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Enterobacter taylorae, a New Opportunistic Pathogen: Report of Four Cases

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Severe nosocomial infections due to *Enterobacter taylorae* (formerly known as CDC Enteric Group 19) are described in four patients. Unlike most members of the *Enterobacter* genus, the isolates were not susceptible to penicillins or cephalosporins. Restriction endonuclease analysis of *E. taylorae* DNA obtained from three patients identified two distinct strains. One strain was found in two patients, suggesting a common source which we were not able to identify. We postulate that in patients harboring *E. taylorae*, the combination of cephalosporin therapy and instrumentation enables this organism to become an opportunistic pathogen.

Enterobacter taylorae (formerly known as CDC Enteric Group 19) has been identified only recently as a separate species (11). Little is known regarding the epidemiology and clinical significance of this organism. To date, there have been three case reports detailing the clinical disease associated with *E. taylorae*, all describing localized infection: osteomyelitis after an open fracture (22), a wound infection after skin abrasion (17), and a community-acquired urinary tract infection (18). These reports suggested an environmental or exogenous source of the organism. We describe four cases of severe infections due to *E. taylorae* which were acquired nosocomially within a 6-month period. The epidemiological, clinical, and microbiological features including restriction endonuclease DNA analysis of the *E. taylorae* strains are described.

CASE REPORTS

Case 1. Bacteremia, cholangitis, and pneumonia due to *E. taylorae*. A 75-year-old white male was admitted for cholestatic jaundice. On day 3 of admission, an obstructed common bile duct (CBD) was cannulated and drained by endoscopic retrograde cholangiopancreatography (ERCP), percutaneous transhepatic cholangiogram (PTC), and balloon papillotomy. Drainage of the biliary tract was maintained by placement of internal and external catheters. Patency of the drainage system was maintained by flushing the catheter with 5 to 10 ml of sterile normal saline once every 8 h. Cefotetan was administered before the procedures and continued thereafter. The next day, because of persistent jaundice, the CBD was reexplored by PTC and insertion of a guide wire. Metronidazole was added.

The next day, the patient developed hematemesis, which was believed to be due to the papillotomy, and a coagulopathy related to liver disease. He developed diffuse pulmonary infiltrates and adult respiratory distress syndrome and required intubation. *E. taylorae* was isolated from blood, sputum, and bile cultures. Antibiotic therapy was changed to trimethoprim-sulfamethoxazole. On day 8 of trimethoprim-sulfamethoxazole treatment, *E. taylorae* was isolated from bile aspirated via the external biliary catheter. Despite appropriate antibiotic therapy and adequate drainage of the biliary tree, the patient had persistent and progressive renal and liver failure with persistent coagulopathy and episodes of hematemesis. Antibiotic therapy was changed to intravenous ciprofloxacin. The patient expired 10 days later.

A postmortem examination revealed a poorly differentiated adenocarcinoma of the gallbladder, extending into the extrahepatic biliary tract, with stenosis of the cystic duct and the second portion of the duodenum. *Candida albicans* was isolated from the gall bladder.

Case 2. Bacteremia and possibly pneumonia due to *E. taylorae*. A 75-year-old white male was admitted for a coronary artery bypass graft and left ventricular aneurysmectomy. Cefuroxime was administered preoperatively. On day 7 postsurgery, the patient was noted to have bilateral pulmonary infiltrates for which empiric treatment with ceftriaxone was administered for 7 days. The patient was stable, and no cultures were obtained.

On day 19 after surgery, because of odynophagia and anemia, the patient underwent an esophagogastroduodenoscopy (EGD), which revealed erosive esophagitis and a duodenal ulcer. A biopsy revealed a benign ulcer. The next day, the patient developed respiratory distress due to aspiration of bloody secretions and required intubation. Two days later, the patient became febrile. A Quinton catheter was removed from a subclavian vein, and empiric therapy with ceftazidime and vancomycin was begun. The following day, the patient developed a new right lung infiltrate. Fever persisted. Three days later, on day 4 of antibiotic treatment, *E. taylorae* was isolated from blood cultures. Two urine cultures were negative. *Staphylococcus epidermidis* was isolated from the Quinton catheter tip. Antibiotics were changed to vancomycin and tobramycin. A gram-negative rod was present in a sputum culture collected 2 days after the

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blood cultures but was not identified because of the small number of organisms, the presence of oral flora, and the large number of squamous epithelial cells in the specimen. Fever and respiratory failure persisted, and the patient expired on day 29 postsurgery. A postmortem examination was not performed.

