Septic Pulmonary Embolism*

Presenting Features and Clinical Course of 14 Patients

Rachel J. Cook, MD; Rendell W. Ashton, MD; Gregory L. Aughenbaugh, MD; and Jay H. Ryu, MD

Background: Septic pulmonary embolism (SPE) is an uncommon disorder with an insidious onset and is difficult to diagnose.
Study objectives: To characterize the presenting features and clinical course of patients with SPE.
Design: Retrospective study.
Setting: Tertiary care, referral medical center.
Patients: Fourteen subjects with SPE diagnosed during a 6-year period between 1996 and 2002.
Interventions: None.

Results: The median age of these patients was 37.5 years (range, 14 to 81 years) and included five women. Presenting symptoms included fever (93%), dyspnea (36%), pleuritic chest pain (29%), cough (14%), and hemoptysis (7%). The median duration of symptoms before diagnosis was 18 days (range, 5 to 180 days). A potential source or underlying condition that predisposed to SPE was identified in all 14 patients and included Lemierre syndrome (4 patients), central venous catheter infection (3 patients), prosthetic cardiac valve (2 patients), and pacemaker infection (2 patients). Two patients had a focal extrapulmonary infection, and one patient was an IV drug user. Most common pathogens were staphylococcal species (eight patients) and fusobacterium (four patients). Chest radiographic presentation was usually nonspecific, but CT was more helpful and revealed multiple nodular opacities peripherally, often with cavitation. Transesophageal echocardiography was performed in eight patients and demonstrated infectious vegetations in four cases. Aside from antimicrobial therapy and removal of infected devices, the management of these patients included cardiac surgery (two patients), thoracoscopic surgery with decortication (one patient), and tube thoracostomy (one patient). All 14 patients recovered from their illness.

Conclusions: We conclude that SPE presents with variable and often nonspecific clinical and radiographic features. The diagnosis is usually suggested by the presence of a predisposing factor, febrile illness, and CT findings of multiple, nodular lung infiltrates peripherally, with or without cavitation.

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Key words: chest CT; infection; lung abscess; pulmonary embolism infection

Abbreviations: CXR = chest radiograph; SPE = septic pulmonary embolism

S eptic pulmonary embolism (SPE) is an uncommon disorder that generally presents with an insidious onset of fever, respiratory symptoms, and lung infiltrates.1,2 Clinical and radiologic features at presentation are usually nonspecific, and the diagnosis of this disorder is frequently delayed. Historically, SPE has been associated with risk factors such as IV drug use, pelvic thrombophlebitis, and suppurative processes in the head and neck.3–5 However, increasing use of indwelling catheters and devices as well as increasing numbers of immunocompromised patients have changed the epidemiology and clinical manifestations of SPE.1,2,6–9

In SPE, the embolic blood clot that leads to an infarction in the pulmonary vasculature also contains microorganisms that incite a focal abscess. Chest radiography may reveal poorly marginated peripheral lung nodules that have a tendency to cavitate but are more often nonspecific in appearance.1,10 CT of the chest can be more helpful in demonstrating peripheral cavitory lesions.1,10 The purpose of this report is to define the epidemiologic, clinical, radiologic, and microbiologic features as well as clinical course in 14 patients with SPE to identify

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features that may facilitate the recognition and diagnosis of this uncommon disorder.

**Materials and Methods**

Using a computer-assisted search, we identified 14 cases of SPE occurring over a 6-year period from July 1, 1996, to June 30, 2002, at the Mayo Clinic in Rochester, MN. We reviewed the medical records and radiologic images of these cases and abstracted the following information: age, sex, and symptoms at presentation, physical examination, and laboratory findings including microbiologic culture results, radiologic findings, echocardiography results, treatment, comorbid medical conditions, and outcome. Microbiologic samples were collected and processed according to well-established, published guidelines.11

Case definition of SPE included the following: (1) focal or multifocal lung infiltrates compatible with septic embolism to the lung, (2) presence of active extrapulmonary infection as potential embolic source, (3) exclusion of other potential explanation for lung infiltrates, and (4) resolution of lung infiltrates with appropriate antimicrobial therapy. This study was approved by the Mayo Foundation Institutional Review Board.

**Results**

The study population consisted of 14 patients, including 5 women (Table 1). The median age of these patients was 37.5 years (range, 14 to 81 years). Thirteen of these patients (93%) were hospitalized at the time of diagnosis.

Presenting symptoms included fever (93%), dyspnea (36%), pleuritic chest pain (29%), sore throat (21%), cough (14%), and hemoptysis (7%). Four patients (29%) described fatigue or malaise. The median duration of symptoms before diagnosis of SPE was 18 days (range, 5 to 180 days). The median duration of hospitalization before the diagnosis of SPE was 3 days (range, 0 to 15 days).

