

LETTERS TO THE EDITOR

Mediastinitis and Thoracic Empyema Caused by *Streptococcus constellatus*

To the editor: Streptococci that colonize the mouth and upper airways are generally considered to be commensal rather than pathogenic. In combination with anaerobics, they can be responsible for pneumonia and empyema in patients with certain predisposing factors. Mortality in such cases is high and complications frequent. We present a case of mediastinitis and thoracic empyema secondary to *Streptococcus constellatus*.

A 63-year-old man who was a moderate drinker with a history of chronic obstructive pulmonary disease and gastroesophageal reflux disease reported cough, dyspnea, odynophagia, and 39°C fever starting 6 days earlier. Physical examination revealed general malaise and crackles at the base of the right lung. Analysis revealed leukocytosis (13 500 μ L) with a left shift, a biochemical profile of cholestasis, and glycemia of 376 mg/dL. Arterial blood gas analysis showed moderate hypoxemia. Chest x-ray showed mediastinal widening with slight cardiomegaly and blunting of the right costophrenic angle. A thoracic computed tomography scan revealed right pleural effusion with perihilar alveolar infiltrates and increased upper mediastinal fat, consistent with mediastinitis (Figure). Thoracentesis revealed a purulent liquid in which *S constellatus* was isolated. A pleural drainage tube was inserted and high doses of penicillin G were administered. Clinical course was good, as the pleural effusion resolved and the mediastinal involvement disappeared.

Streptococcus milleri bacteria, which include *S constellatus*, play a greater role in respiratory infections than was once thought.¹ *S constellatus* is an oropharyngeal commensal which does not cause illness under normal conditions but can do so after intubation (endoscopy, bronchoscopy), surgery, or infection. *S milleri* can cause acute pneumonia, abscesses, and empyema.^{2,3} Their isolation in combination with oral anaerobic bacteria indicate a synergy between these 2 groups. Mortality is then higher, with severe complications such as pulmonary abscesses, empyema, or, as in the case of our patient,

mediastinitis. Mediastinitis caused by *S milleri* without prior intubation or surgery is rare.⁴ Esophageal perforation, cardiothoracic surgery, and head and neck infections are common causes.^{4,6} The clinical picture includes sudden onset fever and chills followed by chest pain and dysphagia. Examination can reveal crackles and edema in the neck and chest. A chest x-ray reveals mediastinal widening, air-fluid levels, and subcutaneous emphysema. Computed tomography permits a more accurate diagnosis. If esophageal perforation is suspected, an esophagogram must be performed with an iodine contrast, which can be administered parenterally. Penicillin G is the treatment of choice given its effectiveness against anaerobic bacteria in the oral cavity. If the patient is allergic to penicillin G, vancomycin and clindamycin can be used, as can third and fourth generation quinolones. Duration of treatment depends on the virulence of the bacteria and factors concerning the host, and varies from weeks to months. Some cases of mediastinitis may need additional surgical drainage. Medical records revealed our patient had undergone an endoscopic examination several weeks before the appearance of the clinical symptoms and the mediastinal involvement and associated pleural empyema probably developed as consequences of that procedure. Thoracic drainage and penicillin G treatment cured the condition.

In conclusion, prior intubation or surgery of the airways and esophagus together with the possible oropharyngeal origin of the bacteria pathogen must be taken into account in respiratory infections in the presence of mediastinitis, pulmonary infiltrates, and pleural effusion, in order for appropriate treatment to be prescribed.

J. Ortiz de Saracho,^a S. Barbancho,^b and J.L. Mostaza^c

^aUnidad de Neumología, Hospital El Bierzo, Ponferrada, León, Spain.

^bMedicina Familiar y Comunitaria, Hospital El Bierzo, Ponferrada, León, Spain.

^cServicio de Medicina Interna, Hospital de León, León, Spain.