Case 3. Urinary tract infection due to *E. taylorae* (and *Pseudomonas aeruginosa*). A 75-year-old white female was admitted for obstructive jaundice. She was suspected of having carcinoma of the head of the pancreas and had undergone placement of an internal stent in the CBD via ERCP 41 days earlier.

On day 5 of admission, an occluded internal stent in the CBD was replaced via ERCP. Cefotetan was administered before the procedure. Twelve days later, the patient developed gastric outlet obstruction. A urinary catheter was inserted to monitor fluid balance. Two days later, the patient developed fever to 102.1°F (38.94°C) and confusion. Urinalysis revealed pyuria, hematuria, and bacteriuria. Empiric therapy with ampicillin was begun. *E. taylorae* (>10^5 organisms per ml) and *Enterococcus* species (10^3 to 10^4 organisms per ml) were isolated from the urine. Three days later, the fever of the patient was 103.2°F (39.56°C). *E. taylorae* (10^4 to 10^5 organisms per ml) and *Pseudomonas aeruginosa* (10^4 to 10^5 organisms per ml) were isolated from the urine; *P. aeruginosa* was also isolated from blood cultures. Antibiotic therapy was changed to ceftazidime and tobramycin. A repeat urine culture on day 2 of tobramycin treatment was negative.

Three weeks later, the patient succumbed to her underlying illness. A postmortem examination was not performed.

Case 4. Urinary tract infection due to *E. taylorae*. An 85-year-old white male underwent a transurethral prostatectomy for benign prostatic hypertrophy at another hospital. No prophylactic antibiotics were administered. There was no complicating infection, and the urinary catheter was removed on day 4 after surgery. On day 12 postsurgery, the patient was transferred to our hospital for an emergency coronary artery bypass graft. Microscopic examination of urine obtained on admission (clean catch specimen) revealed hematuria and pyuria without bacteriuria. A urine culture was not obtained. A urinary catheter was inserted preoperatively. Cefuroxime was administered pre- and postoperatively. Twenty-four hours after surgery, while in the Open Heart Unit, the patient developed hypotension, fever, and macroscopic hematuria with clots. A urine culture was obtained, and the antibiotic treatment was changed to clindamycin. *E. taylorae* (>10^5 organisms per ml) was isolated from the urine culture and from a second urine specimen obtained 2 days later. Blood and sputum cultures were negative. Antibiotic therapy was changed to trimethoprim-sulfamethoxazole. The patient recovered and was discharged.

MATERIALS AND METHODS

Demographics. Saint Francis Hospital & Medical Center is a 700-bed acute-care community teaching hospital. Affected patients were hospitalized on various units and wards between 25 July 1989 and 25 December 1989.

Patient identification. Patients 1 and 2 were monitored in consultation with the Infectious Disease Service. The charts of patients 3 and 4 were reviewed after they had been identified by the microbiology laboratory.

Laboratory methods. Routine identification and susceptibility testing of gram-negative rods was performed by using a Microscan Gram Negative BP-5 panel (Baxter Scientific). Antibiotic susceptibility tests were performed by using Microscan microdilution breakpoint panels and/or Sceptor MIC (Becton Dickinson) microdilution trays. Interpretations of susceptibility tests were made on the basis of published guidelines of the National Committee for Clinical Laboratory Standards. Environmental cultures were obtained by using a swab and inoculating tryptic soy agar with 5% sheep blood and enriched thioglycolate broth.

Isolate identification. Each isolate was originally identified as *E. taylorae* by using Microscan Neg BP Combo 5 panels. Key reactions were confirmed by using standard tubed media. Each isolate had the following characteristics: gram-negative rod; positive for citrate and malonate utilization; positive Voges-Proskauer; positive for *o*-nitrophenyl-β-D-galactopyranoside, arginine dihydrolase, ornithine decarboxylase, and nitrate; acid production from D-glucose, D-mannitol, L-rhamnose, salicin, and L-arabinose; negative for oxidase, lysine decarboxylase, H_2S, urease, tryptophan deaminase, indole, and gelatin; lack of acid production from myo-inositol, melibiose, and D-sorbitol. These reactions are the same as those characteristic of the type species (11).

