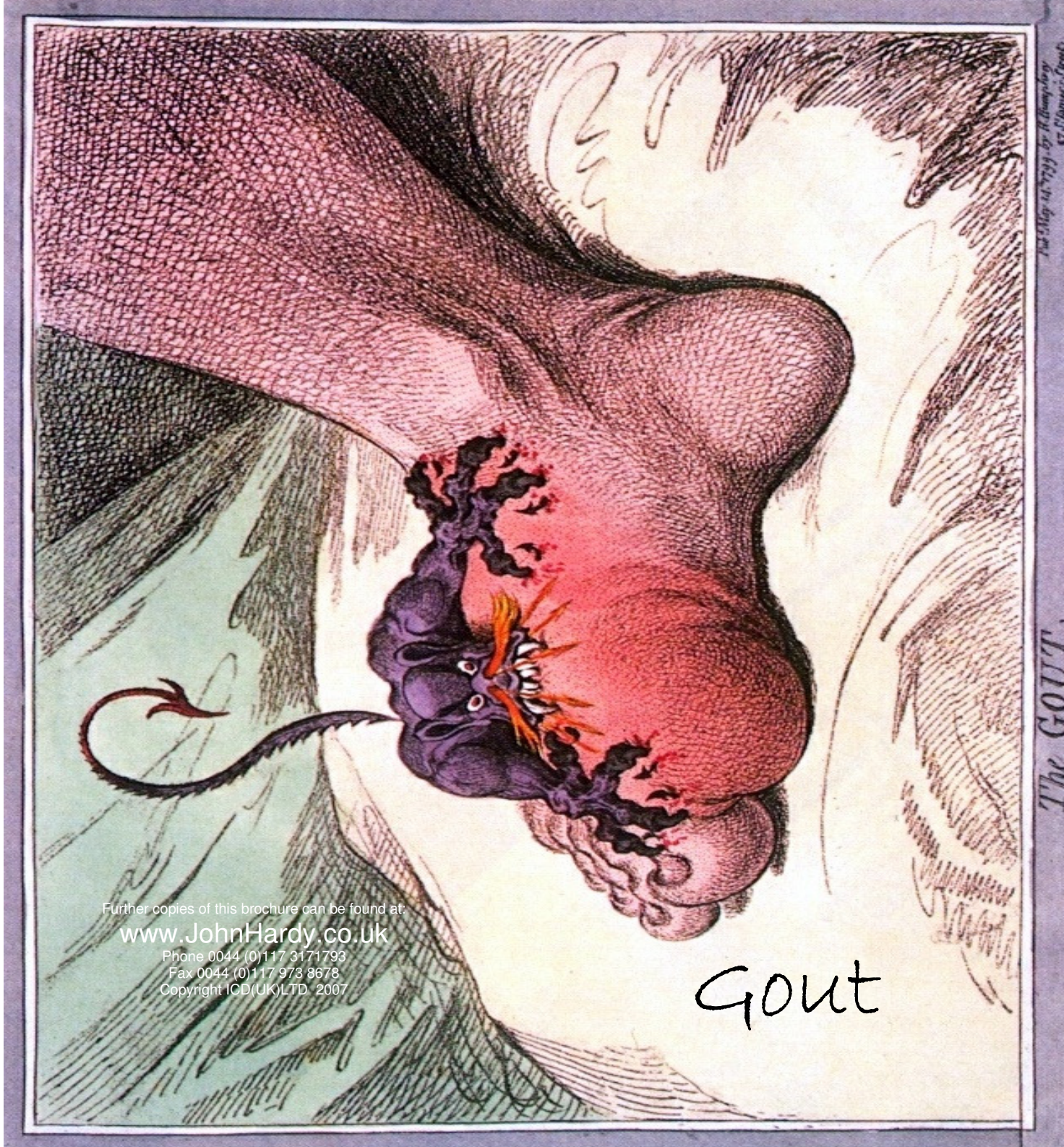
Continued
WHAT ELSE SHOULD I KNOW ABOUT GOUT?

Uric acid is a purine derivative that is the final oxidation product of purine metabolism. It is produced by the enzyme xanthine oxidase, which oxidizes oxypurines such as xanthine into uric acid. Humans and higher primates lack the enzyme uricase which oxidizes uric acid to allantoin. Uric acid is also the end product of nitrogen metabolism in birds and reptiles. In such species, it is excreted in feces as a dry mass. Humans produce only small quantities of uric acid with excess accumulation leading to a type of arthritis known as gout. The loss of uricase in higher primates parallels the similar loss of the ability to synthesize ascorbic acid vitamin C. This may be because in higher primates uric acid partially replaces ascorbic acid[i].

