**Streptococcus suis** Infection Complicated by Purpura Fulminans and Rhabdomyolysis: Case Report and Review

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*Clinical Infectious Diseases* 1997; 24:710–718

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Clinical Infectious Diseases 1997;24:710–2
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1058-4838/97/2404-0022$02.00

"Streptococcus suis" infection, an important zoonotic occupational disease in humans, is associated with meningitis, arthritis, and perceptive deafness. We report a case of severe *S. suis* infection in a previously healthy man who developed purpura fulminans and rhabdomyolysis, complications not previously reported with this disease.

Case Report

A 53-year-old male butcher presented to the hospital with a 2-day history of fever, chills, and rigors as well as diarrhea, vomiting, numbness in both feet, and an erythematous rash on his face, trunk, and hands. His medical history was significant only for an episode of pulmonary tuberculosis, for which he had completed a full course of therapy 5 years before admission. Physical examination revealed stable vital signs as well as diffuse erythema with petechiae and mottled blue extremities. Findings of his cardiorespiratory and abdominal examinations were unremarkable. He had no meningism, and there were no focal findings on the neurological examination. He had no lymphadenopathy.

Laboratory investigations on admission revealed a hemoglobin level of 170 g/L; a WBC count of 22 × 10⁹/L with toxic vacuolation, a left shift, and 90% polymorphonuclear cells; and a platelet count of 15 × 10⁹/L. Screening for disseminated intravascular coagulation was positive with a D-dimer of >2,000 ng/mL; soluble monomer was present, the partial thromboplastin time was 58.2 seconds, and the prothrombin time was prolonged (20.6 seconds). The patient had elevated levels of blood urea nitrogen (46.8 mg/dL) and creatinine (3.0 mg/dL).

Three hours after admission, the patient developed bilateral sensorineural hearing loss. He remained hemodynamically stable but was transferred to the intensive care unit for closer monitoring. Blood samples were taken for culture. The next day, both sets of blood cultures yielded β-hemolytic streptococci that were susceptible to penicillin, ampicillin, cephalaxin, erythromycin, and co-trimoxazole.

The organism was subsequently identified as *S. suis* serotype II by substrate utilization tests with use of the API 20 Strep Identification Strip (bioMérieux, Marcy l’Etoile, France) supplemented with conventional sugar fermentation reactions. The following characteristic features of *S. suis* were observed: growth on nutrient agar and on MacConkey agar; failure to grow in 6.5% sodium chloride; positive biochemical reactions with arginine, glucose, lactose, maltose, salicin, and sucrose and trehalose; and negative biochemical reactions with arabinose, mannitol, raffinose, and sorbitol.

The patient was treated with 24 million units of penicillin G and 240 mg of gentamicin daily. His condition improved and his fever resolved, but he developed features of purpura fulminans, with ecchymoses and bullae spreading over his limbs and trunk. There were areas of necrosis, and he developed gangrene in the toes of both feet.

All other investigations were negative; findings on CT and MRI scans of his head failed to reveal any evidence of meningeal inflammation or collections of fluid. A lumbar puncture was also negative (protein and glucose levels were normal, and no cells were found). Rhabdomyolysis was noted; the levels of creatinine phosphokinase (7,531 U/L, 100% muscle) and aldolase (46.9 U/L; normal range, 0–7.6 U/L) were elevated, and the urine and serum were positive for myoglobin.

Therapy with iv crystalline penicillin and gentamicin was continued for 6 weeks. He also developed reactive thrombocytosis during the recovery phase, with platelet counts reaching 1,000 × 10⁹/L. He had complete renal recovery; he was well when he was discharged from the hospital (he had only residual bilateral hearing loss and bilaterally amputated toes).

Discussion

*S. suis* was first described as an important pathogen in both pigs and humans in the 1960s in Scandinavia [2]. Most of the subsequent cases of *S. suis* infection have been reported from Western Europe [3], and *S. suis* is reportedly a major cause of meningitis in Hong Kong [4]; however, to our knowledge, we...
Table 1. Summary of data from 101 cases of *Streptococcus suis* infection.

