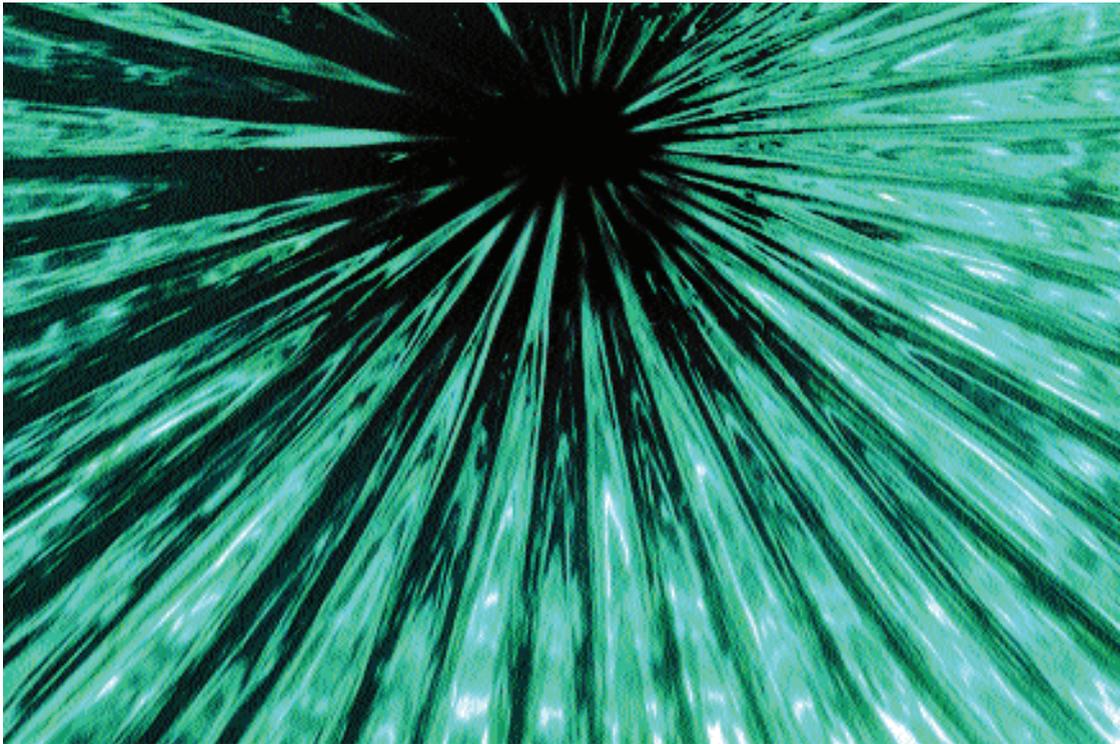




UK Gout Society

All About Gout

A Patient Guide to Managing Gout



Introduction

As early as the **fourth century BC**, **Hippocrates** wrote about gout as an affliction of old men and a product of high living. During the 17th to 19th centuries the links with rich living were a target for laughter, caricature and cartoons. Even the public perception of gout today is not dissimilar – yet nobody who has suffered from this extremely painful condition finds it in the least bit funny!



Gout comes from the Latin gutta – which means ‘drop’ – with reference to the mediaeval “flowing down of humours”

Gout occurs most frequently in **men between the ages of 40 and 60**, particularly in those who are overweight or genetically predisposed. Gout can be associated with diets that are high in animal protein and alcohol but this is by no means always the case. It is less often seen in pre-menopausal women, men under the age of 30, and rarely occurs in children.

Paradoxically, whilst the prevalence of gout has increased over the past two decades, public knowledge about this excruciatingly painful condition remains minimal. Yet, from the medical profession’s perspective, gout is a **20th century medical success story**. Not only are its causes understood but attacks can be effectively treated, the possibility of recurrences greatly reduced and the risks of damage to joints and kidneys largely avoided.

