**Enterococcus cecorum** Empyema Thoracis Successfully Treated with Cefotaxime

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We report the first case of *Enterococcus cecorum* empyema thoracis and spontaneous bacterial peritonitis in a 44-year-old man with underlying cirrhosis. The patient responded to cefotaxime (MIC, 0.25 μg/ml) treatment and drainage of the empyema. Susceptibility of *E. cecorum* to expanded-spectrum cephalosporins could be due to its production of types of penicillin-binding proteins similar to those produced by *Streptococcus* species rather than to those produced by *Enterococcus* species (as predicted by phylogenetic analysis of the 16S rRNA gene sequences).

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**CASE REPORT**

A 44-year-old Chinese man was admitted to a hospital because of fever, shortness of breath, and generalized abdominal pain for 2 days. He had Wilson’s disease complicated by cirrhosis, esophageal varices, portal hypertension, recurrent ascites, recurrent spontaneous bacterial peritonitis, right indirect inguinal hernia, and hemorrhoids. Examination showed an oral temperature of 37.7°C, right pleural effusion, and generalized abdominal tenderness and guarding but minimal ascites. Chest radiograph showed right pleural effusion. The hemoglobin level was 10.6 g/dl, the total white cell count was 13.7 × 10⁹/liter, and the platelet count was 73 × 10⁹/liter. The serum bilirubin level was 76 μmol/liter, the albumin level was 24 g/liter, the alkaline phosphatase level was 249 U/liter, and the alanine aminotransferase level was 40 U/liter. The serum urea and creatinine levels were 15.8 mmol/liter and 111 μmol/liter, respectively. The prothrombin time and activated partial thromboplastin time were 16.6 s (normal range, 11.3 to 13.2 s) and 55.8 s (normal range, 27.6 to 37.6 s), respectively. Blood culture results were negative. The Gram smearing of the pleural fluid revealed the presence of numerous polymorphs. Aerobic and anaerobic cultures of the pleural fluid yielded pure growth of a gram-positive coccus. Blood culture results were negative. The patient responded to cefotaxime treatment and adequate drainage of the empyema and was discharged after 10 days of hospitalization.

*Enterococcus cecorum*, one of the 30 species of the genus *Enterococcus*, was first described in 1983 as *Streptococcus cecorum* (3). Since then, it has been identified as part of the intestinal floras of various animals, including pigs, cattle, chickens, ducks, cats, dogs, and canaries (4). In contrast to the common recovery of *E. cecorum* from animals, human infections associated with *E. cecorum* have been rarely reported, with only three cases described in the English literature (MEDLINE search [1996 to 2003]) (2, 6, 7). Phenotypically, *E. cecorum* is often described as more *Streptococcus*-like than other *Enterococcus* species in that it prefers incubation in atmospheric conditions with CO₂ enrichment, fails to grow on *Enterococcus*-selective medium and in 6.5% NaCl, and grows poorly on bile-esculin agar (10). However, speciation of *E. cecorum* is difficult, especially when commercial kits are used. In the three cases of human infections, the three isolates were identified by whole-cell protein analysis, cellular fatty acid analysis, 16S rRNA gene sequencing, and/or tRNA gene PCR and capillary electrophoresis in addition to the use of conventional phenotypic tests. In this article, we describe the first case of empyema thoracis associated with *E. cecorum*. The epidemiology of *E. cecorum* infections, as well as its susceptibility to cefotaxime in relation to its phylogenetic position as deduced by 16S rRNA gene sequence analysis, are discussed.

**Clinical and microbiological data.** All clinical data were collected prospectively as described in a previous publication by Woo et al. (13). The bacterium was identified by standard conventional biochemical methods (9). Lancefield serogrouping was performed using Streptex (Murex Biotech Ltd., Dartford, United Kingdom). All tests were performed in triplicate with freshly prepared medium on separate occasions. In addition, a Vitek System (GPI) (bioMerieux Vitek, Hazelwood, Mo.), an API system (20 STREP) (bioMerieux Vitek), and an ATB expression system (ID32 STREP) were used for the iden-
The biochemical profile of the isolate is described at both 25 and 37°C. The isolate is nonmotile and resistant to optochin and bacitracin. It is nongroupable with Lancefield group A, B, C, D, F, or G antisera. It also grew well on chocolate and MacConkey agars and in pinpoint colonies after 24 h of incubation at 37°C.

**Antibiotic susceptibility.** Antimicrobial susceptibility was investigated by E-testing, and the results were interpreted according to National Committee for Clinical Laboratory Standards criteria, with *Staphylococcus aureus* (ATCC 29213) and *Streptococcus pneumoniae* (ATCC 49619) as controls. The MICs of penicillin, cefotaxime, and vancomycin were 0.094, 0.25, and 1.5 μg/ml, respectively.

