CASE REPORT

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A 52-year-old man with gouty arthritis and erosive lesion in the hip

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Abstract

The case study of peripheral and axial gouty arthritis is described in a 52-year-old man without concomitant clinical evidence of tophaceus gout on physical evaluation on admission. Gout is a metabolic disorder related to excess of uric acid in the extracellular compartment, and deposition of monosodium urate crystals in the joints and other sites. Arthritis and tophi are major manifestations, which more often involve the peripheral joints asymmetrically. Chronic tophaceous gout commonly develops after a decade of recurrent polyarticular gout. With lower frequency, the axial skeleton (spine and sacroiliac region) may be affected, condition sometimes associated with additional concerns, diagnostic challenges and pitfalls. Higher suspicion index and utilization of novel radiographic tools can settle these matters. Radiographic imaging exams include plain radiography, computed tomography (CT), magnetic resonance imaging (MRI), and bone scintigraphy to show bone erosion and joint lesions characterizing the spectrum of gouty osteoarthropathy.

Keywords: gout, iliac bone, erosions, bone imaging.

Introduction

Gout is a common metabolic disease that is associated with microcrystalline arthropathy, mainly peripheral and monoarticular, with significant prevalence in men of 30–40 years [1–6]. Although axial lesions by monosodium urate (MSU) are considered rare, they actually seem underdiagnosed [3, 4]; gout also causes patellar, spinal, pubis and sacroiliac arthritis, and pathological fracture [2]. Renal failure, obesity, hypertension, diuretic and salicylate (acetylsalicylic acid – ASA) are risk factors of axial gout [3]. Finding of MSU crystals in joint aspirates is the hallmark of tophaceous gout [7]; but the detection by polarizing microscopy may be inconsistent [1, 2]. Pathological study of biopsy sample is an invasive option to be performed in selected cases [3]. Imaging methods are useful to show erosive lesions with marginal over-hanging, in addition to tophi deposition in joints and adjacent tissues, and local inflammatory phenomena [1–4, 6]. Magnetic resonance imaging (MRI), scintigraphy and dual-energy computed tomography (DECT) scans have been used to confirm cases of gouty lesions. MRI may detect tophus, bone marrow edema and inflammation, and DECT can better detect the presence and effects of MSU in joints [1–4, 6].

The middle-aged obese male herein reported presented with arterial hypertension and chronic kidney disease, and was in longstanding regular intake of ASA in low dosage.

The aim of this manuscript is to enhance the suspicion index of workers in primary health care about the sacro-iliac involvement by gout that is considered an uncommon condition.

Case presentation

A 52-year-old man, with hypertension, chronic renal failure and gout, was admitted because of pain in the lower limbs with growing intensity for 10 days, without improvement with common analgesics. He also had pain in low back and right hip areas hindering ambulation. He used atenolol (25 mg/day), amlodipine (20 mg/day), allopurinol (300 mg/day), losartan (50 mg/day), ASA (100 mg/day) and colchicine (0.5 mg/day). He had been a tobacco smoker until two years ago and denied alcoholism. There was familiar antecedent of chronic renal disease and arterial hypertension. His kidney insufficiency was recently diagnosed, and the gout was poorly controlled due to not following an adequate diet. Physical examination showed: body mass index (BMI) 30 kg/m^2; abdominal waist 115 cm; yellowish skin; edema and hyperemia in the right index finger and ankles (Figure 1, A–E); pain at mobilization of right coxofemoral joint, knees, ankles and hallux. No gouty tophus was detected. Opioids and colchicine were needed to control the arthritic symptoms during admission. The routine laboratory tests and controls are showed in Table 1.

Determinations of uricosuria, glucose, calcium, albumin, globulins, immunoglobulins (IgA, IgG, IgM), rheumatoid factor, human leukocyte antigen (HLA B27), antinuclear antibody (ANA), anti-cyclic citrullinated peptide (CCP), direct Coombs test, venereal disease research laboratory (VDRL), parathyroid hormone (PTH), human growth hormone (HGH), thyroid-stimulating hormone (TSH), free thyroxine, TSH receptor antibodies, cortisol, and prostate-specific antigen (PSA) were unremarkable. Repeated urine cultures and blood cultures were negative. Additional

