Serum Calcium and Parathyroid Hormone Derangements in Rhabdomyolysis

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Dear Sir,

Abnormalities in calcium metabolism are frequent in rhabdomyolysis-induced acute renal failure. Marked hypocalcemia, which occurred during the oliguric phase, is a well-recognized biochemical feature of this disorder, and those patients who are initially hypocalcemic, may become hypercalcemic during the diuretic phase of acute renal failure [1–3]. Early studies suggest that the severity of hypocalcemia varies directly with hyperphosphatemia, is related with the intensity of muscle injury and may be due to the rapid deposition of calcium salts in traumatized skeletal muscle and cytoplasmic sequestration [1, 2]. Possible explanations for hypercalcemia include mobilizations of calcium already sequestered in muscle [4, 5], volume depletion and autonomous secretion of parathyroid hormone (PTH) [6, 7]. Llach et al. [7] consider that the hypocalcemia of the oliguric phase may be secondary to decreased synthesis of 1,25(OH)₂-D associated to hyperphosphatemia and skeletal resistance to the calcemic action of PTH and describe high levels of 1,25(OH)₂-D during the recovery phase in addition to elevated PTH, suggesting that repair of the vitamin D abnormality restores bone responsiveness to PTH: For these authors, the most likely cause of the hypercalcemia of the diuretic phase is the increased production of 1,25(OH)₂-D associated to persistent secretion of PTH.

We had the opportunity to study one alcoholic patient with nontraumatic rhabdomyolysis (CPK = 190,000 UI/l) who went into acute renal failure. During the oliguric phase (diuresis < 300 ml/day), which persisted for more than 30 days, he developed hypercalcemia. Peritoneal dialysis was started (calcium concentration of the peritoneal fluid: 3.5 mEq/l).

As shown in figure 1, the lowest level of serum calcium appeared on the 5th day: total calcium 4.4 mg/dl; ionized calcium 1.68 mg/dl; phosphate 14 mg/dl and PTH (1,84-

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Fig. 1. Levels of serum PTH (1,84-COOH), Calcium, phosphorus, creatinine and daily urine volumes, during the course of acute renal failure after rhabdomyolysis. This patient developed hypercalcemia during the oliguric phase of the disease. PD= Peritoneal Dialysis.

COOH) 709 pg/ml (normal: 35 ± 25 pg/ml). From this day, the serum calcium increased reaching the highest level on the 18th day: total calcium 14.2 mg/dl; ionized calcium 7.12 mg/dl; phosphate 7.7 mg/dl. However, at that time the serum level of total PTH was normal: 27.2 pg/ml in spite of renal failure. The patient remained oliguric and peritoneal dialysis was maintained until the 30th day, at which time active diuresis started. Total calcium was then 10 mg/dl and PTH 17 pg/ml.

108

Garcia de Vinuesa/Ahijado/Lur

These data suggest that in rhabdomyolysis-induced acute renal failure the high PTH levels are mainly due to marked hypocalcemia, probably secondary to the rapid deposition of calcium in traumatized skeletal muscle. Severe hyperphosphatemia and renal failure per se may also play a role, but the rapid decrease of PTH values to normal levels in parallel with the increase of serum calcium, even in the oliguric phase, suggests a normal parathyroid function in this patient and excludes the role of the autonomous secretion of PTH as a possible mechanism responsible for the late hypercalcemia of rhabdomyolysis-induced acute failure.

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