DNA isolation and analysis. For plasmid isolation, *E. taylorae* cells were lysed by using a lysozyme–0.1% Triton X-100 procedure as previously described (7). Studies for plasmid DNA isolation were done by cesium chloride-ethidium bromide density gradient centrifugation. Whole-cell DNA was obtained by a lysozyme-20% sodium dodecyl sulfate procedure as described previously (8). Whole-cell DNA was digested by restriction endonucleases *Cla*I and *Hin*dIII as described in manufacturer specifications. Plasmid DNA and whole-cell DNA preparations were analyzed by 0.7% Tris acetate–1 mM EDTA gel electrophoresis on a horizontal gel apparatus, stained with ethidium bromide, and photographed with UV illumination.

To examine the possibility of a plasmid pattern overlying the chromosomal pattern, *E. taylorae* cells were lysed and whole-cell DNA was isolated as previously described (14). *E. taylorae* DNA was digested with *Spe*I as described in manufacturer specifications. Analysis of whole-cell DNA was done by contour-clamped homogeneous electric field electrophoresis, a modification of pulsed-field gel electrophoresis, with a pulse time of 1 to 30 s at 198 V for 18 h at 15°C.

RESULTS

Epidemiologic report. After the identification of case 1, an epidemiological investigation of the ERCP and PTC suites was conducted (by M.T.D.). The ERCP suite was revisited after case 3 was identified. The EGD suite was visited after case 2 was identified. No environmental source of infection was identified. Three PTCs, 130 ERCPs, and 899 EGDs were performed in the same year in which these four cases occurred.

The procedures prior to the *E. taylorae* infection, the number of days between the procedure and the onset of clinical illness, the number of days from admission to first positive culture, and relevant clinical data are detailed in Table 1. A detailed study of the dates of hospitalizations, in-hospital locations, and dates of procedures did not suggest a common source of infection. No two patients were located

TABLE 1. Important clinical features of invasive *E. taylorae* infections—four case reports

Patient no.	Underlying medical illness[a]	Procedure(s) performed (no. of days between procedure and onset of clinical disease)	Antibiotic(s) administered prior to onset of clinical disease	Sources of culture specimens	No. of days from admission to first positive culture	Clinical infectious disease
1	Adenocarcinoma of gall bladder[b]	ERCP + papillotomy + PTC (4); PTC (3)	Cefotetan; metronidazole	Blood	6	Bacteremia
				Bile	6	Cholangitis
				Sputum	6	Pneumonia
2	CABG, ventricular aneurysectomy	EGD + biopsy (3); tracheal intubation (2)	Cefuroxime; ceftriaxone	Blood	23	Bacteremia
				Sputum (?)	25	Pneumonia
3	Carcinoma, head of the pancreas[b]	Urinary catheter (2)	Cefotetan	Urine	22	Urinary tract infection
4	TURP, CABG	Urinary catheter (1)	Cefuroxime	Urine	6	Urinary tract infection

[a] CABG, coronary artery bypass graft; TURP, transurethral prostatectomy.
[b] Patient 1 underwent ERCP, PTC, and papillotomy on 28 July 1989, a second PTC on 29 July 1989, and a third PTC on 9 August 1989. *E. taylorae* grew from blood, bile, and sputum cultures on 31 July 1989 and from bile on 3 and 10 August 1989. Patient 3 was admitted on 1 August 1989, underwent an uneventful ERCP on 4 August 1989, and was discharged on 10 August 1989. The patient was readmitted on 14 September 1989 and underwent an ERCP on 19 August 1989. *E. taylorae* grew from urine on 6 October 1989. Patients were on separate units and were attended by different physicians.

on the same unit at the same time. Patients were attended by different physicians and nurses; no illnesses were reported among the staff members attending the procedures or patients (cultures from staff members were not obtained).

