

CASE REPORT

Recurrent interlobar pneumothorax in an asthmatic patient

C. Gourdon*, A. Dietemann*, C. Beigelman**, B. Sohier*, G. Pauli*

Recurrent interlobar pneumothorax in an asthmatic patient. C. Gourdon, A. Dietemann, C. Beigelman, B. Sohier, G. Pauli. ©ERS Journals Ltd.

ABSTRACT: We report a case of spontaneous interlobar pneumothorax, an uncommon condition. The diagnosis was suspected from the frontal chest X-ray, which showed an incompletely circumscribed air-containing space, with a fluid level. The diagnosis was confirmed by the lateral projections of standard X-ray and tomography, and by thoracic computed tomography (CT). Such air-fluid levels should not be confused with pneumatocele, cystic or cavitory diseases.

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* Service de Pneumologie, Pavillon Laennec, and ** Service de Radiologie Médico-Chirurgicale B, Hopitaux Universitaires de Strasbourg, Hôpital Civil, B.P. 426, 67091 Strasbourg Cedex, France.

Correspondence: G. Pauli, Pavillon Laennec, Hopitaux Universitaires de Strasbourg, Hôpital Civil, B.P. 426, 67091 Strasbourg Cedex, France.

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Spontaneous interlobar pneumothorax is an unusual topographical feature of pneumothoraces [1-3]. Originally described by phthisiologists [4] as a complication of tuberculosis, a few cases of interlobar pneumothorax have been observed by thoracic surgeons following pleurodesis [3]. It does not occur without pleural adhesion. The radiological signs of partial pneumothorax depend upon its volume, its topography, the patient's position, and the previous existence of pleural or pulmonary disease.

Case report

A 61 yr old man was admitted to our clinic with haemoptysis in October 1990. This patient has been suffering from bronchial asthma since 1985. The diagnosis of Samter's syndrome had been made in 1987, based on nasal polyps associated with adult onset asthma, and the results of an aspirin challenge test [5]. This classical triad co-existed with proximal bronchiectasis of the two upper lobes and the middle lobe, which were apparent on computed tomography (CT). The patient did not present any criteria of allergic bronchopulmonary aspergillosis. With treatment (inhaled corticosteroid as 2,000 µg beclomethasone, salbutamol spray and theophylline *per os*) his asthma was mild, with a peak expiratory flow rate (PEFR) greater than 500 l·min⁻¹, and he had no acute attacks requiring clinical treatment after 1987. Pulmonary function tests (July 1990) showed persistent airway obstruction (spirometric values: forced vital capacity (FVC) 5.13 l, 116%; predicted forced expiratory volume in one second FEV₁ 2.48 l; FEV₁/FVC 48%), with hyperinflation (total lung capacity) (TLC) 8.41 l; residual volume (RV) 3.28 l).

One month prior to his hospital admission, the patient had an exacerbation of asthma (PEFR 350 l·min⁻¹) after an upper respiratory infection which responded to antibiotics and prednisolone. However, cough persisted, and he complained of left chest pain during a physiotherapy session. After the pain decreased, haemoptysis appeared, prompting his admission to our clinic. On arrival, he was neither dyspnoeic nor cyanotic, but described left chest pain during inspiration. His temperature was 37°C. Chest auscultation was normal, except for an axillary amphoric auscultatory sound. The PEFR was 500 l·min⁻¹.

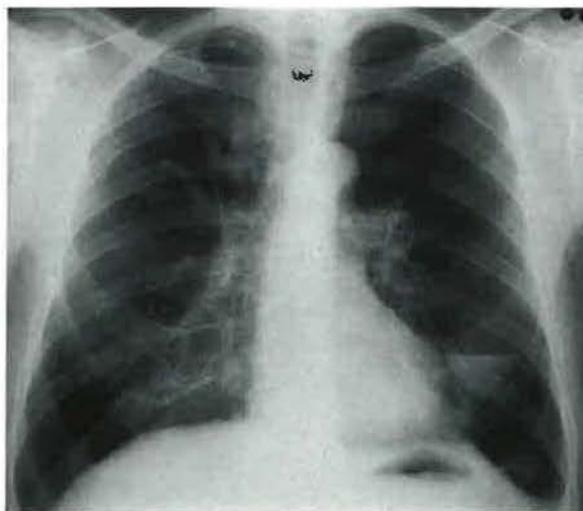


Fig. 1. - Frontal chest X-ray: there is an air-fluid level in the left paracardiac area and a partial lateral pneumothorax in the upper part of the chest.



Fig. 2. - Lateral projection of standard tomography shows an oblong radiolucent area instead of the normal left fissure.

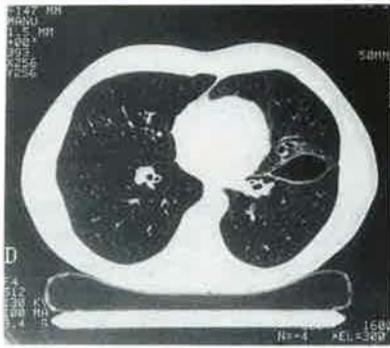


Fig. 3. - Computed tomography (CT) shows a lenticular air space in the left fissure.

Frontal chest X-ray showed an air-fluid level, without a well-circumscribed border, in the left paracardiac area (fig. 1). More distant from the air-fluid level, we noticed a thin, long, linear opacity convex to the external part of the left chest, which had an indistinct superior end in the upper part of the chest. The pulmonary parenchyma external to this line was still in contact with the lateral chest wall. This line could be superimposed with the normal plane of the left fissure. Lateral X-ray projections and standard tomography showed an oblong radiolucent area, instead of the normal left fissure (fig. 2). The air-fluid level corresponded to the inferior portion of this oblong radiolucent area. CT showed a lenticular air space along the normal course of the left fissure (fig. 3). The intrafissural pneumothorax was in contact with bronchiectasis of the upper lobe. The CT films also showed some emphysema and con-

firmed that the pneumothorax was only interlobar. Endoscopic examination was normal.

With rest, the interlobar pneumothorax disappeared after 3 weeks but, six months later, this patient was readmitted for a recurrence of the pneumothorax, again with haemoptysis.

Discussion

Spontaneous pneumothorax is almost always the result of rupture of an air-containing space into the visceral pleura [6]. Air can come from a pneumomediastinum [3], from a rupture of alveolar blebs or bullae, and from cystic spaces, as in our observation.

Intrafissural restriction of the pneumothorax is due to previous fibrous pleural adhesions [1-3]. These prior adhesions could be the result of pleurodesis or pleural diseases. Pleural adhesions prevent air from spreading, as it generally does, into the pleural cavity. We cannot exclude the hypothesis that this particular form of pneumothorax may represent incomplete resorption of a continuous complete pneumothorax. Fluid is due to a secondary pleural effusion.

It is possible that pleural fibrosis associated with bronchiectasis localized the air to the adjacent interlobar fissure. This patient had no other antecedent left-sided pleural disease, although he had fractured the 4th and 5th right ribs.

The roentgenographic abnormalities can involve the entire fissure of the left or right chest. An air-fluid level is uncommon, but facilitates the diagnosis. The oblong form of the radiolucent area, and its location along the course of the fissure, are the most important characteristic features for making the diagnosis.

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