

CASE REPORT

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# Peritoneal dialysis-associated polymicrobial peritonitis with slow onset after root canal treatment: the first case and review of the literature

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## Abstract

**Background** Peritoneal dialysis (PD)-associated peritonitis is linked to an increased risk of mortality and catheter removal, with a higher incidence of these risks observed in polymicrobial peritonitis compared with single-organism infection. In PD patients, invasive procedures can cause peritonitis, typically within 7 days, through transient bacteremia. Although dental procedures are widely recognized as a cause of transient bacteremia, only a limited number of cases involving PD-associated peritonitis after dental procedures, and no cases of polymicrobial peritonitis, have been reported.

**Case presentation** A 60-year-old man undergoing PD presented with acute low abdominal pain, and was diagnosed with PD-associated peritonitis caused by *Streptococcus (S.) oralis*, *S. vestibularis*, and *S. salivarius*. The polymicrobial peritonitis was successfully treated with antibiotics and catheter removal was not required. Medical consultation after admission revealed a history of root canal treatment for dental caries in the right maxillary second molar, and a dental examination during hospitalization confirmed its success.

**Conclusions** We report a case of PD-associated peritonitis caused by co-infection with three species of viridans group streptococci, which developed 9 days after the completion of root canal treatment. This case history suggests that it may be important to carefully observe patients until 10 days after dental procedures, because of the slow onset of peritonitis following such procedures.

**Keywords** Viridans group Streptococci, *Streptococcus oralis*, *Streptococcus vestibularis*, *Streptococcus salivarius*, Peritoneal dialysis, Peritoneal dialysis-associated peritonitis

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## Background

Peritoneal dialysis (PD)-associated peritonitis, a common and serious complication of PD, is associated with a higher risk of mortality [1–3]. It frequently leads to a reduction in peritoneal ultrafiltration capacity, and is the most common cause of permanent hemodialysis transfer [4]. Polymicrobial infection occurs in 16% of patients with PD-associated peritonitis, accounting for 6–8% of peritonitis episodes [5, 6]. Catheter removal is required in 22–33% of polymicrobial infections, resulting in transfer to hemodialysis [5, 6]. Thus, although polymicrobial peritonitis is relatively uncommon, the risk of catheter removal is higher compared with single-organism infection [6].

The end-stage kidney disease (ESKD) population has severe oral disease compared with the general population [7]. Oral diseases represent a potential but preventable cause of poor health outcomes in people with ESKD because of their relationship to infection, inflammation, and malnutrition. A recent large multicenter study revealed that poor oral hygiene was present in a quarter of PD patients and was independently associated with a higher risk of peritonitis and death [8]. Furthermore, dental or oral procedures are widely recognized as a

cause of transient bacteremia. In PD patients, transient bacteremia after invasive procedures can lead to peritonitis, usually within 7 days [9–13]. To date, a limited number of cases involving PD-associated peritonitis after dental procedures have been reported [14, 15]; however, none of these cases has developed peritonitis after root canal treatment, a known cause of transient bacteremia [16].

Approximately 5–10% of all PD-associated peritonitis cases are caused by *Streptococcus* species, which are the most frequently isolated organisms from odontogenic bacteremic patients after dental procedures [9, 17]. *Streptococcus* (*S.*) *viridans* is the predominant organism observed in PD patients with post-dental procedure peritonitis [9, 15] because viridans group streptococci (VGS) normally reside in the oral cavity. Although some cases exhibited polymicrobial peritonitis with *S. viridans*, no cases have been reported after oral or dental procedures. We report a case of PD-associated peritonitis caused by co-infection with three species of VGS, *S. oralis*, *S. vestibularis*, and *S. salivarius*, 9 days after the completion of root canal treatment. This polymicrobial peritonitis was successfully treated with antibiotics.