A potential source or predisposing condition for SPE was identified in all 14 patients, including Lemierre syndrome in 4 patients (29%), central venous catheter infection in 3 patients (21%), prosthetic cardiac valve endocarditis in 2 patients (14%),

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, yr/Sex</th>
<th>Cause</th>
<th>Pathogens</th>
<th>Culture Source</th>
<th>Procedures</th>
<th>Hospital Stay, d</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>18/F</td>
<td>Lemierre syndrome</td>
<td>Fusobacterium, <em>arcanobacterium</em></td>
<td>Blood</td>
<td>Thoracentesis</td>
<td>14</td>
</tr>
<tr>
<td>2</td>
<td>21/M</td>
<td>Lemierre syndrome</td>
<td>Fusobacterium</td>
<td>Blood, pleural fluid</td>
<td>Tube thoracostomy</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>18/F</td>
<td>Lemierre syndrome</td>
<td>Fusobacterium</td>
<td>Blood, maxillary sinus aspirate</td>
<td>Thoracentesis</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>19/M</td>
<td>Lemierre syndrome</td>
<td>Fusobacterium, coagulase-negative Staphylococcus, a Gram-negative anaerobe</td>
<td>Blood</td>
<td>Thoracentesis</td>
<td>17</td>
</tr>
<tr>
<td>5</td>
<td>49/M</td>
<td>Infected central venous catheter</td>
<td>Methicillin-resistant <em>Staphylococcus aureus</em></td>
<td>Blood</td>
<td>Central venous catheter removed</td>
<td>44</td>
</tr>
<tr>
<td>6</td>
<td>40/F</td>
<td>Infected central venous catheter</td>
<td>Oxacillin-sensitive <em>S aureus</em></td>
<td>Blood, pleural fluid, lung biopsy</td>
<td>Central venous catheter removed; thoracoscopic decortication, tricuspid and mitral valve replacement</td>
<td>97</td>
</tr>
<tr>
<td>7</td>
<td>53/M</td>
<td>Infected central venous catheter</td>
<td>Corynebacterium, Klebsiella oxytoca, Coagulase-negative Staphylococcus</td>
<td>Blood, central intravenous catheter</td>
<td>Central venous catheter removed</td>
<td>9</td>
</tr>
<tr>
<td>8</td>
<td>35/F</td>
<td>Prosthetic pulmonary valve endocarditis</td>
<td><em>S aureus</em></td>
<td>Blood</td>
<td>Mechanical ventilation, bronchoscopy</td>
<td>44</td>
</tr>
<tr>
<td>9</td>
<td>14/M</td>
<td>Prosthetic pulmonary valve endocarditis</td>
<td><em>Streptococcus pneumoniae</em></td>
<td>Blood</td>
<td>Reconstruction of right ventricular outflow tract</td>
<td>26</td>
</tr>
<tr>
<td>10</td>
<td>81/M</td>
<td>Infected pacemaker</td>
<td>Coagulase-negative Staphylococcus</td>
<td>Blood</td>
<td>Pacing system removed</td>
<td>15</td>
</tr>
<tr>
<td>11</td>
<td>51/M</td>
<td>Infected pacemaker lead wires</td>
<td>Coagulase-negative Staphylococcus</td>
<td>Blood, pacemaker leads</td>
<td>Pacing system removed</td>
<td>17</td>
</tr>
<tr>
<td>12</td>
<td>65/M</td>
<td>Dental abscess</td>
<td>Not isolated</td>
<td>NA</td>
<td>Bronchoscopy</td>
<td>0</td>
</tr>
<tr>
<td>13</td>
<td>64/F</td>
<td>Perinephric abscess</td>
<td>Ampicillin-resistant <em>Escheria coli</em>, lactobacillus</td>
<td>Blood, perinephric fluid</td>
<td>Percutaneous drainage of perinephric abscess</td>
<td>11</td>
</tr>
<tr>
<td>14</td>
<td>22/M</td>
<td>IV drug abuse</td>
<td>Methicillin-sensitive <em>S aureus</em></td>
<td>Blood</td>
<td>NA</td>
<td>7</td>
</tr>
</tbody>
</table>

*M = male; F = female; NA = not applicable.*
and infected pacemaker or lead wire in 2 patients (14%) [Table 1]. Dental and perinephric abscess were the embolic sources in one patient each. One remaining patient was an IV drug user. All three patients with central venous catheter infections were immunocompromised due to immunosuppressive therapy after liver transplantation (one patient), recent autologous stem-cell transplantation for acute myelogenous leukemia (one patient), and long-term corticosteroid therapy for ulcerative colitis (one patient).