The UK Gout Society is a national charity dedicated to raising public awareness of gout. If you would like more information about gout, please log-on to our website: www.ukgoutsociety.org or contact us at: **PO Box 527, London, WC1V 7YP** or email us at: info@ukgoutsociety.org

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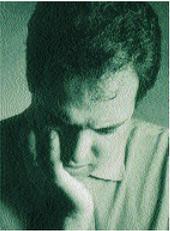
What is gout?

Gout is a metabolic disorder that causes acute, intermittent and painful attacks of arthritis in the joints of the foot, knee, ankle, hand and wrist – especially the big toe. If the underlying condition is left untreated, attacks become more frequent, more prolonged and more generalised.

Facts and Figures

- Gout is the most common cause of inflammatory joint disease in men over 40 years old
- A joint in the big toe is the first joint affected in more than half of cases
- In most patients only one joint is affected while in up to 10% of patients it affects more than one joint
- In a typical UK general practice of 2000 patients there may be 17 men and three women with gout

What causes gout?



Gout occurs as a result of excess uric acid (urate) in the blood and tissues. After prolonged supersaturation of the tissues, crystals of urate can form in and around the joints and kidneys. If uric acid crystals enter the joint, they may trigger the development of inflammation. The affected joint becomes red, swollen and extremely painful and tender. Infrequently, stones (calculi) may form in the kidneys. Most patients with gout have high levels of urate in their blood because they do not pass enough in their urine. In most cases this is caused by an inherited peculiarity of the kidneys, which is in other respects harmless. It can also be caused by high levels of uric acid in the diet or by some drugs eg. diuretics. Much less commonly, patients produce too much uric acid in the first place, owing to an inherited metabolic abnormality or to one of the disorders associated with greatly increased production of cells in the body.

What is uric acid?

Uric acid is the end product of purine nucleotide (cell nucleus nucleoprotein) metabolism in all cells of the body. The levels normally found in the blood and tissues are derived from both the breakdown of old cells and from the degradation of purine-containing foods in normal diets.

Who is at risk?

Gout is a common joint disease affecting over five times more men than women. It is rare in children. In men, it can occur any time after puberty, whereas in women it is uncommon before the menopause. In around 10% of cases there is a family history of the disorder. Men and women who are older, overweight, have high blood pressure, eat diets rich in shellfish and animal protein and drink large quantities of alcohol or sugar-sweetened soft drinks, have an increased risk of developing gout.

Can I prevent gout?



As gout can be triggered by environmental factors, there are some basic steps you can take to help prevent gout occurring in the first place or help prevent recurrent attacks.

Environmental trigger factors

- Diets containing high levels of purine (see below)
- Alcohol – especially beer and fortified wine
- Drugs – including diuretics and antibiotics which can interfere with the normal excretion of uric acid
- Excess weight
- Crash dieting
- Surgical operations
- Severe illness
- Prolonged stress
- Injury
- Unusual physical exercise

Dietary measures

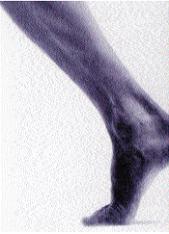
There are several things sufferers can do to help prevent a recurrence of gout. As uric acid is a by-product of purine metabolism, reducing your intake of dietary purines can be helpful. A diet very low in purines is actually difficult to stick to, but foods that are high in animal and seafood purines should be avoided if possible, or at least reduced in amount. These include:

- meat – particularly red meat and offal, such as liver and kidneys
- game
- seafood – especially mussels, herrings, sardines
- alcoholic drinks – including beer, lager, port and some red wines
- sugar-sweetened soft drinks- including fruit drinks, carbonated fizzy drinks and colas.

(Alcohol containing beverages and sugar-sweetened soft drinks which contain fructose raise uric levels even though they do not have a high purine content).

