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Case report

Mucoid impaction of bronchi

A scanning electron microscopic study*

S. Richard[1], J. De Blic[2], A. Pfister[1], A. Lellouch-Tubiana[1], P. Scheinmann[2], and C. Da Lage[1]

[1] Laboratoire d'Histologie et Embryologie, Faculté de Médecine Necker-Enfants Malades, 156, rue de Vaugirard, F-75015 Paris, France
[2] Service d'Immuno-Allergologie Infantile, Hôpital Necker-Enfants Malades, 149, rue de Sèvres, F-75015 Paris, France

Summary. An example of mucoid impaction in the culminal bronchus in a potentially asthmatic child is related. Histological sections of the plug showed the characteristic pattern with clusters of eosinophils and other cells within the impacted mucus, which consisted of mainly sulfated glycoproteins. Scanning electron microscopy revealed new data on the three-dimensional structure of the plug. Ten to fifteen μm thick fascicles of partially rolled-up mucous fibres surrounded wide meshes filled with eosinophils and other components. As far as can be determined, this is the first reported ultrastructural description of this particular lesion.

Key words: Bronchi – Mucoid impaction – Mucus – Plugs – Scanning electron microscopy

Production of respiratory mucus is affected in several lung diseases. At the worst, bronchi may be sometimes filled with real plugs of mucus resulting from hypersecretion and accumulation of viscid, tenacious material. Diseases as varied as asthma, chronic bronchitis, cystic fibrosis or allergic bronchopulmonary aspergillosis may produce these changes (Irwin and Thomas 1973; Katzenstein et al. 1975). The term "mucoid impaction of the bronchi", introduced by Shaw in 1951, is commonly used to designate this severe complication. The microscopic structure of bronchial plugs is well documented (for a review see Katzenstein and Askin 1982) but no interest has been taken in their ultrastructural appearance. This paper reports a mucous plug in a child studied by scanning electron microscopy.

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Offprint requests to: S. Richard at the above address

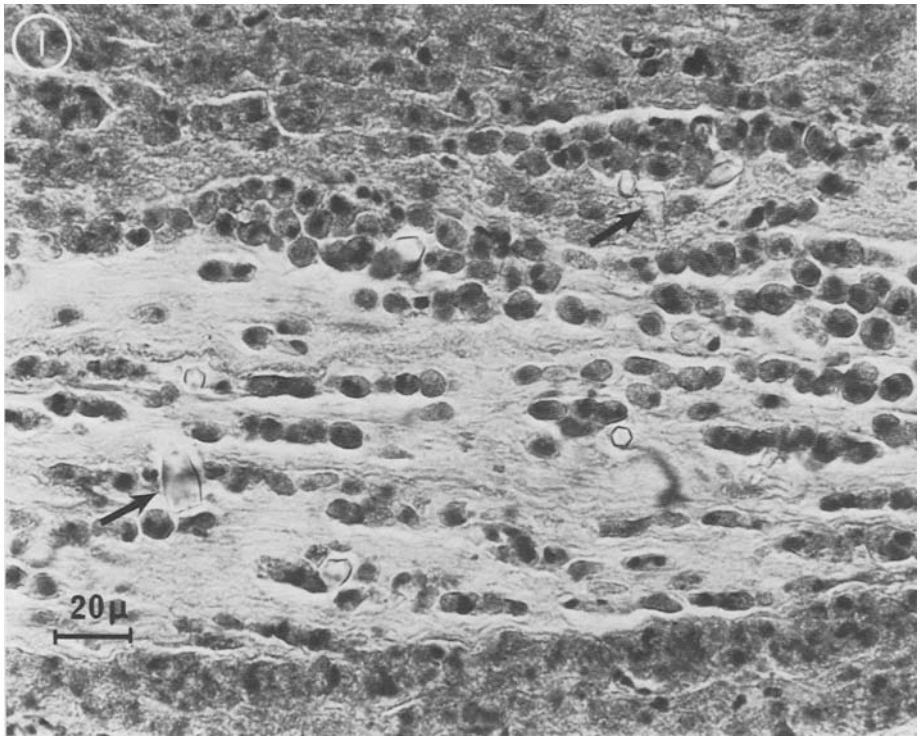

Fig. 1. Section through the plug showing laminated bands of impacted mucus with mainly eosinophils in various stages of necrosis. Charcot-Leyden crystals (→). (H.E. × 500)