Microbiologic studies yielded likely responsible pathogens in all but one of the cases (Table 1). These pathogens were isolated from the blood in all 13 patients; most common pathogens were staphylococcal species (8 patients) and fusobacterium (4 patients). Additional sources of positive culture findings included pleural fluid (two patients), lung biopsy (one patient), maxillary sinus aspirate (one patient), perinephric fluid (one patient), central venous catheter (one patient), and pacemaker leads (one patient). In the remaining patient with negative culture findings, bronchoscopic lung biopsy demonstrated acute and organizing pneumonia with features of botryomycosis.

Abnormalities were noted in all 13 patients in whom chest radiographs (CXRs) were available for current review and demonstrated patchy infiltrates and/or nodules in all cases; these were bilateral in 10 patients (Fig 1). Cavitation was apparent in three cases (23%). Unilateral or bilateral effusions were present in seven patients (54%), but no adenopathy was appreciated. One patient without a CXR had abnormalities noted on CT. None of the original CXR interpretations mentioned SPE in the differential diagnosis of radiographic findings.

CT studies demonstrated abnormalities in all 13 patients with studies available for current review (Table 2). Nodular opacities were seen in all 13 patients, with other nonnodular infiltrates also seen in 9 patients. Parenchymal opacities were bilateral in all 13 scans, and cavitation was demonstrated in 11 scans (85%) [Fig 2, top, A]. “Feeding vessels” were appreciated on approximately one half of CT examinations but were associated with a minority of the parenchymal lesions (Fig 2, bottom, B). Unilateral or bilateral pleural effusion was demonstrated in nine patients (69%), and hilar or mediastinal lymphadenopathy was noted in four patients (31%). Possible diagnosis of SPE had been mentioned in the original interpretations for 7 of the 13 scans (54%).

The majority of patients had both upper and lower lobe involvement demonstrated on CXR and/or CT study. Upper lobes of both lungs were involved in 10 of 14 patients (71%). Peripheral or subpleural zones were most commonly affected (92% by CT), but central lesions were also seen (23% by CT).

Transesophageal echocardiography was performed in eight patients (57%). Four of these studies demonstrated right-sided vegetations involving the tricuspid valve (two patients), prosthetic pulmonary valve (one patient), and pacemaker leads (one patient).

The diagnosis of SPE was derived primarily from the CT scan report when this diagnosis was mentioned as a possibility, as in seven of our patients. In the remaining seven patients, the diagnosis was reached by the clinicians (internists and consulting subspecialists) based on the combination of evolving pulmonary lesions in the context of persistent bacteremia, potential embolic source such as vegetations and septic thrombophlebitis, and extrapulmonary embolic manifestations (eg, emboli to the brain).

All but one patient received parenteral antimicrobial therapy; the remaining patient was treated with oral antimicrobial therapy only. The duration of total antimicrobial therapy ranged from 4 to 8 weeks. Two patients required cardiac surgery: tricuspid and mitral valve replacement in a patient with native valve endocarditis, and right ventricular outflow tract reconstruction in a patient with tetralogy of Fallot and

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**Table 2—CT Findings of SPE in 13 Patients**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bilateral abnormalities</strong></td>
<td>13 (100)</td>
</tr>
<tr>
<td>Nodular opacities</td>
<td>13 (100)</td>
</tr>
<tr>
<td>Nonnodular infiltrates</td>
<td>9 (69)</td>
</tr>
<tr>
<td>Cavitation</td>
<td>11 (85)</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>9 (69)</td>
</tr>
<tr>
<td>Lymphadenopathy</td>
<td>4 (31)</td>
</tr>
</tbody>
</table>

*CT of the chest was not available for current review in one patient.
prosthetic pulmonary valve endocarditis. One patient required tube thoracostomy for drainage of an infected pleural effusion, and another patient underwent thoracoscopic surgery for drainage of empyema and decortication. Thoracoscopic lung biopsy in the latter patient demonstrated infarcted lung parenchyma with microabscess formation and pleuritis. One additional patient experienced massive hemoptysis caused by septic infarctions in the lung and required mechanical ventilation for 7 days.

All patients recovered from their illness. The median duration of hospitalization was 14.5 days (range, 0 to 97 days). Follow-up CXR or CT scan demonstrated improvement following antimicrobial therapy. There were no identifiable long-term complications resulting from SPE. Median follow-up duration was 26 months (range, 2 to 61 months) for 12 patients; 2 patients were unavailable for follow-up after dismissal from the hospital.