## Case presentation

A 60-year-old Japanese man with ESKD caused by autosomal dominant polycystic kidney disease had been undergoing PD for 1 year, and was admitted to our hospital with acute low abdominal pain. He was being treated with continuous cycling PD: automated PD during the night using 1.5% glucose solution (Reguneal LCa 1.5%; Baxter, Tokyo, Japan) and daytime use of 7.5% icodextrin solution (Extraneal; Baxter). He used an ultraviolet light-based tubing welder (Tsunagu; Baxter) to connect and disconnect dialysate bags. His medical history included hypertension, dyslipidemia, and ischemic heart disease, but not diabetes mellitus. He was regularly taking diltiazem, alfacalcidol, sacubitril valsartan, vonoprazan, atorvastatin, furosemide, torsemide, and tolvaptan. One year before presentation, he had PD-associated peritonitis caused by *Enterobacter cloacae*, which was successfully treated with antibiotics. His daily living activities were unremarkable, and he did not smoke or regularly consume alcohol.

On admission, his blood pressure was 102/64 mmHg, respiratory rate 12 per minute, heart rate 76 per minute, temperature 36.3 °C, and oxygen saturation 98% on ambient air. His height was 170 cm, weight 60.8 kg, and body mass index 21. The physical examination revealed abdominal distension and tenderness throughout, with no apparent muscular defense or recoil pain. There were no findings suggestive of an exit site or tunnel infection. Laboratory findings are shown in Table 1. His white blood cell count was 5,690/ $\mu$ L, and serum C-reactive

**Table 1** Laboratory characteristics of the patient on admission

Parameter	Value	(normal range)
(Blood)		
White blood cell ( $\mu$ L)	5690	(3040–8540)
Neutrophil (%)	64.4	(38.3–71.1)
Lymphocyte (%)	25.5	(21.3–50.3)
Monocyte (%)	6.9	(2.7–7.6)
Red blood cell ( $10^4$ / $\mu$ L)	382	(378–499)
Hemoglobin (g/dL)	13.5	(10.8–14.9)
Hematocrit (%)	39.6	(35.6–45.4)
Platelet ( $10^4$ / $\mu$ L)	33	(15.0–36.0)
Aspartate transaminase (U/L)	21	(13–33)
Alanine transaminase (U/L)	23	(8–42)
Lactate dehydrogenase (U/L)	332	(124–222)
Alkaline phosphatase (U/L)	127	(38–113)
$\gamma$ -Glutamyltransferase (U/L)	49	(13–64)
Total bilirubin (mg/dL)	0.3	(0.4–1.5)
Serum albumin (g/dL)	2.6	(4.0–5.0)
Blood urea nitrogen (mg/dL)	64.6	(8–20)
Creatinine (mg/dL)	7.37	(0.40–0.70)
Na (mmol/L)	133	(138–146)
K (mmol/L)	3.8	(3.6–4.9)
Cl (mmol/L)	97	(99–109)
Calcium (mg/dL)	9.8	(8.6–10.4)
Phosphate (mg/dL)	5.8	(2.5–4.7)
Uric acid (mg/dL)	7.1	2.3–7.0)
C-reactive protein (mg/dL)	0.05	(< 0.20)
(Peritoneal dialysis effluent)		
White cell count ( $\mu$ L)	7500	Negative
Neutrophil (%)	93.2	Negative

protein (CRP) concentration was 0.05 mg/dL. The PD effluent was cloudy, and the white cell count was elevated at 7,500/ $\mu$ L (neutrophils, 93.2%), resulting in a diagnosis of PD-associated peritonitis. Because the previous peritonitis caused by *Enterobacter cloacae* was resistant to third-generation cephalosporins, intravenous administration of cefepime was started. The patient's white blood cell count (9,170/ $\mu$ L) and CRP level (23.03 mg/dL) were elevated on treatment day 2, but decreased to 6,000/ $\mu$ L and 5.20 mg/dL on treatment day 6, respectively (Fig. 1). Similarly, the white cell count in the PD effluent was elevated at 9,750/ $\mu$ L on treatment day 2, but decreased to 230/ $\mu$ L on treatment day 6 (Fig. 1). Contrast-enhanced computed tomography of the abdomen did not reveal any abscesses in the abdominal cavity or around the PD catheter. Transthoracic echocardiography revealed no vegetation or valvular regurgitation.