The four cases were identified between July 1989 and December 1989. These were the first cases identified in our hospital. Of all microbiological specimens obtained from in-hospital patients during that time, *Enterobacter* species other than *E. taylorae* were considered microbiologically significant in 42 patients. No additional nosocomial cases of *E. taylorae* were identified between July 1989 and June 1990.

Microbiology results. All four isolates had the same antibiogram (Table 2). All were resistant to cephalosporins, resistant or intermediately susceptible to penicillins, and

TABLE 2. *E. taylorae* susceptibilities to antibiotics

Antibiotic	Susceptibility to antibiotic[a] (MIC [μg/ml])								
	Cases reported in this study				Referenced case reports				
	Isolate 1 (bile)	Isolate 2 (blood)	Isolate 3 (urine)	Isolate 4 (urine)	Farmer et al. (11) (102 isolates)[b]	Reina et al. (17) (1 isolate)	Westblom (22) (1 isolate)	Reina et al. (18) (1 isolate)	Muytjens et al. (15) (10 isolates)[c]
Trimethoprim-sulfamethoxazole	S (<1/19)	S (≤1/19)	S (<1/19)	S (<1/19)		S (<20)		S (<10)	S (4)
Ciprofloxacin	S (≤1)		S (≤1)	S (≤1)				S (<0.5)	S (≤0.06)
Gentamicin	S (≤0.25)	S (≤0.25)	S (≤0.25)	S (≤0.25)	S (97%)	S (<1)	S (<1)	S (<0.5)	S (0.5)
Tobramycin	S (≤4)	S (≤4)	S (≤4)	S (≤4)		S (<1)	S (<1)	S (<0.5)	
Amikacin	S (≤4)	S (≤4)	S (≤4)	S (≤4)		S (<1)		S (<2)	
Chloramphenicol	S[d]	S[d]	S[d]	S[d]	S (100%)	S (<1)		S (8)	S (8)
Tetracycline					S (81%)	S (<1)	S (2)		S (4)
Aztreonam	R[e]			R[e]				R (>32)	
Ampicillin	R (>16)	R (>16)	R (>16)	R (>16)	R (53%)	R (>32)	S (4)	R (>32)	R (>128)
Mezlocillin	I (64)	I (64)	I (64)	I (64)				R (>256)	
Ticarcillin	R (256)	R (256)	R (256)				S (<8)	S (32)	
Piperacillin	I (64)	I (64)	I (64)				S (<8)	R (>128)	S (2)
Carbenicillin					S (69%)	S (<16)		R (64)	
Cefazolin	R (>64)	R (>64)	R (>64)	R (>64)	R (3%)		R (>16)	R (>32)	R (>128)
Cefotetan	R[e]			R[e]					R (>128)
Cefamandole						I (16)			R (64)
Cefotaxime	R (>64)	R (>64)	R (>64)				S (<2)	S (8)	S (0.25)
Ceftriaxone	R (>128)	R (>128)	R (>128)					R (64)	

[a] S, susceptible; R, resistant; I, intermediate.
[b] Expressed as percentage of isolates susceptible by the Kirby-Bauer disc diffusion method.
[c] MIC, 90%.
[d] Determined by Kirby-Bauer disc diffusion method.
[e] Determined by using Microscan microdilution breakpoint panels.

