Sustained Metabolic Alkalosis Associated with Development of the Milk-Alkali Syndrome

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Dear Sir,

The milk-alkali syndrome (MAS) has been described as hypercalcemia and metabolic alkalosis from the treatment for peptic ulcer with a high calcium and absorbable alkali intake in any form, usually as calcium carbonate. After the introduction of H₂ Mockers has altered the basis of treatment of peptic ulcer, MAS may still occur with the ingestion of a smaller amount of calcium and alkali [1-3]. The pathogenesis of MAS has not been fully understood. The evidence below suggests that the sustained alkalosis might be related to MAS. (a) Patients with accelerated acid excretion or suppressed alkali excretion, i.e., duodenal ulcer or renal complication, are more inclined to develop MAS and to a much greater degree [4]. (b) Increased tubular reabsorption of calcium is maintained in chronic metabolic alkalosis [5, 6]. We saw a patient with MAS suggesting that only sustained metabolic alkalosis could develop MAS.

This 74-year-old man had a history of cerebral infarction (middle cerebral artery region), and improved activity during the course of rehabilitation. The patient had been prescribed magnesium oxide (2 g/day = 35.5 mEq alkali) for chronic constipation and ingested milk (200 ml = 0.22 g calcium) and ice cream (145 g = 0.19 g calcium) every day for 4 months. He had several episodes of aspiration pneumonia and elevated body temperature and was treated with minocycline and ofloxacin. When the patient began to feel severe nausea and anorexia, hypercalcemia (serum Ca 14.3 mg/dl), hypernatremia (serum Na 161 mEq/l), metabolic alkalosis (HCO₃⁻ 37.4 mEq/l), and renal insufficiency (serum creatinine 2.34 mg/dl) were observed. A predisposition to hypercalcemia could not be shown by any known factors. On cessation of therapy and correction of dehydration, the serum Ca rapidly returned to normal within 1 week. TRP on diagnosing this disorder was 50.5%, which suggested hyperparathyroidism, and returned to a 90% normal range when serum Ca was restored to its normal range. Although the present case showed triads of MAS, hypercalcemia, metabolic alkalosis, and renal insufficiency, it could be distinguished from any other case previously reported with regard to the small amount of calcium (0.4 g/day) and alkali (35.5 mEq/day) intake and the two clinical situations outlined below. The patient did not have the complication of peptic

ulcer, so absorbable alkali was administered not for peptic ulcer but for chronic constipation. The existence of hypertonic dehydration and contraction alkalosis was suspected from the high serum Na concentration. Tubular calcium reabsorption was demonstrated to be enhanced by metabolic alkalosis independently of parathyroid hormone [5]. It has occasionally been noted that MAS is associated with the increased secretion of parathyroid hormone [1, 6, 7]. In the present case, the metabolic alkalosis induced by hypertonic dehydration and enhanced by absorbable alkali intake could cause an increase in renal tubular reabsorption of calcium and a decrease in ionized calcium which might produce increased secretion of parathyroid hormone followed by

vitamin D³ activation and increased Ca absorption from the gut.

The sustained metabolic alkalosis might be essential to the development of MAS without a high calcium and absorbable alkali intake. MAS is rare but not insignificant. Hypercalcemia should be suspected in patients with sustained metabolic alkalosis, which is usually seen in dehydration of elderly patients.

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