abnormal tests (normal ranges) were erythrocyte sedimentation rate 76 (<15) mm/h, C-reactive protein 12.7 (0.5–0.9) mg/dL, clearance of creatinine 26 (≥90) mL/min/1.73 m^2, β_2-microglobulin 4.63 (1.09–2.53) mg/L, proteinuria 639 (<150) mg/24 h, adrenocorticotropic hormone (ACTH) 3.46 (7.2–63.3) pg/mL, ferritin 1203 (30–400) ng/mL, transferrin 160.7 (200–360) mg/dL, vitamin D 20.16 (>30) ng/mL, and vitamin B_{12} 1037 (211–946) pg/mL. Synovial fluid of right finger was turbid; pH 7; nucleated cells 390/mm^3 (85% neutrophils); red cells 2240/mm^3; glucose 59 mg/dL; lactate dehydrogenase (LDH) 2272 IU/dL; absence of crystals; absence of Gram-stained microorganisms, and negative cultures. On D5, a CT of the lumbosacral spine showed an erosive lesion in the left iliac bone with medial thinning and sclerosis in its most lateral aspect, extending to the proximal sacroiliac joint (Figure 2A). Incipient anterior osteophytes were seen in the L3–L5 bodies; the intervertebral discs, conjugate foramina, dural sac, nerve roots, spinal canal, and epidural fat were unremarkable. MRI of the pelvis revealed edema in the mid-substance of the soft tissues adjacent to the right gluteal tendons, associated with focal edema in the large femoral tuberosity and net distension of the trochanteric bursa and the related synovial bursae. Edema of subcortical bone in the iliac margin of the left sacroiliac joint, and erosion and irregularity of the cortical in the lower margin of the iliac bone, typical of osteolytic injury; bilateral distention of the iliopsoas bursa, and normal aspect of gluteal tendons as well as the relationship of acetabulum with femoral head (Figure 2B). Bone scintigraphy of the pelvis showed a lithic area with sclerotic borders in the lower left iliac region near the sacroiliac joint, with a slight increase in osteoblastic activity (Figure 2C). The patient evolved with significant improvement of the signs of arthritis (Figure 1F) after the schedule with ceftriaxone (14 days) and oxacillin (21 days). On D33, he was discharged and referred to specialized follow-up under the outpatient care of the Nephrology and Rheumatology teams.

Table 1 – ***Laboratory tests of a 52-year-old man with chronic renal failure and gout***

Parameters (normal ranges)	**D1**	**D8**	**D14**	**D29**	**D33**
Hemoglobin (13.5–18 g/dL)	14.4	**13**	13.8	**11.5**	**12.5**
Hematocrit (42–52%)	**41.3**	**37.9**	**39**	**34**	**35.5**
Leukocytes (4–11×10^9/L)	**20.7**	**16.4**	**18.4**	8.7	8.9
Neutrophils (40–70%)	**90**	**84**	**85**	**73**	70
Band forms (0–3%)	**5**	**4**	1	**6**	3
Platelets (140–450×10^9/L)	327	318	406	285	397
Uric acid (3.4–7 mg/dL)	–	–	**8.9**	**10.3**	9
Sodium (135–145 mmol/L)	136	144	137	139	140
Potassium (3.5–5.2 mmol/L)	**6.4**	**5.8**	5.2	4.5	4.5
Urea (10–50 mg/dL)	**239**	**180.6**	**108.6**	**88.5**	**90**
Creatinine (0.7–1.3 mg/dL)	**4.8**	**3.3**	**2.5**	**2.6**	**2.6**

D: Day of hospitalization; D1: Admission; D33: Discharge. Abnormal data are showed in bold.

Discussion

The male patient had history of hyperuricemia and classical chronic gouty arthritis, manifested by recurrent asymmetric podagra well controlled by colchicine and allopurinol [7]. Four years ago, a unique gouty tophus was transitorily observed on the right index finger. This obese man had hypertension and chronic renal disease, and utilized ASA 100 mg daily, and was admitted with acute polyarticular involvement that included sacral and lumbar pain. The acute episodes of gout are characteristically peripheral, monoarticular or oligoarticular; however, in the chronic phase of disease, the involvement may become polyarticular [1–3, 5, 7]. MSU tophi more often develop in chronic gout, but they can be the first sign of disease [5]. Although the axial gout is uncommon, chronic renal failure, obesity, arterial hypertension, se of diuretics, and use of salicylates in low dosage constitute the main risk factors of this entity [3]. Criteria of axial gout are bone erosions, joints or disk calcification, and skeletal tophi [4].

