Purulent Pleurisy Caused by *Campylobacter lari*

B. Bruneau, L. Burc, C. Bizet, N. Lambert-Zechovsky, C. Branger

**Abstract** An 80-year-old debilitated patient developed purulent pleurisy caused by a *Campylobacter lari* isolate. The patient underwent surgical drainage and received antibiotic therapy with amoxicillin/clavulanic acid and ofloxacin. Antibiotic susceptibility data showed that the isolate was fully sensitive to clarithromycin, tetracycline, aminoglycosides, and ciprofloxacin. Imipenem and amoxicillin plus clavulanic acid were the most active β-lactam agents.

**Key words** *Campylobacter lari* · Pleurisy · Antibiotic therapy

**Introduction**

The organism known as *Campylobacter lari* was first described by Skirrow and Benjamin [1] in 1980 as a nalidixic acid-resistant, thermophilic campylobacter. This species has been isolated from humans and a variety of birds and animals [1-3]. Cases of human illness associated with this organism have been recognized infrequently [4-9]. Enteritis is the most common syndrome caused by *Campylobacter lari* both in healthy and in immunocompromised patients [4, 6, 9], and rare cases of systemic infection have been described [4, 6, 8]. We report a case of purulent pleurisy caused by *Campylobacter lari*. Detailed antibiotic susceptibility data for this isolate are also presented.

**Case Report**

An 80-year-old man with Willebrand disease, a history of alcoholism, and cardiac deficiency was admitted to the hospital in November 1995 with fever (38.5°C), chest and abdominal pain, productive cough, and subicterus. A chest radiograph and computed tomography scan showed a large pleural effusion on the left side. The peripheral leukocyte count was 19000/mm³ with 92% neutrophilic polymorhnuclear cells. Before therapy began, blood samples were cultured (Bioargos aer medium; Sanofi Pasteur, France) and pleural fluid was inoculated onto blood agar (incubated aerobically and anaerobically) and chocolate agar (incubated in 5% CO₂). After 72 h of incubation at 37°C, blood cultures were positive on Bioargos aer medium, and growth of tiny pinpoint colonies was present on blood agar incubated anaerobically and on chocolate agar. Gram-stained smears revealed spiral-shaped gram-negative bacilli. Reactions for catalase and oxydase were positive. The isolate was presumed to be *Campylobacter* spp.

The patient underwent thoracic drainage three times and intrapleural fibrinolysis with streptokinase twice. He was also treated intravenously with amoxicillin/clavulanic acid (CA) (1 g t.i.d.) and ofloxacin (200 mg b.i.d.) for five days. After the antimicrobial susceptibility of the isolate was determined, amoxicillin (1 g t.i.d.) was administered for 13 days. The patient became afebrile. Follow-up cultures of blood and pleural fluid remained negative for *Campylobacter* spp. The patient did not have diarrhea, and stool cultures (Campylosel; bioMérieux, France) incubated at 42°C were negative for *Campylobacter* spp. An abdominal sonogram showed only a dysmorphic liver. On day 19 of hospitalization, the patient became febrile (39°C) again and his condition deteriorated due to hepatocellular deficiency and bronchitic congestion. He died 46 days after admission.

The *Campylobacter* isolate grew at 37°C and 42°C, but not at 25°C, was resistant to nalidixic acid and cephalothin, and did not hydrolyze hippurate. It was also tolerant to 1.5% NaCl but not 3.5% NaCl [5, 10]. It was therefore identified as *Campylobacter lari*. The identification of the strain was subsequently confirmed by the Laboratoire des Identifications, Institut Pasteur, Paris, France (Dr. Kiredjian).
Since little was known about the antibiotic susceptibility of Campylobacter lari, the minimal inhibitory concentrations (MICs) of 23 antibiotics, three β-lactamase inhibitors, and amoxicillin/CA (2 μg/ml) (Table 1) were determined using an agar dilution method with Mueller-Hinton agar plates supplemented with 5% sheep blood (Sanofi Pasteur). Plates were inoculated with $10^5$ cfu per spot using a multipoint inoculator, and incubated for 48 h, at 37°C in a microaerophilic atmosphere. Staphylococcus aureus ATCC 25923 was included as a control strain. The MIC was defined as the lowest concentration of drug that completely inhibited visible growth.

Susceptibility breakpoints for each antimicrobial agent were determined according to the guidelines of the Antibiogram Committee of the French Society for Microbiology [11]. Two reference strains of Campylobacter lari (CIP 102722 type strain and CIP 102723) obtained from the Collection de Bacteries, Institut Pasteur, Paris, were also tested. Cefinase disks (bioMérieux) were used according to the manufacturer's recommendations for detection of β-lactamase. Staphylococcus aureus ATCC 25923 was used as a positive control and Staphylococcus aureus ATCC 29213 as a negative control. The test was positive when a red coloration developed in 30 min [12]. The reference strain, Campylobacter lari CIP 102722, was the only strain found to produce β-lactamase.

The patient's isolate and the two reference strains had antibiotic susceptibility patterns (Table 1) similar to those obtained in previous studies of Campylobacter jejuni and Campylobacter coli [13, 14]. The strains showed resistance to rifampin and piperacillin, resistance or moderate sensitivity to cephalosporins, moderate sensitivity to erythromycin, and sensitivity to amoxicillin/CA, imipenem, aminoglycosides, clarithromycin, and tetracycline. Clavulanic acid (2 μg/ml) significantly decreased the MICs of amoxicillin, whether the strain tested was positive or negative for β-lactamase production. The MICs of clavulanic acid, tazobactam, and sulbactam were similar to those obtained in previous studies of Campylobacter jejuni and Campylobacter coli [14]. The patient's isolate was sensitive to fluoroquinolones, whereas the two reference strains were resistant to these antibiotics.