

Arch. Dis. Child. 52: 157-159
Feb., 1977

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Smith for performing the diagnostic radiological studies, and to Dr. Pamela Davies for her help in the preparation of this report.

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Powder aspiration in children

Report of two cases

Acute aspiration of zinc or talc powder is a very dangerous condition in childhood, and several fatalities have been reported. Recently we saw 2 cases. The first showed the classical course with delayed respiratory distress, and extreme measures were needed to save the baby. Using an aggressive therapeutic approach we were able to avoid acute sequelae in the second patient. We report our experiences and give clinical recommendations.

Case reports

Case 1. A 7½-month-old girl was admitted to hospital 6 hours after aspiration of Fissan Baby powder (containing talc, zinc oxide, and other substances).

The child was found choking, her face covered with powder. The mother cleaned the upper airways, and normal breathing returned. About 2½ hours after the accident she developed steadily increasing respiratory distress and was admitted to hospital.

Physical examination (6 hours after the accident) showed a well-nourished and well-developed baby in severe respiratory distress (pulse 164/min, respirations 64/min, temperature 38° C). The upper airways were clear. Expiration was prolonged with scarce fine rhonchi over both lungs which were hyper-resonant to percussion. The liver could be felt 2 cm below the costal margin.

Laboratory results showed a capillary P_{CO_2} of 42 mmHg and a leucocytosis of 15 400/mm³ ($15.4 \times 10^9/l$) with a strong shift to the left. The first x-ray film of the chest showed fine and diffuse alveolar infiltrates on both sides, partially confluent, a barely visible interlobar fissure on the right, and a small pleural effusion on the left (Fig. 1).

Treatment was begun with intravenous infusions, ampicillin, cloxacillin, and prednisone. During the next 3 hours respiratory distress increased, the P_{CO_2} rising to 58 mmHg requiring nasal intubation and controlled ventilation under neuromuscular blockade with pancuronium (Loosco Baby Respirator, IPNV with maximal inspiratory pressure). Repeated vigorous bronchial washing with normal saline still showed, even after many hours, viscous whitish material. Besides the antibiotics and prednisone, aminophylline, orciprenaline, and potassium iodide were administered. However, it was impossible to ventilate the child sufficiently because of



Fig. 1 Case 1. Chest x-ray 7 hours after powder aspiration. Note the alveolar infiltrates in both lungs with partial confluence, and the pleural effusion on the left.

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tremendous airway resistance (arterial blood gases 12 hours after aspiration: P_{aO_2} 63 mmHg with an F_{iO_2} of 0.8, pH 6.9, P_{aCO_2} 99 mmHg, bicarbonate 18.5 mEq/l, BE -17.6 mEq/l). At this time signs of hypercapnia were most impressive with tachycardia, hypertension with wide pulse pressure, flushed and sweating skin, and a bulging and bounding fontanelle.

About 6 hours after starting mechanical ventilation the P_{aCO_2} dropped to 60-65 mmHg and the airways obstruction improved. Despite hypoventilation the baby was never hypoxaemic because of high F_{iO_2} . The following day, signs of right heart failure developed (massive weight gain, tachycardia, hepatomegaly), and digitalis and frusemide were given. Chest x-ray showed early atelectasis of the right upper lobe and bilateral basal emphysema. Air-bronchograms were seen at different levels.

On day 6 after the accident (still with maximal respiratory pressure) a right-sided pneumothorax developed which was treated by insertion of a chest tube and suction. 2 days later the baby again had fever and a leucocytosis with a strong shift to the left; the tracheal aspirate grew *Pseudomonas aeruginosa*. The antibiotics were changed to gentamicin and Cephapirin. On day 9 we were able to change to assisted ventilation, and the following day the child was extubated. She remained another 10 days in hospital, all treatment being gradually stopped.

At examination before discharge, she was in good general health with no neurological deficit. The lungs were clear. Capillary O_2 -saturation (breathing room air and crying) was 92%, P_{CO_2} 30-35 mmHg. Chest x-ray showed some confluent perihilar densities, bronchial wall thickening and a small area of atelectasis in the posterior segment of the right upper lobe (Fig. 2).

On clinical follow-up after 3½ months we found no symptoms and signs of respiratory tract or pulmonary disease. The lungs were clear, no cardiac abnormality was detected. Capillary O_2 -saturation was 93%. The x-ray still showed some regressing right hilar densities and discrete peribronchial infiltrations. An electrocardiogram was normal.

Case 2. A 13-month-old boy was admitted to hospital half an hour after aspiration of Merfen powder (containing talc and borate of phenylmercury). No signs of respiratory distress were visible, but over the left lung some rhonchi were heard. The initial chest x-ray had the same characteristics as seen in the first patient (mainly alveolar infiltrates).