<table>
<thead>
<tr>
<th>Reference</th>
<th>No. of cases of <em>S. suis</em> infection</th>
<th>No. who had contact with pigs or handled pork</th>
<th>No. with hearing loss</th>
<th>Skin manifestations</th>
<th>Other manifestations</th>
<th>No. of survivors</th>
</tr>
</thead>
<tbody>
<tr>
<td>[3]</td>
<td>60</td>
<td>53</td>
<td>33</td>
<td>Petechiae, gangrene</td>
<td>Arthritis, endophthalmitis, uveitis, macular bleeding, neuritis, pneumonia, respiratory distress, shock, relapse</td>
<td>56</td>
</tr>
<tr>
<td>[7]</td>
<td>25</td>
<td>15</td>
<td>16</td>
<td>None</td>
<td>Arthritis, diarrhea, endocarditis, sinusitis, pneumonia, shock</td>
<td>24</td>
</tr>
<tr>
<td>[8]</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>None</td>
<td>None</td>
<td>2</td>
</tr>
<tr>
<td>[9]</td>
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<td>1</td>
<td>0</td>
<td>Arteritic lesions</td>
<td>Endocarditis</td>
<td>1</td>
</tr>
<tr>
<td>[10]</td>
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<td>None</td>
<td>Pneumonia</td>
<td>0</td>
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<tr>
<td>[11]</td>
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<td>1</td>
<td>0</td>
<td>Ecchymoses</td>
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<td>None</td>
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<td>None</td>
<td>Endocarditis, arthritis</td>
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</tr>
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<td>1</td>
<td>None</td>
<td>None</td>
<td>1</td>
</tr>
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<td>[5]</td>
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<td>Central ophthalmoplegia</td>
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<td>Endophthalmitis, uveitis</td>
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<td>None</td>
<td>1</td>
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<tr>
<td>[20]</td>
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<td>Pneumonia, arthritis</td>
<td>1</td>
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</tbody>
</table>

NOTE. NS = not stated.

report the first case of human *S. suis* bacteremia in Singapore or Malaysia. *S. suis* infection has been strongly associated with intensive contact with pigs or pork [3], and, as our patient was a butcher, he had such contact; it has been suggested that people who are in daily contact with pork and pigs minimize skin trauma and use protective handwear, as the skin is thought to be the main portal of entry of this bacterium [5].

*S. suis* infection in humans mainly causes meningoencephalitis, and early eighth cranial nerve damage manifested as hearing loss and/or vestibular dysfunction is often a prominent feature of the disease [6]. In our patient's case, this loss occurred barely 30 minutes after administration of gentamicin, too early to be aminoglycoside ototoxicity.

*S. suis* infections in humans were most recently reviewed in 1988 by Arends and Zanen [3]. Four of their sixty patients had petechiae, and one of these had gangrene of the fingers. Since then, there have been 17 reports (including 41 cases) of *S. suis* infection in the English-language literature, and none of these has featured patients with petechiae, purpura, gangrene, or major skin manifestations. Other clinical manifestations of *S. suis* infection have included endophthalmitis, pneumonia, diarrhea, and arthritis (table 1).

Purpura fulminans has been reported as a complication of overwhelming infection with *Neisseria meningitidis* or varicella and more recently of *Streptococcus pneumoniae* [21]. Although this condition occurs mainly in children, severe cases have been reported in adults, especially in splenectomized patients [22].

The pathogenesis of purpura fulminans with digital gangrene in severe streptococcal infections is unclear and thought to be related to the development of disseminated intravascular coagulation [21]. We did not determine our patient's protein C levels at the time that he developed purpura fulminans, although induced protein C deficiency has recently been reported as a cause of purpura fulminans in children with meningococcemia [23].

Rhabdomyolysis has been reported in association with a variety of infectious agents. Influenza virus and HIV are the most important viral agents associated with rhabdomyolysis, and *Legionella* species, *Streptococcus* species, and *Salmonella* species are the most common bacterial causes of this condition [24]. To our knowledge, we report the first case of *S. suis* infection causing rhabdomyolysis.

To date, *S. suis* infections have, with one exception [6], been susceptible to penicillin, which remains the drug of choice for treating this illness. Our patient was fortunate that his condition was diagnosed early and that he received prompt appropriate therapy that left him with only minor deficits from a potentially fatal infection.

References