Indwelling Device-Related Bacteremia Caused by Serum-Susceptible *Campylobacter coli*

PO-REN HSUEH,[1,2] LEE-JENE TENG,[1,3] PAN-CHYR YANG,[2] SHEN-WU HO,[1,3] AND KWEN-TAY LUH[1,2]*

Departments of Laboratory Medicine[1] and Internal Medicine,[2] National Taiwan University Hospital, and School of Medical Technology, National Taiwan University Medical College,[3] Taipei, Taiwan

Two isolates of serum-susceptible *Campylobacter coli* were recovered in a 7-day interval from blood from a patient with hepatocellular carcinoma and liver cirrhosis whose peritoneal-caval (Denver's) shunt malfunctioned. Identical random amplified polymorphic DNA fingerprints, cellular fatty acid chromatograms, and antibiograms of the two isolates indicate that *C. coli* has the ability to cause catheter-related bacteremia following its colonization of the catheter.

Campylobacter coli and *Campylobacter jejuni* are recognized as the leading bacterial causes of infectious diarrhea in humans (3, 16). Extraintestinal infections or bacteremia caused by *C. coli* are rare (3, 5, 12, 16). Serum susceptibility has been observed to inversely correlate with prolonged bacteremia or systemic infection in *C. jejuni* infections, except in immunocompromised hosts (4, 5). We report here two episodes of bacteremia due to a serum-susceptible strain of *C. coli* in a patient with liver cirrhosis and hepatocellular carcinoma who had a peritoneal-caval shunt implanted.

Case report. A 56-year-old man with liver cirrhosis and hepatocellular carcinoma was hospitalized; he had had a fever for 3 days and progressive abdominal fullness for 2 weeks. He had undergone a segmental hepatectomy and implantation of a peritoneal-caval (Denver's) shunt 5 years earlier. During the 3 years prior to admission, he had experienced several episodes of spontaneous bacterial peritonitis that were successfully treated with intravenous antibiotics. On admission to the hospital, he appeared pale and chronically ill. His temperature was 37.3°C. Physical examination revealed hepatosplenomegaly and signs compatible with peritonitis. An abdominal sonography disclosed liver cirrhosis and massive ascites. The ascitic fluid contained 100 leukocytes per mm³, 56% of which were lymphocytes. Bacterial and mycobacterial cultures were negative.

A clinical diagnosis of sepsis and dysfunction of peritoneal-caval shunt was made, and the patient was treated initially with cefmetazole (3 g/day [administered in three doses]). With this treatment, defervescence and rapid clinical improvement were observed by the third day after admission to the hospital. The patient underwent surgical manipulation of the shunt on the seventh day of his hospital stay. The valve between the peritoneal and venous limbs of the shunt was completely occluded with fibrin-like material. Unfortunately, the patient developed a chilly sensation several hours after the surgical manipulation; a temperature of 39.4°C accompanying shaking chillness developed the day after the operation. Two sets of blood cultures drawn at that time as well as those drawn on admission grew *C. coli*. A culture of the removed valve yielded the same organism (this isolate was not preserved for further study). Therapy was changed to intravenous ciprofloxacin (800 mg/day [adminis-

tered in two doses]) and netilmicin (300 mg/day [in two doses]) for 1 week and switched to oral ciprofloxacin (1 g/day [in two doses]) for an additional 10 days.

Microbiology. All three isolates were curved, oxidase-positive, gram-negative rods. Colonies on the CAMPY BAP (BBL Microbiology Systems, Cockeysville, Md.) were moist and spreading. They were negative for hippurate reaction, positive for catalase activity, and resistant to cephalothin (30-μg disk) and grew well in a microaerophilic atmosphere (5% O_2, 10% CO_2, and 85% N_2) at 42°C. The biochemical profiles of these isolates generated by the API CAMPY system (bioMerieux Vitek, Inc., Hazelwood, Mo.) were identical and were in agreement with those of *C. coli* (16).