In addition, yeast products, oatmeal, mushrooms, asparagus, spinach and some pulses – especially lentils – can all increase the level of uric acid in the body and should be eaten only in moderate quantities. “Quick fix” slimming regimes, particularly crash and ‘yo-yo’ dieting and fasting, should be avoided. They can lead to uric acid retention by the kidney. This is due to lactic acid production and ketosis as a consequence of fasting. The solubility of uric acid decreases in acid urine, and therefore treatment with alkalis may be useful for the minority of gout patients who produce more than average amounts of uric acid. In order to decrease the tendency for stone formation a high intake of fluid (8–10 glasses of water each day) will also decrease the likelihood of crystals being deposited in the kidneys as calculi. There are other aspects of the diet which may require more detailed discussion with a health professional. For further information on gout and diet, please see the UK Gout Society’s *All About Gout and Diet* fact sheet at www.ukgoutsociety.org

How do I know if I have gout?



Few things are as painful as a severe attack of gout. Gout is usually diagnosed on the basis of its distinctive symptoms and an examination of the joint. An acute attack of gout often develops during the night or early hours of the morning and reaches a peak within a few hours so that, with an affected foot, it is impossible to tolerate even the touch of bed clothes. The skin may be red and shiny and the inflammation may be so severe that the skin may peel. A mild fever, a loss of appetite and a feeling of tiredness can also accompany acute attacks of gout. An untreated attack generally lasts for a few days, then dies down and the joint gradually returns to normal. Some people never experience another attack. If the uric acid level remains high most will have a second attack between six months and two years after the first. Subsequently, attacks become more frequent and more prolonged and may result in joint damage if the uric acid level is not controlled.

Will my doctor do any tests?

You may not have any investigations until the acute attack has subsided, after which a blood test may help to clarify the diagnosis. High levels of uric acid are found in patients with gout at some time or another but the blood level is frequently not raised during an acute attack. If there is suspicion of gout the blood test may be repeated more than once. You should realize that the finding of a raised blood level does not prove that an attack of joint pain is due to gout: the majority of people with slightly raised blood uric acid levels do not develop gout. However, it is true that the higher the level, the more likely it is that sooner or later gout will develop. A more specific test that may be performed at the time of acute gout is the analysis of the fluid from the affected joint. Fluid from the joint space is aspirated (removed through a needle and syringe) and examined under a special type of microscope. The presence of needle-like, uric acid crystals confirms the diagnosis of gout. However, this approach may not be practical if the joint is a small one. Another important reason for doing medical tests is that gout patients have greater than average risks of having, or of developing, medical problems such as diabetes and cardiovascular diseases, including heart attacks and strokes. Therefore when someone develops gout, this should be taken as an opportunity to consider other aspects of his or her health – such as blood pressure, obesity, cholesterol and sugar levels – all of which can be associated with increased risks of vascular disorders.

How is gout treated?



Having been diagnosed by your GP, the goals of treatment are threefold: The first step is to reduce the pain and inflammation of the acute attack. The second is to reduce the likelihood of future attacks by lifestyle modification and the third is to lower urate levels and so prevent the development of the complications such as joint damage and kidney disease.

Step one – treatment of the acute attack

During the actual attack, the most important thing is to start treatment as soon as possible by controlling the inflammation and pain and also by immobilising the joint. Currently non-steroidal anti-inflammatory drugs (NSAIDs) such as diclofenac, naproxen or indomethacin are usually used as first-line therapy. NSAIDs with a

short half-life, such as indomethacin and ibuprofen are frequently recommended because of their rapid onset of action – although their use should be avoided in patients with a history of peptic ulceration, gastric bleeding or renal insufficiency. Ice packs on the affected joint may be helpful in reducing pain: most patients soon find out themselves how to avoid the worst pain by reducing movement and pressure. Occasionally, when NSAIDs cannot be given because of the danger of aggravating existing gastric or kidney disease, an injection or tablets of a corticosteroid drug such as prednisolone can be used to treat acute gout.

