

***Enterococcus casseliflavus* and *Enterococcus gallinarum* as causative agents of spontaneous bacterial peritonitis**

Janaína Luz Narciso-Schiavon, Ariane Borgonovo, Paula Couto Marques, Débora Tonon, Emilia Tiemi Oshiro Bansho, Dariana Carla Maggi, Esther Buzaglo Dantas-Corrêa, Leonardo de Lucca Schiavon

Department of Internal Medicine, Gastroenterology Division, Federal University of Santa Catarina, Florianopolis, SC, Brazil.

ABSTRACT

Infection by multidrug resistant bacteria is arousing as a relevant issue among hospitalized subjects and is of particular interest in patients with cirrhosis given the frequent use of broad spectrum antibiotics and their altered immune response. We report the first case report of spontaneous bacterial peritonitis (SBP) caused by *Enterococcus casseliflavus* and the sixth case of SBP caused by *Enterococcus gallinarum*.

Key words. VanC protein. *Enterococcus*. Ascites. Liver cirrhosis.

INTRODUCTION

Infection by multidrug resistant bacteria is arousing as a relevant issue among hospitalized subjects and is of particular interest in patients with cirrhosis given the frequent use of broad spectrum antibiotics and their altered immune response. We recently assisted two patients with positive ascitic fluid culture for the vancomycin-resistant enterococci (VanC genotype) *Enterococcus casseliflavus* and *Enterococcus gallinarum*. Although positive blood cultures for this *Enterococci* have been reported in cirrhotic individuals,¹ this is the first case report of spontaneous bacterial peritonitis (SBP) caused by *Enterococcus casseliflavus* and the sixth case of SBP caused by *Enterococcus gallinarum*.²⁻⁵

CASE REPORT

A 56-year-old man with alcoholic cirrhosis Child-Pugh C, MELD 17, was admitted to the gas-

troenterology ward for malnutrition. He had been submitted to hepatocellular carcinoma resection ten months before and had a malfunctioning transjugular portosystemic shunt. He had received in-hospital vancomycin and meropenem for positive blood cultures for *Staphylococcus epidermidis* and *Serratia marcescens*, respectively. On the fiftieth day of admission he presented both ascitic fluid and blood cultures positive for *Enterococcus casseliflavus*. The following laboratory results were obtained:

- Hemoglobin 10.3 g/dL.
- Leukocytes 1,8150/mm³.
- Neutrophils 15,826/mm³.
- Platelet count 172,000/mm³.
- Total bilirubin 0.7 mg/dL.
- Creatinine 1.8 mg/dL, and international normalized ratio 1.51.
- Albumin 2.6 g/L.
- Sodium 131 mEq/L.

On the ascitic fluid, albumin was 1.8 g/L, neutrophils 3,571/mm³. As meropenem had been empirically introduced before culture results and 48-h ascitic fluid total cell count was of 46 cells/mm³, the antibiotic was maintained and the patient underwent liver transplantation soon after. His recovery after liver transplantation was uneventful and he is now on outpatient follow-up.

A 63-year-old woman with cirrhosis related to non-alcoholic steatohepatitis, Child-Pugh C, MELD

Correspondence and reprint request: Prof. Janaína Luz Narciso-Schiavon, M.D., Ph.D.

Departamento de Clínica Médica. Hospital Universitário Polydoro Ernani de São Thiago, 3º andar. Universidade Federal de Santa Catarina (UFSC), Rua Professora Maria Flora Pausewang s/no, Trindade, Florianópolis-SC, 88040-900, Brazil.

Ph.: (+55 48) 37219149. Fax: (+55 48) 37219040
E-mail: janaína.narciso@uol.com.br

Manuscript received: July 13, 2014.

Manuscript accepted: August 18, 2014.

15, was admitted to the gastroenterology ward for abdominal pain and worsening of the ascites. On admission she presented positive ascitic fluid culture for *Enterococcus gallinarum*. The following laboratory results were obtained:

- Hemoglobin 12.7 g/dL.
- Leukocytes 9,810/mm³.
- Neutrophils 7,269/mm³.
- Platelet count 132,000/mm³.
- Total bilirubin 3.1 mg/dL.
- Creatinine 0.9 mg/dL
- International normalized ratio 1.52.
- Albumin 1.9 g/L.
- Sodium 132 mEq/L.

On the ascitic fluid, albumin was 0.3 g/L, neutrophils 2,610/mm³. Patient received gentamicin and ampicilin for 7 days and was discharged for outpatient follow-up.