Intubation was performed under fluothane anaesthesia with intensive bronchial wash-out. Plenty of whitish material was aspirated. After-



Fig. 2 Case 1. Chest x-ray 14 days after powder aspiration. Note the confluent perihilar densities, bronchial wall thickening, and atelectasis of the posterior segment of the right upper lobe.

wards the boy received prednisone, ampicillin, humidified air, and N-acetyl cysteine by inhalation. The course was uneventful and the patient recovered completely within 4 days.

Discussion

Until now 22 cases of powder aspiration have been reported (Cless and Anger, 1954; Gould and Barnardo, 1972; Gouvea *et al.*, 1966; Heiman and Aschner, 1922; Hughes and Kalmer, 1966; Jenkins, 1963; Lund and Feldt-Rasmussen, 1969; Molnar *et al.*, 1962; Tortorolo and Romano, 1966). The patients were mainly children around 1 year of age. The mortality rate was 23%. Nearly always there is a characteristic silent period of several hours between the initial event and the beginning of severe respiratory distress. This asymptomatic period can lead to wrong medical decisions. The respiratory distress is due mainly to bronchiolar obstruction by the aspirated powder and massive bronchitis and bronchiolitis with pulmonary oedema, atelectasis, and compensatory emphysema.

Our first case presented most severe lower airway obstruction, compatible with the pathoanatomical findings described in the literature. Acute right heart failure, pneumothorax, and pseudomonas infection were secondary to the pulmonary disease or to our treatment.

The results obtained by Gouvea *et al.* (1966) and those in our Case 2 justify an aggressive therapeutic approach. We recommend therefore early bronchial wash-out or lavage in a child who has aspirated powder (especially talc or zinc), even

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if this child presents without any signs of respiratory distress. Children already in respiratory distress should have optimal medical treatment with forced intravenous fluids, corticosteroids in high doses, and bronchodilators. Depending on the clinical course and blood gas analysis, mechanical ventilation may be required. Very high airway resistance must be overcome, and respiratory adjustment should be done as in severe bronchial asthma. Hypoxaemia can be avoided by increasing F_{iO_2} . Respiratory acidosis may be corrected by infusion of THAM (not sodium bicarbonate), especially when high $Paco_2$ interferes with vital functions.

Summary

Two cases of powder aspiration are reported. A 7½-month-old girl showed a classical course with an asymptomatic period of 3–4 hours, then severe respiratory distress developed. Acute respiratory insufficiency made tracheal intubation and mechanical ventilation necessary for 10 days. Complications included insufficient alveolar ventilation, atelectasis, pneumothorax, and superinfection. But the baby recovered with some residual radiological changes in the lungs. A 13-month-old boy was treated immediately after massive powder aspiration by tracheal intubation and bronchial wash-out. The post-operative course was uneventful and no respiratory distress developed.

Powder aspiration leads to severe bronchiolar obstruction with a delay of several hours and has a high mortality rate. The best results in treatment are obtained by immediate intubation and bronchial wash-out, even in the absence of respiratory symptoms. Artificial ventilation may be necessary with the special problem of overcoming very high airway resistance. Corticosteroids and bronchodilators may be helpful.

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Pyogenic meningitis in chronic gastroenteritis and marasmus

We report a series of cases of infants presenting with a severe illness with gastroenteritic symptoms in whom the initial cerebrospinal fluid (CSF) was normal but pyogenic meningitis developed during the course of the illness. Clinical presentation was atypical and the development of meningitis was unexpected and unpredictable. This unusual combination prompted this paper.

Case reports

Case 1. A male aged 3 months was readmitted 10 days after a previous episode of chronic gastroenteritis, with a history of diarrhoea for 2 days. He was 5% dehydrated, opisthotonic, and after rehydration was below the 3rd centile for weight. Lumbar puncture yielded sterile CSF (Table). Enteropathic *E. coli* O127/B8 was cultured from the stool.

Opisthotonus persisted; 2 weeks after admission the diarrhoea was severe, he was pyrexial, a lumbar puncture again yielded sterile CSF (Table). A week later the diarrhoea was blood-stained, pyrexia and opisthotonus persisted, a third lumbar puncture yielded sterile CSF (Table). X-rays of chest and abdomen were normal. Improvement occurred until 6 weeks after admission, another pyrexial episode prompted a fourth lumbar puncture which now yielded purulent CSF (Table). After treatment for meningitis total nerve deafness was diagnosed.

Case 2. A male aged 8 months was admitted with a history of diarrhoea for the previous month. He was

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