DOI: 10.5472/MMJ.2012.02189.0

A 53-year-old Man with Disabling Arthropathy and Acute Renal Failure

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A 53-year-old Brazilian man came to our hospital because of intense oliguria beginning three days before admission. He was a chronic alcohol abuser, with previous diagnosis of type 2 diabetes, dyslipidemia, arterial hypertension, and deforming arthropathy. For one week, he had taken high dosages of indomethacin for bilateral arthralgia on the ankles, wrists, and metacarpophalangeal, metatarsophalangeal and interphalangeal joints. On admission, his BMI was 22.8 Kg/m^2, and he presented with signs of bilateral acute arthritis in association with numerous asymmetric hard nodules on the periarticular soft tissues. Except for scars on the elbows and right knee, no change was found by examination of cutaneous or mucous surfaces, and nails. There were conspicuous deforming osteoarticular and subcutaneous changes (Figure). Laboratory data: urea 97.6 mg/dL, creatinine 2.4 mg/dL, uric acid 12.2 mg/dL, ionized calcium 1.06 mmol/L, sodium 135 mEq/L, potassium 3.5 mEq/L, magnesium 1.4 mg/dL, glucose 136 mg/dL, ALT 184.8 U/L, AST 291 U/L, albumin 3.66 g/dL, globulins 3.1 g/dL, total bilirubin 0.34 mg/dL, prothrombin activity 89%, INR 1.07; red cells 3.17x10^{12}/L, hemoglobin 8.6 g/dL, hematocrit 26.5%, MCV 84 fL, white cells 4.2x10^9/L, platelets 490 x 10^9/L. Laboratory data after hydration and nutritional support: urea 42.5 mg/dL, creatinine 0.9 mg/dL, uric acid 8.1 mg/dL, ALT 85.2 U/L, AST 40.1 U/L; red cells 3.37x10^{12}/L, hemoglobin 9.3 g/dl, hematocrit 29%, MCV 86 fL, white cells 5.8x10^3/L, platelets 717 x10^9/L; cholesterol 159 mg/dL, LDL 93 mg/dL, HDL 18 mg/dL. The images of the echographic study of the kidneys were unremarkable. With clinical improvement, the patient was referred to the Rheumatology outpatient surveillance.

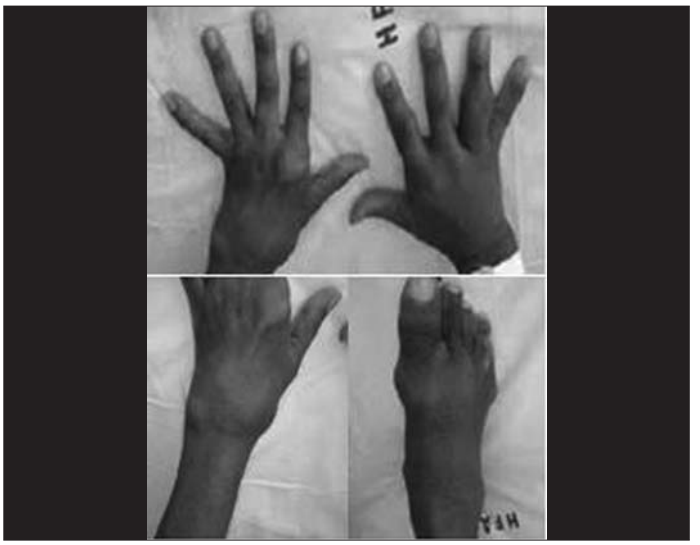

Figure 1

What is your diagnosis?

