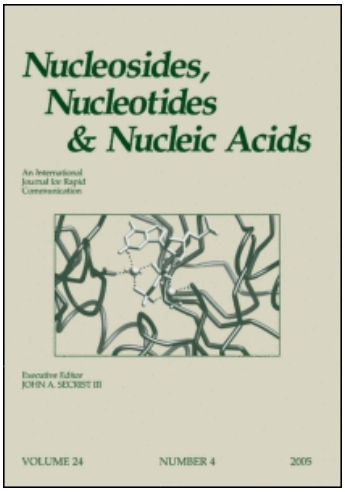

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AN UNUSUAL PATIENT WITH HYPOTHYROIDISM, TOPHACEOUS GOUT, AND MARKED JOINT DESTRUCTION

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□ *This report describes a 75-year-old Caucasian man with extensive urate deposits and severe gouty arthropathy that confined him to a wheelchair. Since age 50, he suffered multiple acute gout flares and progressive deformities in his hands, feet, knees, and elbows (tophi). Serum creatinine was 1.4 mg/dL and serum urate 9.4 mg/dL. Conditions known to increase uric acid production (psoriasis, chronic bronchitis) and to decrease uric acid excretion (hypothyroidism, metabolic syndrome, and nephroangiosclerosis) may operate in a single patient, illustrating the dramatic clinical course of untreated gout.*

Keywords Uric acid; hyperuricemia; tophus; gout; hypothyroidism

INTRODUCTION

Tophaceous gout with marked joint impairment is currently uncommon. Its presentation should elicit the investigation of secondary causes of hyperuricemia. We present a patient with huge urate deposits and severe gouty arthropathy that confined him to a wheelchair. Several pathogenic factors were identified known to cause uric acid overproduction and renal underexcretion. The coincidence of both pathomechanisms in a single patient may well explain this very unusual case of tophaceous gout.

CLINICAL DESCRIPTION

A 75-year-old Caucasian man came to our attention as a "resistant case of tophaceous gout." He reported being an active smoker and a regular alcohol drinker in the past (20 g of alcohol daily). He had been diagnosed as having chronic bronchitis, psoriasis, arterial hypertension, obesity grade 1, and diabetes mellitus type 2. He had been treated with oral hypoglycaemic

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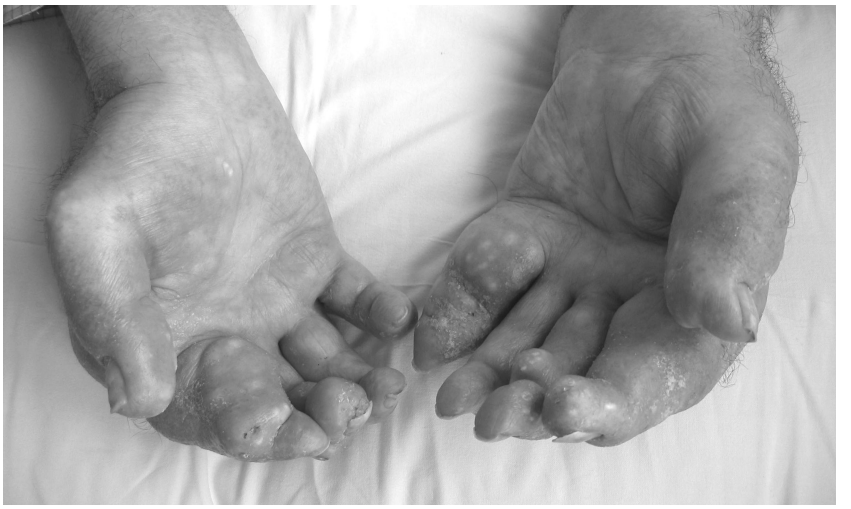

FIGURE 1 Bilateral hand tophaceous deposits in a 75-year-old man with hypothyroidism and several concomitant pathomechanisms leading to uric acid overproduction and underexcretion.

