Septic Pulmonary Embolism Ascribed to Periapical Periodontitis: A Case Report and Review of the Literature

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Abstract: Septic pulmonary embolism is a rare condition, the common sources of which are bacterial endocarditis and infected venous catheters. Septic pulmonary embolism ascribed to dental infection is extremely rare. The present report describes a case of septic pulmonary embolism ascribed to periapical periodontitis in a 39-year-old patient, who was admitted with persistent toothache, fever, and chest pain. Chest CT showed a feeding vessel sign and a target sign. A panoramic radiograph of the left maxillary bone showed alveolar bone loss in a horizontal pattern typical of periodontitis and periapical periodontitis. The patient was treated with intravenous antibiotics and teeth extraction. To our knowledge, only 4 cases of septic pulmonary embolism ascribed to dental infection have been reported in the English literature. These patients repeatedly developed dental infections but did not receive appropriate dental treatment including extraction of the causative teeth, leading to the development of septic pulmonary embolism. This paper may contribute to the limited clinical knowledge base regarding septic pulmonary embolism ascribed to periapical periodontitis. Effective oral care and removal of the primary source of infection, including extraction of the causative teeth, are important to prevent septic pulmonary embolism.

Key words: Septic pulmonary embolism, Multiple peripheral nodules, Periapical periodontitis, Streptococcus milleri

Introduction

Septic pulmonary embolism is a rare condition, with the most common sources being tricuspid valve endocarditis and the peripheral veins. Less common sources of septic pulmonary emboli include thrombophlebitis of the pelvis, postanginal sepsis, and infected central venous catheters1–3. Septic pulmonary embolism ascribed to dental infection is an extremely rare occurrence. To our knowledge, only 4 cases of septic pulmonary embolism ascribed to dental infection have been reported in the English literature4–6. The present report describes a case of septic pulmonary embolism attributable to periapical periodontitis.
Report of a case

A 39-year-old male presented to the Department of Internal Medicine in our hospital with fever and chest pain on June 30, 2003. The patient was referred to the Department of Oral and Maxillofacial Surgery on July 4. He had been well until 3 years previously when he developed toothache and was treated with antibiotics for several days. Six months before the patient presented to us, he developed toothache and swelling of the left maxillary gingiva and recurrent toothache and swelling every 1 to 2 months thereafter. On June 20, 2003, the patient had developed toothache and swelling of the same region and had taken antibiotics (cefcapene pivoxil hydrochloride). On June 22, he developed fever and chills. Two days later, he experienced right-sided chest pain and left-sided backache.

At the time of first presentation, the patient was febrile (38.5℃). Chest X-ray showed multiple nodular shadows. A chest CT image through the lung bases showed a target sign and a patchy pleural-based area of consolidation with a feeding vessel sign (Fig. 1). Peripheral blood examination was as follows: C-reactive protein, 13.81 mg/dL; white blood cells (WBC), 12,000/μL (70% neutrophils, 22 lymphocytes, 5% monocytes, 2% eosinophils, and 1% basophils); and erythrocyte sedimentation rate, 82 mm/h. Arterial blood gas analysis in room air yielded normal values: pH, 7.43; arterial oxygen tension, 96.4 mmHg; and arterial carbon dioxide tension, 38.4 mmHg. Septum culture yielded Neisseria sp. and α-haemolytic Streptococcus sp. Septum cultures were negative for tuberculosis and acid-fast bacillus. Repeated blood cultures were negative. Intraoral examination showed toothache and swelling of the left maxillary gingiva. A panoramic radiograph of the left maxillary bone showed alveolar bone loss in a horizontal and vertical pattern typical of periodontitis and periapical periodontitis (Fig. 2). Pus from the left maxillary gingiva yielded Neisseria sp. and α-haemolytic Streptococcus sp. No potential sources of septic pulmonary embolism were present except for the dental infection.

The patient was treated initially with gatifloxacin hydrate (200 mg/day) and levofloxacin (400 mg/day) before admission. However, the fever continued to rise, therefore the patient was treated with biapenem (600 mg/day) administered intravenously. On the sixth hospital day, laboratory examination showed a WBC count of 5,600/μL and a C-reactive protein concentration of 1.29 mg/dL. The patient underwent extraction of the left maxillary first and second molars. On July 15, the patient was discharged. In the subsequent 3 years of follow-up, the patient has remained asymptomatic and clinically stable.