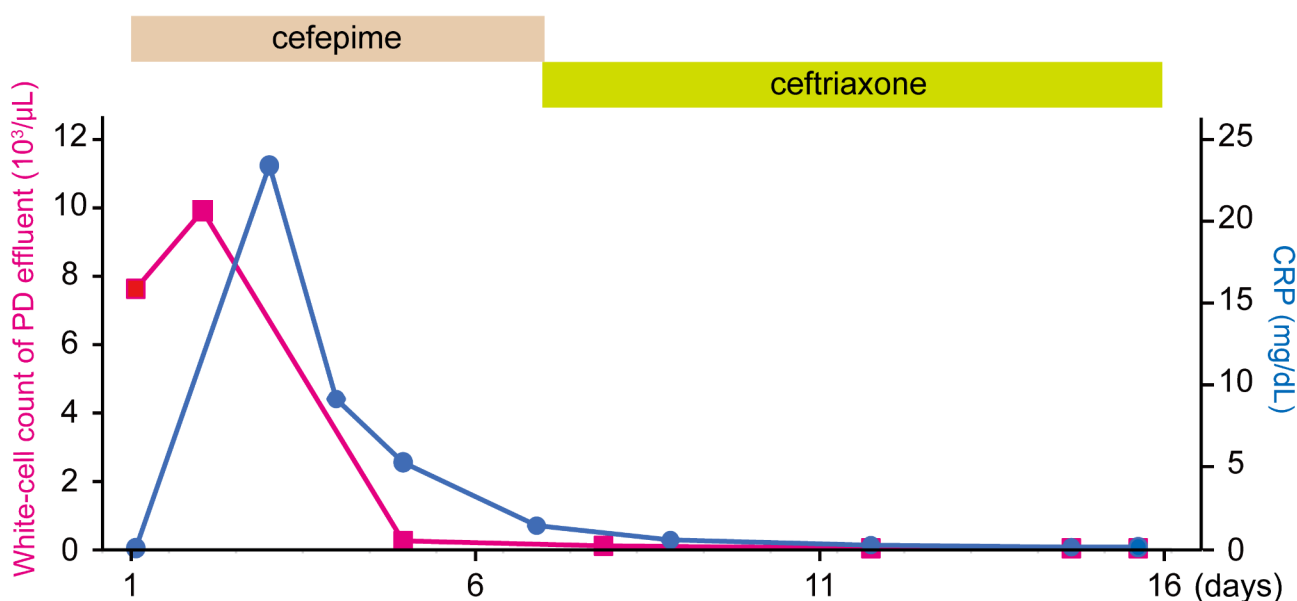
*S. oralis* and *S. salivarius* were detected on treatment day 1, and *S. vestibularis* was detected on treatment day 5 in the PD effluent culture. Two sets of blood cultures obtained before starting the antibiotic therapy tested negative. To elucidate the infection route, the patient was asked about his dental history. He revealed that 2 weeks before the hospitalization, he had visited a dentist because of toothache in the right maxillary second molar; root canal treatment had been initiated and was completed 9 days before admission. No prophylactic antibiotics had been administered. A dental examination during hospitalization confirmed the oral cavity was clean, with no observed dental caries and the success of the root canal treatment. Because the three *Streptococcus* species are susceptible to a wide range of antibiotics (Table 2), cefepime was de-escalated to ceftriaxone on day 8

(Fig. 1). The white cell count in the PD effluent decreased to less than 100  $\mu$ L on day 8, and did not worsen thereafter (Fig. 1). In line with the recommendations of the 2022 International Society for Peritoneal Dialysis (ISPD) guidelines [18], the patient was treated with antibiotics for a total of 2 weeks. No deterioration in the clinical findings was observed, and he was discharged on day 16.

