gutta* or "drop", podagra being thought to arise as a result of the bodily humours falling to the affected body part. Although our current understanding of the pathogenesis of gout is dramatically distant from humoral theory, these observations concerning the intimate relationship between gout and the foot have been reinforced over the centuries and continue today. This review will consider the ways in which gout affects the foot and discuss potential mechanisms underlying this relationship.

After an often prolonged period of asymptomatic hyperuricaemia, the initial manifestation of gout is usually an acute attack of synovitis affecting a single peripheral joint, most commonly the first metatarsophalangeal joint (MTPJ). Other commonly affected joints include the mid-tarsal joints, ankles, knees, fingers, wrists and elbows Such attacks are characterised by sudden onset of excruciating joint pain, typically taking less than 24 hours from symptom onset to reach peak intensity, with associated joint swelling, overlying erythema and exquisite tenderness to touch. Although acute gout should be treated rapidly with a non-steroidal anti-inflammatory drug (NSAID) or colchicine, it usually resolves completely over a period of two to three weeks even without treatment. A variable period of time then elapses until the patient experiences a further attack (the "intercritical period"). With time, attacks may increase in severity and frequency, involve different joint sites, and may become oligo- or polyarticular. Eventually, without treatment, the patient may develop chronic tophaceous gout, characterised by chonic pain and stiffness, joint damage and erosive arthropathy, and clinically evident subcutaneous nodular deposits of MSU crystals (tophi) which can occur at the toes, Achilles' tendons, pre-patellar tendons, fingers, olecranon processes, and less commonly, the ears.

Making a diagnosis of gout usually involves the doctor interviewing the patient to understand the history of their joint pain, a physical exam, X-rays of affected joints, blood testing (especially for uric acid), and extraction of joint fluid to test for deposits of excess uric acid.

The history of the joint pain will include learning which joints are involved, whether joint swelling is present, whether the painful joints get red, and whether the inflammation happens in one or in many joints at a time. The physical exam of the joints will help determine which joints are affected, whether there is redness, heat or swelling, and whether there are any bumps (such as over the toe or elbow) that might be collections of uric acid crystals. This type of bump is called are called a “tophus” (plural, "tophi"), which is Latin for "stone."

Making a final diagnosis of gout requires putting together the findings from these questions and tests. Gout is often under-diagnosed. For example, a gout flare may be mistaken for an injury, such as an ankle sprain. But it in other instances, it is also over-diagnosed. For example, a hot, red and swollen

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knee may look like a gout flare, but it could be pseudogout (due to calcium crystals instead of uric acid), or an infection that is unrelated to gout or pseudogout.

The key message for patients is that gout is a treatable disease and it is very reasonable to expect to be gout-free in the future if you stay with your uric acid-lowering medication. Uric-acid-lowering medication isn't a "cure" − since you need to keep taking it to reap its benefits – but in the vast majority of cases it can completely control gout. If you stop the uric-acid-lowering medication, the gout can and likely will come back. Uric acid-lowering medications can also markedly reduce the risk of uric-acid-related kidney stones (although they don't prevent calcium-related stones).

When you first start a uric-acid-lowering medication, this leads to uric acid crystals being removed from the joint lining via the bloodstream. As the crystals are released, they can cause gout flares. These flares early in uric-acid-lowering treatment (such as allopurinol) are *not* a reason to stop the uric-acid-lowering medication. For the first six months after starting a medication such as allopurinol, low-dose colchicine is often added to help reduce early flares. People are also advised to be especially careful in the first six months after starting allopurinol (or other uric acid-lowering medication). Down the road, the diet won't be as crucial after many of the crystals have been removed from the joints via the bloodstream.

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