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Destructive pulmonary disease due to mixed anaerobic infection

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Pulmonary infection is one of the commonest infections in childhood and is responsible for considerable mortality and morbidity, especially in Nigeria. Most infections are caused by *Staphylococcus pyogenes, Streptococcus pneumoniae,* and *Haemophilus influenzae,* though occasionally other organisms such as *Klebsiella pneumoniae,* coliform, and salmonella are responsible. Rarely does one encounter mixed infection with *Clostridium welchii,* coliform, and anaerobic streptococci organisms which are the causative organisms in the case reported here. The child presented with features of a bronchopneumonia and empyema, and was later shown to have destructive lung disease due to unusual organisms. This diagnosis was reached on the basis of the radiological finding and bacteriological examination of the sputum. The child was treated with appropriate antibiotics and postural drainage and, when the condition had improved, was subjected to thoracotomy and excision of the diseased lung. He made an uneventful recovery.

Pulmonary infection with anaerobic organisms is a well-recognized entity. Often these infections occur secondary to penetrating wounds of the chest. Reports of primary infections with anaerobic streptococci or *Clostridium welchii* are scanty in the literature. Welch (1941) described the first cases of pulmonary infection with *Cl. welchii.* Since then there have been two further reports of pulmonary infection with the same organism (O'Donnell, 1952; Sweeting and Rosenberg, 1959). Three cases of necrotizing pneumonitis and empyema due to microaerophilic streptococci were reported by Finegold, Smolens, Cohen, Hewitt, Miller, and Davis (1965). These reports described the condition in adults.

The purpose of the present communication is to report an unusual case of lung infection with mixed anaerobic organisms in a 2-year-old boy.

CASE REPORT

The patient (U.C.H. No. 177833), a 2-year-old boy, was referred to the Children's Emergency Room, University College Hospital, Ibadan with six weeks' history of fever, cough, loss of weight, and soreness of the mouth. Past medical and family history were non-contributory.

On examination he was a miserable looking child, weighing 18 lb. 6 oz. (8·5 kg.). The eyes were puffy and there was pedal oedema. There were sores in the mouth and angular stomatitis. The heart rate and respiratory rate were 140 and 40 per minute respectively. The trachea was shifted to the right. Chest movement was diminished on the left side and the percussion note on the same side was markedly impaired. Air entry on the left side was diminished and bronchial breathing was heard in the left upper zone. From the clinical findings it was concluded that the child had pneumonia with secondary empyema, and he was admitted.

Investigations included a chest radiograph which showed complete consolidation of the left lower lobe with collapse in the left upper lobe. There was no superior mediastinal or hilar adenopathy. A penetrating film (Fig. 1a, b) revealed some mottling due to small cavities in the left lung. This appearance was interpreted by the radiologist as indicating a highly destructive form of pneumonic process probably due to an unusual organism. Diagnostic thoracentesis yielded 10 ml. of blood-stained pus. A mixture of Gram-positive cocci and a few bacilli were seen on the Gram stain, but culture of the material was sterile. However, culture of three specimens of sputum yielded a heavy growth of *Cl. welchii,* coliforms, and anaerobic streptococci at three different times. The haematocrit (P.C.V.) was 22% and the haemoglobin genotype was AA.

MANAGEMENT The child was treated with a combination of penicillin, polymixin, and cloxacillin, to which the different organisms were sensitive. Anti-gas-gangrene serum was also administered. Postural drainage was instituted. After two weeks' treatment there was little change either in the chest findings or in the repeat chest films. There was, however, marked clinical improvement; the peripheral oedema disappeared and the child gained weight.

Since there was no significant improvement in either the chest findings or the chest radiographs, it