## Discussion and conclusions

Viridans group streptococci are divided into five different groups: mutans, anginosus, mitis, sanguinis, and salivarius [19]. *S. oralis*, a virulent member of the mitis group, is known to be a common pathogen of endocarditis, while *S. salivarius* and *S. vestibularis*, members of salivarius group, are less common but can be isolated in patients with endocarditis [20–22]. To date, eleven cases of PD-associated peritonitis caused by mono-infection of *S. oralis*, *S. salivarius*, or *S. vestibularis* have been reported (Table 3), and among them, only one case was peritonitis following a dental procedure [14, 23–31]. Therefore, this is the first reported case of PD-associated polymicrobial peritonitis caused by co-infection with *S. oralis*, *S. salivarius*, and *S. vestibularis*.

Although touch contamination is the main cause of PD-associated peritonitis due to VGS [32], other sources, including oral, respiratory, and gastrointestinal routes, should also be considered, especially in patients with dental disease or recent procedures [11, 27]. In this case, the patient developed peritonitis after the completion of root canal treatment for dental caries, and dental examination confirmed its success. While two sets of blood cultures were negative, bacteria that enter the bloodstream during dental procedures are known to be rapidly



**Fig. 1** Clinical course of the patient. Changes in the white-cell count of PD effluent (pink lines) and C-reactive protein (CRP, blue lines)

**Table 2** Antibiotic susceptibility test results of three Streptococcal subspecies

Antibiotic	<i>S. oralis</i>		<i>S. salivarius</i>		<i>S. vestibularis</i>	
	MIC (μg/mL)	Susceptibility	MIC (μg/mL)	Susceptibility	MIC (μg/mL)	Susceptibility
Penicillin G	≤ 0.03	S	0.5	I	0.5	I
Ampicillin	≤ 0.12	S	0.5	I	0.5	I
Cefazolin	0.12	S	1	I	1	I
Cefotiam	0.5	S	> 2	R	> 2	R
Ceftriaxone	≤ 0.06	S	1	S	1	S
Imipenem	≤ 0.12	S	≤ 0.12	S	≤ 0.12	S
Meropenem	≤ 0.03	S	0.12	S	0.12	S
Sulbactam/Ampicilin	≤ 0.12	S	0.5	I	0.5	I
Clarithromycin	1	R	2	R	2	R
Clindamycin	0.12	S	0.06	S	≤ 0.03	S
Vancomycin	≤ 0.5	S	≤ 0.5	S	≤ 0.5	S
Ciprofloxacin	> 2	R	2	I	1	S
Levofloxacin	2	S	2	S	1	S

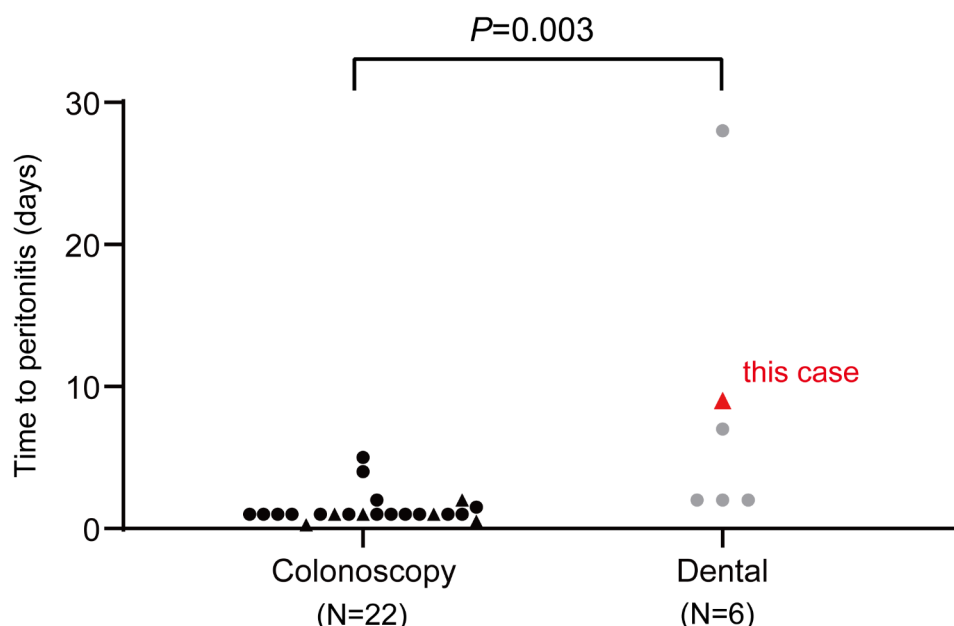
**Table 3** Clinical outcome of three Streptococcal subspecies