The cellular fatty acid methyl esters of the two blood isolates were analyzed as previously described (9). As shown in Fig. 1, both isolates consisted of high percentages of hexadecanoic acid (16:0), octadecenoic acid (18:1 *w*7c), 19-carbon cyclopropane fatty acid (19:0 cyc), tetradecanoic acid (14:0), and 3-hydroxytetradecanoic acid (3-OH-14:0) and low percentages (≤3%) of hexadecenoic acid (16:1 *w*7c) and octadecanoic acid (18:0). The ratio of the relative percentages of 16:0 and 16:1 *w*7c was ≥4. The chromatograms of the two isolates both corresponded to the pattern for *C. coli* and exhibited the group B fatty acid profile of *Campylobacter* species designated by Lambert et al. (15).

Susceptibilities of the two blood isolates to cefametazole were determined by the disk diffusion method with a 30-μg cefmetazole disk. An additional 12 antimicrobial agents were tested by using the Etest (PDM Epsilometer; AB Biodisk, Solna, Sweden) on CAMPY BAP (BBL Microbiology Systems), and the results were read after 24 h of incubation at 37°C in a microaerophilic atmosphere (9, 16). Antibiograms of the two isolates were considered identical because Etest MICs of all antimicrobial agents tested were identical or within one MIC gradient discrepancy (Table 1). The two isolates were presumptively considered to be resistant to cefmetazole (diameters of inhibition zone, 8 and 10 mm) if we applied the cefmetazole zone diameter breakpoints of members of the family *Enterobacteriaceae* to *Campylobacter* species (17). Both isolates were β-lactamase positive, as indicated by a cefinase disk (BBL Microbiology Systems).

Serum susceptibility testing. The serum bactericidal test of the first blood isolate was performed as previously described (4). The isolate had 93% inhibition of growth 60 min after exposure to 10% normal pooled serum (from two men and two women who had not been treated with antibiotics 2 weeks prior

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* Corresponding author. Mailing address: Department of Laboratory Medicine, National Taiwan University Hospital, Number 7, Chung-Shan South Rd., Taipei, Taiwan. Phone: 886-3-3562150. Fax: 886-2-3224263.
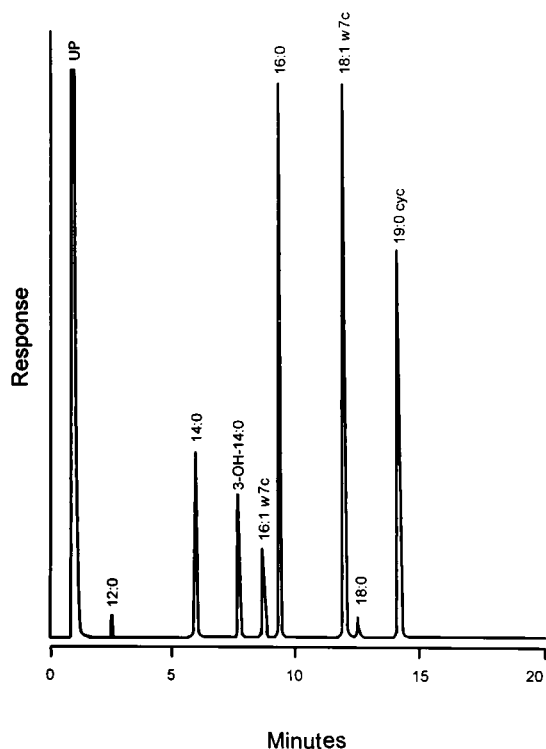
FIG. 1. Gas chromatogram of methylated fatty acids of *C. coli*. See the text for explanations of compound abbreviations and designations. UP, unknown peak.

to blood collection) and was considered to be serum susceptible (>90% inhibition) as defined by Blaser et al. (4).

Molecular typing. The procedure for random amplified polymorphic DNA (RAPD) assay of the two blood isolates, generated by arbitrarily primed PCR (APPCR), was followed as described previously (7, 8). The following five arbitrary oligonucleotide primers were used: M13 (5′-TTATGTAAAA CGACGGCCAGT-3′) and four 10-mer primers, i.e., OPA-9 (5′-GGGTAACGCC-3′), OPA-11 (5′-CAATCGCCGT-3′), OPA-13 (5′-CAGCACCCAC-3′), and OPB-12 (5′-CCTTGA CGCA-3′) (Operon Technologies, Inc., Alameda, Calif.). As shown in Fig. 2, the RAPD patterns found with the five primers of the two isolates were identical.