DISCUSSION

E. gallinarum and *E. casseliflavus* strains have a specific chromosomal vanC gene that leads to low-level vancomycin resistance,⁴ and their clinical significance is not yet fully established.⁶ They are infrequently recovered from clinical specimens but may cause serious invasive infections.⁴ *E. gallinarum* and *E. casseliflavus* are components of the normal intestinal flora of the general population; this hinders the identification of risk factors for their transmission.^{4,7} Nevertheless, the use of antibiotics such as third-generation cephalosporins, quinolones, penicillins, carbapenems, aminoglycosides and vancomycin may play a role in increasing colonization with these organisms.^{4,7} In the intensive care unit, colonization by *E. gallinarum* and *E. casseliflavus* has been reported in up to 23% of the patients, and is associated with prior carbapenem use and the presence of nephropathy.⁸ Patients often present serious underlying diseases, and manifest severe invasive illness with variable mortality rates, ranging from 13 to 40%.^{2,6} As VanC VREs are not usually transmitted between patients, contact precautions are not routinely recommended.² A study assessing the antimicrobial *in vitro* susceptibility of enterococcus showed that both *E. gallinarum* and *E. casseliflavus* were sensitive to penicillin, ampicilin, imipenem and teicoplanin.⁹ Enterococcus isolated in both of our cases exhibited *in vitro* sensitivity to ampicilin and gentamicin (with high MIC). The

other previous reported cases of SBP and bacteascites by *E. gallinarum* were treated with amoxicilin³ and ampicilin,² respectively.

In conclusion, *E. gallinarum* and *E. casseliflavus* are uncommon but important agents involved in SBP and bacteascites. Clinicians need to be alerted to the possibility that vancomycin may not be effective against *E. gallinarum* and *E. casseliflavus* and antibiotics choice should be based on antibiogram results.

DISCLOSURE STATEMENT

The authors have no conflicts of interest to declare.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

FINANCIAL SUPPORT

No financial support.

REFERENCES

1. Choi SH, Lee SO, Kim TH, Chung JW, Choo EJ, Kwak YG, Kim MN, et al. Clinical features and outcomes of bacteraemia caused by *Enterococcus casseliflavus* and *Enterococcus gallinarum*: analysis of 56 cases. *Clin Infect Dis* 2004; 38: 53-61.
2. Alvarez MA, Domenech E, Rosinach M, Lorenzo-Zuniga V, Montoliu S, Planas R. *Enterococcus gallinarum* bacteascites in a patient with active tuberculosis and HCV cirrhosis. *Am J Gastroenterol* 2002; 97: 2681-2.
3. Redondo-Cerezo E, Lopez FN, Tapia M, Blanco FS, Garcia RM, Martin-Vivaldi R. *Enterococcus gallinarum* spontaneous bacterial peritonitis in an HCV cirrhotic woman. *Am J Gastroenterol* 2002; 97: 214-5.
4. Toye B, Shymanski J, Bobrowska M, Woods W, Ramotar K. Clinical and epidemiological significance of enterococci intrinsically resistant to vancomycin (possessing the vanC genotype). *J Clin Microbiol* 1997; 35: 3166-70.
5. Reuken PA, Pletz MW, Baier M, Pfister W, Stallmach A, Bruns T. Emergence of spontaneous bacterial peritonitis due to enterococci-risk factors and outcome in a 12-year retrospective study. *Aliment Pharmacol Ther* 2012; 35: 1199-208.
6. Reid KC, Cockerill IF, Patel R. Clinical and epidemiological features of *Enterococcus casseliflavus/flavescens* and *Enterococcus gallinarum* bacteraemia: a report of 20 cases. *Clin Infect Dis* 2001; 32: 1540-6.
7. Van Horn KG, Rodney KM. Colonization and microbiology of the motile enterococci in a patient population. *Diagn Microbiol Infect Dis* 1998; 31: 525-30.
8. Batistao DW, Gontijo-Filho PP, Conceicao N, Oliveira AG, Ribas RM. Risk factors for vancomycin-resistant enter-

cocci colonisation in critically ill patients. *Mem Inst Oswaldo Cruz* 2012; 107: 57-63.

9. Gordon S, Swenson JM, Hill BC, Pigott NE, Facklam RR, Cooksey RC, Thornsberry C, et al. Antimicrobial susceptibility patterns of common and unusual species of enterococci causing infections in the United States. Enterococcal Study Group. *J Clin Microbiol* 1992; 30: 2373-8.