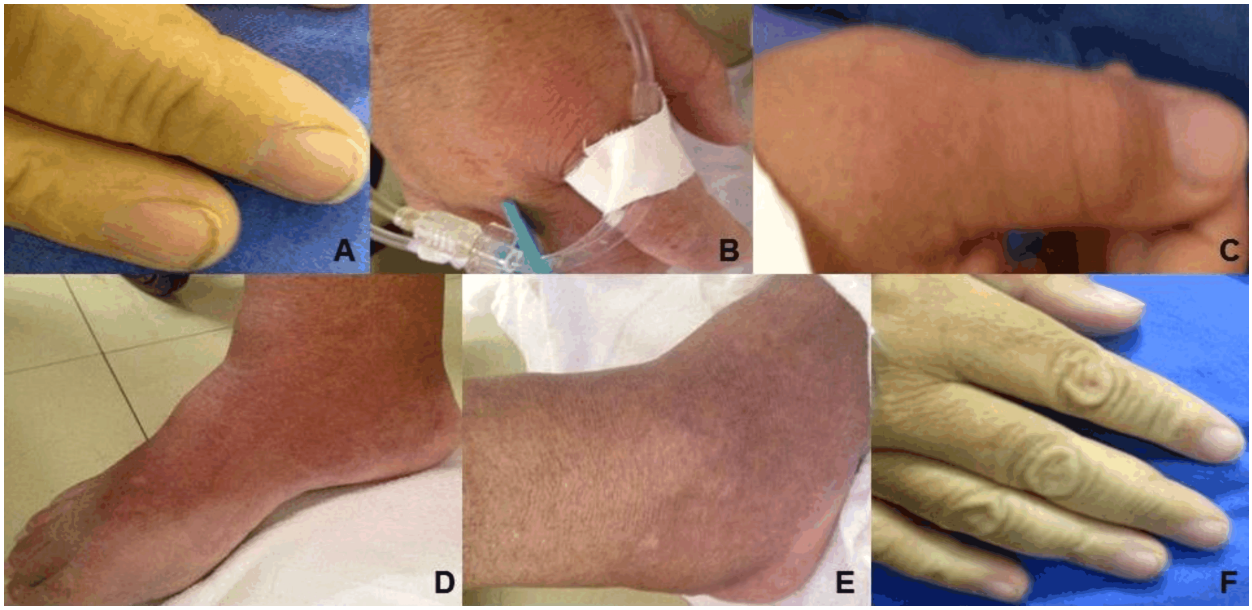

Figure 1 – ***(A) Hyperemia and edema in the joints of the right index finger; (B) Yellowish skin, and nails with indistinct lunula and discrete longitudinal ridges; (C) Acute inflammatory signs of gouty arthritis in a hand finger; (D and E) Bilateral perimalleolar edema; (F) Absence of inflammatory signs after the complete antibiotic course.***