agents and various anti-hypentensive drugs, including diuretics. At age 50, he suffered his first gouty attack (podagra). Since then he has had multiple acute gout flares in his feet, hands, elbows and knees. In 1991, he noted the appearance of multiple tophi localized to his hands, feet and elbows. Tophi progressively increased in size and in recent years limited his joint movements to the point that he could barely walk or eat unaided. He was told that at this stage no medication could help him. Physical examination disclosed an obese man (BMI 30.2 kg/m^2), with no goiter, and marked deformities of his hands (Figure 1), feet, knees, and elbows.. Multiple tophi of different sizes could be identified, with severe joint inflammation. Several hand and feet tophi produced a white-chalk material and ulcerations,. Blood analyses showed a haemoglobin level of 12.3 g/dL, and leucocytes 14700 per μL. Serum creatinine was 1.4 mg/dL and creatinine clearance (Cockroft formula) 48 mL/minute. Urinary protein was 2.28 g/day. Serum urate was 9.4 mg/dL. Thyroid stimulating hormone (TSH) was 74.7 μUI/mL (normal 0.25–5.00 μUI/mL) and free T4 0.52 ng/dL (normal 0.7–1.6 ng/dL). Antithyroid antibodies were negative.

FOLLOW UP

The patient has been followed up for 12 months. He was initially treated with thyroid hormone (25 μg/day), which was gradually increased to 200 μg/day, according to TSH levels. Serum TSH progressively decreased to normal levels (TSH, 2.23 μUI/mL). Due to his huge urate deposits, low dose allopurinol was initiated at 50 mg/day and gradually increased to

200 mg/day with prophylactic colchicine. Low dose steroids (prednisone, 4 mg/day) were also prescribed to reduce marked joint inflammation. Serum urate progressively decreased to 7.1 mg/dL at 6 months, but we could not ascertain if this decrease was related to thyroid replacement therapy since he was treated with allopurinol. At 12 months, serum urate was 7.7 mg/dL and serum creatinine remained stable at 1.4 mg/dL. For the last 12 months, no substantial changes were observed in his tophi deposits. He lost 10 kg of weight, and his subjective sense of well being improved, probably due to euthyroidism and gouty flares being controlled. For the last 12 months the patient had not had an acute gouty attack.

COMMENTS

Several pathogenic factors of hyperuricemia and gout could be identified in this patient. He reported being an active alcohol drinker,[1–3] and he suffered from psoriasis[4,5] and chronic bronchitis,[6] three conditions associated to uric acid overproduction. Psoriatic arthritis is often confused with gouty arthritis,[7,8] but in this case the presence of huge tophi and the absence of radiologic characteristics of psoriatic arthritis made the latter diagnostic unlikely.

Several conditions associated to uric acid underexcretion were also identified. Arterial hypertension,[9,10] the use of thiazides and loop diuretics,[11,12] obesity,[13,14] and a high alcohol intake,[1–3] may also have contributed in an additive manner to the unusual tophaceous deposits. Moderate, stage 3, chronic renal failure was also diagnosed, possibly due to nephroangioesclerosis, diabetic nephropathy or both. Remarkably, the patient reported also had severe hypothyroidism, which is a frequently unrecognized secondary cause of gout.[15,16]

We could not ascertain the contribution of hypothyroidism to this unusual case of tophaceous gout since, in our opinion, his clinical situation precluded any long-term clinical investigation without hypouricemic therapy. Thus, the patient was simultaneously treated with thyroid hormone and allopurinol,[17,18] and we could not determine whether thyroid therapy by itself would have normalized uric acid metabolism.

This unusual case of tophaceous gout exemplifies the dramatic clinical course of untreated gout due to several pathogenetic factors. In addition, this report highlights that secondary causes of hyperuricemia, including hypothyroidism, should be identified to offer the best available therapy, considering all pathogenic circumstances.

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