Renal Failure

ISSN: 0886-022X (Print) 1525-6049 (Online) Journal homepage: informahealthcare.com/journals/irnf20

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To cite this article: Shang-Feng Tsai & Kuo-Hsiung Shu (2010) CAPD peritonitis caused by *Alcaligenes xylosoxidans* in a diabetic cirrhosis patient, Renal Failure, 32:7, 899-901, DOI: 10.3109/0886022X.2010.494796

To link to this article: https://doi.org/10.3109/0886022X.2010.494796

Published online: 21 Jul 2010.

Renal Failure, 32, 899–901, 2010

ISSN: 0886-022X print / 1525-6049 online
DOI: 10.3109/0886022X.2010.494796

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CASE REPORT

CAPD peritonitis caused by *Alcaligenes xylosoxidans* in a diabetic cirrhosis patient

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ABSTRACT

Despite the advancement of disconnecting system, peritonitis remains one of the most common causes in patients treated with continuous ambulatory peritoneal dialysis (CAPD). We report a cirrhosis patient undergoing CAPD who suffered from an episode of peritonitis caused by a rare pathogen, *Alcaligenes xylosoxidans* (AX). This organism rarely induces CAPD peritonitis and only nine cases were reported in the literature. This is the first case of AX-related CAPD peritonitis in cirrhosis patient. Infection with AX is more common in immunocompromised patients and is associated with a high mortality rate. Appropriate antibiotics and early removal of CAPD catheter may be life-saving in such rare infection.

Keywords: *Alcaligenes xylosoxidans*; continuous ambulatory peritoneal dialysis; peritonitis; cirrhosis; immunocompromised

Received 6 May 2010; accepted 11 May 2010
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INTRODUCTION

Although the peritonitis rate of continuous ambulatory peritoneal dialysis (CAPD) declined steadily in the past two decades, peritonitis remains to be an important cause of technique failure, only second to ultrafiltration failure in our series. The decline of Gram-positive peritonitis exceeds that of Gram-negative peritonitis, making an increasing proportion of Gram-positive peritonitis in the modern era. More importantly, some of the Gram-negative pathogens are associated with a remarkable mortality and morbidity. Of them, a rare Gram-negative pathogen called *Alcaligenes xylosoxidans* (AX) has only been reported in nine cases treated with CAPD. We herein report such a case in a cirrhosis patient undergoing CAPD. AX is also rare in cirrhosis patients who suffered from spontaneous bacterial peritonitis (SBP). To our knowledge, this is the first case in patients with both uremia and cirrhosis.

CASE REPORT

The patient is a 51-year-old female with long-term history of type 2 diabetes mellitus (DM), hypertension, and hepatitis C and Child's B-related cirrhosis. She developed progressive deterioration of renal failure, which was most likely due to diabetic nephropathy. Meanwhile, progressive distension of abdomen was noted which was later confirmed to be cirrhosis-related ascites. By 2008, she reached end-stage renal failure. Renal replacement therapy was suggested and she chose to receive CAPD, taking the advantage of both dialysis therapy and intermittent ascites drainage. The catheter was inserted on 20 October 2008.

Unfortunately, she experienced four episodes of CAPD peritonitis before the final admission. She insisted to continue CAPD instead of catheter removal because of rapid resolution of these episodes and personal preference. The first episode of peritonitis due to *Streptococcus bovis* responded to treatment with cefazolin and ceftazidime rapidly in 2 weeks. The second episode occurred 1 month later. This episode also responded quickly to cefazolin and ceftazidime and the organism was *Klebsiella pneumoniae*. Then the third episode occurred 1 month after the second one. However, the peritonitis with negative culture also responded rapidly to cefazolin and ceftazidime. Despite successful treatment for 14 days, the fourth episode occurred 2 weeks later which was due to *Escherichia coli*, with extended spectrum beta lactamase (ESBL). Ertapenem 500 mg was prescribed for 5 days followed by ciprofloxacin for

another 9 days. The patient finally agreed to stop CAPD and shifted to hemodialysis but asked to retain the catheter for the drainage of ascites.

Unfortunately, 90 days after hemodialysis, she suffered from the fifth peritonitis. Studies of ascites revealed the following: white blood cell (WBC) was 7030/mm^3 with neutrophil predominant (88%), protein 1600 mg/dL, amylase 24 U/L, and lactate dehydrogenase (LDH) 344 U/L. The high protein and LDH concentration was not in favor of cirrhosis-related SBP. The biochemical data were as follows: prothrombin time = 11.4 seconds, albumin = 2.1 mg/dL, total bilirubin = 0.6 mg/dL, potassium = 3.3 mEq/L, WBC = 17,000/mm^3, neutrophil : lymphocyte = 90 : 6. Therefore, the catheter was removed immediately and the patient was treated with intravenous cefazolin and ceftazidime. Four days later, despite an improvement of cell count of ascites (WBC: 970/mm^3), the abdominal pain persisted. Ascites culture yielded AX for three sets. The antibiotics were shifted to ampicillin with sulbactam to cover AX for 7 days and followed by ciprofloxacin for 2 days. At this time, hospital-acquired pneumonia appeared. Therefore, ciprofloxacin was further shifted to cefepime to cover both AX-related CAPD peritonitis and hospital-acquired pneumonia. After antibiotics treatment for 1 month, she completely recovered and remains well on hemodialysis till now. The clinical course is summarized in Table 1.

