Acute Uric Acid Nephropathy following Angiography and Coronary Artery Bypass Surgery

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Fig. 1. Progression of some biochemical values before, during hospitalization and after discharge. Cath = Coronary angiography; Adm = admission; HD = hemodialysis; D/C = discontinued; # = creatinine; 0 = uric acid; ▲ =BUN. The shaded area depicts the span of normal range of values.

Dear Sir,

Acute uric acid nephropathy (AUAN) is not uncommon in patients with lymphomas and hematologic disorders, particularly after chemotherapy or radiation therapy [1]. Hyperuricemia and acute renal failure (ARF) can also occur in states of severe muscle damage from exertion, seizures, or heat-related disorders. Isolated cases of AUAN have been reported in association with the use of non-steroidal anti-inflammatory drugs [2], phencyclidine [3] and thalassemia [4]. We describe an unusual case of AUAN in a patient following angiography and coronary artery bypass surgery (CABG).

A 77-year old man presented with a history of angina. An exercise stress test was positive, and coronary angiography was performed 2 days prior to his admission. CABG was done 1 day after his admission. There was a past history of prostate cancer 14 years earlier, but there was no prior history of gout, hyperuricemia, nephrolithiasis, diuretic or large-dose aspirin usage, renal insufficiency or abnormal urinalysis.

Physical examination revealed an irregular pulse of 120/min and blood pressure of 110/70 mm Hg. The neck veins were flat, lungs clear and heart sounds normal. The rest of the physical exam was unremarkable. Hemodynamic monitoring revealed a central venous pressure of 4-5 mm Hg and capillary wedge pressure of 4-8 mm Hg. All other indices were normal.

Laboratory data showed a normal admission urinalysis. A repeat urinalysis 2 days after admission showed hyaline casts, blood, trace protein and many uric acid and amorphous urate crystals. Pertinent laboratory values on admission included blood urea nitrogen of 9.3 mmol/l (26 mg/dl), serum creatinine 114.9 µmol/l (1.3 mg/dl), uric acid 357 µmol/l (6.0 mg/dl). Creatine kinase (CK) and lactate dehydrogenase peaked at 700 and 555 IU/l, respectively, other laboratory data were normal. A spot urine uric acid to urine creatinine ratio (UUA:UCr) was 1.5 and no eosinophiluria was detected. Urinary excretion of uric acid was 58,310 µmol/l (980 mg/24 h;
normal (less than 47,600 µmol/l; 800 mg/24 h). Renal sonography was normal. Aggressive hydration with isotonic fluids was attempted but his renal function progressively deteriorated. On the 6th postoperative day, he developed mild congestive heart failure, and hemodialysis was initiated. He was dialyzed daily for 3 consecutive days and his renal function dramatically improved (fig. 1). Computed tomography of his abdomen showed bilateral nephrolithiasis with 1-mm radiolucent stones in both renal pelves.

The most common cause of hyperuricemia is impaired urate excretion. Conditions associated with both overproduction and hyperexcretion of uric acid include hereditary disorders of purine metabolism, the tumor lysis syndrome and gout in which 20% of patients are hyperexcretors of uric acid. These conditions may cause a UUA:UCr > 1.0 which has been reported to be diagnostic of AUAN [5]. However, similar ratios have been reported following the administration of radiocontrast agents [6] and in hypercatabolic ARF with jaundice [7]. Several radiocontrast agents have been reported to cause uricosuria which may persist for up to 5-6 days. This effect is secondary to enhanced uric acid secretion by the proximal tubules [8]. Pathologic changes consistent with AUAN on kidney biopsy have been reported in 1 patient with hyperuricemia and renal insufficiency who developed ARF following intravenous urography [9].

Hemodynamically induced renal ischemia is the most common cause of ARF following cardiac surgery, but sepsis, embolic phenomenon, nephrotoxic drugs and fluorinated anesthetic agents may also be causative. Some of the biochemical findings in our patient may be seen with rhabdomyolysis. Postoperative rhabdomyolysis has recently been reported in 3 patients following head and neck surgery [10], however, those patients had clinical evidence of muscle damage at anatomic sites remote from the operative site and had high CK levels. Our patient had no evidence of muscle damage, pigmented casts or myoglobinuria.

Hyperuricemia disproportionate to the degree of renal failure, hyperuricosuria, a UUA:UCr of 1.5 and the ease of reversibility of the ARF are supportive of the diagnosis of AUAN. A kidney biopsy would have been confirmatory, but was deemed unnecessary. It is conceivable that he was primarily an overexcretor of uric acid judging from the degree of hyperuricosuria at the time when he had ARF. We postulate that in asymptomatic overexcretors of uric acid, the combination of radiocontrast-dye-induced hyperuricosuria, the trauma of open heart surgery with evidence of mild volume depletion and hypercatabolism can result in AUAN as manifested by our patient.

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References


124

Obialo/Hagerty

AUAN after Angiography and CABG