Case report

A 4-year-old girl without previous pulmonary disease was admitted on April 24, 1984 with a suspicion of foreign body inhalation. A short time after ingestion of peanuts, she suddenly developed dyspneoa, a productive cough and fever. Left upper lobe atelectasis was detected on a chest radiograph. Initial bronchoscopy revealed diffuse oedema only and abundant secretions. Laboratory data showed a blood leukocyte count of 8800/mm^3 with 97% neutrophils. Blood cultures were negative. Under treatment including erythromycin, steroids and physical therapy, the temperature soon receded to normal but the left superior lobe collapse persisted. On April 19 fiberoptic bronchoscopy revealed copious purulent secretions and a 2,5 × 0,4 cm viscid, grey, branched mucous plug obstructing the culminal bronchus. A control chest radiograph following plug suctioning showed almost immediate re-aeration of the culmen and lingula. The patient's convalescence was uneventful. Additional laboratory results revealed a peripheral eosinophilia at 616/mm^3, normal sweat test for cystic fibrosis and no specific allergy. She was last seen in November 1984 at which time she was in good health.

Materials and methods

The plug, immediately fixed in 10% – formalin, was divided into two parts. The first one, embedded in paraffin, was sectioned at 4 microns. Sections were stained with Haemalum-Eosin, Grocott's methenamin silver or with Alcian blue pH 2.6-periodic acid-Schiff and Alcian blue pH 1 for mucins.

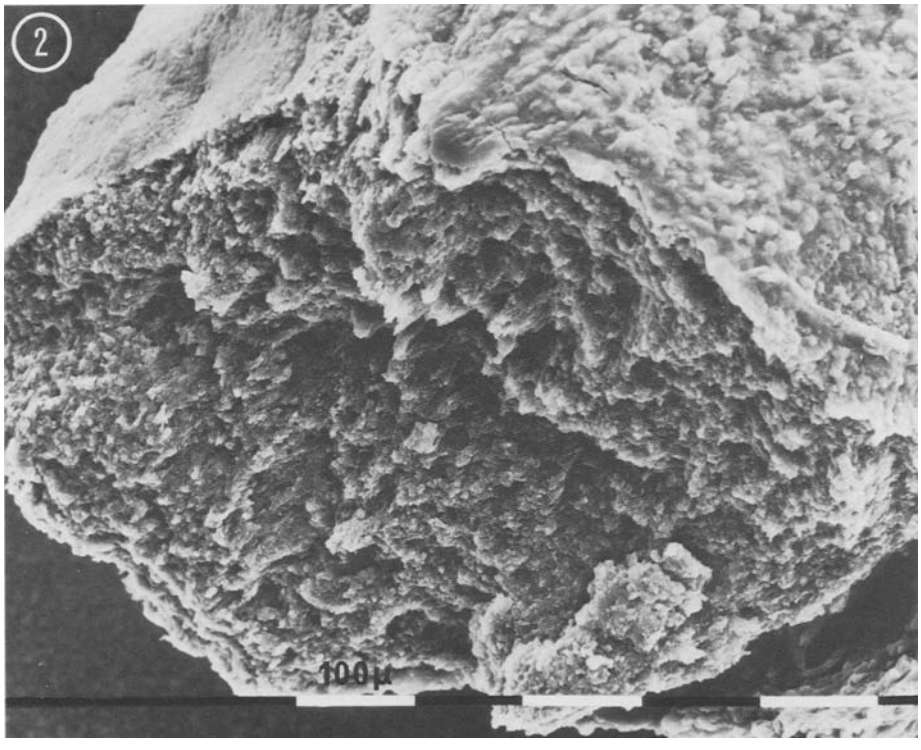

Fig. 2. Transverse section through the plug. Note the compact concentric structure coated with fresh mucus. (S.E.M. ×160)

The other fragment was dehydrated in acetone followed by critical point drying in CO_2. After coating with gold (Polaron E 5100), samples were observed with a Philips Scanning Electron Microscope 505.

Results

Light microscopy

Histological sections through the plug revealed numerous clusters of various cells including neutrophils, columnar cells and, predominantly, eosinophils in various stages of necrosis, separated from each other by laminated bands of mucus. Charcot-Leyden crystals were often observed (Fig. 1). Histochemical staining demonstrated mainly sulfated mucins in impacted mucus. No fungal elements were identified in sections stained with Grocott's methenamin silver.

