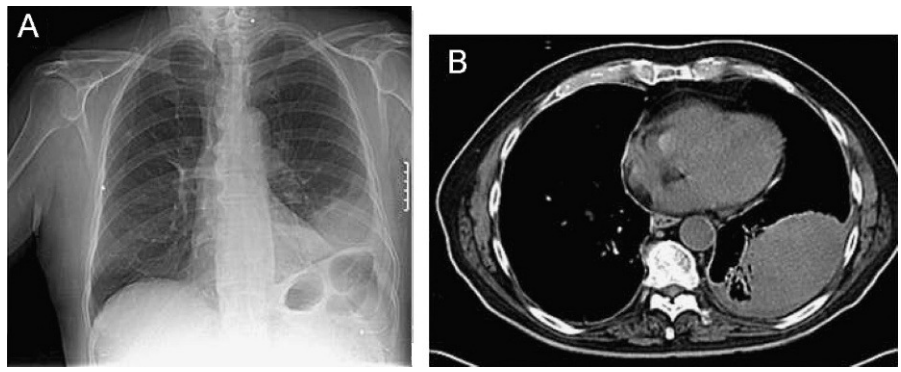

Figure. *(A)* Chest radiograph showing an 8-cm, rounded parenchymal lesion with well-defined contours located posteriorly in the left lower lobe, and pleural effusion. *(B)* Computed tomography scan showing pulmonary abscess due to *Leuconostoc* species.

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.

Although local anesthetics are considered safe, they can lead to cardiovascular and neurologic toxicity and allergic reactions. Neurologic toxicity caused by lidocaine is well documented, especially after spinal anesthesia,[2] and it manifests uncommonly as convulsions, lethargy, or confusion.[3] Ataxia following nasal or oropharyngeal administration of lidocaine in procedures such as bronchoscopy or transesophageal echocardiogram is exceptional and resolves spontaneously after a few hours.[4] There have been no reports of ataxia after local administration for pleural procedures.

The neurologic effect observed in our patient could be due to rapid systemic absorption of an excessive quantity of lidocaine across a hypervascularized inflamed pleura. Some in vitro studies suggest that mepivacaine has potentially less neurologic toxicity than lidocaine.[5] If lidocaine is used, the water-based 2% solution should be chosen.

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