

and impaired CSF resorptive capacity, corticosteroid therapy may be effective, but it was not in this case. Because of the persistence of elevated CSF pressure, a lumbarperitoneal diversionary shunt procedure was successful; removal of the large vascular malformation was not attempted.

SUMMARY

A vascular malformation caused prominent intracranial hypertension by impairing CSF resorptive capacity rather than by its mass effect or ventricular obstruction. It was successfully treated by diversionary

shunt.

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Pulmonary Talcosis as a Result of Massive Aspiration of Baby Powder*

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In 1954 Cless and Anger¹ reported a fatal case of massive aspiration of baby powder. In 1962 Molnar and associates² added to the literature another instance, and in 1963 Jenkins³ reported a third case in which death resulted shortly after aspiration. Hughes and Kalmer,⁴ in 1966, recorded survival of an infant in whom recovery was associated with administration of dexamethasone; this patient was asymptomatic, with a normal chest film, two weeks after the acute episode. The purpose of this paper is to record progressive inflammatory disease of the lung as a sequel to massive aspiration of baby powder.

CASE REPORT

A 10-year-old white girl was admitted to LeBonheur Children's Hospital in November 1966 for evaluation of lung disease noted by an orthopedic surgeon who had discovered the changes of hypertrophic osteoarthropathy while treating her for a bruise of the left foot.

On admission to the hospital the patient appeared chronically ill, but in no distress. There was no cyanosis, but clubbing of the fingers and toes was noticed. The only other significant physical finding was a loud pulmonary second sound.

The child had apparently been in good health until about three years before admission, when she began having frequent sore throats and "colds." Approximately 2½ years before admission she was hospitalized for pneumonia. Her parents were later told that the infection was cured, but that her lungs were "scarred"; they were told that this was of no consequence. The child continued to have frequent respiratory infections, accompanied by low-grade fever, cough, and moderate dyspnea until January, 1966; since that time her condition appeared to improve. It had been observed that the girl tired more easily than her playmates. Neither cyanosis nor edema was ever noted. The child had a poor appetite, with frequent early morning vomiting. Chest discomfort was not present, and a morning cough was not productive. Several years before the apparent onset of the present illness there were possible episodes of measles, pneumonia, and acute rheumatic fever.

Admission laboratory values were within normal limits. PPD skin test was negative, as were complement fixation tests for histoplasmosis, blastomycosis, and coccidioidomycosis.

The roentgenogram of the chest on admission (Fig 1) showed

widespread chronic inflammatory disease of the lungs. Slow progression of the process was discerned by comparison with previous films.

Results of bronchoscopic examination, bronchograms, and electrocardiogram were essentially within normal limits.

An open biopsy of the lingula of the left lung revealed multiple nonconfluent nodules throughout the lung. The impression at the time of the thoracotomy was that these nodules were "cystic." The cultures from the lung tissue for acid-fast bacilli, fungi, and pyogens were negative. The postoperative course was relatively uneventful, and the patient was discharged nine days after operation.

The surgical specimen was a 1.5 × 1.2 × 1.0 cm irregular mass of firm gray tissue with small areas of aerated parenchyma. An impression of ill-defined nodularity was given.

Microscopic examination revealed extensive confluent fibrosis and hyalinization, with large areas in which pulmonary parenchyma was obliterated (Fig 2). Recognizable small arteries and



FIGURE 1. Admission chest film demonstrating diffuse bilateral chronic inflammatory infiltrate.

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FIGURE 2. Photomicrograph showing severe peribronchiolar fibrosis. (H & E, original magnification $\times 40$)

arterioles had thickened walls and very small lumina. There was a tendency for peribronchiolar distribution of changes, and at the periphery of the nodules were occasional aggregates of foreign-body-type multinucleated giant cells. There was prominent inflammatory infiltrate, mostly mononuclear. Eosinophils were relatively conspicuous. Within many of the giant cells were clefts filled with pale-staining structures which appeared to be crystalline (Fig 3). On polarization these foci were easily identified as doubly refractile bodies (Fig 4). The size varied, with a length of up to approximately 100μ and a width of up to approximately 20μ .

After intensive questioning concerning the possibility of aspiration of foreign material, the mother remembered an episode when the patient, then approximately 2 years of age, "got into a can of baby powder and aspirated quite a bit." She was seen by a physician, at which time hospitalization was not thought indicated. There were no apparent sequelae.