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Employment Prospects for Young Spanish Pneumologists in the Association of Pneumologists of the South (NEUMOSUR)

To the editor: Several studies have looked into the situation of pneumologist in Spain,¹ and reflected on the future that awaits us.^{2,3} The Association of Pneumologists of the South (NEUMOSUR) has created the so-called "pneumological maps" to better understand the circumstances of our specialty.^{4,5} To look beyond those maps to find out more about the real situation of newly graduated pneumologists, the association cooperated in distributing a questionnaire by ordinary and electronic mail to specialists belonging to the Communities of Andalusia and Extremadura who finished their specialty training in 2000-2003. Questions sought information on when and where specialist studies were completed, present employment and when it started, and place and duration of employment. NEUMOSUR provided a list showing 51 pneumology residents who had trained over a period of 4 years covered by the study. Fifty questionnaires were returned (98.04% participation). Twenty-one respondents had never worked as pneumologists since finishing residency training, and 30 had worked under grants from various sources with a mean (SD) duration of 11.17(8.98) months. Twenty had worked temporarily for emergency services. Three had occasionally worked as primary care physicians. Two had worked only in private medicine. Three worked abroad (in Portugal), and 3 others had changed specialty (to anesthesiology and postoperative recovery care). Three pneumologists were unemployed at the time of the survey. Of the 29 who worked or had worked as pneumologists, only 11 had stable contracts as specialists. Thirty-one were working or had worked under temporary contracts or arrangements to take calls. A large percentage of pneumologists, therefore, do not practice their profession after obtaining a specialty degree, a situation pointed out some time ago by Rosell et al⁶ and one that they found to be worsening over time. We

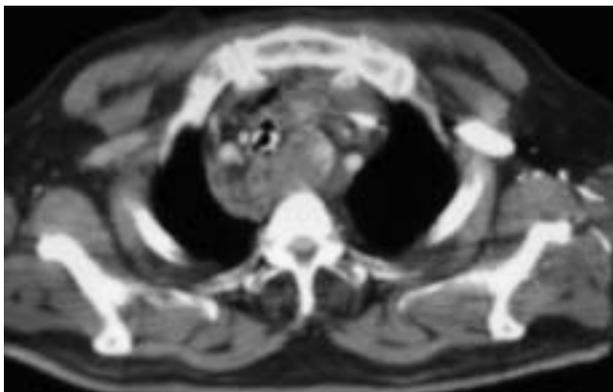


Figure. Thoracic computed tomography scan revealing mediastinal involvement.

consider that the percentage of pneumologists working under grants in our context is high at rates similar to the aforementioned study of the situation in Catalonia.⁶ Pneumologists in our society must put up with precarious contractual conditions, and the situation calls for our leaders to put greater pressure on public administrators and to insist on answering a question that has been posed before: Are there too many or too few pneumologists?³

J.M. Díez Piña

Servicio de Neumología,
Hospital Universitario Virgen de las Nieves,
Granada, Spain.

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Spontaneous Hemothorax Due to Rupture of Pulmonary Artery Aneurysm in Rendu-Osler-Weber Syndrome

To the Editor: Pulmonary arteriovenous malformations appear in 2 to 3 per 100 000 population. Hereditary hemorrhagic telangiectasia, or Rendu-Osler-Weber syndrome, is a hereditary autosomal dominant disorder frequently associated with such malformations.¹ About 95% of these malformations are found in pulmonary circulation and only about 5% are systemic.² Spontaneous hemothorax is a rare manifestation of the disease.

We report the case of a 36-year old woman, smoker of 16 pack-years, with aspirin intolerance and an otherwise unremarkable history, except for the fact that her father had recently been diagnosed with Rendu-Osler-Weber syndrome. She came to the emergency department complaining of dyspnea and right-sided pleuritic pain developing over the course of only a few hours. Upon arrival she was pale, with signs of central cyanosis, tachypnea (28 breaths/min), and difficulty breathing evidenced by use of accessory muscles. Baseline oxygen saturation was 80%, blood pressure, 95/50 mm Hg,