Discussion

The diagnostic criteria of septic pulmonary embolism include recurrent, persistent dental infection of the same region; preceding symptoms of dental infection such as toothache, gingival swelling, and pain; and subsequent high fever, cough, chest pain, and respiratory failure⁴⁻⁶.
Imaging diagnosis by CT is useful, because it presents characteristic findings such as feeding vessels and target signs. When the above findings are present, and no source of infection other than dental infection is identified by a thorough systemic examination, septic pulmonary embolism associated with dental infection should be suspected. The oral indigenous *S. milleri* group has been reported as an etiologic agent of septic pulmonary embolism. There are 3 species within the *S. milleri* group: *S. constellatus*, *S. intermedius*, and *S. auginous*. Oral indigenous bacteria were the etiologic agents in about half the pneumonia cases, and 13% of these reportedly belonged to the *S. milleri* group. However, the *S. milleri* group is not frequently identified on culture tests in septic pulmonary embolism cases, which may be due to the fact that antibiotic treatment is previously given in many cases and detection by the standard culture test is difficult because the bacteria are microaerophilic. The patient described in the present report experienced recurrent swelling and pain in the left maxillary molar region for several years. Swelling and pain in the same region occurred at the time of onset, and later, high fever, chest pain, cough, and respiratory failure developed. No typical hard wedge-shaped shadows representing multiple nodules (feeding vessel sign), target sign, or atelectasis were noted on chest CT, but patchy shadows were present. On bacterial examination, only oral indigenous bacteria such as *Neisseria sp.* and α-haemolytic *Streptococcus sp.* were detected in sputum and the pus-draining region in the mouth. Both specimens were negative on anaerobic culture. Blood culture was also negative, which may have been due to the antibiotics that the patient was administered before admission. On CT, pulmonary lesions included peripheral nodules accompanying vascular inflow, while no chronic pulmonary lesion or aspiration was present, suggesting that although an airway infection was not present, a hematogenous infection was likely. On close examination of the patient’s entire body, there was no other potential source of infection other than the dental infection. Based on these findings, this case is assumed to represent septic pulmonary embolism caused by dental infection. Septic pulmonary embolism associated with dental lesions, such as periodontal disease and periapical periodontal disease, is considered extremely rare, but it has been reported that many patients with septic pulmonary embolism have dental problems. The importance of oral care has been increasingly recognized, but its practice is still insufficient. In the previously reported cases, few patients had underlying diseases, such as diabetes and heart disease, and septic pulmonary embolism subsequently developed in those who showed recurrent dental infection but were treated only with antibiotics for inflammation without specific periodontal treatment or tooth extraction. The course of the present case was similar. If the causative teeth had been appropriately treated in the patients described previously, septic pulmonary embolism would likely have been avoided in most cases. For prevention, in addition to the importance of oral care, removal of the source of infection (extraction of the causative teeth) may be most effective. Since treatment could be con-

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<th>Ref.</th>
<th>Age/Sex</th>
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<th>Treatment</th>
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<td>Christensen et al.</td>
<td>56/M</td>
<td>-</td>
<td><em>S. intermedius</em></td>
<td>Negative</td>
<td>Mandible and maxilla</td>
<td>Antibiotics</td>
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<td>Russi et al.</td>
<td>50/M</td>
<td>Osler-Weber-Rendu Disease</td>
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<td>Negative</td>
<td>Maxilla</td>
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<td>Shiota et al.</td>
<td>53/M</td>
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<td>Negative</td>
<td>Negative</td>
<td>Right mandibular first molar</td>
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<td>Shiota et al.</td>
<td>67/M</td>
<td>Bronchial asthma</td>
<td>Negative</td>
<td>Negative</td>
<td>Mandible and maxilla</td>
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ducted without identifying dental disease as the cause of septic pulmonary embolism due to a lack of recognition by physicians in many cases, it should be kept in mind during both the diagnosis and treatment that dental disease may cause such a condition.

References