typical subtype is 82%–87% and that for the atypical subtype is 56%–75%.3

This case is of interest because, despite the advanced age of onset (69 years) and a diagnosis of typical carcinoid tumor, there was local recurrence after the patient was disease-free for more than 4 years. The tumor showed no necrotic element but the mitotic index was high, so the final diagnosis was atypical carcinoid carcinoma. The central location of the recurrence prevented radical treatment. The patient is currently asymptomatic, leading a normal life and attending regular check-ups.

References


Empyema Necessitatis Following Chest Trauma

Dear Editor,

Empyema necessitatis is a rare complication that can occur when pleural infections are treated late or inadequately. It consists of the penetration of pus from the pleural cavity through the adjacent tissues to form an abscess in the chest wall, sometimes even forming a skin fistula.1 Chest trauma is a very rare cause of empyema necessitatis, so a case recently treated at our center is of interest.

A morbidly obese 49-year-old male, current smoker, with poorly controlled diabetes mellitus type 2, presented due to left costal blunt trauma caused by an impact from the horn lateral surface of a charging bull, 30 days before his admission to the hospital. The patient did not attend emergency services at the time of trauma. He had a hematoma in the left chest wall and chest pain, which did not improve with standard analgesia. On arrival to the emergency room, blood pressure was 140/86 mmHg, heart rate 110 bpm, oxygen saturation 96%, and temperature 36.5 °C. Physical examination revealed a hematoma in left lateral chest wall with no surrounding cellulitis and decreased breath sounds in the left hemithorax. Blood tests showed leukocytosis of 32,400/mm³ with neutrophilia (92.2%), hemoglobin 10 g/dl, glucose 487 mg/dl, and C-reactive protein 40.3 mg/dl. Left pleural effusion was detected on the chest X-ray and no rib fractures were observed. A computed tomography (CT) scan completed the study, revealing left pleural effusion that connected with a collection in the left anterolateral chest wall (Fig. 1). Empiric antibiotic therapy with piperacillin-tazobactam was initiated (4/0.5 mg IV every 8 h) and both collections were drained by percutaneous puncture of the chest wall and a chest tube, from which abundant purulent material was obtained. Good lung re-expansion was observed on X-ray. After 72 h of admission, multiple orifices were observed in the left lateral thoracic wall, surrounded by purulent necrotic areas without muscle involvement, so debridement and lavage were performed under general anesthesia. Microbiological results from both the pleural fluid and thoracic abscess were positive for Streptococcus agalactiae, that was sensitive to the prescribed antibiotic. The patient was hospitalized for 40 days, during which surgical wound care continued without the need for additional interventions.

The most common location of an empyema necessitatis is, as in this case, the anterior chest wall between the midclavicular and anterior axillary line. Other locations less frequently described are the abdominal wall, the paravertebral space, the mediastinum, the breast or the diaphragm.1,2 Before the antibiotic era, most cases were caused by Mycobacterium tuberculosis and the mortality rate was 66%. The incidence has fallen significantly since antibiotics were introduced, and the most common etiologic agents have become Actinomyces israelii, Streptococcus pneumoniae, Staphylococcus aureus or Pseudomonas cepacia.3,4 In our case, the causative agent was Streptococcus agalactiae, occurring secondary to chest trauma, features that are both rare in these circumstances. The clinical presentation can vary widely and includes chest pain, soft tissue mass, cough or dyspnea. Depending on the age of the patient and his/her morbidity, it may progress to septic shock. Our case was an obese patient with poorly controlled diabetes. Both of these conditions are favorable for the development of infections with atypical etiologies and locations, due to changes in the immune system response to invading microorganisms, principally fungi and bacteria. Diagnosis is based on imaging techniques, mainly CT, revealing the continuity between the pleural collection and the abscess in the chest wall. The differential diagnosis must consider other diseases such as lymphoma, mesothelioma or endocarditis.4 Although treatment should be tailored for each patient, both antibiogram-

![Fig. 1](image-url) Computed tomography: left pleural effusion communicating with collection in the left anterolateral chest wall.

adjusted antibiotic treatment, when possible, and surgical drainage are crucial. Drainage is required to evacuate the accumulated pus and to sterilize and close the pleural cavity, thus allowing good pulmonary mobility. Different procedures may be employed, such as closed drainage systems by tube thoracostomy, or partial costectomy, or open systems, such as open thoracostomy with the creation of a pleuropulmonary fistula that allows drainage without a tube. The timing of these treatments is not standardized, but the intravenous antibiotic should be maintained for a week and then oral treatment should be continued for 1–3 weeks, depending on the patient’s clinical response. With the combination of both treatments, a high cure rate is achieved with a low mortality rate (less than 5%); death, if it occurs, is usually secondary to the confluence of several mechanisms, such as respiratory failure, heart failure, mediastinitis, hematogenous spread or renal failure.1