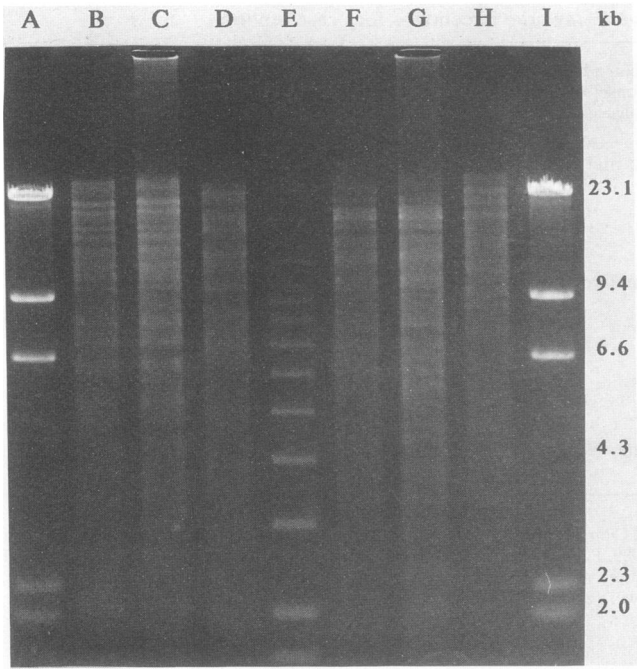

FIG. 1. DNA typing of *E. taylorae*. The results of agarose gel electrophoresis of purified *E. taylorae* whole-cell DNA digested with restriction endonuclease *Cla*I (lanes B to D) and *Hin*dIII (lanes F to H) are shown. Lanes A and I show bacteriophage lambda *Hin*dIII fragments for size reference. DNAs from case 1 and case 3 isolates are shown in lanes B and C and lanes F and G, respectively. DNA from a case 2 isolate is shown in lanes D and H. Lane E shows a molecular weight reference ladder.

uniformly susceptible to trimethoprim-sulfamethoxazole and aminoglycosides. Three isolates were tested for ciprofloxacin and found to be susceptible. Two isolates were tested for aztreonam and found to be resistant.

DNA typing results. The results of DNA typing are shown in Fig. 1. The restriction endonuclease digestion patterns of whole-cell DNA of *E. taylorae* isolates from cases 1 and 3 were the same; DNA from the isolate from case 2 was distinct. In addition, a single plasmid was isolated from the isolate from case 2; no plasmid was identified in the isolates from cases 1 and 3 (data not shown). The isolate from case 4 was not available for study.

Results with pulsed-field gel electrophoresis confirmed these results. Fragment sizes ranged from 366 to 34 kb (data not shown).

DISCUSSION

E. taylorae, formerly known as CDC Enteric Group 19, was defined as a separate species in 1985 (11). The organism has been isolated from blood, spinal fluid, wounds, urine, the respiratory tract, and stool specimens obtained from animals and humans (10, 11), but little is known regarding the epidemiology or clinical significance of this organism. Three case reports, of osteomyelitis, a wound infection, and a urinary tract infection, associate *E. taylorae* infection with an environmental or exogenous source (17, 18, 22). We describe severe nosocomial infections in four patients, acquired between July 1989 and December 1989. These four cases share several features. (i) Cephalosporin therapy had been administered before surgery or an invasive procedure and preceded the *E. taylorae* infection. The cephalosporins used had no activity against *E. taylorae*. (ii) The patients underwent instrumentation or an invasive procedure that potentially enabled bacterial invasion (ERCP plus papillotomy plus PTC, urinary catheter, EGD plus biopsy, or tracheal intubation). (iii) *E. taylorae* was responsible for severe morbidity and probably contributed to the mortality of patients 1 and 2. (iv) Patients were 75 or more years old. (v) No clear environmental or common source of infection was identified. DNA typing studies suggested that patients 1 and 3 were infected with the same strain, while patient 2 was infected by a distinct strain (Fig. 1); all four isolates had the same antibiogram (Table 2).

Sources of nosocomial infections are either endogenous (i.e., the patient harbors the pathogen prior to and during the hospitalization) or exogenous (i.e., the patient acquires the pathogen in the hospital). Contaminated parenteral solutions (13) or contaminated instrumentation used in EGD, ERCP, and PTC (1, 5, 21) are two of the various sources associated with exogenous acquisition of nosocomial pathogens. PTC has been associated with sepsis and cholangitis caused by organisms found in the gut flora of patients, including *Enterobacter* species (2, 6). We were not able to identify an exogenous source of *E. taylorae* in the two patients who underwent these procedures.

The skin, upper airways, and gastrointestinal tracts of hospitalized patients harbor potential opportunistic pathogens associated with endogenously acquired nosocomial infections. Hospitalization for more than 48 h and the use of antibiotics have both been implicated in "selecting out" endogenous gram-negative rods. This has been recently observed in *Enterobacter* species after cephalosporin treatment. Flynn et al. demonstrated that a narrow-spectrum cephalosporin (cefazolin) given prophylactically for cardiac surgery led to increased colonization of *Enterobacter* species in the intestinal flora of the patients. Colonizing strains of *Enterobacter aerogenes* and *Enterobacter cloacae*, unique to each patient, were identified as the pathogens in subsequent nosocomial infections. No environmental source of transmission was found (12). We postulate that our patients were harboring *E. taylorae* and that the instrumentations and/or invasive procedures they underwent provided a portal of entry for this organism.

