CHRONIC, LONG-LASTING, AND UNTREATED GOUT WITH CONCOMITANT DILATED CARDIOMYOPATHY AND EXCEPTIONALLY VAST ANASARCA: CASE REPORT

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SUMMARY – Gout is the most common type of inflammatory arthritis in man caused by deposition of urate crystals into the joints as the result of elevated serum urate levels. A case of a 59-year-old patient with untreated, long-lasting gout and clinical manifestation of decompensated global dilated cardiomyopathy is presented. Examination revealed generalized pitting edema extending from both lower extremities to the sacrum, abdominal, and thoracic wall, with scrotal swelling and upper extremity involvement, an exceptionally vast generalized edema, i.e. anasarca. Proximal and distal interphalangeal joints of the hands and feet were swollen and deformed, with marked yellow tophi nodules. Laboratory studies revealed high serum uric acid concentration (546 μmol/L), decreased creatinine clearance (0.8 mL/s) and albumin concentration (27.4 g/L), as well as increased total urine protein mass (0.35 g/24 h). X-rays of the affected feet and fists showed punched-out lesions of the subchondral bone with overhanging bony margins in the first metatarsophalangeal, proximal, and distal interphalangeal joints of both hands. The extreme clinical presentation resolved upon intravenous administration of diuretics and pleurocentesis, followed by oral medications including furosemide, angiotensin-converting enzyme inhibitor, spironolactone and digoxin. Since serum urate level has been identified as an independent risk factor for the development of ischemic heart and chronic kidney disease, regulation of urate concentration is necessary, especially in patients diagnosed with gout.

Key words: *Gout; Arthritis, gouty; Cardiomyopathy, dilated*

Introduction

Gout is the most common type of inflammatory arthritis in man[1]. It is caused by deposition of urate crystals into the joints as a result of an elevated serum urate level[2]. Uric acid is formed by catabolism of the purine bases adenine and guanine. Approximately two-thirds of the uric acid is excreted through the kidneys and one-third through the gastrointestinal tract. Hyperuricemia can result from overproduction or reduced excretion of uric acid, or both[3].

Herein, we report on a patient with untreated, long-lasting chronic gout presenting with signs and symptoms of decompensated global dilated cardiomyopathy and exceptionally vast generalized edema, i.e. anasarca.

Case Report

A 59-year-old Caucasian male presented to the Emergency Room complaining of worsening dyspnea at minimal exertion, orthopnea, anuria, and marked abdominal distension with generalized scrotal and limb edema (anasarca). Previous medical history in-

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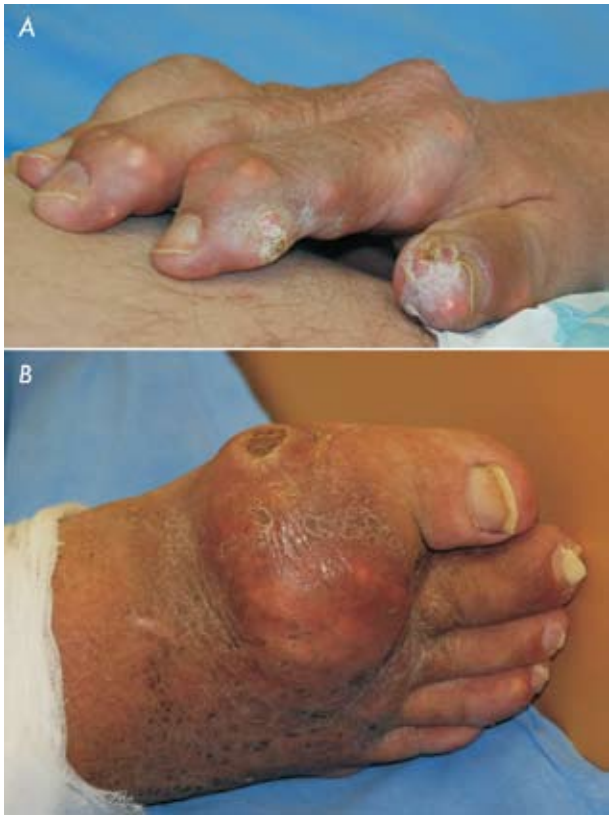

Fig. 1. Marked yellow tophi nodules on both hands (A), and feet (B).

