

clear beneficial effect.⁵ In fact, corticosteroids seem to have a paradoxical effect, as they have been considered to play a role in the genesis of FES in patients under chronic treatment with these agents.⁵

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Bilateral Pneumothorax after Pleural Drainage

Neumotórax bilateral tras la colocación de un drenaje pleural

To the Editor:

Bilateral primary spontaneous pneumothorax is a rare disease (<1%)¹ that requires urgent diagnosis and treatment. We report a case of presentation that occurred after draining a pneumothorax in the contralateral hemithorax.

A 30-year-old man with a 3-month history of right unilateral pneumothorax presented with dyspnea and pain in the left-hemithorax lasting 3 hours. Physical examination revealed tachypnea, oxygen saturation of 93%, and stable vital signs. A chest x-ray showed a left pneumothorax with contralateral mediastinal shift. During insertion of an 18-F chest tube, the patient complained of moderate pleuritic chest pain; a follow-up chest x-ray was made (Figure) and the right hemithorax was drained with no complications. The new radiologic test showed reexpansion of both lungs. A computed tomography scan (CT) revealed bilateral apical paramediastinal bullae, mainly on the left side. On completion of the preoperative evaluation, bullectomy and mechanical pleurodesis of both lungs were performed in a 2-stage operation. Postoperative recovery was satisfactory and the patient was discharged a week later.

The incidence of bilateral spontaneous pneumothorax is low and is more common in patients with an etiologic trigger (human immunodeficiency virus infection, tuberculosis, sarcoidosis, etc).¹ Although patients with bilateral spontaneous pneumothorax have a lower body mass index than those with unilateral pneumothorax, there are no differences regarding age, sex, or smoking. However, patients with bilateral pneumothorax do present a greater incidence of bullae, which are considered an independent risk factor in the development of bilateral pneumothorax.^{1,2}

We emphasize the importance of this case because the contralateral pneumothorax was only detected in the chest x-ray made after the chest tube was inserted; there was no evidence of air accumulation in the first x-ray study. Although the first imaging test revealed no pneumothorax on the contralateral side, the follow-up chest x-ray made after the chest tube was inserted led to its detection. This prompts us to raise the following points:

The contralateral pneumothorax may have been a complication of the reexpansion of the pre-existing pneumothorax. To date, pulmonary edema after abrupt reexpansion is the only known complication of spontaneous pneumothorax. However, if a patient

presents with an underlying disease such as bullae, air aspirated through a chest tube from the pleural cavity of one of the hemithoraces would exert sufficient traction on the contralateral pleura and the bullae to trigger a new pneumothorax.

The images might also be due to the effects of pressure; that is, a simultaneous bilateral pneumothorax might have occurred in our patient but gone undetected in the first imaging test because more pressure was exerted on the left side. This would have led to the compression of the right pneumothorax, which would only have become apparent when the former resolved.

Finally, a pleural window may have communicated with both pleural spaces.³ The window might have been embryonic in origin (due to a defect in the fusion of the pleuropericardial folds in the fifth week of development), or a complication of cardiac and thoracic surgery. In patients with unilateral pneumothorax, this window permits the passage of air from one side to the next, causing bilateral collapse. Our patient had bilateral bullae, however, and pleural windows are typically found in patients with unilateral disease. Although the pleural defects were predominantly left-sided in our

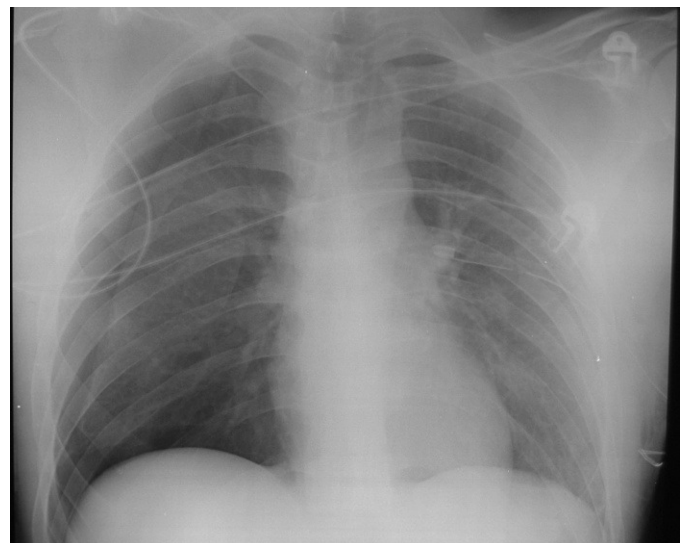


Figure. Posteroanterior chest x-ray: chest tube in the left hemithorax with complete reexpansion of the left lung and appearance of a new right pneumothorax over 3 cm long.

case, any of the possibilities outlined above could be valid, including that of the pleural window.