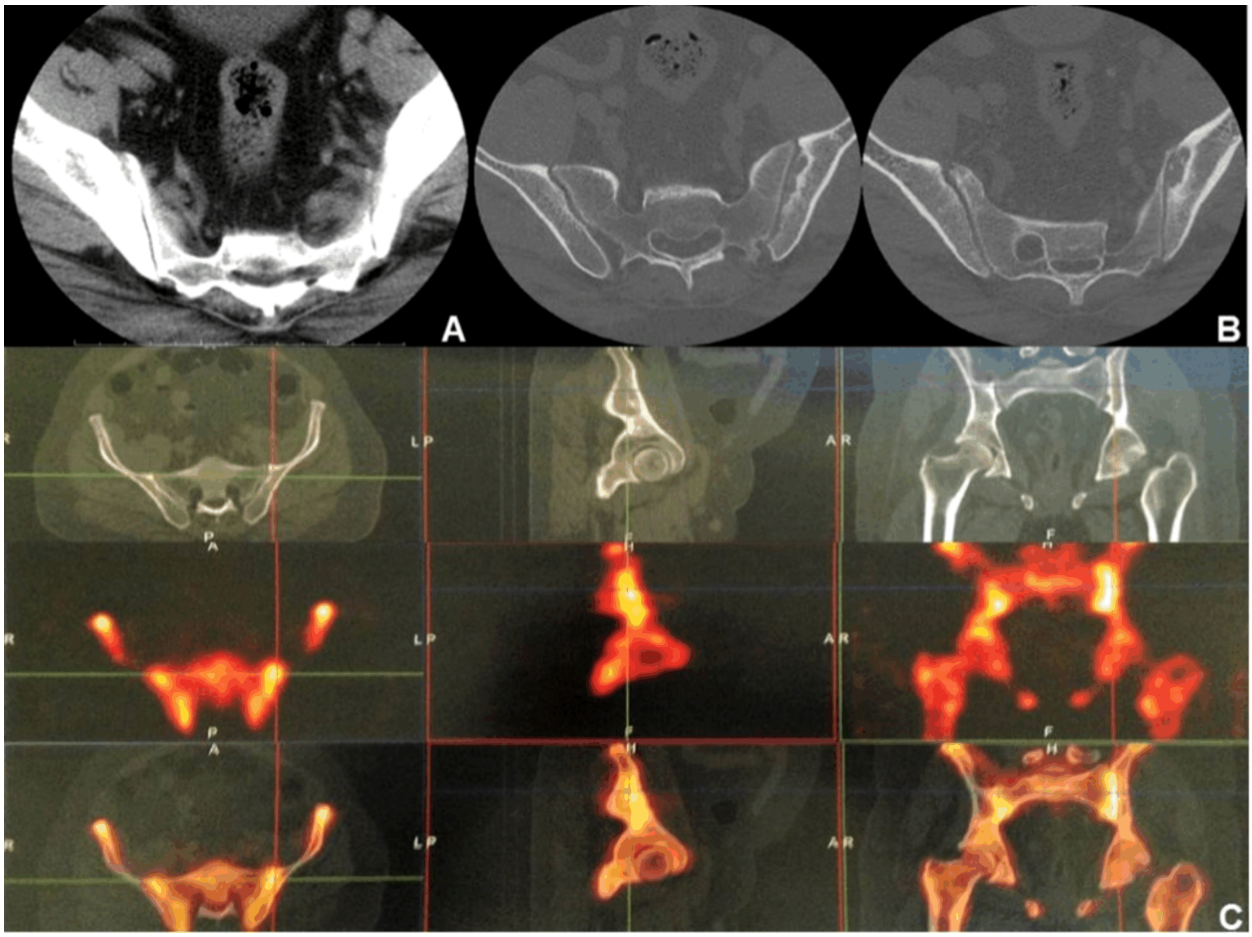

Figure 2 – ***(A) CT of the lumbosacral area showing an erosive lesion in the left iliac bone with medial thinning and lateral sclerosis extending to the proximal sacroiliac joint; (B) MRI of the pelvis revealing edema of the soft tissues adjacent to the right gluteal tendons, focal edema in the large femoral tuberosity and distension of the trochanteric bursa and related synovial bursae. Edema of subcortical bone in the iliac margin of the left sacroiliac joint, erosion and irregularity of the cortical at lower margin of iliac bone, indicative of osteolytic injury; (C) Bone scintigraphy of the pelvis showing a lithic area with sclerotic borders and slight increase of osteoblastic activity in the inferior region of the left iliac bone near to the sacroiliac joint.***

Osteoarticular changes are due to tophus pressure, proinflammatory cytokines, chemokines, and matrix-degrading enzymes produced by inflammatory and immune cells from tophi [6]. Radiographic imaging tools including plain radiography, CT, DECT, MRI, and scintigraphy may show the MSU crystals and tophi that can contribute to bone and joint lesions of gout [6]. CT evaluation of 42 gouty patients showed axial gout in 29%, axial tophi in 12%, erosions or calcifications in 17% of cases. The frequencies of lumbar, thoracic and sacroiliac lesions were 100%, 42% and 18%, respectively; and higher prevalence was related to peripheral tophi [4]. Plain radiograph evaluation of 290 gouty patients with less than 10 years of disease evolution yielded additional evidence that the development of tophus plays a role in bone erosions [8, 9]. MRI evaluation of 40 gouty patients was performed for edema, erosions, tophi and synovitis, whereas DECT was utilized to evaluate a subgroup of 10 gouty individuals. Erosions were found in 63% of cases and showed a significant association with the presence of tophi; however, there was no relationship between the erosions and the presence of bone edema [9]. Plain radiograph and DECT evaluation of 92 patients with tophaceous gout found that both MSU and soft tissue components of tophus are strongly associated with bone erosions [10]. Differing from previous data, peripheral tophi did not occurred in the patient herein described; the MRI osteolytic findings of this case study were edema of subcortical bone at iliac margin of the left sacroiliac joint, and erosion of the cortical in the lower margin of the iliac bone. The radiographic prevalence of gouty sacroiliac arthropathy is estimated between 13% and 17%, and the axial involvement as a whole may be up to 35%; notwithstanding, two major differential diagnoses that should be ruled out are infectious arthritis and osteomyelitis [3]. However, the diagnosis of gout coexistent infectious arthritis was not discarded in the present case because microbiological investigation was done after the beginning of antibiotic schedule. The rapid improvement of acute arthritis after antibiotic therapy is on favor of this hypothesis; however, the possibility of gout causing lesions that mimic infection was also considered [5]. MSU crystals and tophi play a role in bone erosion and damage in the joints affected by gout, but these changes can develop without tophi in late stage or after recurrent attacks of gout [6]. Worthy of note, advanced renal disease is an immunosuppressive condition and may change typical phenomena of gout or infectious arthritis, like in coexistent rheumatoid arthritis [7]. This may involve inhibition of connective tissue and synovial fluid changes of both diseases, with interference

in crystal deposition, phagocytosis, and MSU–neutrophil interactions [7].

Conclusions

The axial (spinal and sacroiliac) gouty arthritis has been considered a very uncommon or rare condition; but it seems to be underdiagnosed and/or under reported. The authors strongly believe that higher awareness about this diagnostic possibility followed by utilization of novel radiographic tools would better settle this challenging matter. Case studies might also enhance the suspicion index of non-specialists about concomitant disorders with gout.

Conflict of interests

The authors had full freedom of manuscript preparation and there was no potential conflict of interests.

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