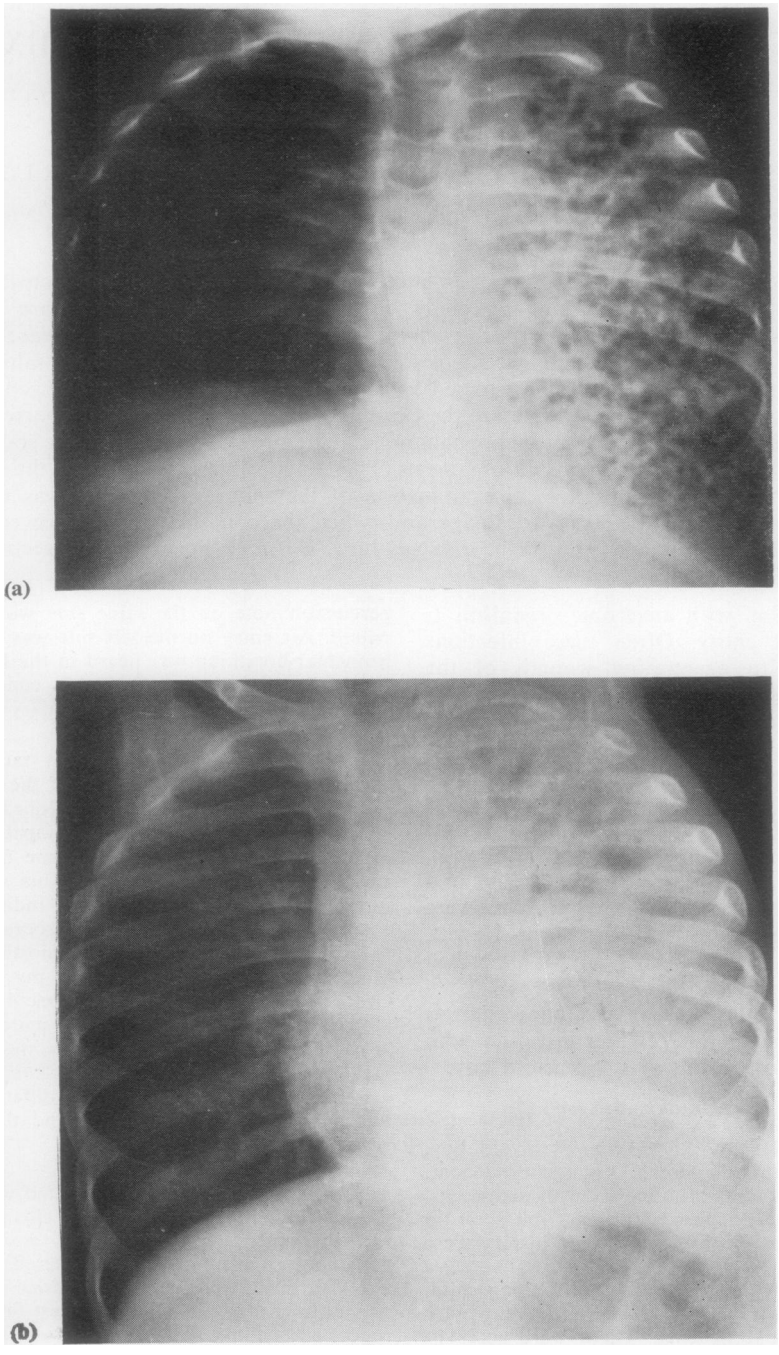

FIG. 1. *(a) Penetrating film of chest showing multiple air cysts in the left lung field. (b) Chest film taken immediately after thoracotomy and resection of the left lung.*

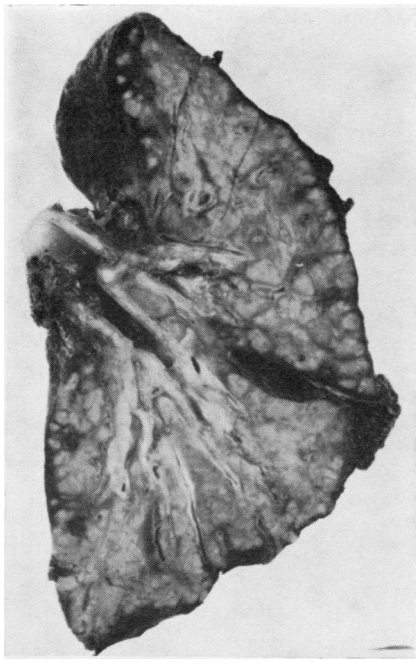

FIG. 2. *Cut surface of resected lung.*

was considered that the lung was destroyed and that the child would benefit from a pneumonectomy. Accordingly, a thoracotomy was carried out. The entire left lung was found to be destroyed and there were hilar adenopathy and adhesions. Pneumonectomy and lymph-gland biopsy were performed. The post-operative course was unremarkable. He gained 7 lb. (3·18 kg.) in weight before discharge. He has remained well and continues to gain weight satisfactorily six months after discharge.

PATHOLOGY The resected lung weighed 96 g. A few hilar nodes were slightly enlarged. The pleura was slightly thickened and there were fibrous tags indicating adhesions. The cut surface of the lung showed a rather striking appearance. Almost the whole parenchyma was consolidated, being replaced by multiple yellowish nodules measuring 0·3–1·5 cm. in diameter (Fig. 2). The nodules were surrounded by whitish fibrous bands which subdivided a nodule further into micronodules. The main bronchus was moderately dilated and its major branches were also slightly dilated and contained mucoid material. The gross appearance was suggestive of confluent tuberculous bronchopneumonia or an unresolved bacterial pneumonia.