cluded established diagnosis of gouty arthritis twenty years before, without regular medical treatment during the last fifteen years. On admission, the patient was alert, with central type cyanosis and inability to walk due to anasarca. He was afebrile, with blood pressure 160/100 mm Hg and arterial oxygen saturation 50% on room air. Physical examination revealed tachycardia, gallop rhythm with soft heart sounds, and expiratory wheezing with evidence of percussion dullness at the basis of both lungs. The abdomen was distended and tense, therefore hepato- or splenomegaly could not be assessed. Generalized pitting edema extending from both lower extremities to the sacrum, abdominal and thoracic wall, with scrotal swelling and upper extremity involvement was present. Proximal and distal interphalangeal joints of the hands and feet were swollen and deformed, with marked yellow tophi nodules (Fig. 1).

Laboratory studies on admission revealed normal complete blood count, increased sodium (145 mmol/L) and decreased potassium level (3.6 mmol/L), and compensated hypochloremic metabolic alkalosis with hyperkapnia. Liver enzymes, PT, and APTT were normal, with increased fibrinogen (6.0 g/L) and D-dimers (4.83 mg/L), low LDL and HDL (1.7 mmol/L and 0.9 mmol/L, respectively), and high serum uric acid concentration (546 μmol/L). Serum protein electrophoresis revealed decreased total protein (56 g/L) and albumin concentration (27.4 g/L), while immunoelectrophoresis was normal. Blood urea and creatinine concentrations were normal, while creatinine clearance was decreased (0.8 mL/s), with increased total urine protein mass (0.35 g/24 h). Serum TSH concentration was increased (10 mIU/L), with low T3 and T4 concentrations.

Radiography of the chest showed cardiomegaly, hilar congestion, and bilateral pleural effusions. Electrocardiography showed atrial fibrillation with uncontrolled ventricular response. The echocardiogram showed left and right ventricular dilatation and dysfunction with fractional shortening (28% and 25%, respectively), second grade mitral and tricuspid valve regurgitation. X-rays of the affected feet and fists showed punched-out lesions of the subchondral bone

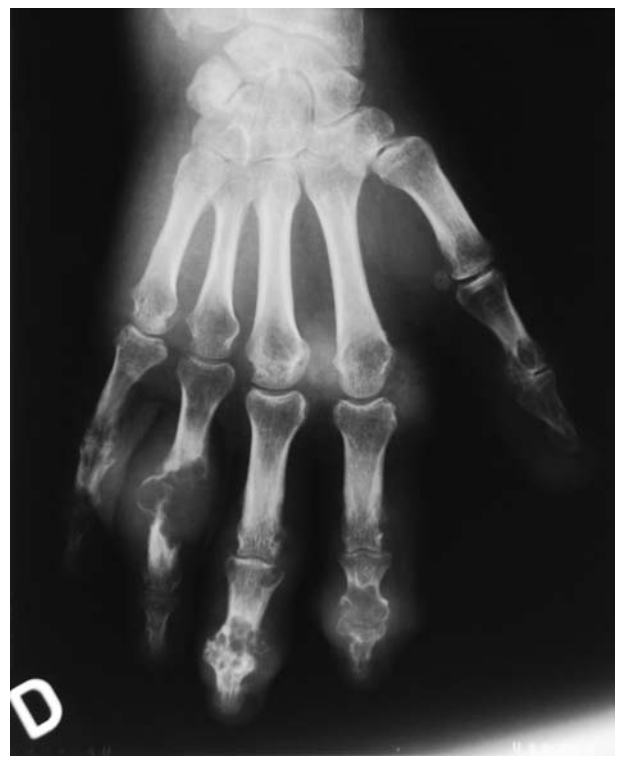

Fig. 2. Punched-out lesions of subchondral bone of proximal and distal interphalangeal joints of the right hand.