**16S rRNA gene sequencing and phylogenetic characterization.** PCR amplification and DNA sequencing of the 16S rRNA gene of the isolate were performed as previously described (15, 16). LPWSS (5'-AGTTTGATCTCTGCTACCA-3') and LPW205 (5'-CCGGTACCCTGCTACCA-3') (Gibco BRL, Rockville, Md.) were used as the PCR and sequencing primers. *E. faecalis* (ATCC 29212) was used as a control. The sequences of the PCR products were compared with known GenBank 16S rRNA gene sequences by multiple sequence alignment using the CLUSTAL W program (11). Phylogenetic relationships of the isolate recovered from the patient with other *Enterococcus* species, other representative *Streptococcus* species (S. pyogenes, S. mitis, S. agalactiae, S. intermedius, S. mutans, and S. bovis), *Granulicatella adiacens*, and *Abiotrophia defectiva* were determined using PileUp and the neighbor-joining method with GrowTree (Genetics Computer Group, Inc.).

A total of 1,235 nucleotide positions of the 16S rRNA genes were included in the analysis. PCR of the 16S rRNA gene of the pleural fluid isolate showed a band at about 1,460 bp. There were two (0.1%) base differences between the 16S rRNA gene sequence of the isolate and that of *E. cecorum* (GenBank accession no. AF061009), 33 (2.3%) base differences between the 16S rRNA gene sequence of the isolate and that of *E. columbae* (GenBank accession no. Y18275), and 55 (3.8%) base differences between the 16S rRNA gene sequence of the isolate and that of *E. gallinarum* (GenBank accession no. AF277567), indicating that the isolate was a strain of *E. cecorum* (Fig. 1).

We report the first case of empyema thoracis caused by *E. cecorum*. The patient probably had concomitant spontaneous bacterial peritonitis, as he had abdominal pain, generalized abdominal tenderness, and guarding, although abdominal...
paracentesis was not performed because of minimal ascites. Including the present case, four cases of *E. cecorum* infections have been reported in the English literature (2, 6, 7). Three patients were males, and one was female. All four patients had underlying diseases. Three of them had cirrhosis of the liver, which seems to be a major underlying disease predisposing patients to *E. cecorum* infections. Two patients had bacteremia and two had peritonitis. Overall, one of the four patients died.

The portal of entry for *E. cecorum* in the present case, as well as in the cases of the other two patients with *E. cecorum* peritonitis, was probably the gastrointestinal tract. It is well known that decompensated liver cirrhosis with portal hypertension and ascites is associated with the presence of intestinal mucosal edema and local immunosuppression secondary to portal venous congestive vasculopathy. Therefore, the gastrointestinal tracts of patients with liver cirrhosis are often the primary site of infection or the portal of entry for extraintestinal infections. According to one recently published article on spontaneous bacterial empyema in cirrhotic patients, 13 of the 18 patients with positive cultures from pleural fluid had pathogens (*Escherichia coli* in eight patients, *Enterococcus* species in three, and *Klebsiella pneumoniae* in two) that likely originated from the gastrointestinal tract (17). None of these bacteria are conventional pathogens causing pneumonia, bone and joint infections, or skin and soft tissue infections. Interestingly, *Laribacter hongkongensis*, a recently described organism which has been recovered from fecal specimens of patients with gastroenteritis, was also originally discovered in the blood and empyema pus cultures of a patient with alcoholic liver cirrhosis (8, 14, 18).

Sensitivity of *E. cecorum* to expanded-spectrum cephalosporins could be due to its production of types of penicillin-binding proteins (PBPs) different from those produced by other *Enterococcus* species but similar to the PBPs produced by other *Streptococcus* species. Enterococci, as opposed to streptococci, are intrinsically resistant to the cephalosporins. This differential susceptibility to cephalosporins in these two closely related genera is related to the production of different types of PBPs.
of which PBP 5, the major PBP in \textit{E. faecalis} and \textit{E. faecium}, is demonstrated to have a low affinity to cephalosporins (1, 5, 12). However, in the present case and in another case reported in the literature (2), \textit{E. cecorum} has been noted to be susceptible to cefotaxime and ceftriaxone. We speculate that this unexpected finding was due to the presence of types of PBPs in \textit{E. cecorum} different from those found with \textit{E. faecalis} and \textit{E. faecium}. In fact, \textit{E. cecorum} and \textit{E. columbae}, with their 16S rRNA gene sequences making up the first branch in the phylogenetic tree among the \textit{Enterococcus} species (Fig. 1), are probably the ancestors of the other \textit{Enterococcus} species and are also more closely related to the \textit{Streptococcus} species than the other \textit{Enterococcus} species. This is in line with the more \textit{Streptococcus}-like culture characteristics of \textit{E. cecorum} (10). Therefore, it is not surprising that the PBPs of \textit{E. cecorum}, which render \textit{E. cecorum} susceptible to cefotaxime and ceftriaxone, might be closely related to those of the \textit{Streptococcus} species.

**Nucleotide sequence accession number.** The 16S rRNA gene sequence of the isolate has been deposited in GenBank (accession no. AY365054).

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**REFERENCES**