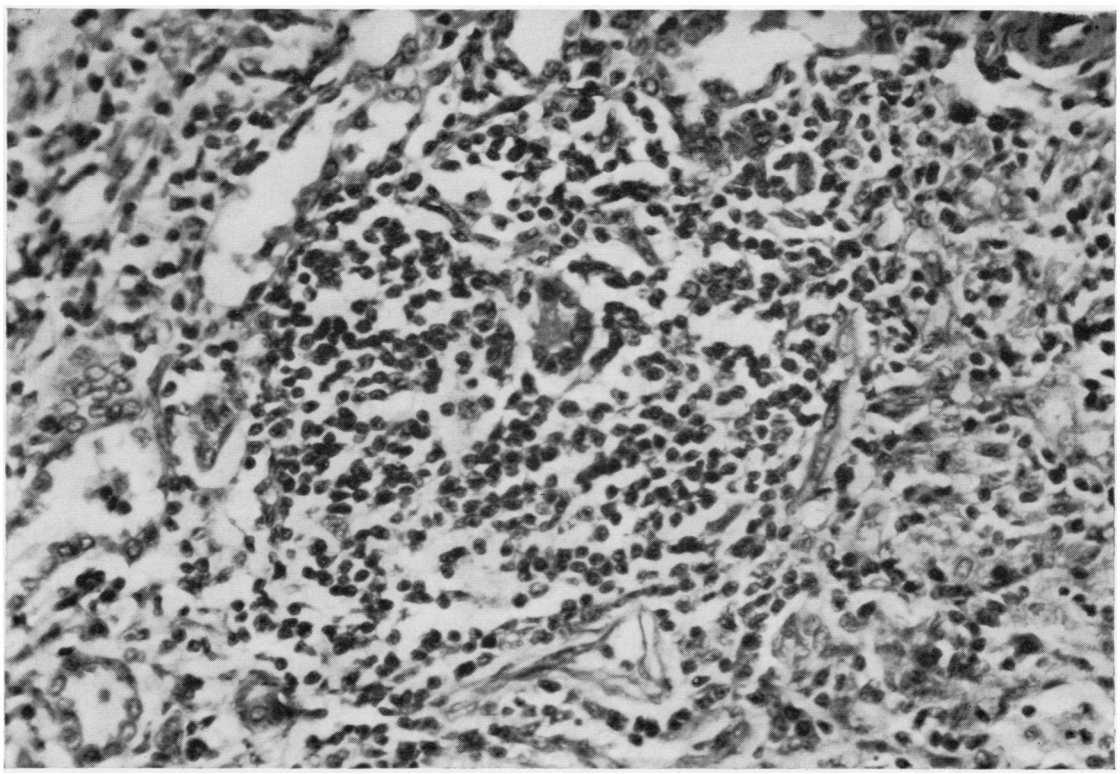

FIG. 3. *Granulation tissue showing mononuclear cells and giant cells* (× 345, *H. & E.*).