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Submitted/Başvuru Tarihi: 03.06.2012 **Accepted/Kabul Tarihi:** 18.07.2012

ANSWER to PHOTO QUIZ

Chronic tophaceus gout and reversible acute renal insufficiency due to nonsteroidal antiinflammatory drugs (NSAID)

Discussion

The patient was an alcohol abuser with metabolic syndrome diagnosed with hyperuricemia since 1984. He had suffered several episodes of gouty arthritis[1]. Laboratory panels for collagen diseases were unremarkable, and radiography features showed typical findings of tophaceous gout. He did not adhere to the medical guidelines for gout, and developed disabling bilateral deforming arthropathy on all the extremities. In 2009, gouty tophi were excised from his elbows, and there was a septic arthritis at the right knee. Indomethacin and colchicine were often used as self-medication for episodes of acute arthritis, and the actual occurrence of acute renal insufficiency followed the high dosages of NSAID. The alternative hypothesis of acute worsening of a chronic renal insufficiency was ruled out, based on the urea and creatinine levels before hospitalization (28.2 and 1.0 mg/dL, respectively). Moreover, the high levels found at admission showed a rapid improvement following hydration and nutritional management (42.5 and 0.9 mg/dL, respectively). The risk of renal failure in patients with gout include: dehydration, hypoalbuminemia, infection, obstructive uropathy, interstitial nephritis by urate crystals, hypercalcemia of immobilization, and drug adverse effects[1-3]. Renal collateral effects of NSAIDs mainly occur after the age of 50 years[4], and transitory or permanently can affect the renal function[1,3]. Furthermore, oliguria and arterial hypertension can also occur abruptly[1]. Worth of note is the already confirmed association between hyperuricemia and metabolic syndrome, in addition to chronic alcoholism[1]. Hyperuricemia may inhibit the nitric oxide system in the kidneys and enhance the concentration of endothelin-1 as well; these phenomena originate in renal vasoconstriction with reduction of the arterial flux to the medullar region, propitiating the development of an acute renal insufficiency[5]. The changes observed in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels can be related to the alcohol abuse, which plays additional role in renal dysfunction associated with low serum albumin and use of NSAID[3]. Disabling chronic tophaceous gout developed because the non-adherent patient neglected the ominous consequences of untreated hyperuricemia for a long period[6]. Therefore, deposits of urate crystals occurred on cartilages, joints, tendons and soft tissues, causing dysfunctions and irreversible deformities[6]. Hyperuricemia is an independent factor associated with enhanced risk of acute renal injury, and high mortality among inpatients submitted to coronary intervention[5]. High serum levels of acid uric constitute a main risk in gout. However, it is amenable to prevention and control. If changes in lifestyle (e.g. adequate diet, control of hypertension, and abstention of alcohol) can be adopted as early as possible, the best outcomes are achieved. In addition to preventive measures, routine treatment of gout often includes allopurinol, NSAIDs, colchicine, corticosteroids, benzbromarone and probenecid. New alternative drugs are available or under investigation (e.g. febuxostat, pegloticase, rasburicase, interleukin-1 (IL-2) antagonists, and urate transporter-1 (URAT-1) inhibitors)[1,4]. At first, psoriatic arthropathy and rheumatoid arthritis could be considered as alternative diagnoses. This patient with severe polyarticular involvement had low serum levels of total bilirubin and cutaneous scars on the extensor surfaces of both elbows and on the right knee. Skin sequels of psoriasis often occur bilaterally at elbows and knees[7], but the scars seen in our patient were due to surgery (removal of tophi and drainage of septic arthritis). Moreover, nail changes (e.g. trasversal striae, pitting, and subungueal hyperkeratosis)[7] very frequently associated with psoriatic arthopathy were not found in the patient here reported. Worth of note, low levels of bilirrubin were related to the severity of rheumatoid arthritis[8]. This inverse relation is based on the anti-oxidant and anti-inflammatory properties of bilirubin (binding to serum albumin, clearing peroxyl radicals, decreasing oxidative stresses), and reduction of the levels of IL-2 and promotion of cytoprotection by biliverdin[8]. Hypothetically, similar phenomena might have a role in this severe tophaceous osteoarthopathy.

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