Iron lung: bronchoscopic and pathological consequences of aspiration of ferrous sulphate

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Abstract
Acute bronchial damage was caused by aspiration of a ferrous sulphate tablet, early histological changes (unlike in the few previously reported cases) being observed in the biopsy specimens.

Foreign body aspiration occurs mainly in children but may occur in adults, particularly in the presence of oesophageal abnormalities. We describe the radiological, bronchoscopic, and histological findings in an elderly woman with a hiatus hernia who aspirated a ferrous sulphate tablet.

Case report
An 84 year old woman, a non-smoker, suddenly developed vigorous coughing, which produced a cupful of blood. The cough persisted for four weeks until presentation, and right pleuritic chest pain developed. The patient had a hiatus hernia but no previous history of dysphagia, and was taking ferrous sulphate tablets, 200 mg thrice daily, for chronic blood loss from the hiatus hernia. She could not recall a specific episode of aspiration.

A chest radiograph showed an incarcerated hiatus hernia and patchy opacification in the right mid and lower zones. Thoracic computed tomography showed concentric narrowing of the right lower lobe bronchus and patchy linear subsegmental collapse in the posteromedial aspect of the right lower lobe.

At bronchoscopy the right main and intermediate bronchi were lined with greenish brown necrotic material. The left main bronchus was entirely normal. The right intermediate bronchus was partially occluded by a hard polypoid mass covered with similar necrotic material. A bronchial biopsy specimen was taken, which histologically consisted of proteinaceous debris and elements of partially necrotic fibroconnective tissue. Fragments of disrupted bronchial epithelium were clearly identifiable and these, together with the subepithelial connective tissue, were encrusted with golden brown pigment that showed a very strong reaction for ferric iron (Prussian blue). Iron had precipitated particularly heavily on elastic fibres and bronchial epithelial cells, including their cilia (fig 1).

Overlying some of the degenerate tissue was a reactive metaplastic squamous epithelium, devoid of iron precipitate.

Repeat bronchoscopy, seven days later, showed similar appearances but the polypoidal lesion was no longer visible. The


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bronchial biopsy specimen showed necrotic collagenous tissue and a mild acute inflammatory reaction. Brown iron pigment was still abundant within respiratory epithelium, particularly concentrated along epithelial and capillary endothelial basement membranes.

The patient was treated with nebulised lignocaine (4% solution) to reduce the frequency of cough. After two months the chest radiograph had returned to normal, but after four months she developed a recurrence of cough and wheeze and of radiographic abnormality. Repeat bronchoscopy showed irregular, friable inflamed tissue almost occluding the right intermediate bronchus just distal to the origin of the upper lobe bronchus. Bronchial biopsy showed further evolution of the airway injury and repair. Maturing granulation tissue was abundant and contained many haemosiderin laden macrophages. The bronchial cartilage was eroded with perichondral scarring, squamous metaplasia was widespread, and iron deposition was prominent around small vessels (fig 2).

Discussion

This patient did not recall inhaling an iron tablet; but the history of sudden cough, haemoptysis, and abnormal chest findings on auscultation is typical of aspiration, as is the site of the foreign body in the right lung.1

Reports of aspiration of an iron tablet are rare, but the late occurrence of bronchial stenosis has been described.45 Reports of the histological appearances in these cases are from biopsy specimens taken several months after the insult, when bronchial stenosis was established. The findings at that time are predominantly those of fibrosis, small or moderate amounts of haemosiderin within macrophages, and perhaps an associated foreign body type granulomatous reaction. In this case we observed earlier changes, including acute mucosal damage, widespread iron precipitation on pre-existing mucosal elements, and squamous metaplasia over part of the ulcerated surface. The metaplastic squamous epithelium was completely devoid of iron, suggesting that it developed after the episode of iron deposition. The first two biopsy specimens show very extensive submucosal tissue necrosis, whereas the third shows much scarring and granulation tissue.

It is not surprising therefore that bronchial stenosis may be a late consequence of aspiration of iron tablets.

The presence of appreciable iron (haemosiderin) deposition in lung biopsy material suggests a differential diagnosis of pulmonary haemorrhage, with or without infarction or, rarely, Goodpasture's syndrome or idiopathic pulmonary haemosiderosis. In these circumstances, the haemosiderin is largely within alveolar macrophages with less lying in the interstitium. Iron deposition in the bronchus, as seen in this case, would be negligible. Bronchial mucosal iron deposits may rarely be seen in haemochromatosis but the deposition then is intracellular and without associated tissue damage. The pattern and density of deposition and the associated tissue damage in our case are unlike those seen in any of these conditions.

In one previously published case4 mucosal appearances returned to normal during 15 months of follow up, though bronchial stenosis persisted; in the other severe bronchial stenosis necessitated right middle and lower lobectomies.5 Removal of the tablet at the initial bronchoscopy was not possible in our patient but should clearly be attempted wherever possible to minimise tissue damage and subsequent complications.6

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