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 defect[6] 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 (creatine, 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, PaO_2 of 80.9 mm Hg, $PaCO_2$ 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

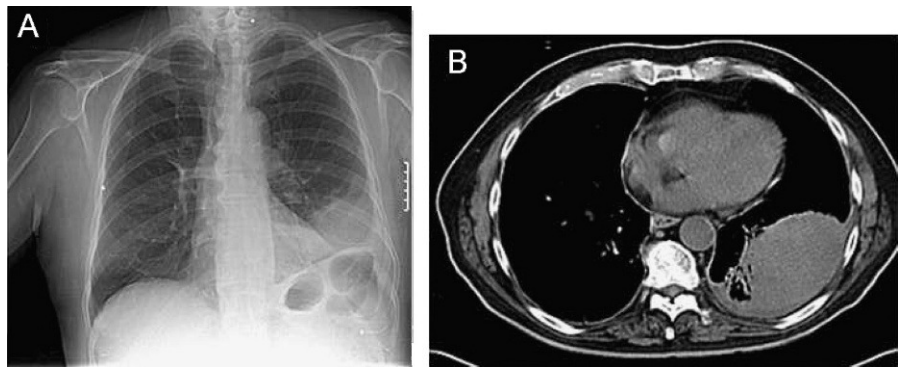

Figure. *(A)* Chest radiograph showing an 8-cm, rounded parenchymal lesion with well-defined contours located posteriorly in the left lower lobe, and pleural effusion. *(B)* Computed tomography scan showing pulmonary abscess due to *Leuconostoc* species.

bacteremia, catheter-related infections, meningitis, urinary tract infections, osteomyelitis, or liver dysfunction, among other problems.[3,4] They rarely cause pleuropulmonary disease, although pneumonia was reported in a patient with human immunodeficiency virus infection[5] and pleural empyema in an immunocompromised patient.[6] To date, no cases of pulmonary abscess in immunocompetent patients have been reported.

Risk factors for infection by *Leuconostoc* species are placement of central venous catheters, a history of use of vancomycin, liver failure, chronic renal insufficiency treated with hemodialysis, extensive burns, and compromised immunity. No deaths related to *Leuconostoc* species infection have been reported.

The peculiarity of our case is that the patient presented neither compromised immunity nor other risk factors for infection by this microorganism. The clinical importance of infection by *Leuconostoc* species in immunocompetent patients is unclear owing to the lack of reports of such cases.

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Acute Transient Ataxia Caused by Local Lidocaine Injection During Insertion of a Pleural Catheter

Ataxia aguda transitoria por lidocaína local durante la inserción de un catéter pleural

To the Editor:

Drainage of the pleural cavity by inserting an intercostal catheter or tube is a common therapeutic procedure in patients with empyema and complicated parapneumonic pleural effusion. Potential complications during the insertion of a small-caliber pleural catheter are rare and include pain, vasovagal reflex, bleeding caused by injury to the vessels of the chest wall, and pneumothorax.[1] The patient seldom experiences adverse effects caused by local anesthesia, as in the case we report.

A 46-year-old man was admitted to hospital with fever, cough, and acute left pleuritic pain. His history was remarkable only in that he had type 1 diabetes mellitus. Radiology revealed condensation at the base of the left lung. He was diagnosed with community-acquired pneumonia and empiric treatment with ceftriaxone and azithromycin was started. A massive left pleural effusion was observed after 48 hours; therefore, a 12F pleural drainage tube was inserted using the Seldinger technique. The patient was not premedicated with atropine. The procedure began with a 10-mL injection of 5% lidocaine (500 mg) into the skin and subcutaneous tissue at the level of the fourth intercostal space at the left midaxillary line. A further 15 mL (750 mg) of lidocaine was injected to anesthetize the parietal pleura. Purulent pleural liquid was obtained and the procedure continued with the insertion of dilators followed by the drain. The tube drain was inserted to a depth of 12 cm below the skin, although no further liquid was obtained. At this point, approximately 2 minutes after administration of the local anesthetic to the parietal pleura, the patient presented dysarthria, followed immediately by binocular diplopia. His blood pressure was 145/85 mm Hg, his pulse was regular at 85 bpm, and capillary blood glucose was 350 mg/dL. There was neither motor loss nor sensory loss, nor impaired level of consciousness; however, the patient did present noticeable global ataxia, which manifested as adiadochokinesia and impaired performance in the finger-nose test and heel-knee test. The drain was withdrawn and no attempt was made to reinsert it due to the patient's acute neurologic status. There was a substantial spontaneous improvement in the cerebral symptoms over the next 30 minutes and these had resolved completely 1 hour later. Twelve hours later, a new drain was inserted using 20 mL of 2% mepivacaine after first verifying that no reaction occurred with a 0.5-mL injection. There were no complications. The patient had never received a local anesthetic before.