Discussion

SPE is an uncommon but serious disorder that is difficult to recognize. Our results demonstrate that the characteristic features of SPE are a febrile illness and lung infiltrates associated with an active focus of extrapulmonary infection, often involving indwelling catheters or devices. Three of our patients were immunocompromised. Although the abnormalities detected by CXR are nonspecific, CT scanning demonstrates bilateral nodules or multifocal infiltrates, commonly involving peripheral lung zones, often associated with cavitiation.

In 1978, MacMillan and colleagues described 60 patients with SPE encountered over a 5-year period. Drug users comprised 78% of their cohort, and tricuspid endocarditis was the embolic source in 53% of the cases. Twelve of their patients (20%) required thoracotomy for management of lung abscess, bronchopleural fistula, and empyema. One of these surgically treated patients (8%) died, while seven deaths (15%) occurred in the medically treated group. Since this report, there have been relatively few published studies of SPE, with most of the available literature consisting of isolated case reports.

Results of our study indicate that the epidemiology and outcome of patients with SPE have changed over the past 30 years. Most of our cases were related to infected intravascular devices or catheters and soft-tissue infections. Only one of our patients was an IV drug user, although this may partly reflect the characteristics of the referral population to our medical center. However, other authors have observed that SPE has become an uncommon complication of IV drug use, presumably due to greater awareness of needle hygiene.6 Our study also demonstrates improved outcomes for patients with SPE with all patients recovering from their illness. This may be attributable, in part, to earlier diagnosis, more effective antimicrobial therapy, improvements in surgical management, and better supportive care.

As the use of intravascular catheters and intracardiac devices has increased, the incidence of infections related to these appliances has also increased.6–9 Accordingly, infections related to these devices are responsible for an increasing portion of cases of SPE, as seen in our study.

Lemierre syndrome was initially described in 1936 and represents anaerobic thrombophlebitis of the internal jugular vein with metastatic infection.12 Eighteen of 20 patients in this initial report died of their illness. As seen in our cohort, most of the patients affected with this illness are adolescents or young adults in whom the inciting infection is tonsillopharyngitis, odontogenic infection, mastoiditis, or sinusitis. Extension of the infection to the adjacent...
lateral pharyngeal tissue containing the internal jugular vein results in the characteristic manifestations. Vascular involvement is followed by hemogenous spread to other organs, most commonly the lungs, causing metastatic abscesses. Pulmonary involvement in Lemierre syndrome has been reported in up to 97% of cases and has included SPE with lung abscesses, pleural effusion, empyema, and pneumothorax. In an adolescent or young adult presenting with sore throat or neck pain, fever, and lung infiltrates, the possibility of Lemierre syndrome should be considered.

Although findings on CXR tend to be nonspecific, CT may yield helpful clues that may suggest the diagnosis of SPE. Parenchymal lesions related to SPE are usually multiple and nodular with a peripheral distribution and a tendency for cavitation. These features associated with an extrapulmonary focus of infection should lead to consideration of SPE as the cause. Although other authors have described a “feeding vessel” sign (a vessel leading to a peripheral lung lesion) as a characteristic feature of SPE, we were able to identify this feature associated with only a minority of parenchymal lesions and did not find it particularly helpful in the recognition of SPE.

In patients with SPE who are suspected of having infective endocarditis, echocardiography is helpful not only in diagnosing the valvular infection but also in detecting complications such as valvular insufficiency or dehiscence, congestive heart failure, and paravalvular abscesses. A transesophageal approach provides greater spatial resolution compared to transthoracic imaging and is a superior method for imaging small vegetations, abscesses, and leaflet perforations that are < 5 mm in size. Diagnostic accuracy is also better for transesophageal echocardiography in cases of prosthetic valve endocarditis.

SPE will continue to pose a diagnostic challenge. Definitive criteria for the diagnosis of SPE are difficult to formulate since histopathologic confirmation of this diagnosis has been uncommon in previous studies or in clinical practice. The case definition used for identifying patients with SPE in the current study was based on diagnostic criteria used by other authors and our own experience. The recognition of SPE relies heavily on the presence of typical radiologic features, particularly on CT, but it is possible that SPE may be associated with a broader spectrum of radiologic findings than what is currently believed. Our study has demonstrated potential clinical contexts in which SPE may occur in the current era as well as characteristic clinical and radiologic features at presentation. Blood cultures, CT of the chest, and echocardiography are invaluable in the evaluation of a patient with suspected SPE. With early diagnosis, appropriate antimicrobial therapy, and control of the infectious source, resolution of the illness can be expected for most patients with avoidance of potential complications.

REFERENCES
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