Immobilization Hypercalcemia and Acute Kidney Injury in Chronic Tophaceous Gout

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The commonly encountered causes of hypercalcemia are malignancy (70%), primary hyperparathyroidism (20%), excess intake of vitamin D and calcium supplements, and chronic granulomatous disorders. Immobilization is still under appreciated due to vague clinical features and a rare cause of acute kidney injury. In most previous reports, immobilization hypercalcemia occurred with normal renal functions. There is a paucity of literature describing immobilization hypercalcemia with acute kidney injury, in chronic tophaceous gout. A 66-year-old male who has been suffering from chronic tophaceous gout of multiple, large joints for ten years, presented to our emergency room with complaints of general weakness, polyarthralgia, generalized myalgia, and generalized edema. The patient was diagnosed acute kidney injury and immobilization hypercalcemia, and successfully treated with fluid and diuretics, rehabilitative exercises, and bisphosphonate. Herein, we report that a patient with chronic tophaceous gout was developed hypercalcemia and acute kidney injury following longstanding immobilization due to pain and limitation of motion on multiple large joints with tophi.

Hepatic portal venous gas due to nonocclusive mesenteric ischemia in a hemodialysis patient

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A 70-year-old man with abdominal distension for 3 day had been taking hemodialysis (HD) since 2 years ago due to end stage renal disease with membranous glomerulonephritis. He experienced frequent hypotensive episodes during dialysis session. His abdomen was tense and rigid with generalized tenderness. A plain abdominal radiography showed diffuse gaseous distension of the small bowel, and hepatic portal venous gas. A contrast-enhanced CT confirmed multiple dilated loop of bowel with pneumatisis intestinalis of small bowel and colon. Extensive hepatic portal venous gas (HPVG) (Fig. 1) and advanced atherosclerosis along the abdominal aorta was also noted (Fig. 2). Free peritoneal air was also present. On the emergency exploratory laparotomy, there were total necrosis of small bowel, but no definite thrombosis of mesenteric artery. Bowel ischemia and/or infarction is the primary etiology of HPVG, result in damage to the mucosal barrier which, in association with over-distension of the bowel and gas-forming bacterial proliferation, leads to gas moving from the intestinal lumen to the mesenteric veins and flowing through it to the portal system. Nonocclusive mesenteric ischemia (NOMI) appears more commonly among HD patients. The predisposing causes for the development of NOMI in HD patients are hypotension, hypovolemia, mesenteric vasoconstriction, and atherosclerosis. He had severe atherosclerosis, suffered from intradialytic hypotension frequently. Massive NOMI should be considered in HD patients presenting abdominal symptoms with HPVG.