A Case of Fulminant Peritonitis Caused by *Streptococcus mitis* in a Patient on Peritoneal Dialysis

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Abstract

A 54-year-old woman on peritoneal dialysis (PD) was hospitalized with peritonitis with a high body temperature, abdominal pain and cloudy peritoneal fluid. She progressively fell into septic-like shock within only 6 hours after onset. The causative bacteria were *Streptococcus mitis* (*S. mitis*), part of the normal flora of oral cavity, intestine, female genital tract and upper respiratory tract. *S. mitis* shows pathogenicity for diseases such as endocarditis, brain abscesses and sepsis in children with malignancy or transplantation. However, *S. mitis* rarely shows severe pathogenic responses in adults. We report herein a case of fulminant peritonitis caused by *S. mitis* in an adult PD patient.

Key words: fulminant peritonitis, peritoneal dialysis, *Streptococcus mitis*

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Introduction

Peritonitis is a major complication on peritoneal dialysis (PD) therapy and is an important reason for withdrawals in PD patients (1). PD-related infectious peritonitis is usually resolved using adequate anti-bacterial therapies. However, some microorganisms such as *Pseudomonas aeruginosa*, *Staphylococcus aureus* and fungus cause severe PD-related peritonitis. Some cases of peritonitis progress to life-threatening encapsular sclerosing peritonitis (2).

Viridans group streptococci (VGS) are known as normal resident bacterial flora of the upper respiratory tract, mouth, intestine, skin and female genital tract. These bacteria usually have weak pathogenic effects in adults. However, those microorganisms can occur endocarditis and life-threatening infections resulting in viridans-related septic shock in children, especially, with malignancy or bone marrow transplantation (3-5). Although *Streptococcus mitis* (*S. mitis*) is a VGS, few reports have described severe infections caused by *S. mitis* in adults (6). Here, we report a case of fulminant PD-related peritonitis caused by *S. mitis* in an adult PD patient.

Case Report

A 54-year-old woman developed end-stage renal disease due to chronic glomerulonephritis, and had been maintained on continuous ambulatory PD (CAPD) for 9 years, except for 1 year of hemodialysis treatment in place of PD during high-dose steroid treatment for bullous pemphigoid at age 46 years old. The PD bag-exchanges were 1.5% glucose solution (Dianeal-N PD-4 1.5%™; Baxter, Tokyo, Japan) 1.8 L ×3 times/daytime and icodextrin solution (Extraneal™; Baxter) 1.6 L/overnight. To change PD fluid (PDF) bags, she used a sterile connecting device system UV flash device, “Cline flash”™ (Baxter). Medication history comprised 10 mg/day of prednisolone as maintenance therapy for bullous pemphigoid and medications for hypertension. The patient had allergies to antibiotics such as minocycline hydrochloride, penicillin, ciprofloxacin and tobramycin, and had no history of peritonitis.

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Due to a high body temperature (BT) of 38°C and mild abdominal discomfort, she came to the emergency room. Peritoneal fluid from the last PDF exchange at 6 hours before coming to the hospital was macroscopically clear. On arrival in the emergency room, physical examination showed: BT, 38.5°C; blood pressure (BP), 130/74 mmHg; and heart rate, 117/min in sinus rhythm. During the primary examination, her general condition was seen to rapidly deteriorate to BP 85/40 mmHg, severe abdominal pain with rebound tenderness and muscle defense, watery diarrhea and a decreased oxygen saturation value of ~90% (room air). As she entered a state of shock within 6 hours after onset, norepinephrine and dopamine hydrochloride were administered, along with oxygen (oxygen mask; 3 L/min) and volume re-suscitation. When PDF exchange was performed in the hospital, we observed that the peritoneal fluid was cloudy. She did not have exit site infection.

On admission, clinical laboratory data were as follows: peripheral white blood cell (WBC) count, 4,400 cells/mm³ (polymorphonuclear neutrophils, 76.3%; lymphocytes, 19.6%); blood hemoglobin level, 10.4 g/dL; and C-reactive protein level, 0.21 mg/dL. WBC count from the first cloudy PDF was 9,420 cells/mm³ (polymorphonuclear neutrophils, 80%; mononuclear cells, 20%). In contrast, exchanged PDF from the previous night was macroscopically clear and WBC count was <10 cells/mm³, suggesting that no peritonitis was present before the previous exchange of PDF.