Scanning electron microscopy

Transverse sections showed the compact laminated structure of the plug and its composition of necrotic cells within impacted mucus. The plug was

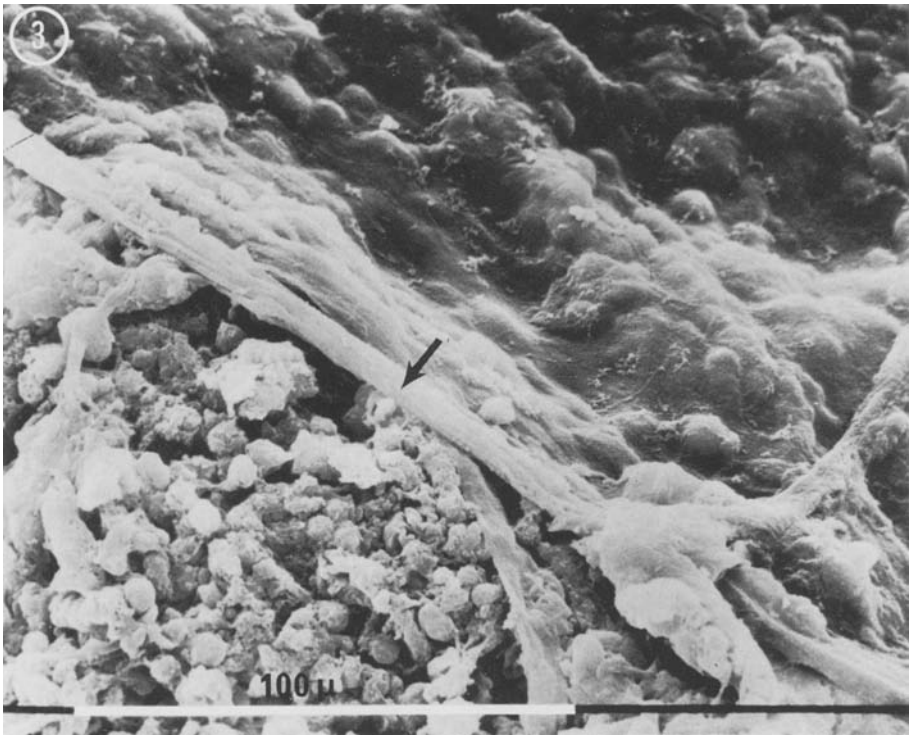

Fig. 3. Low-magnification photomicrograph of longitudinal section through the plug. Thick fascicles of mucous fibres (→) delimitate wide meshes filled with cells and debris. (S.E.M. × 700)