Author	Year	Age	Sex	Species	Dental procedures	Catheter loss	Recurrence
Koruk ST [25]	2005	N/A	N/A	<i>S. oralis</i>	-	-	-
Amirou M [26]	2012	77	M	<i>S. oralis/mitis</i>	-	-	+ <i>S. parasanguis</i>
Kotani A [27]	2021	77	M	<i>S. oralis</i>	-	-	-
Mihara Y [14]	2023	60	M	<i>S. oralis</i>	+	-	-
Mizuno M [31]	2011	54	F	<i>S. mitis</i>	-	-	-
Mert M [30]	2021	32	F	<i>S. mitis</i>	-	-	-
Mert M [30]	2021	72	F	<i>S. mitis</i>	-	-	-
Yilmaz F [29]	2017	58	F	<i>S. vestibularis</i>	-	-	-
Kasıkçı E [28]	2018	49	M	<i>S. vestibularis</i>	-	-	-
Barajas-Colon E [24]	2021	0	M	<i>S. salivarius</i>	-	-	-
Chaker H [23]	2021	42	M	<i>S. salivarius</i>	-	-	-

cleared within 15 min [33], leaving the possibility that transient bacteremia following root canal therapy may have caused the PD-associated peritonitis. This possibility is supported by the isolation of *S. salivarius* from the PD effluent culture because this microorganism is rarely responsible for any infections in adults except dental caries [19]. In this case, during PD exchanges, the patient had used not only a mask, gloves, but also an ultraviolet light-based PD catheter connection system, which produces a germicidal effect [34] and significantly decreases the incidence of peritonitis [35]. This is consistent with a recent report showing that PD-associated peritonitis caused by *S. oralis* developed after a tooth extraction [14]. Although the possibility of contamination by saliva droplets cannot be completely excluded, we hypothesize that, in our case, transient bacteremia occurred following root canal therapy, leading to PD-associated polymicrobial peritonitis with three species of VGS.

Our patient developed PD-associated peritonitis 9 days after the final dental procedure. In previous cases of PD-associated peritonitis, no cases occurred within 1 day of a dental procedure (Fig. 2) [14, 15]. In contrast, PD-associated peritonitis often occurs within 1 day of colonoscopy

(Fig. 2) [12, 13, 36–43]. Thus, the onset of peritonitis after dental procedures is slower than that after colonoscopy (Fig. 2). The present case can be considered to be unique in that the onset was slower than that of other cases of peritonitis caused by dental procedures. Although one case reported the development of peritonitis 4 weeks after a tooth extraction, prophylactic antibiotic treatment had been administered from 2 days before the procedure. Additionally, wound healing was delayed until the peritonitis developed [14]; thus, delayed wound healing might be a more reasonable explanation for the development of peritonitis than the tooth extraction itself. Similarly, we speculate that transient bacteremia occurred during the wound healing process in this case, although dental procedures had been completed at the dental examination. Colonoscopy-related peritonitis is defined as peritonitis developing within 1 day or 1 week of colonoscopy [44–46]. Therefore, it is important to carefully observe PD patients for up to 10 days after dental procedures, even if the wound is healing well. Furthermore, although the onset of dental procedure-related peritonitis is slow, if peritonitis occurs more than 10 days after the



**Fig. 2** Late onset of peritonitis after dental procedures. The data of time to peritonitis were obtained from the previous reports showing PD-associated peritonitis after colonoscopy [12, 13, 36–43] or dental procedures [14, 15]. Comparison was analyzed using the two-tailed t test. Cases of polymicrobial peritonitis were plotted as closed tri-angles. Cases of peritonitis with single-organism or culture-negative were plotted as closed circles. This case is indicated by red closed tri-angle

dental procedure, other reasons for the infection should be investigated.

