Milk-Alkali Syndrome in an Aged Patient with Osteoporosis and Fractures

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Dear Sir,

Milk-alkali syndrome is caused by the excessive intake of calcium and absorbable alkali and is characterized by the triad of metabolic alkalosis, hypercalcemia and renal failure. With the development of better β-blockers and antacids, the number of cases of milk-alkali syndrome has markedly decreased in the past two decades. Recently reported cases have been limited to new and variable etiologies of this hypercalcemic disorder [1,2]. We present a case of milk-alkali syndrome resulting from the use of calcium carbonate and calcitriol for the treatment of osteoporosis and bony fractures.

A 70-year-old Asian female with no pertinent medical history sustained accidental fractures of her pelvis and right humerus 1 month prior to admission. At that time, closed reduction of the pelvis and right humerus was performed. X-rays revealed severe osteoporosis, and the patient was started on calcium carbonate, 1,250 mg three times daily and Rocaltrol, Roche [1,25-dihydroxycholecalciferol, calcitriol, 1,25(OH)2D3] 0.25 µg twice daily to enhance bone formation. On admission, the patient presented with anorexia, nausea, lethargy and an altered level of consciousness. On physical examination, her supine blood pressure was 114/70 mm Hg and her heart rate was 100/min. Skin turgor was reduced, oral mucosa was dry, and the jugular veins were not distended. Slow and shallow respiration was observed. Cardiac, pulmonary and abdominal examinations were unremarkable. Neurological examination revealed decreased alertness, slow recall, and bilateral hyporeflexia.

Laboratory data showed normal peripheral blood pictures. Urinalysis showed pH 8.0, granular cast and trace proteinuria. Biochemistry indicated sodium 155 mEq/l, potassium 3.1 mEq/l, chloride 114 mEq/l, total calcium was 15.9 mg/dl, free calcium 7.7 mg/dl, inorganic phosphate 1.9 mg/dl, glucose 88 mg/dl, albumin 3.3 g/dl, uric acid 14.8 mg/dl, blood urea nitrogen 77 mg/dl, creatinine 3.7 mg/dl. Arterial blood gas showed metabolic alkalosis with pH 7.51, PCO2 52.0 mm Hg, PO2 66.4 mm Hg, and HCO3 40.6 mEq/l. Chest roentgenography and KUB were normal without soft tissue calcification. Abdominal sonography showed normal kidney size without nephro-calcinosis. At this point milk-alkali syndrome was diagnosed. Hypercalcemia, metabolic alkalosis and renal failure completely resolved within 1 week after withdrawal of calcium carbonate and Rocaltrol, coupled with fluid supplementation consisting of normal saline and 5% glucose.
Milk-alkali syndrome can be classified into three clinical manifestations according to the duration and magnitude of calcium and alkali ingestion: acute (toxemia), intermediate (Cope’s syndrome), and chronic irreversible (Burnett’s syndrome) [1]. The acute form usually appears within 2-30 days of ingestion and is characterized by symptoms of hypercalcemia and metabolic alkalosis, including weakness, apathy, myalgia, irritability, headache, vertigo, nausea and vomiting. Renal dysfunction occurs early in the course of the disorder. Renal failure, hypercalcemia, and metabolic alkalosis resolve within days after withdrawal of the offending medication. Our patient presented typically with the acute form of milk-alkali syndrome, based on the acute onset of symptoms and rapid reversal of renal dysfunction, hypercalcemia and metabolic alkalosis after cessation of the offending medications coupled with vigorous hydration. In contrast to hyperphosphatemia, which is commonly found in milk-alkali syndrome, hypophosphatemia was observed in our patient. This may be attributed to decreased absorption of phosphate in the intestine due to the use of calcium carbonate, phosphate mobilization to the healing bone, possible soft tissue calcification, or poor intake. Hypophosphatemia, if present, may increase the serum calcium level by decreasing calcium uptake in the bone and increasing intestinal absorption [3].

Supplementation of calcium and/or vitamin D has been used increasingly as a means of enhancing bone formation and forestalling the development of osteoporosis [4, 5], as with the patient presented in this case. The patient subsequently developed the triad of metabolic alkalosis, hypercalcemia and renal failure and was thus diagnosed as having milk-alkali syndrome. We therefore consider calcium supplementation to be generally not required in such patients receiving calcitriol. In the event of hypercalcemia, calcitriol should be discontinued until the serum calcium levels normalize. In addition, the findings of renal failure and hypercalcemia in the presence of metabolic alkalosis should alert the clinician to this diagnosis when these medications are used.

References
Announcement
Seminar on Hereditary Kidney Diseases
Update on Phenotype-Genotype Relationships and Therapeutic Perspectives San Donata Milanese, September 28, 1996
The Associazione Amici della Nefrologia have organized a ‘Seminar on hereditary kidney diseases’. The meeting will be held at the Forte Crest Hotel, San Donato Milanese, Italy.
Deadline for receipt of abstracts is July 27, 1996. The languages of the seminar will be English and Italian. For further information and submission of abstracts, please contact:
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