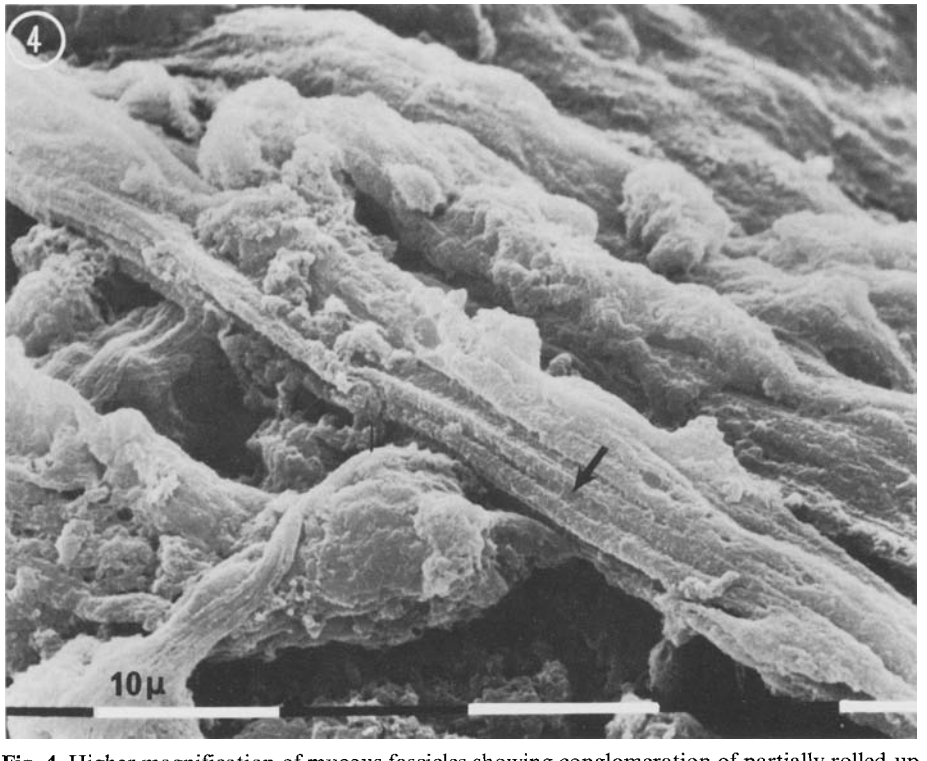

Fig. 4. Higher magnification of mucous fascicles showing conglomeration of partially rolled-up 1–2 µm fibres (→). (S.E.M. × 2,600)

completely coated with a delicate film of fresh mucus (Fig. 2). The internal framework of the plug was clearly revealed by longitudinal sections. It consisted of 10–15 μm thick fascicles of linked mucus fibres which delimited wide irregular meshes filled with clusters of cells and various debris (Fig. 3). Higher magnification of the mucus fascicles revealed conglomeration of partially rolled-up 1–2 μm fibres (Fig. 4).

Discussion

Mucus plugging is frequent in various lung diseases. Mucoid impaction occurring in bronchi, distal to an area of bronchial obstruction (neoplasm, foreign body, tuberculosis...), is quite common (Felson 1979). Mucous plugs may also be produced in quadriplegic subjects or other patients who have an ineffective cough (Dee et al. 1984).

In contrast, mucoid impaction of bronchi is a rare, distinct clinical entity characterized by mucus plugging which is unrelated to obstruction and which occurs predominantly in patients with underlying lung disease. The most frequently involved disease are asthma, chronic bronchitis, cystic fibrosis and allergic bronchopulmonary aspergillosis, but mucoid impaction may be found in association with bronchocentric granulomatosis or eosinophilic pneumonia (Sanerkin et al. 1966; Katzenstein et al. 1975).

Pathologically, the segmental bronchi are filled and distended with viscid, laminated mucous plugs which expand by concentric accretions (diameters as large as 2.5 cm have been reported). A predilection for upper lobes is usual. The histological structure of these mucus plugs consists of varying amounts of mucus, fibrin, epithelial cells, polymorphonuclear cells, eosinophils and necrotic debris (Urschell et al. 1966; Irwin and Thomas 1973; Katzenstein et al. 1975). Charcot-Leyden crystals, probably resulting from the agglutination of intracytoplasmic granules of necrotic eosinophils, are often numerous (Sanerkin et al. 1966). Clusters of eosinophils, which are usually absent from postobstruction impactions, are arranged in layers separated from each other by radial bands of mucus. Great attention must be directed to a search for fungal elements in the eosinophils clusters and mucus. Jelihovsky (1983) reported that fungal hyphae were frequent in mucoid impaction and were more readily seen among the eosinophils than in impacted mucus. This finding has been the first clue to diagnosis in several unsuspected cases of allergic aspergillosis (Katzenstein and Askin 1982).

The gross and microscopical appearance of the related mucous plug were very similar to those of previous reports. However no underlying disease was discovered. A viral affection was suspected but not proved. The presence of numerous eosinophils both in blood and in the impacted mucus despite the absence of other allergic signs, suggests that the patient is a potential asthmatic.

However, scanning electron microscopy of the mucus plug provided interesting results. The core of the plug consisted a fibrous network with wide meshes filled with other components. The fibrous fascicles themselves

were composed of agglomerations of smaller filaments. Although ultrastructural histochemistry is necessary for a precise investigation of the real composition of thick mucous fibres, it seems clear that they result from an increased bridging between abundant acidic mucins, especially sulphated glycoproteins. Other products such as lipids, hydrophobic peptides or DNA might be associated. This view is consistent with biochemical studies of bronchial mucus from patients with chronic disease (Lopez-Vidriero and Reid 1978; Roussel et al. 1984). Thus, production of abnormal viscid material may represent the first stage of mucus plugging. Cell accumulation, fibrin deposition, mucociliary transport failure and excessive water reabsorption could be involved later.

Scanning electron microscopy of small-sized plugs obtained post mortem or from surgical operation may provide interesting information in the future on the initial steps of this particular lesion.

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