This young woman is now a 19-year-old college student, reportedly carrying on usual activities without difficulty nine years after thoracotomy. Chest x-ray film taken last year (Fig 5), 16 years after the episode of aspiration, resembles that taken eight years previously. There is prominence in the region of the pulmonary conus, suggestive of pulmonary hypertension.

DISCUSSION

The major ingredient in most brands of baby powder is talc, and we believe our patient had talc pneumoconiosis. This disease is encountered as an occupational hazard in the mining and processing of talc, as well as

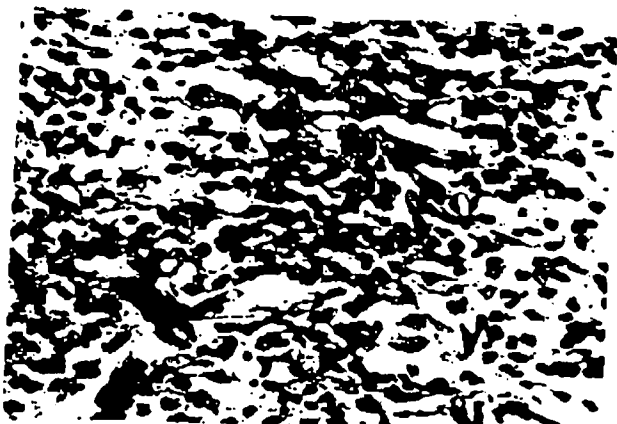


FIGURE 3. Photomicrograph showing chronic granulomatous reaction associated with the presence of several cleft-like spaces containing poorly staining amorphous material. (H & E, original magnification $\times 400$) Compare with Figure 4.



FIGURE 4. Photomicrograph of same field as Figure 3. Polarization dramatically emphasizes the birefractile fibers present. (H & E, original magnification $\times 400$, partially polarized)

in numerous industries in which talc is used. The term "talc pneumoconiosis" may be a misnomer, as it is likely that impurities such as tremolite or silica are the fibrogenic agents responsible.

Talcosis is generally characterized by gray nodules concentrated within the lower lobes of the lungs. Microscopically the foreign material is incorporated in the more fixed parts of the alveolar walls, especially around terminal air passages, smaller blood vessels, septa, and beneath the pleura. Fibers resembling asbestos bodies are often found.

In the case reported by Molnar et al,² autopsy disclosed an acute bronchitis and bronchiolitis, with bronchial lumina filled with granular material containing talc. The changes were interpreted as those of early bronchiolitis obliterans.

Despite the wide use of baby powder, aspiration of

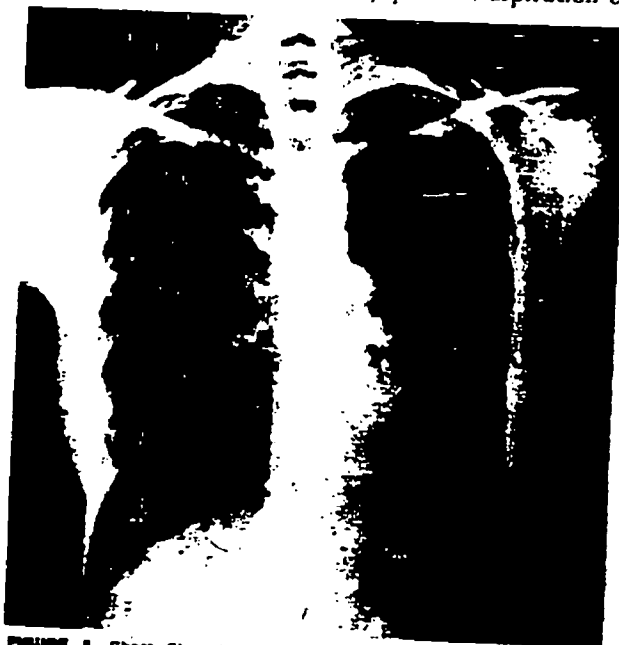


FIGURE 5. Chest film nine years after admission. Extensive fibrosis, predominantly within the lower lung fields, is present; there is perhaps increased radiolucency of the upper lung fields. The pulmonary conus is prominent.

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 Gracey⁵ 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 50/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 0.3 mEq/liter (Fig 1, B). On the fourth day, the patient's heart rate was 80/min.

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) = $109/257 = 0.40$, as compared with a predicted value of $97/260 = 0.36$. There were no abnormalities on his vector, 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 conduc-