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 hyperphosphataemia, 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 hyperphosphataemia 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/1) who went into acute renal failure. During the oliguric phase (diuresis < 300 cmVday), 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-
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.


References