# Gout and its Treatment

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***Key points***

- What is Gout?
- Effect of gout on body systems
- Treatment of gout

Gout is one of the most prevailing inflammatory arthritis diseases that arises due to hyperuricemia and elevation in serum urate levels. This concludes in super saturation of urate in body tissues. This further leads to the formation and deposition of monosodium urate crystals in and around the joints. Recent reports of the prevalence and gout incidents vary from a range <1% to 6.8%. Gout is more prevalent in men than in women, with increasing age, and in some ethnic groups. Gout risk factors include obesity, dietary factors and comorbid conditions. As well as a firmly established increased risk of cardiovascular disease and chronic kidney disease in those with gout. Also, novel associations of gout with other comorbidities have been reported which include erectile dysfunction, atrial fibrillation, obstructive sleep apnea (temporary cessation of breathing), osteoporosis and venous thromboembolism. Discrete patterns of comorbidity clustering in individuals with gout have been reported. Consequently, increasing the prevalence and the rising burden of gout in individuals.1

**Treatment**

1.Urate-lowering therapies

All rheumatologic society guidelines endorse the principle that treatment of established gout requires urate lowering. This position is in partial disagreement with the guidelines of the American College of Physicians (ACP), who recommend a “treat to prevent symptoms” approach that has been roundly rejected by gout experts can effectively reduce the frequency of attacks internationally. The primary problem is the

formation.1 When ULT was discontinued from a group of gout patients who had been attack-free for 5 years, flares recurred if the serum urate level returned to a level above 7.0 mg/dL. The higher the serum urate, the more rapidly flares returned. Moreover, if gouty symptoms are prevented with anti-inflammatory agents without concurrently lowering the serum urate level, the relentless process of occult urate crystal deposition will eventually set the patient up for worse, more treatment-resistant gout. Recent studies, not available at the time the ACP guidelines were written, support that prolonged urate lowering.1,2

**Lifestyle**

Diet has long been considered a remediable risk factor in gout, with emphasis placed on reducing intake of purine rich foods and alcoholic beverages, both of which are associated with increases in serum urate levels.1 Fructose consumption notably increases the metabolic production of urate, and obesity, which is an independent risk factor for hyperuricemia. On the other hand, the amount of urate lowering that can be accomplished with dietary intervention alone is unlikely to allow a patient to achieve normal urate levels in the absence of medication, since the major contributor to most patients' hyperuricemia is the genetically determined low efficiency of urate excretion by the kidneys and intestine. Moreover, an unhealthy diet or lifestyle is more likely to lead to non-compliance with gout treatment. Accordingly, while physicians should encourage healthy and tolerable dietary modification, they should be careful not to introduce intolerable dietary restrictions. A good patient-physician relationship may be the most potent gout treatment in the physician's armamentarium.2

Some foods, perhaps by modifying the diet may intensify the inflammatory tone, reflected in a more vigorous Il-1β response to crystals. Thus, dietary habits and alcohol may have an impact on flare rates independent of their direct effects on the serum urate level.2

**Medication**

Several medications can stabilize serum urate levels by either increasing or decreasing them. Drugs like Colchicine and low-dose aspirin raise the serum urate level only slightly; given the need to aggressively control hypertension and provide secondary coronary event prophylaxis to many patients with gout. These increases in the serum urate can generally be managed with attentive monitoring and adjustment of the ULT. In selecting an it is useful to know that is unique among the (ARBs) and antihypertensives. Other drugs such as the antagonists have greater elevating effects on serum urate, but are often harder to replace.2

**References**

1. Dehlin M, Jacobsson L, Roddy E. Global epidemiology of gout: prevalence, incidence, treatment patterns and risk factors. Nature Reviews Rheumatology. 2020 Jul;16(7):380-90.
2. Pillinger MH, Mandell BF. Therapeutic approaches in the treatment of gout. InSeminars in Arthritis and Rheumatism 2020 Jun 1 (Vol. 50, No. 3, pp. S24-S30). WB Saunders.