Sarcoidosis with Extreme Hypercalcemia

To the Editor:

A 29-year-old white man came to the University of California Davis-Sacramento Medical Center with anorexia, nausea, lethargy, epigastric distress, and polyuria of several months' duration. He had been consuming one to two quarts of milk daily but had taken no vitamins or antacids. Evaluation revealed the following levels: calcium, 17.4 mg/100 ml; phosphorus, 3.3 mg/100 ml; alkaline phosphatase, 85 International units (IU) (normal, 30 to 85 IU); and creatinine, 2.0 mg/100 ml. Saline diuresis and therapy with corticosteroids caused a prompt return of the serum level of calcium to normal and reversal of all symptoms. Levels of parathyroid hormone of 324 pg/ml and 359 pg/ml (normal, 200 to 600 pg/ml) were obtained while the patient had mild hypercalcemia. Bilateral hilar adenopathy was present on the chest x-ray film, and a biopsy of a cervical node showed noncaseating granulomas (Fig 1). The findings from a skeletal survey were normal. Therapy with corticosteroids was gradually tapered over a six-month period, during which time a minimal regression of the hilar adenopathy was noted and the serum levels of calcium remained normal.

A review of the literature indicates that hypercalcemia accompanies sarcoidosis in from less than 2 percent1-4 to over 60 percent5 of the cases, with the frequency dropping over the past few decades, corresponding to the general availability of corticosteroids. Most reports describe a mild hypercalcemia, although two cases feature levels of calcium of 19 mg/100 ml6 and 20 mg/100 ml,6 however, both occurred in patients with severe, widespread clinically apparent disease.

Although other causes of hypercalcemia were ruled out in this patient, the substantial ingestion of milk may have been contributory, in view of the hyperresponsiveness to vitamin D, which is thought to be the cause of hypercalcemia in sarcoidosis.6 Despite the rarity of profound hypercalcemia due to sarcoidosis, it must be remembered that it does occur and even in early, otherwise asymptomatic disease.

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Ages ranged from 26 to 44 weeks. Six infants were premature, and two were postmature. Weights at birth ranged from 750 to 5,580 gm (1 lb 10 oz to 12 lb 5 oz). Seven infants died. Four of the seven died with severe respiratory distress syndrome, one had a diaphragmatic hernia, one had trisomy E, and one large (5,580-gm) postmature infant died with a massive pulmonary hemorrhage. Of the five infants who survived, two had respiratory distress syndrome, two had meconium aspiration, and one had a diaphragmatic hernia.

The patients were given tolazoline (1 to 3 mg/kg of body weight) intravenously over a vein in the scalp over five minutes, with continuous monitoring of arterial blood pressure. Three infants were also given a continuous infusion at 2 mg/kg/hr.

Eleven of our infants sustained an increase in PaO₂ of 15 mm Hg or more within 50 minutes of the infusion of tolazoline. Five (45 percent) of the 11 infants who responded survived. Their response to therapy with tolazoline was three to five times greater than that of infants who died (Fig 1). Mortality was higher in infants with the respiratory distress syndrome. A decrease in blood pressure occurred within one to five minutes of the infusion, and six infants required replacement of volume for systemic hypotension. A cutaneous flush was seen in most infants. None of our patients developed gastric bleeding or distention, thrombocytopenia, or hematuria.

We agree with recent reports3,4 that suggest that tolazoline, because of its effect on vascular smooth muscle in dilating an actively constricted pulmonary vascular bed, would seem to have a definite role in the management of infants with severe pulmonary disease and pulmonary hypoperfusion. Because of the potential effect of therapy with tolazoline on systemic blood pressure, it is essential that the systemic arterial pressure be monitored continually during administration of tolazoline and that volume expanders and epinephrine be readily available during infusion.

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