NSAIDs are all associated to a greater or lesser extent with a risk of gastric bleeding. This risk can be reduced by the use of some of the newer more selective NSAIDs known as coxibs or by co-prescription of a gastroprotective drug. However all NSAIDs, including coxibs, are contraindicated in patients with heart failure and renal insufficiency and must be used with great care in frail and elderly patients and in patients with risk factors for coronary heart disease and strokes. Colchicine therapy was formerly the treatment of choice and still has a part to play where NSAIDs are contraindicated. However, it frequently causes gastrointestinal side effects (diarrhoea) and has to be used carefully. If only one or two joints are affected and patients have multiple medical problems or are unable to take oral medications, a corticosteroid suspension may be injected through a needle into the joint space in order to reduce inflammation. Very occasionally, steroid drugs (usually prednisolone or prednisone) are used for longer term treatment, where NSAIDs are strongly contra-indicated. For further information on gout and treatment, please see the UK Gout Society's *All About Gout and Treatment* fact sheet at www.ukgoutsociety.org



Step two – reducing the likelihood of recurrences by lifestyle modification

Once the attack has passed, the next step is to help prevent recurrences by addressing trigger factors that can be modified. Patients are best advised to lose weight gradually and progressively if they are obese, to reduce alcohol consumption (especially beer) and intake of sugar-sweetened soft drinks, and to eat smaller amounts of purine-rich food. Such life style modification can work if these environmental risk factors are present and prescription of regular urate-lowering medication is not always necessary. However, patients should be aware that if the blood uric acid levels remain high and if acute attacks continue, then long term joint damage is very likely.

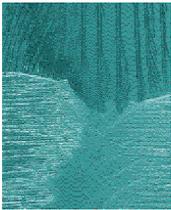
Step three – lowering uric acid levels

In most patients who have repeated attacks, long-term treatment to lower the level of uric acid in the blood below the level at which crystals form in the tissues is required. This is best started when the acute attack has settled. The treatment is then usually continued indefinitely to prevent further attacks. This is essential as the risk of recurrent attacks in a patient prone to gout persists throughout life. The drugs that are used to lower blood uric acid include:

- **Allopurinol**, a drug that reduces production of uric acid in the body. This is especially helpful to people who have abnormally high uric acid levels and kidney stones or damage, but it must be used with considerable caution, and usually in much lower doses, in patients with kidney failure.

- **Uricosuric drugs**, which lower urate levels in the blood by increasing the excretion of uric acid in the urine. These drugs include probenecid and sulfipyrazone, which may not be effective in patients with kidney failure. Benzbromarone is a uricosuric drug that can be used in patients with mild or moderate kidney disease. However, it has been associated with side effects, and so is kept in reserve for patients who are unable to tolerate allopurinol.
- It is important not to start treatment with uric acid lowering drugs until the acute attack has been settled for at least a week. There is an increased likelihood of developing further acute attacks of gout following the initiation of treatment with uric acid lowering agents such as allopurinol or uricosuric drugs. This risk may be present for many months after starting the urate-lowering drug, so small doses of colchicine or NSAIDs are usually prescribed along with the uric acid lowering drug for at least three months and usually longer, to prevent recurring attacks.
- A new drug, **febuxostat**, with a mode of action similar to allopurinol, has recently been given marketing authorisation by the drug regulatory authorities in Europe. The UK National Institute for Health and Clinical Excellence (NICE) has recommended that it may be used to treat gout patients who can't take allopurinol for medical reasons or where the side effects of allopurinol are so bad that the person either has to stop taking it or can't be given the most effective dose. Recommendations for the use of febuxostat for NHS patients with gout in Scotland must await guidance from the Scottish Medicines Consortium (SMC). Supplies of febuxostat should become available for prescription by GPs and hospital specialists in the UK towards the end of 2009.

Does gout recur?



Some people never experience another attack. If the uric acid level remains high most will have a second attack between six months and two years after the first. Subsequently, attacks become more frequent and more prolonged and may result in joint damage if the uric acid level is not controlled.

How can the UK Gout Society help?

The UK Gout Society raises awareness about the challenges of living with gout. It also provides educational materials for people with gout and their families. For further information on the work of the Society, and information on gout, please contact: PO Box 90, HINDHEAD GU27 9FW or email us at: info@ukgoutsociety.org



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