**First Report of *Laribacter hongkongensis*
Peritonitis in Continuous Ambulatory
Peritoneal Dialysis**

The most common pathogens associated with peritonitis in patients on continuous ambulatory peritoneal dialysis (CAPD) are gram-positive bacteria, which constitute 60 – 80% of all isolates.

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With the advancement of molecular technologies for bacterial identification, cases of CAPD-related peritonitis caused by bacteria previously not known to be associated with this clinical condition have been reported. Here we report the first case of CAPD-related peritonitis due to *Laribacter hongkongensis*.

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A 79-year-old Chinese wheelchair-bound male nursing home resident was admitted to hospital in July 2014 because of fever and cloudy dialysis effluent for 1 day. He had end-stage renal disease, for which continuous ambulatory peritoneal dialysis (CAPD) had been commenced 3 months prior. He also had histories of hypertension, a transient ischemic attack in 2008 as a result of carotid artery stenosis, and myocardial infarction with coronary artery bypass graft performed in 2014. He had no abdominal pain or diarrhea. On admission, he was febrile (38.7°C) with turbid dialysis effluent, but no abdominal tenderness. His hemoglobin was 7.4 g/dL, total white cell count $7.5 \times 10^9/L$, with neutrophils $5.8 \times 10^9/L$, lymphocytes $1.4 \times 10^9/L$, monocytes $0.3 \times 10^9/L$, and platelet count $119 \times 10^9/L$. Serum urea and creatinine levels were 17.2 mmol/L and 414 $\mu\text{mol/L}$, respectively, with normal levels of liver enzymes. The total leukocyte count of the dialysis fluid was $919 \times 10^6/L$, with 74% neutrophils and 26% lymphocytes. Gram stain of the dialysis effluent after centrifugation revealed only numerous leukocytes, but no microorganisms were seen. Intraperitoneal cefazolin and amikacin were started for empirical treatment of CAPD peritonitis. Culture of the dialysis effluent obtained on admission yielded pure growth of a gram-negative, S-shaped, motile bacillus. The bacterium was identified by 16S rRNA gene sequencing and matrix-assisted laser desorption ionization-time of flight mass spectrometry as *Laribacter hongkongensis*, using protocols previously described (1,2). Multilocus sequence typing showed that the sequence of the isolate belonged to sequence type-63 (3,30,3,19,14,3,15) (3). Blood culture taken on admission was negative for bacteria after 5 days of incubation. All subsequent cultures of the dialysis effluent were negative and the patient received 10 days of intraperitoneal cefazolin and amikacin. In the following 2 months, no relapse of peritonitis occurred.

The portal of entry in our patient with *L. hongkongensis*-CAPD-related peritonitis is likely to be the gastrointestinal tract. Pathogens causing CAPD-related peritonitis originated from 2 major sources. Bacteria of the skin flora, such as *Staphylococcus aureus* and *S. epidermis*, as well as environmental microbes, such as environmental fungi (4) entered the peritoneal cavity through the Tenckhoff catheter *in-situ*. On the other hand, bacteria from the gastrointestinal tract, including both the normal flora such as *Escherichia coli* or *S. bovis* (5) and occasionally gastrointestinal pathogens such as *Campylobacter jejuni*, *Salmonella enteritidis*, *Shigella sonnei* and *Plesimonas shigelloides*, reached the peritoneal cavity by translocating the intestinal wall (6–8). *Laribacter hongkongensis* was first

isolated from the blood and thoracic empyema of an alcoholic liver cirrhosis patient in Hong Kong (9). In addition to invasive bacteremic infections (9,10), *L. hongkongensis* is also associated with freshwater fish-borne community-acquired gastroenteritis and traveler's diarrhea (1). *Laribacter hongkongensis* has been found in up to 60% of intestines of commonly consumed freshwater fish of the carp family (1). Similar to most patients with *L. hongkongensis* gastroenteritis, the patient in this study gave a positive history of freshwater fish consumption within 3 days prior to the development of CAPD-related peritonitis, although he did not have gastrointestinal symptoms. We speculate that our patient developed CAPD-related peritonitis because he consumed freshwater fish harboring the *L. hongkongensis* bacterium, which had translocated through the intestinal wall into the peritoneal cavity, causing peritonitis. To prevent *L. hongkongensis* infections, all freshwater fish should be thoroughly cooked before consumption, as the bacterium should be killed by standard cooking.

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DISCLOSURES

The authors have no financial conflicts of interest to declare.

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