In addition to selecting out particular organisms, the use of antibiotics has been implicated in the development of gram-negative organisms with resistance patterns unique to each hospital environment. The β-lactamases identified in *Enterobacter* species have been both plasmid mediated and chromosomally located, both inducible and constitutive, and of the Richmond-Sykes classes I and IV (3, 9, 20). More disturbingly, a novel plasmid β-lactamase (CTX-1) originally found in *Klebsiella pneumoniae*, has been recently identified in both *E. aerogenes* and *E. cloacae*, rendering them resistant to cefotaxime, ceftazidime, and aztreonam, antibiotics to which these organisms are usually susceptible (16). Resistant mutants that produce constitutive class I β-lactamases usually exist in small numbers, but they can emerge and predominate after β-lactam therapy by virtue of selection and do not necessarily require the presence of a β-lactam inducer. This has been demonstrated for both *Enterobacter* species and *P. aeruginosa* (19). Resistance due to production of β-lactamase and selection of resistant clones have both been recently described in clinical isolates of the *Enterobacter* genus by Chow et al. (4), who demonstrated (i)

that patients who had received previous antibiotics were more likely to have a multiresistant *Enterobacter* bacteremia than patients who had not received antibiotics and (ii) that therapy with broad-spectrum cephalosporins was associated with the emergence of *Enterobacter* isolates that had acquired resistance to the same cephalosporins as well as resistance to other classes of antibiotics. (One case of infection due to *E. taylorae* was noted, but no clinical or microbiological data were detailed.) We did not observe a change in the resistance patterns in our four isolates. Of importance is the disturbing observation that, unlike most members of the *Enterobacter* genus, our four isolates were uniformly resistant to expanded- and broad-spectrum cephalosporins. Furthermore, the resistance of our isolates to broad-spectrum cephalosporins (i.e., cefotaxime and ceftriaxone) has not been observed by others (15, 18, 22) (Table 2). Of note, 3% of the 102 *E. taylorae* isolates tested by Farmer et al. (11) were susceptible to cefazolin (a narrow-spectrum cephalosporin). However, no clinical information regarding these isolates is given. Unlike some of the isolates studied by others (11, 15, 18, 22), all of our isolates were resistant or intermediately susceptible to penicillins (Table 2). While only one of the strains contained a plasmid, all four isolates had the same antibiogram. These observations suggest that the resistance pattern we identified in our isolates was an inherent and not an inducible characteristic of the organism.

Our cases were separated over time, occurred in separate locations, involved different procedures, and did not involve the same nursing and medical staff. The similarity between the strains infecting patients 1 and 3 and the clustering of cases over a limited period of 6 months may suggest an in-hospital common source, such as colonized hospital staff or hospitalized patients, or contaminated solutions, which we were not able to identify. In the absence of information regarding the DNA patterns of *E. taylorae* and the number or frequency of existing strains, we are unable to state whether the similar strain found in two patients represents interpatient transmission.

We postulate that the administration of cephalosporins in combination with instrumentation and/or invasive procedures enabled the development of nosocomial infections in patients harboring *E. taylorae*. We have identified *E. taylorae* as a new opportunistic pathogen in our institution. Of concern is the fact that the currently recommended surgical prophylactic regimens using narrow- or expanded-spectrum cephalosporins appear to be ineffective against *E. taylorae*. We would urge physicians to limit the duration of prophylactic regimens to avoid the emergence of multiresistant opportunistic pathogens from within the patient or hospital microflora. The use of cephalosporins outside the hospital may also contribute to the emergence and proliferation of this organism in the community, thus providing an additional avenue of introduction into the hospital microflora.

We provide clinical, microbiological, and epidemiological data describing severe nosocomial infections due to a newly identified organism, *E. taylorae*. We postulate that in some patients the organism is part of their endogenous flora and that the combination of cephalosporins and instrumentations enables this organism to become an opportunistic pathogen. Additional studies are necessary to establish the carrier rate, resistance patterns, and clinical significance of this new opportunistic pathogen.

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