DIET AND GOUT

People who are obese should consult with their doctor to decide on a reasonable weight-loss program. Fasting or severe dieting can actually raise uric acid levels and cause gout to worsen. Usually people can eat what they like within limits. People who have kidney stones due to uric acid may need to actually eliminate purine-rich foods from their diet because those foods can raise their uric acid level. Consuming alcohol can raise uric acid levels and provoke an episode of gout. Coffee and tea are not a problem. Drinking at least 10-12 glasses of non-alcoholic fluids every day is recommended, especially for people with kidney stones, to help flush the uric acid crystals from the body.

Foods very high in purines include: hearts, herring, mussels, yeast, smelt, sardines, sweetbreads. Foods moderately high in purines include: anchovies, grouse, mutton, veal, bacon, liver, salmon, turkey, kidneys, partridge, trout, goose, haddock, pheasant, scallops.

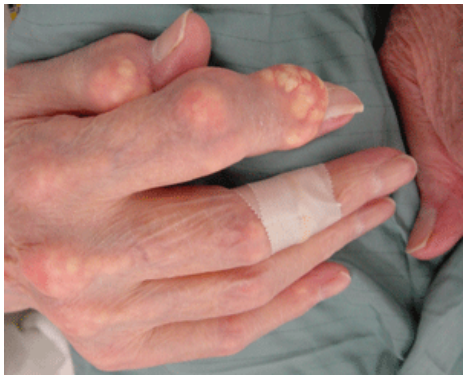


GOUT (HYPERURACAEMIA)

INTRODUCTION

Gout is an inflammation of soft tissue caused by the deposition of monosodium urate crystals in joints and soft tissues.

There are four phases of gout including: asymptomatic hyperuricemia, acute gouty arthritis, intercritical gout and chronic tophaceous gout seen in the image below.



It is a common condition often underdiagnosed. The peak incidence occurs in patients 30 to 50 years old. It occurs in men after puberty and women after the menopause. It is much more common in men than in women.

HOW DO I KNOW I HAVE GOUT?

Acute gout most commonly affects the first metatarsal joint of the foot, but other joints like the knees and tendons like tennis elbow are also commonly involved.

The typical presenting history is of cyclical symptoms of pain in a joint or tendon. Pain is often excrucating and not associated with injury. It increases over a few hours to the point where the limb cannot be used. It responds surprising well to oral non-seroidal anti-inflammatories. Examination shows surprisingly little heat or redness though there is tenderness, swelling and of course pain.

Definitive diagnosis requires either a confirmatory blood test or joint aspiration with demonstration of birefringent crystals in the synovial fluid under a polarized light microscope. Hyperuricaemia (high uric acid in the blood) is a major risk factor for gout and may be a useful diagnostic marker when defined by the normal range of the local population, although some gouty patients may have normal serum uric acid concentrations at the time of investigation.

Radiographs have little role in the diagnosis of gout. However, in late or severe gout radiographic changes of asymmetrical swelling and subcortical cysts without erosion may be useful to differentiate chronic gout from other joint conditions. The condition is occasionally associated with the florid growth of osteophytes which are growth of cartilage and bone at the margin of joint surfaces.

WHAT CAUSES GOUT?

Gout seems to be associated with changes in hormonal balance towards high testosterone. There is a hereditary influence to the disease and certain conditions predispose to it. In addition, there are known risk factors (sex, thiazide diuretics, purine-rich foods, alcohol, lead) and co-morbidities (cardiovascular diseases, hypertension, diabetes, obesity, and chronic renal failure) associated with gout.

WHAT IS NEW IN THE TREATMENT OF GOUT?

Patients with asymptomatic hyperuricemia do not require treatment, but efforts should be made to lower their urate levels by encouraging them to make changes in diet or lifestyle.

Gout attacks are debilitating can cause joint destruction through secondary osteoarthritis and end up with joint deformity. Key therapeutic goals require the serum uric acid levels to be kept below 6mg/dl (360 µmol/L). At this level existing crystal deposition is dissolved, inflammation from crystal deposition is abated and in the stage 4 patient the tophi dissappear[ii].

The importance of patient education, modification of adverse lifestyle (weight loss if obese; reduced alcohol consumption; low animal purine diet) and treatment of associated comorbidity and risk factors. When gout is associated with the use of diuretics, the diuretic should be stopped if possible. Treatment includes nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, corticosteroids and analgesics. In patients without comorbidity, NSAID therapy is preferred.

Long term treatment should be considered for patient with marked joint damage and recurrent attacks. The introduction of allopurinol undercover of a course of NSAID's with a normal serum uric acid level is the recommended way forward.

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[i] Zhang W et al. EULAR evidence based recommendations for gout. Ann Rheum Dis. 2006 Oct;65(10):1301-11.

[ii] Perez-Ruiz F, Lioté F. Lowering serum uric acid levels: what is the optimal target for improving clinical outcomes in gout? Arthritis Rheum. 2007 Oct 15;57(7):1324-8.