Although cefazolin sodium and ceftazidime are first-line antibiotics in our hospital, in accordance with the recommendations of the 2005 guidelines of the International Society for Peritoneal Dialysis, vancomycin hydrochloride and aztreonam were chosen as empiric therapy of antibiotics because of the patient’s history of allergy to multiple antibiotics. The 2010 guideline of International Society for Peritoneal Dialysis, which was recently published, also recommended the first-line of antibiotics. Clindamycin was also added because the peritonitis was severe and gram-positive cocci were detected on smear sample from her peritoneal fluid, suspecting toxic shock syndrome induced by group A streptococcus (Fig. 1). It was reported that administration of clindamycin improved the toxic shock-like syndrome (9). Administration of aztreonam was stopped because the causative microorganisms were diagnosed as alpha-hemolytic streptococci in culture. S. mitis was finally defined by biochemical characteristics and 16S rRNA gene-sequence analysis. The patient recovered from shock state on day 3. WBC count in peritoneal fluid had decreased <50 cells/mm³ by day 6. The findings of echocardiography showed slight myocardial hypertrophy but not vegetation. On day11 she could be discharged. Before her discharge, we carefully checked and re-educated the total procedure of PD therapy, including her bag exchange technique. She did not have another peritonitis and continuously keeps CAPD for the renal replacement therapy at 10 months after this peritonitis episode. Her clinical course after admission is shown in Fig. 2.

**Discussion**

We encountered an adult PD patient who rapidly fell into fulminant shock after onset of severe peritonitis. The causative microorganism was S. mitis that is a VGS. In the present case, contamination during her bag exchange was suspected as her infection route. Colonization of the cardiovascular endothelium by VGS can result in infective endocarditis, progressing to circulation failure. However, the opportunity for pathogenicity may be dependent on the species of VGS. For example, S. sanguis in VGS has high invasive ability in human endothelial cells, but S. mitis does not (10). S. mitis is occasionally found on the skin, particularly of infants and children, but usually has only weak pathogenicity.

Peritonitis is a common complication of PD and an important cause of mortality. Fontan et al reported that mortality associated with Streptococcus sp. infection was not high in PD peritonitis and fatal Streptococcal peritonitis was accompanied by fungal infections (11). Among Streptococci sp. infections, the streptococcal pyrogenic exotoxins from group A streptococci are well known as pathogens inducing toxic shock-like syndrome. We did not know why this patient showed severe and sudden-onset toxic shock-like syndrome because we did not detect causative toxins to induce shock. There was little information to induce severe illness caused by the virulent factors of S. mitis. As an explanation, a 34-kDa protein from some S. mitis colonies has recently been reported, showing a different composition from the streptococcal pyrogenic exotoxins (12) and the potential to induce severe illness. In the present case of long-term PD therapy the usage of medication of 10 mg/day of prednisolone might have enhanced the compromised condition.

PD-related peritonitis caused by VGS is not rare and most patients do not develop severe illness. Common signs and symptoms of PD-related peritonitis are cloudy peritoneal fluid, abdominal pain, and gastrointestinal symptoms and fever, but not shock state. Appropriate antibiotics can resolve
Figure 2. Clinical course in the present case. This figure shows the time course of blood temperature (BT), C-reactive protein (CRP), white blood cell counts in peripheral blood (WBC) and in peritoneal fluid (PD WBC). The top of this figure indicates the usage of antibiotics. VCM: 1 g/time of vancomycin hydrochloride, CLDM: 2,700 mg/day of clindamycin, AZT: 1.5 g/day of aztreonam, DOA/NAD: dopamine hydrochloride/norepinephrine.

Most cases of PD-peritonitis, although generalized peritonitis from perforation of gastrointestinal, such as peptic ulcer, appendicitis, diverticulitis, cholecystitis, ischemic colitis and malignancy, is usually severe and sometimes requires emergency operation. However, the present case suggests that PD-related peritonitis could be a severe, emergency complication among patients on PD. We therefore have to recognize that PD-related peritonitis may result in a fulminant state shortly after onset, and a quick diagnosis and immediate start of appropriate pharmacotherapy is essential to optimize the prognosis in PD patients.

The authors state that they have no Conflict of Interest (COI).

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References


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