

Hyperphosphatemia and Hypocalcemia in Myeloproliferative Disorder

TO THE EDITOR: Recent reports of hyperphosphatemia and hypocalcemia as a complication of antileukemic therapy in children (1-3) have prompted us to report this occurrence in a 33-year-old man with an acute myeloproliferative disorder.

He presented initially with severe hypercalcemia, which required repeated courses of mithramycin therapy until the hematological disorder was diagnosed. Prednisone therapy was then started in a dose of 120 mg per day, which had no effect on the serum calcium or phosphorus concentrations. After 3 days of prednisone therapy, thioguanine (25 mg/kg body weight) and cytosine arabinoside (3.0 mg/kg body weight) were added. Sixteen hours later the phosphorus level had risen from 3.5 mg/100 ml to 16mg/100 ml and the calcium level had decreased from 12.4 to 6.2 mg/100 ml. The patient died 10 hours later of acute pulmonary edema and respiratory failure.

The hyperphosphatemia could not be explained by his mild renal disease because the serum creatinine concentration was 2.5 mg/100 ml. Although the posttherapy course was too short to allow further investigation, the similarity of our case to those reported by Zusman, Brown, and Nesbit (1) suggests that the severe hyperphosphatemia is probably caused by the acute release of phosphorus from the leukemic cells, and this causes the secondary hypocalcemia. Besides the potential hazard of hyperuricemia, we should be aware of the possibility of severe hyperphosphatemia and hypocalcemia when treating any patient with a myeloproliferative disorder.

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