Gout in the Elderly
Clinical Presentation and Treatment

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Abstract
Gout in the elderly differs from classical gout found in middle-aged men in several respects: it has a more equal gender distribution, frequent polyarticular presentation with involvement of the joints of the upper extremities, fewer acute gouty episodes, a more indolent chronic clinical course, and an increased incidence of tophi. Long term diuretic use in patients with hypertension or congestive cardiac failure, renal insufficiency, prophylactic low dose aspirin (acetylsalicylic acid), and alcohol (ethanol) abuse (particularly by men) are factors associated with the development of hyperuricaemia and gout in the elderly.

Extreme caution is necessary when prescribing nonsteroidal anti-inflammatory drugs (NSAIDs) for the treatment of acute gouty arthritis in the elderly. NSAIDs with short plasma half-life (such as diclofenac and ketoprofen) are preferred, but these drugs are not recommended in patients with peptic ulcer disease, renal failure, uncontrolled hypertension or cardiac failure. Colchicine is poorly tolerated in the elderly and is best avoided. Intra-articular and systemic corticosteroids are increasingly being used for treating acute gouty flares in aged patients with medical disorders contraindicating NSAID therapy.

Urate-lowering drugs are indicated for the treatment of hyperuricaemia and chronic gouty arthritis. Uricosuric drugs are poorly tolerated and the frequent presence of renal impairment in the elderly renders these drugs ineffective. Allopurinol is the urate-lowering drug of choice, but its use in the aged is associated with an increased incidence of both cutaneous and severe hypersensitivity reac-
tions. To minimise this risk, allopurinol dose must be kept low. A starting dose of allopurinal 50 to 100mg on alternate days, to a maximum daily dose of about 100 to 300mg, based upon the patient’s creatinine clearance and serum urate level, is recommended. Asymptomatic hyperuricaemia is not an indication for long term urate-lowering therapy; the risks of drug toxicity often outweigh any benefit.

Gout is the most frequent microcrystalline arthritis, affecting approximately 0.5 to 2.8% of adult men and 0.1 to 0.6% of adult women, with an overall prevalence of about 0.3 to 1% depending on the population studied.\textsuperscript{[1-3]} It chiefly affects adult men, with a peak incidence in the fifth decade of life. The incidence of gout increases in women in the postmenopausal period. Approximately 10 to 25% of patients have a family history of gout. Because of increasing longevity, changes in diet, lifestyle and the frequent long term use of thiazide diuretics and prophylactic low dose aspirin (acetylsalicylic acid), the prevalence of gout is rising in countries with a high standard of living.

Hyperuricaemia is present in about 5% of asymptomatic adults and about 10% of hospitalised patients. However, fewer than 1 in 4 individuals with hyperuricaemia will develop gouty arthritis after a period of years, and the incidence of the disease increases with increasing serum urate levels.\textsuperscript{[2,3]}

Gout is one of the better understood of the rheumatic diseases and is characterised by chronic hyperuricaemia, recurrent attacks of acute arthritis provoked by the release of monosodium urate crystals into joint cavities, and the development in some patients of gross urate deposits (tophi).\textsuperscript{[1-3]} The natural history of untreated gout consists of 3 overlapping phases occurring over a period of 20 to 40 years: a long phase of asymptomatic hyperuricaemia, a period of recurrent acute gouty attacks separated by symptom-free intervals (interval gout), followed in about 10% of patients by the development of chronic tophaceous gouty arthritis.\textsuperscript{[1-3]}

In the majority of patients, gout is a primary disorder. Less commonly, hyperuricaemia and gout are secondary to purine enzyme defects, myelo- or lymphoproliferative disorders, renal failure, drugs or other conditions such as haemolytic anaemia, psoriasis or metabolic acidosis (table I). The measurement of 24-hour urinary uric acid excretion in younger patients with primary gout can often indicate whether the hyperuricaemia is the result of the overproduction or underexcretion of urate.\textsuperscript{[4,5]}

In most patients (80 to 90%), hyperuricaemia is caused by a renal tubular defect in the excretion of uric acid (gouty underexcretors).\textsuperscript{[1,4]} These patients excrete either normal or reduced amounts of uric acid (<600mg or 3.6 mmol/day on a purine-restricted diet), and have a urate clearance lower than that found in healthy individuals (<6 ml/min, compared with 6 to 11 ml/min). They have a limited capacity to eliminate a urate (purine) load, and excretion of normal amounts of uric acid is accomplished only at inappropriately high serum urate levels.\textsuperscript{[4]}

In a minority of patients with primary gout (10 to 20%), hyperuricaemia is caused by the overproduction of uric acid. These patients also excrete excessive quantities of uric acid (>800 to 1000mg or > 4.5 to 5.5 mmol/day while on a regular diet), and are classified as gouty overproducer overexcretors. In these patients, urate clearance is often normal and studies using isotope-labelled urate invariably demonstrate an increased rate of de novo purine (and uric acid) biosynthesis.\textsuperscript{[1,4,5]}

1. Clinical Features

1.1 Classical Gout

Gout chiefly affects middle-aged men, but the incidence in women increases after menopause. Familial occurrence, caused by polygenic inheritance, is reported in up to 25% of patients.\textsuperscript{[1-3]} Acute gouty arthritis commonly affects the joints of the lower extremity, particularly the metatarso-phalangeal (MTP) joint of the great toe (acute podagra). The intertarsal, ankle, knee, elbow and