the material is either infrequent or infrequently reported within the United States. This case illustrates the serious consequences that may result, although the patient has recovered from the acute episode and has been asymptomatic for several years.

One of the most bizarre cases of talc pneumoconiosis was reported by Nam and Gracey[5] in 1972. Although death was from an unrelated disease, the patient had developed extensive talcosis as a result of liberal use of cosmetic talcum powder over a period of 20 years. The authors stated that they had been unable to find in the literature a case of pulmonary talcosis associated with the use of cosmetic dusting powder.

SUMMARY

Progressive diffuse pulmonary fibrosis as a sequel to massive aspiration of baby powder (talc) is documented. The diagnosis should be considered in "idiopathic" pulmonary fibrosis in childhood and adult life.

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Cardiac Arrhythmia and Hypopotassemia in Association With Lithium Carbonate Overdose

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FOR OVER 100 YEARS, lithium salts have been used in the treatment of various disorders including rheumatism, gout, hyperthyroidism, diabetes mellitus, and as a salt substitute in patients with congestive heart failure. These uses, however, proved unrewarding and were associated with significant toxic effects. Therapeutic efficacy of lithium salts in the treatment of manic-depressive psychosis recently has been appreciated. Lithium has a narrow therapeutic range and multiple side effects may develop when the serum concentration reaches or exceeds upper therapeutic levels (2.0 to 2.5 mEq/liter).[1] Toxic serum levels of lithium can prove fatal. Therapeutic serum lithium levels do not appear to have significant cardiotoxic effects, but at toxic levels serious cardiac disorders and arrhythmias may develop which require particular attention.

CASE REPORT

A 30-year-old man with no history of cardiovascular disease was taking 300 mg of lithium carbonate b.i.d. for the treatment of a psychiatric disorder. Four hours before admission, he took 120 tablets (36 gm) of this preparation. On arrival at the hospital he was in no acute distress. The skin and mucous membranes were well hydrated; temperature was normal; lungs were clear, and blood pressure was 125/80 mm Hg. The heart rate was 75/min with regular sinus rhythm. There were no cardiac abnormalities on palpation or auscultation. Except for lateral gaze nystagmus there were no abnormal neurologic findings. The serum BUN, hemoglobin, and hematocrit values were normal. Chest roentgenogram was negative. The serum lithium level was 3 mEq/liter one hour after admission; three hours after admission it had fallen to 2.2 mEq/liter. Other pertinent laboratory and clinical data are outlined in the Table.

Hospital Course

On admission to the hospital, gastric lavage was instituted. This was followed by oral administration of 60 ml of Fleet Phospho-Soda (sodium biphosphate and sodium phosphate) and activated charcoal. Intravenous infusion of 125 mg of aminophylline in 500 ml of normal saline at a rate of 100 ml/hour was begun and continued for 38 hours. Twelve hours after admission, the serum lithium concentration dropped to 0.66 mEq/liter and the potassium to 2.4 mEq/liter. This was associated with sinus bradycardia at a rate of 44 to 56/min and periods of ventricular escape (idioventricular rhythm) at a rate of 52/min without significant clinical symptoms (Fig 1, A). The idioventricular rhythm had a left bundle branch block pattern. This pattern lasted a few hours and terminated following intravenous infusion of potassium chloride solution. The sinus bradycardia persisted for over 38 hours in spite of a return of the serum potassium to 4.7 mEq/liter and a lithium level of 0.8 mEq/liter (Fig 1, B). On the fourth day, the patient's heart rate was 80/minute.

A month later, while the patient was taking 150 mg of imipramine hydrochloride (Tofranil) per day, noninvasive cardiac evaluation was performed. The systolic time intervals revealed a prolonged pre-ejection phase/left ventricular ejection time (PEP/LVET = 105/257 = 0.401, as compared with a predicted value of 97/269 = 0.361). There were no abnormalities on his vectro-, apex-, phono-, and echocardiograms.

DISCUSSION

The development of sinus bradycardia and idioventricular rhythm in the presence of marked hypokalemia in this case was rather unusual. This occurred when the lithium level was at a low therapeutic range of 0.66 mEq/liter. The patient did not receive diuretics during the course of treatment and the drop in the serum potassium from 4.3 mEq/liter to 2.4 mEq/liter within 12 hours could not be explained by gastric lavage and intravenous fluid infusion alone. Hypokalemia due to gastric lavage and loss of hydrochloric acid is associated with hypochloremic alkalosis.[2] This patient had a serum pH of 7.46 and the serum chloride levels were normal (Table). The development of hypokalemia may have been in part due to the toxic effects of lithium on the kidney and excessive potassium excretion.[3] Low serum potassium causes increased automaticity, especially of the latent pacemaker fibers. This is probably due to decreased potassium conductance[4] with consequent decreased efflux of potassium in the face of sodium influx. Low serum potassium, by causing increased excitability, tends to lead to

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