A CT scan of the chest is indicated prior to surgical treatment.⁴ In fact, several studies advocate this procedure for patients with unilateral pneumothorax to check for the presence of bullae in both lungs and evaluate the risk of recurrence.⁵ A CT scan enables the exact localization of the pleural defect⁶ prior to definitive treatment. The treatment of choice for bilateral pneumothorax is video-assisted thoracoscopic bullectomy with pleural abrasion.⁴

In short, doubts still remain about the origin of bilateral primary spontaneous pneumothorax, which requires close study in order to treat the condition adequately by video-assisted thoracoscopic surgery. We therefore support the use of chest CT scanning in both patients with bilateral primary spontaneous pneumothorax and patients with unilateral spontaneous pneumothorax with lung collapse when there is a suspicion of bullous lung disease.⁴

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Pulmonary Abscess Due to *Leuconostoc* Species in an Immunocompetent Patient

Absceso pulmonar por Leuconostoc spp. en un paciente no inmunodeprimido

To the Editor:

Leuconostoc species are gram-positive, catalase-negative bacteria resistant to vancomycin. Although their common habitat is the natural environment, they occasionally cause infection in humans, especially in immunocompromised patients. We present a case of pulmonary abscess caused by *Leuconostoc* species in a patient with no risk factors. To our knowledge this is the first reported case of a lung abscess due to *Leuconostoc* species.¹

The patient was a 75-year-old man, an ex-smoker (40 pack-years) who had quit smoking 40 years earlier. He had chronic renal insufficiency (creatinine, 1.8 mg/dL; creatinine clearance, 27.6 mL/min) and a 10-year history of chronic obstructive pulmonary disease under treatment with bronchodilators. He was hospitalized owing to a weight loss of 10 kg during the previous month and a cough with purulent sputum lasting 15 days but no fever. Physical examination, which revealed the presence of crepitations in the lower third of the left hemithorax, was otherwise normal. A blood workup showed a leukocyte count of 15 600/mL with 82% neutrophils; hemoglobin, 11g/dL; iron, 50 µg/dL; transferrin, 122 mg/dL (saturation, 32%); ferritin, 1385 ng/mL (morphology, normal); 65 000 platelets/mL; C-reactive protein, 18.22 mg/dL; and normal levels of carcinoembryonic antigen, carbohydrate antigen 19-9, and α-fetoprotein. Arterial blood gas analysis breathing a fraction of inspired oxygen of 0.21 revealed a pH of 7.43, PaO₂ of 80.9 mm Hg, PaCO₂ of 34.2 mm Hg, and a bicarbonate concentration of 22.5 mmol/L. Chest radiography and computed tomography with no intravenous contrast showed a round parenchymal lesion of approximately 8 cm with well-defined contours in the posterior portion of the left lower lobe and a small pleural effusion (Figure).

The patient started treatment with levofloxacin (500 mg/12 h). On the third day of hospitalization, fiberoptic bronchoscopy showed a reduction in the diameter of segments in the left base and signs of

inflammation, but no visible endobronchial tumors or other findings. Cytology of the bronchial aspirate and brushings indicated extensive acute inflammation; transbronchial biopsy revealed focal squamous cell metaplasia and chronic inflammation in the lamina propria, as well as indications of bronchiolitis obliterans organizing pneumonia in fragments of lung parenchyma. Samples obtained by bronchial aspiration and through a protected telescopic catheter showed gram-positive cocci.

Three milliliters of foul smelling pus obtained by fine-needle aspiration of the lesion under radiological guidance revealed gram-positive cocci of the *Leuconostoc* species, sensitive to penicillin and cephalosporin. *Leuconostoc* species were also isolated in the cultures from the bronchial aspirate and protected brush.

The patient's fever peaked on the seventh day of hospitalization, when blood cultures were negative. After receiving the result of the antibiogram, we prescribed cefditoren pivoxil 400 mg/12 h for 30 days. Treatment led to clinical improvement and showed radiologic resolution.

Leuconostoc species are gram-positive, catalase-negative, anaerobic cocci that form pairs or chains and produce carbon dioxide from glucose. They are resistant to vancomycin and frequently difficult to differentiate from pathogens such as *Enterococcus* and *Lactococcus* species, and *Streptococcus viridans*. When identifying *Leuconostoc* species, it is important to remember that they are the only gram-positive, catalase-negative cocci that are also pyrrolidonyl arylamidase and leucin aminopeptidase negative and vancomycin resistant.¹ They are commonly found in the natural environment— plant material, green plants, and roots— which is their ecological niche, and in milk products and other fresh foods; they are used in the production of wine, cheese, milk products, and sugars.²

These microbes are usually sensitive to ampicillin, clindamycin, erythromycin, and aminoglycosides, and have intermediate sensitivity to cephalosporins and imipenem.¹ They occasionally cause infection in humans although reports have been few and have involved few patients.^{3,4} They can cause opportunistic infection at any age although infection is most frequently seen in infants and the elderly.

The skin and digestive tract are believed to play important roles as routes of entry into the body. *Leuconostoc* species can cause