References

Idiopathic Lipoid Pneumonia Findings in Chest High Resolution Computed Tomography. A Case Report

Hallazgos en la tomografía computarizada de tórax de alta resolución de la neumonía lipoidea idiopática. A propósito de un caso

To the Editor,

Lipoid pneumonia (LP) is an uncommon condition that occurs as a result of the deposit of exogenous or endogenous fatty materials in the lung tissue. The incidence of LP is between 1% and 2.5% according to some series of autopsies.1

We report the case of a 15-year-old male patient with a history of jaundice at the age of 4 days, febrile seizure at one year and 4 months, scarlatiniform syndrome at 5 years, erysipelas rash at 7 years of age, and hip synovitis. He has been monitored by the pediatric department since he was 8 years old due to delayed growth and development, with episodes of coughing and exertional dyspnea. He was diagnosed with mild episodic asthma, and was treated with montelukast and albuterol as rescue medication, without improvement. The following analyses were performed: ANA, C3, C4, CD3, NK, IgE, eosinophils, granulocyte function, immunoglobulins, TSH, test for celiac disease, sweat test, skin prick and radioallergosorbent test for food allergies; all results were normal. He has had contact with 2 canary birds from an early age, and with spray paints at 11–12 years of age (graffiti).

Upon admission to the pulmonology unit, the patient had dyspnea grade I–II. Physical examination revealed good coloration of the skin and mucous membranes, and finger clubbing. Baseline O2 saturation was 93% and good vesicular breath sounds were perceived upon auscultation, with no added noises. Spirometry indicated FEV1: 44%, FVC: 47%, FEV1/FVC: 79, MMEF: 29%, although patient cooperation was incomplete. The chest X-ray showed an extensive bilateral alveolointerstitial pattern with multilobar involvement. The examination was completed during hospitalization with high-resolution computed tomography (HRCT), in which marked alteration of the lung parenchyma was observed, with large ground-glass fields in all lobes, but predominantly basal. There was overlapping septal thickening, forming a crazy-paving pattern (Fig. 1). Furthermore, honeycombing areas were visible in the subpleural lung parenchyma at the level of both upper lobes, although predominantly in the right side.

In view of these findings, a transbronchial biopsy was performed. The histological study showed giant cells phagocytosing foreign body cholesterol crystals in the alveolar interstitium and alveolar spaces, along with both isolated and accumulated macrophages with microvacuolated cytoplasm. There was occasional eosinophilic pseudo-proteinaceous material in the alveolar spaces that was negative on PAS and Congo red staining. No microorganisms were observed. Histological findings were compatible with LP.

Currently the patient is receiving oral and inhaled corticosteroids, which have resulted in a clinical improvement, although the radiographic findings have shown no change.

In the absence of frequent use of laxatives, balms, oils or other lubricants, other causes of endogenous pneumonia should be assessed,1 such as bronchial obstruction produced by a pulmonary tumor (clearly ruled out in our case), repeated episodes of respiratory fungal infection (not recorded in the patient’s medical history nor suggested by histological or laboratory data), alveolar proteinosis (ruled out by pathology) and Niemann-Pick disease (mainly type B), in which crazy-paving pattern has also been described.2 However, the patient did not present hepatosplenomegaly or any laboratory abnormality suggesting this possibility. Thus, in the absence of any other identifiable cause, this case was classified as idiopathic LP.3

The most common HRCT radiological findings in LP4,5 are the presence of condensation with low attenuation coefficient (between −75 and −35 HU) that is very specific and prevalent in endogenous LP,1 ground-glass opacities, crazy-paving pattern, and presence of centrilobular opacities. In addition, lung involvement is mainly basal.5

The crazy-paving pattern was initially described in alveolar proteinosis, although it was later shown to appear in


Susana Pérez-Brú, a, * David Martínez-Ramos, b José Luis Salvador-Sanchís b

a Servicio de Cirugía General y del Aparato Digestivo, Hospital Universitario Sant Joan, San Juan de Alicant, Alicante, Spain
b Servicio de Cirugía General y del Aparato Digestivo, Hospital General de Castellón, Castellón, Spain

*Corresponding author.
E-mail addresses: susanaperezbru@gmail.com, susanabr_83@hotmail.com (S. Pérez-Brú).