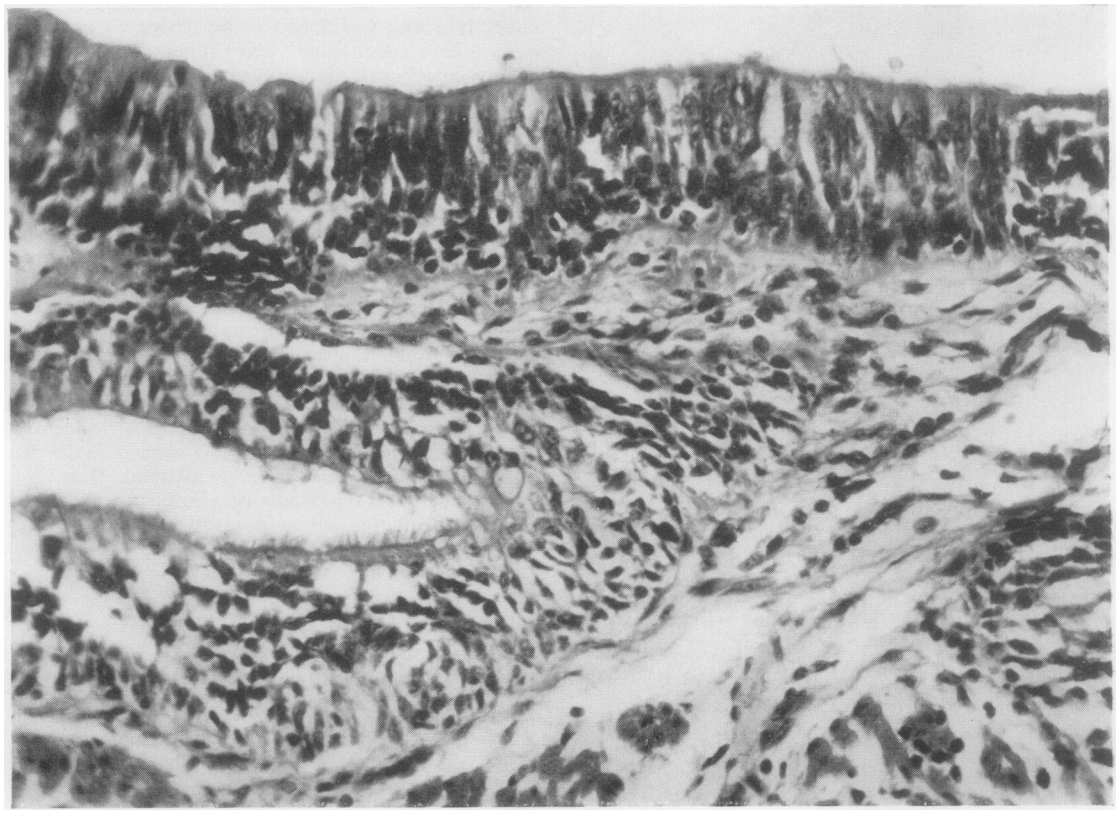

FIG. 4. *Hyperplasia of bronchial epithelium* (× 345, *H. & E.*).

Histological examination of sections from different areas showed a well-vascularized granulation tissue on the pleura. The normal alveolar pattern of the lung was destroyed and was replaced by poorly defined confluent areas of consolidation which were composed of relatively mature granulation tissue admixed with varying proportions of plasma cells, mononuclear cells, lymphocytes, and syncytial giant cells (Fig. 3). There was no caseation necrosis. The cellular infiltrates were predominantly around the bronchioles, and intervening surviving alveoli appeared either collapsed or filled with foamy macrophages. There were several foci of bronchial proliferation and hyperplasia of bronchial epithelium (Fig. 4). There was bronchiectasis, and some bronchioles contained polymorphonuclear leucocytes. Focal squamous metaplasia of bronchiolar epithelium was also present. There was interstitial fibrosis and an increase of interlobular fibrous tissue. Special stains for inclusion bodies in the hyperplastic bronchial epithelium and giant cells were negative and staining for bacteria was also negative. These changes were consistent with an unresolved bronchopneumonia of some duration.

DISCUSSION

The predisposing factors for anaerobic lung infection are a period of unconsciousness with aspiration of gastric content, aspiration from the throat during dental extraction, foreign bodies, tuberculosis, cavitation, and carcinomatosis. In the patient described in the present report there was no history of either vomiting or aspiration, although this could not be completely excluded, since in childhood vomiting is a common symptom and regurgitation of feeds during infancy was a possibility. Poor oral hygiene in this child may have been a contributory factor. Tuberculosis was excluded by the negative tuberculin skin tests and by the failure to recover tubercle bacilli from the sputum. Neither the history nor the findings at operation suggested a foreign body as a predisposing factor. It therefore remains conjectural what the underlying factor was in this patient.

The differential diagnosis was a bronchopneumonia with empyema and lung abscess. Without

a penetrating film it was impossible to entertain the diagnosis of a destroyed lung caused by an unusual organism. The special radiological examination together with bacteriological examination of the sputum clinched the correct diagnosis. Although anaerobic organisms are known to inhabit the throat, the organisms isolated from sputum are not considered to be contaminants.

Management of the present case with appropriate antibiotics and postural drainage was successful only in preventing spread of the infection to the other lung. Surgical resection of the affected lung seems to be the treatment of choice, since no reparative process could restore the lung to normal. In the case reported by Hewlett, Bitner, and Moraca (1959) surgical resection of the affected lung was employed.

The difficulty in arriving at a diagnosis in this case suggests that this infection might be more common than the literature indicates. Pulmonary infection with anaerobic organisms in children may be suspected when there is protracted illness resembling pulmonary tuberculosis which does not respond to anti-tuberculosis treatment. If there is a history of vomiting or regurgitation with possible aspiration in a child already debilitated from malnutrition, then this type of infection should be considered in the differential diagnosis. Laboratory confirmation using a special culture technique is absolutely necessary.

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