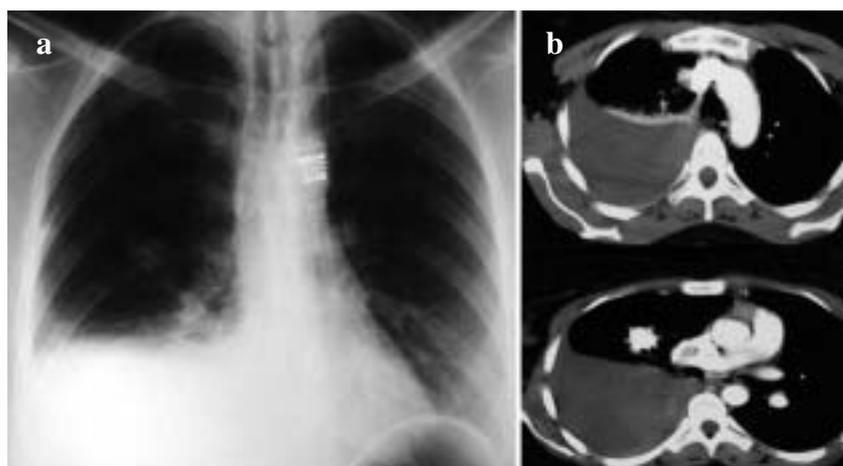


Figure 1. Chest x-ray showing right pleural effusion and increased density in the right parahilar region (a), and computed tomography of the chest showing an aneurysm of the right middle lobe artery and contrast in the right pleural cavity (b).

and temperature 36°C. On lung auscultation, vesicular sounds were absent and the others were diminished in the lower half of the right hemithorax. Complete blood count, biochemistry, and coagulation tests were normal.

A chest x-ray showed right pleural effusion and increased density in the right parahilar region (Figure 1a). Contrast-enhanced computed tomography of the chest (Figure 1b) showed a vascular malformation arising from the right pulmonary artery and contrast extravasation from the malformation into the right pleural cavity.

Selective pulmonary arteriography showed a saccular dilation of the interlobar portion of the right middle lobe artery. This was interpreted as an aneurysmal malformation that could not be embolized because of the high rate of blood flow in the artery. In view of the patient's hemodynamic instability and the characteristics of the aneurysm, an emergency thoracotomy was performed.

During surgery a hemothorax occupying nearly the entire chest cavity and an aneurysm of the interlobar portion of the right middle lobe artery were detected. The aneurysm was dissected and ligated, with atypical resection of the middle lobe without section of the venous branches.

The patient progressed satisfactorily during the postoperative period, with no further hemorrhages or complications, and was discharged on the seventh day following surgery.

Spontaneous hemothorax without pneumothorax is an uncommon entity of which hemorrhages due to pulmonary arteriovenous malformations are an unusual cause.³ The most serious clinical manifestations of Rendu-Osler-Weber syndrome are paradoxical embolisms having repercussions on the central nervous system, massive hemoptysis, and hemothorax.^{2,4} Arteriovenous malformations are usually diagnosed by computed tomography, and arteriography is a necessary diagnostic and therapeutic tool.⁴ Currently, angiographic embolization techniques seem to be replacing surgery, as they are associated with lower rates of morbidity and mortality and allow lung function to be preserved.^{1,5} In some patients (19%-60%), however, residual shunts persisting after embolization contribute to continued hypoxemia, thus rendering

ineffective the attempt to preserve the parenchyma. Surgery remains the treatment of choice in large arteriovenous malformations requiring lung resection when embolization fails, when blood vessel rupture is suspected, when there is a high risk of hemorrhage due to the rupture of an aneurysm, and when there are recurrent hemorrhages.^{1,6}

P. Ausín Herrero,^a A. Gómez-Caro Andrés,^b and F.J. Moradiellos Díez^b

^aServicio de Neumología, Hospital Universitario 12 de Octubre, Madrid, Spain.

^bServicio de Cirugía Torácica, Hospital Universitario 12 de Octubre, Madrid, Spain.

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