Prophylactic antibiotic treatment was not administered in our case, although root canal therapy is a known cause of transient bacteremia [16]. One possible reason is that the exact frequency of peritonitis associated with the transient bacteremia from dental procedures and the efficacy of antibiotic prophylaxis to prevent peritonitis have not yet been definitively established. This contrasts with the increasing evidence supporting antibiotic prophylaxis in PD patients undergoing colonoscopy [12, 18, 44]. Indeed, the 2022 ISPD guidelines state only that clinical trials are required to assess the benefits and harms of antibiotic prophylaxis before dental procedures [18]. Similarly, the American Dental Association currently lacks specific guidelines for antibiotic prophylaxis in PD patients, as discussed in a previous review [47]. Transient bacteremia after dental procedures is common and antibiotic prophylaxis has been shown to reduce its frequency [48], thereby decreasing persistent bacteremia and endocarditis, especially in patients at high risk [49]. In PD patients, transient bacteremia following dental disease or recent procedures can lead to PD-associated peritonitis even without persistent bacteremia [11, 27], highlighting the importance of reducing its occurrence through antibiotic prophylaxis, as PD patients are at high risk of peritonitis. Therefore, it seems reasonable for PD patients to be given antibiotic prophylaxis before undergoing dental surgery, as stated in the 2016 ISPD guidelines [50]. Only 6 cases of PD-associated peritonitis have

been associated with dental or oral procedures, including our case [9, 14]. Hence, further case reports and clinical studies are required to evaluate the precise frequency and the efficacy of antibiotic prophylaxis prior to dental procedures in PD patients.

In our patient, polymicrobial peritonitis was clearly improved by antibiotics, and catheter removal was not required. Similarly, there have been no reported cases of catheter removal and relapse/recurrence in patients with PD-associated peritonitis caused by *S. orlans*, *S. salivarius*, or *S. vestibularis* (Table 3) [14, 23–31]. This is consistent with a previous paper showing a low risk of catheter loss (2.6%) in patients with single-organism VGS group infection [51]. Although catheter loss overall was higher in the polymicrobial peritonitis group with VGS (16.1%) [51], this finding is similar to another study demonstrating that 17% of patients with polymicrobial peritonitis with pure gram-positive organisms required catheter removal [52]. Therefore, it is crucial to closely monitor patients with polymicrobial peritonitis compared with those with single-organism VGS, although pure gram-positive polymicrobial peritonitis has less impact on catheter removal than other types of polymicrobial peritonitis, including mixed gram-negative and gram-positive, pure gram-negative, or mixed bacterial and fungal organisms [52].

In conclusion, we report a case of PD-associated polymicrobial peritonitis with *S. oralis*, *S. salivarius*, and *S. vestibularis*, which may have been caused by transient bacteremia after a dental procedure. Because the onset of peritonitis after dental procedures is slower than that



after colonoscopy, it is important to carefully observe the patient until 10 days after the dental procedure. This case supports the need for further studies evaluating the use of prophylactic antibiotics prior to dental procedures in PD patients.

# Abbreviations

CRP	C-reactive protein
ESKD	End stage kidney disease
PD	Peritoneal dialysis
S.	Streptococcus
UV	Ultraviolet
VGS	Viridans group streptococci

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# Author contributions

S.K., Y.M., K.O., R.Y., and A.T. treated the patient. S.K. and Y.M. drafted the manuscript. S.K., Y.M., A.T., M.Y., N.I., Y.O., K.S. and T.M. revised the manuscript critically. All authors read and approved the final manuscripts.

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# Data availability

Data is provided within the manuscript.

# Declarations

# Ethics approval and consent to participate

This study was conducted according to the guidelines laid down in the Declaration of Helsinki. Informed consent was obtained from the patient described in this case report, and the consent allowed their data to be stored, as required by the Hiroshima University Hospital.

# Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

# Competing interests

The authors declare no competing interests.

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