DISCUSSION

AX infections are rare and meningitis, pneumonia, ear infections, respiratory infection have been reported. AX-related peritonitis is also rare in CAPD patients. Up to date, only nine cases were reported in the literature. AX-related SBP in cirrhosis patients is even rarer and only two cases have been reported. Our case represented the first case of AX-related peritonitis in cirrhosis CAPD patient. There were several risk factors in our patient. First, she had DM which seems to be a significant risk factor for AX-related peritonitis. Including ours, out of the 10 cases of AX-related CAPD peritonitis, 4 had type 2 DM.[1–3] Second, AX-related infection always occurred in immunocompromised host,[4] such as patients with HIV infection, neutropenia, and hyper-IgM syndrome. Our case had both uremia and Child's B cirrhosis that made her immunocompromised and prone to opportunistic infection. Third, the remarkably low-level serum albumin implies advanced cirrhosis and/or malnutrition. For each drainage of ascites, the relief of abdominal distention also accompanied with the loss of albumin. Malnutrition may predispose to infectious complications. Fourth, frequent use of broad-spectrum antibiotics may select multidrug-resistant pathogen, such as *Pseudomonas aeruginosa, Acinetobacter baumannii,* and AX. Our patient experienced four episodes of peritonitis in a quite short period

TABLE 1. Bacteriology and management of peritonitis.

Episode	Time past CAPD (20 October 2008)	Ascites data		Ascites culture	Blood routine		Management	*Outcome*
		WBC	N/L		WBC	N (%)		
First	44th day	3,040	96/3	*S. bovis*	20,900	90	Cefazolin 250 mg qid IRR for 14 days + ceftazidime 250 mg IRR for 14 days	
Second	89th day	24,750	94/0	*K.P*	12,000	89.1		
Third	136th day	12,280	86/3	Negative	34,000	98		
Fourth	180th day	5,525	90/8	*E. coli*	13,300	89	Cefazolin 250 mg qid IRR for 3 days + ceftazidime 250 mg qid IRR for 3 days → ertapenem 500 mg iv for 3 days → ciprofloxacin 200 mg q12 h for 9 days	*Resolved*
367th day: Shift to hemodialysis, but catheter retained for ascites drainage								
Fifth	457th day	7,031	88/4	AX*3	17,000	90	Cefazolin 250 mg qid IRR for 4 days + ceftazidime 250 mg qid IRR for 4days → ampicillin + sulbactam iv for 7 days → ciprofloxacin for 2 days → cefepime for 7 day	*Removed catheter*

Notes: *S. bovis* = *Streptococcus bovis*; *K.P* = *Klebsiella pneumoniae*; *E. coli* = *Escherichia coli*; AX = *Alcaligenes xylosoxidans*; IRR: irrigation; N/L = neutrophil/lymphocyte.

before the onset of AX infection. Strong antibiotics were used and all resulted in complete remission. We speculated that the frequent use of strong antibiotics may have predisposed to the appearance of rare pathogen like AX.

Whether the uremic cirrhosis patient can receive CAPD is still a subject of controversy.[5] CAPD has the advantage of ascites drainage, stable hemodynamics, and preservation of residual renal function. However, cirrhotic patients are at risk of developing SBP themselves and the loss of albumin from CAPD drainage may be abundant. Our patient had the lowest level of albumin (2.1 g/dL) in all the 10 cases of AX-related CAPD peritonitis because of advanced cirrhosis and frequent drainage of albumin-abundant ascites. Other cases also suffered from hypoalbuminemia (e.g., 2.5 g/dL[2,4] and 3.6 g/dL[4]). Furthermore, cirrhotic patients are prone to intestinal bacterial overgrowth.[6] In our previous study, DM and hypokalemia in CAPD patients are significant factors that are associated with intestinal bacterial overgrowth. The organisms in such patients are usually enterobacteria instead of epidermal bacteria. Our patient suffered from five episodes of peritonitis of which three were from enterobacteria and only one episode was from epidermal bacteria. Taken together, our patient has several risk factors for the development of AX and probably should have been advised to discontinue CAPD with prolonged course of antibiotics when the first episode of peritonitis took place. Unfortunately, this was not carried out under the patient's strong willingness to continue CAPD.

AX-related peritonitis is detrimental to CAPD and only nearly one-third (3 out of 10) were cured without the removal of catheter. AX may form biofilm on plastic material like CAPD catheter and is difficult to be eradicated. Keeping CAPD catheter and flushing could mechanically shear the biofilm and cause detachment of cells or aggregates. Half cases (5 out of 10) recovered with the removal of CAPD catheter. Moreover, AX may produce β-lactamase, making it resistant to the first- and second-generation cephalosporin, aminoglycosides, and narrow-spectrum penicillins.[7] Our case was successfully treated with prompt catheter removal and ertapenem.

In conclusion, AX is a rare cause of CAPD peritonitis. We have reported such a case with several risk factors. CAPD should probably be discouraged in such patients although the evidence is still insufficient because of the rarity of this infection.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

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