TABLE 1. Susceptibilities of two blood isolates of *C. coli* determined by the Etest

Antimicrobial agent	MIC (μg/ml) for isolate[a]:	
	C1	C2
Amoxicillin	1.0	0.75
Cephalothin	>256	>256
Cefoxitin	>256	>256
Cefotaxime	>32	>32
Ceftriaxone	>32	>32
Imipenem	0.094	0.125
Ciprofloxacin	0.032	0.064
Gentamicin	1.0	1.5
Minocycline	12	8
Erythromycin	>256	>256
Clarithromycin	>256	>256

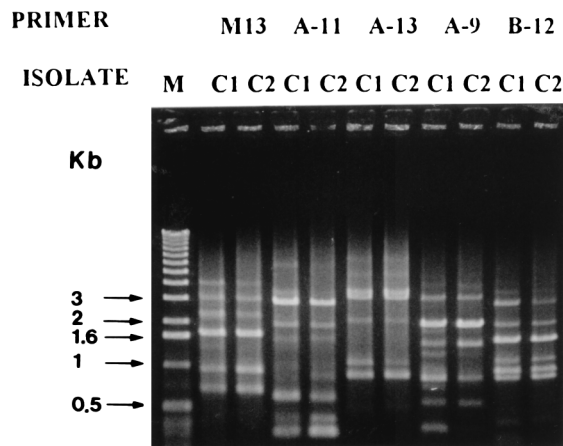[a] C1, first blood isolate; C2, second blood isolate.

FIG. 2. RAPD patterns of the two isolates of *C. coli* obtained with primers. The first blood isolate (C1) and second blood isolate (C2) of *C. coli* obtained with primers M13, OPA-9 (A-9), OPA-11 (A-11), OPA-13 (A-13), and OPB-12 (B-12) are shown. Molecular size markers (lane M) (1-kb ladder; Gibco BRL). Molecular sizes are indicated in kilobase pairs to the left of the gel.

In the present study, identical RAPD patterns and antiobiograms as well as the same *C. coli* cellular fatty acid chromatogram group for the two blood isolates indicate that both episodes of bacteremia were caused by a single clone of *C. coli*, which probably persisted in the shunt during the interval between the two episodes of bacteremia.

For *Campylobacter* species, 118 RAPD types analyzed by a random 10-mer primer (OPA-11) were found among 178 isolates studied by Hernandez et al. (7). The high genetic diversity of *Campylobacter* species revealed by the APPCR method has also been demonstrated by other molecular typing methods (6). The RAPD patterns of our two isolates of *C. coli* by five different primers were identical but did not belong to any RAPD profile group designated by Hernandez et al. (7).

Though antimicrobial susceptibility testing and the MIC breakpoints for susceptibility and resistance are not standardized for *Campylobacter* species (9), the Etest for *Campylobacter* species performed satisfactorily compared to the agar dilution method (2, 10). The results of susceptibility testing our isolates were in accord with the previous findings (13, 14, 18). Interestingly, our patient responded favorably to cefmetazole treatment initially, which might be inactive in vitro against the isolate. The serum susceptibility of the isolates may partly explain this phenomenon (4, 5), though the bactericidal activity of the patient's serum in response to the isolate and the immune status of the patient were not investigated.

The portal of entry of *C. coli* in our patient, as well as in most previous reports regarding bacteremia caused by *Campylobacter* species, is difficult to evaluate (1, 5). The patient had neither experienced a preceding gastrointestinal illness nor had septic thrombophlebitis before the bacteremic event (1, 5, 11). Bacteremia caused by *C. jejuni* and *C. coli* has been documented as the result of entry of these enteric pathogens into the bloodstream as a subpopulation of organisms (serum-resistant strains) or in a host with compromised immunity (1, 4, 5). It is possible that the bacteria seeded the fibrin-like material coating the valve of the shunt during the first episode of bacteremia and, in turn, reentered the bloodstream following surgical manipulation.

In summary, this report demonstrates the capacity of *C. coli* to cause catheter-related bacteremia following its colonization of the catheter and emphasizes that this organism should be

included in the differential diagnosis of bacteremia associated with the use of indwelling devices.

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