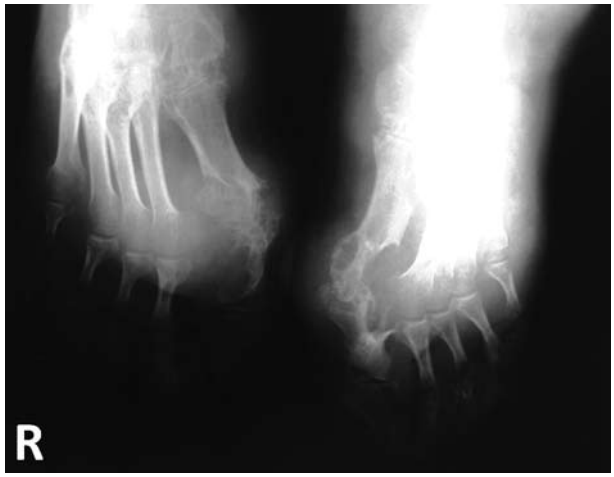

Fig. 3. Punched-out lesions of subchondral bone with overhanging bony margins in the first metatarsophalangeal joints of both feet.

with overhanging bony margins in the first metatarsophalangeal joints, as well as proximal and distal interphalangeal joints of both hands (Figs. 2 and 3).

The patient was admitted and started on intravenous furosemide. Given the likelihood of gastrointestinal edema with impaired absorption, oral medications were withheld. Pleurocentesis yielded 1250 mL of pleural effusion indicative of transudate. Over the next three weeks, the patient lost some 20 kg of the initial weight. During the treatment, oral medications, including furosemide, angiotensin-converting enzyme inhibitor, spironolactone, and digoxin were resumed. The patient was administered substitution levothyroxine.

Discussion

The case is presented of a patient with chronic, severely advanced, and untreated gout, in whom dilated cardiomyopathy was decompensated resulting in large pleural effusions, ascites, and exceptionally vast presentation of anasarca. The prolonged course of untreated gout resulted in deformities of fingers with notable tophi on both extremities. In spite of the reported elevated serum urate concentration in patients with severe left heart disease[3], the etiology of evaluated cardiomyopathy still remains unrecognized. To our knowledge, cardiomyopathy was associated with gout in only two cases but always with concomitant neurological abnormalities presenting in the third decade of life[4,5]. Since hyperuricemia has been established as a well-known risk factor for the development of ischemic heart disease, aggressive management of the cardiovascular risk factor is advisable[6]. Finally, recent epidemiologic studies have identified serum urate elevations as an independent risk factor for chronic kidney disease, which yields regulation of urate concentration necessary[7].

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Sažetak

KRONIČNI DUGOTRAJNI NELIJEČENI GIHT S ISTODOBNOM DILATACIJSKOM KARDIOMIOPATIJOM I IZNIMNO OPSEŽNOM ANASARKOM

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Giht je najčešći tip upalnog artritisa, a uzrokovan je nakupljanjem kristala urata u zglobovima kao rezultat povišene razine urata u serumu. Prikazan je slučaj 59-godišnjeg bolesnika s neliječenim uričnim artritisom i kliničkom manifestacijom globalno dekompenzirane dilatacijske kardiomiopatije. Kliničkim pregledom dominirala je anasarka, tj. opsežan generalizirani tjestasti edem oba gornja i donja ekstremiteta, skrotalni otok, edem trbušne i prsne stijenke. Proksimalni i distalni interfalangealni zglobovi šaka i stopala bili su otečeni i deformirani, uz vidljive žute čvoričaste tofe. Laboratorijski testovi pokazali su visoku koncentraciju mokraćne kiseline u serumu (546 μmol/L), smanjeni klirens kreatinina (0,8 mL/s) i koncentraciju albumina (27,4 g/L), te povišenu ukupnu masu bjelančevina u mokraći (0,35 g/24 h). Rendgenske snimke zahvaćenih stopala i šaka pokazale su destrukcije i rubne uzure s uzdignućem korteksa u prvom metatarzofalangealnom, proksimalnom i distalnom interfalangealnom zglobu obiju šaka. Ova ekstremna klinička slika riješena je intravenskom primjenom diuretika i pleurocentezom, nakon čega je slijedila oralna terapija uključujući furosemid, inhibitor enzima za konverziju angiotenzina, spironolakton i digoksin. Kako su serumske razine urata utvrđene kao neovisan čimbenik rizika za razvoj ishemijske srčane i kronične bubrežne bolesti, regulacija koncentracije urata je neophodna, naročito kod bolesnika s dijagnosticiranim gihtom.

Ključne riječi: *Urični artritis – giht; Kardiomiopatija, dilatacijska*