Hypertensive Emergency due to Hypercalcemia after Acute Renal Failure Secondary to Rhabdomyolysis

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Dear Sir,

Acute hypercalcemia may induce a sharp elevation in systolic and diastolic blood pressure (BP), and this effect seems to be particularly pronounced in the presence of renal insufficiency [1]. The mechanisms responsible for the rise in BP are not clear. In up to one third of patients suffering from hyperparathyroidism the hypertension can be attributed to renal parenchymal damage due to nephrolithiasis and nephrocalcinosis (2). However, also in the absence of parathyroid hyperactivity, hypercalcemia can induce hypertension probably by means of a direct vasoconstrictive effect. We present a case of critical hypertension in a patient with acute hypercalcemia due to remobilization of calcium deposits from the injured muscles after ischemic rhabdomyolysis.

A 67-year-old man was admitted to hospital in February 1993 for anuric acute renal failure (ARF) due to nontraumatic rhabdomyolysis after a bifurcated aortofemoral bypass graft. On admission the patient was immediately submitted to dialytic treatment (short daily dialysis with bicarbonate buffer and calcium dialysate concentration of 1.75 mmol/l) and to intravenous injections of calcium (3-4 g/day of calcium gluconate) until the normalization of calcemia. Most significant clinical parameters are reported in figure 1.

After 18 days, dialytic treatment was interrupted because of recovery of the diuresis and progressive improvement of the renal function. During this first period from the admission BP values ranged from 140/90 to 160/95 mm Hg without antihypertensive therapy. Seven days later, during the polyuric phase, the patient showed some calcific soft

* Serum creatinine

• Serum calcium

■ Serum phosphorus

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Dialysis

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2 Calcium infusions ↓

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• Systolic BP □ Diastolic BP

65 70 75

10 15 20 25 30 35 Days

Fig. 1. Early and late behavior of the serum calcium, phosphorus and creatinine concentrations during both the oliguric and diuretic phase of ARF due to rhabdomyolysis. Delayed hypercalcemia (after 65 days of the disease) induced hypertensive emergency. The arrow indicates the time in which soft tissue calcium deposits became evident.

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tissue deposits. Serum calcium and phosphorus concentrations became elevated with a calcium-phosphorus product of 90; the serum parathyroid hormone level was normal. His BP was slightly increased (170/100 mm Hg). Intravenous calcitonin infusions and oral administration of aluminium hydroxide favored the normalization of calcemia and phosphatemia. Ten days later the patient was discharged in good clinical condition even though a mild renal insufficiency (serum creatinine 2.5 mg/dl) and some soft tissue deposits of calcium persisted. A month later (April 1993) the patient was again admitted due to a hypertensive emergency (240/130 mm Hg) with a state of moderate mental confusion. Blood chemistry studies disclosed severe hypercalcemia (15.4 mg/dl) and a slight reduction of renal function (serum creatinine 2.2 mg/dl), while serum concentrations of phosphorus, parathyroid hormone and 1,25-dihydroxyvitamin D₃ were normal. After recovery from the sharp BP elevation by means of calcium antagonists (sublingual administration of nifedipine) intravenous infusions of calcitonin and disodium clodronate for some days permitted the quick correction of hypercalcemia. Only after the disappearance of the hypercalcemia did BP return to normal (150/90 mm Hg). In the following weeks the patient had two further transient episodes of mild hypercalcemia without significant rises of his BP, probably because of preventive medication by calcium antagonists. The hypercalcemia of the diuretic phase of ARF seems to arise quite exclusively in patients with rhabdomyolysis: this suggests the existence of a cause-and-effect relationship [3]. The most probable etiopathogenetic mechanism is represented by the reabsorption of calcium soft tissue deposits [4, 5].

On the basis of our observation the increase in calcemia could be very important in these conditions, mainly during the period of recovery from ARF, at least in the presence of a persistent mild renal insufficiency and so of a reduced excretion of calcium.

In the absence of demonstrable favorable factors (i.e. hyperparathyroidism or increased production of calcitriol), even if we

cannot exclude the interference of other pressor substances, i.e. renin and catechol-amine, whose release is calcium dependent [6-8], it is very probable that hypercalcemia 'itself was the cause of the critical BP rise. In fact it is known that acute hypercalcemia can induce a vasoconstrictive effect with a subsequent increase in peripheral vascular resistance [2, 9, 10]. Moreover, studies in rats [10] and humans [8] demonstrated that the pressor response to the acute hypercalcemia was mediated by a direct action of calcium ion on smooth muscle. So, according to Massry et al. [9] and Berl et al. [10], we can confirm that: (1) there is a clinically significant correlation between the rise in both systolic and diastolic BP and the increment in serum calcium; (2) pre-treatment with calcium antagonists can favor the block or the reduction of the pressor hypercalcemia-related effect in patients with delayed resorption of soft tissue deposits of calcium due to rhabdomyolysis.

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