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CASE REPORT

Vancomycin-resistant enterococci peritonitis development after shifting from hemodialysis to peritoneal dialysis

Chih-Ming Chang, Chih-Chia Liang, Hung-Chieh Yeh, Ya-Fei Yang, I-Kuan Wang, Hsin-Hung Lin, and Chiu-Ching Huang

Department of Medicine, Division of Nephrology and Kidney Institute, China Medical University Hospital, Taichung, Taiwan

Abstract

Although hemodialysis (HD) patients could develop vancomycin-resistant enterococci (VRE) infection, limited studies of VRE-related peritoneal dialysis (PD) peritonitis have been reported. Here, we document a patient who developed peritonitis for the first time with VRE infection after shifting from HD to PD over 4 weeks. We suggest that physicians consider VRE a possible cause of PD peritonitis in patients who have recently shifted from HD to PD, especially in cases involving previous vancomycin exposure.

Keywords: peritoneal dialysis, hemodialysis, peritonitis, vancomycin, vancomycin-resistant enterococci

INTRODUCTION

Vancomycin-resistant enterococci (VRE) is a key source of nosocomial infection. Although hemodialysis (HD) patients could develop VRE infection, limited studies of VRE-related peritoneal dialysis (PD) peritonitis have been reported.[1–4] Here, we document a patient who developed peritonitis for the first time with VRE infection after shifting from HD to PD over 4 weeks. We suggest that physicians consider VRE a possible cause of PD peritonitis in patients who have recently shifted from HD to PD, especially in cases involving previous vancomycin exposure.

CASE REPORT

A 66-year-old woman with a history of end-stage renal disease, who had been on HD for 2 years, was admitted in September 2009. She presented with fever along with aspiration pneumonia and suspected arteriovenous fistula infection. During hospitalization, intravenous vancomycin (1 g/5 days) was given from day 6 to day 22. During this interval, ertapenem was given for 1 week, along with Tazocin for 10 days and cefepime for another 1 week applied intravenously. Blood and sputum cultures did not yield bacteria. Because of chronic intradialytic hypotension, she underwent Tenckhoff catheter implantation on day 12 while she was afebrile. She was shifted from HD to PD 5 days later and was discharged on day 25 with smooth continuous ambulatory peritoneal dialysis (CAPD) therapy for 10 days.

About 2 weeks later, she was admitted for PD peritonitis with shock. Laboratory data showed systemic leukocytosis (WBC 18,250/μL) with 86.6% segment form of white cells. PD dialysate showed WBC 2637/μL with 88% polymononuclear neutrophils (PMN). Empiric antibiotics (vancomycin: 1 dose, 1 g, intravenously; cefepime: 1 g intravenous per day) were used. Another dose of vancomycin (1 g, intraperitoneal) was applied on day 3. Because of increased leukocytosis in dialysate (WBC: 8227/μL, PMN: 97%) on day 4, daptomycin (6 m/kg, every other day, intravenous) was given and the CAPD therapy was halted and shifted to sustained low-efficiency daily diafiltration. Bacterial culture of the dialysate and blood yielded vancomycin-resistant *Enterococcus faecium* (VREF). The Tenckhoff catheter was removed on day 7. The patient expired on day 10 because of profound septic shock. Bacterial culture from the removed catheter also yielded VREF.

Address correspondence to Hsin-Hung Lin, MD, Department of Medicine, Division of Nephrology and Kidney Institute, China Medical University Hospital, No. 2 Yuh-Der Road, Taichung 404, Taiwan. Tel.: 886-4-2251-8965; fax: 886-4-2203-8883; E-mail: lsh3056m@ms27.hinet.net

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DISCUSSION

Limited studies of VRE-related PD peritonitis have been reported.[1–4] A cross-sectional survey of 320 patients[1] on long-term dialysis therapy showed the prevalence of VRE was 14.4% from positive fecal culture. There were significant associations between VRE and the dialysis type (HD) and the number of hospitalizations. Factors that predispose for VRE colonization or infection include a high percentage of hospital days receiving antimicrobial therapy (especially vancomycin), HD, and abdominal surgery.[1,5] In our reported case, the patient had risk factors for VRE including prolonged hospital stay under antimicrobial therapy, particularly vancomycin exposure, previous HD dependence, and recent abdominal surgery with Tenckhoff catheter implantation. In general, VRE-related PD peritonitis always developed after intraperitoneal antibiotics for previous PD peritonitis.[2] However, our patient developed peritonitis for the first time with VRE infection after shifting from HD to PD. VRE in the stool culture was found in this patient. It might be reasonable to consider that the previous exposure of vancomycin in this chronic HD patient made the VRE colonization in her gastrointestinal tract. We think the possible infection route of the VRE peritonitis was through the gastrointestinal tract translocation. Should we examine VRE colonization on patients before or after shifting from HD to PD therapy, especially if previously exposed to vancomycin? Further study may be considered.

VRE-related PD peritonitis outcome may be poor in regard to mortality and PD therapy continuation. One case study of nine patients[2] showed high patient mortality rate (55.5%) and PD discontinuation rate (77.7%), though the case number was too small. The International Society for Peritoneal Dialysis (ISPD) guidelines in 2005 described VRE-related PD peritonitis was uncommon and limited data were available for proper management.[6] Intravenous linezolid[3] or intraperitoneal daptomycin[4] was ever reported to be effective to treat it.

In conclusion, we suggest that physicians note VRE as a possible pathogen for PD peritonitis in patients who have just shifted from HD to PD, especially if previous exposure to vancomycin is involved.

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

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