

***Streptococcus salivarius* spontaneous bacterial peritonitis in a HIV/HCV-co-infected patient treated with direct antiviral agents[☆]**



Peritonitis bacteriana espontánea por Streptococcus salivarius en paciente con coinfección por VIH-VHC en tratamiento con antivirales de acción directa

Streptococcus salivarius, which is a member of the *Streptococcus* genus, *Viridans* group, is a typical component of the oral microbiota. It is generally considered to be a contaminant, although it also plays a pathogenic role in infective endocarditis as well as some cases of meningitis and bacteraemia in neutropenic patients.¹ We present a clinical case diagnosed within hydropic decompensation.

The case involves a 49-year-old woman with a history of injected drug abuse diagnosed with HIV-1 infection in the 1990s. She received antiretroviral treatment with efavirenz, zidovudine and didanosine, with good immunovirological management (687 CD4⁺/mcl and HIV viral load [VL] < 20 copies/ml). She also had coinfection with hepatitis C virus (HCV) genotype 4, which could never be treated with interferon due to an anxiety-depressive syndrome and a personality disorder. Her liver disease was in a stage of cirrhosis, with an elasticity index of 72 kPa, in follow-up on the cirrhosis unit at the site, with prior episodes of decompensation in the form of uncomplicated ascites (in June 2013) and upper gastrointestinal bleeding due to oesophageal varices (in March 2015). For this reason, she had treatment with spironolactone, furosemide, propranolol and lactulose; she did not receive primary prophylaxis with norfloxacin.

In June 2015, she started treatment with sofosbuvir and ledipasvir for 24 weeks. After 4 weeks, the HCV VL was undetectable, with improvement in the laboratory parameters for hepatocellular dysfunction: albuminaemia 4.4 g/dl, GPT 26 IU/l, GOT 41 IU/l and prothrombin activity 84%. However, the patient reported an increase in waist circumference during the previous week, with a weight gain of 5 kg, without fever and with spontaneous diffuse abdominal pain. Examination revealed signs of ascites and a systemic blood pressure of 64/40 mmHg. The patient was admitted and underwent paracentesis with fluid extraction. Analysis of this fluid showed 800 leukocytes/mcl (60% polymorphonuclear), glucose 129 mg/dl, total proteins 1.03 g/dl and LDH 136 U/l (analysis of the plasma showed glycaemia 118 mg/dl, total proteinemia 6.2 g/dl and plasma LDH 275 U/l). These findings were consistent with spontaneous bacterial peritonitis (SBP). Empirical antibiotic therapy with ceftriaxone was started without suspending direct antiviral agents (DAAs), with gradual management of the patient's haemodynamic parameters and symptoms. *S. salivarius* sensitive to ceftriaxone and quinolones was identified in the blood agar culture of the ascitic fluid using the MALDI-TOF system. Therefore, 5 days of antibiotic treatment were completed, and secondary prophylaxis with norfloxacin was prescribed. From then on, the patient remained stable and achieved a viral response at the end of treatment with DAAs, with optimal immunovirological HIV infection management at all times.

To date, only 8 cases of SBP with isolation of *S. salivarius* have been identified in the literature.^{1–3} All of them involved chronic liver disease of various aetiologies (including HCV infection).² Translocation of the microorganism from the oro-gastrointestinal tract to the peritoneal space with ascites has been postulated as a mechanism by some authors¹; in our patient, the pres-

ence of oesophageal varices with bleeding, and the fact that oesophagoscopy had been performed a few weeks before, may have been more significant.²

Furthermore, the case that we report has some unique features: the event coincided with an improvement in hepatocellular function laboratory data (Child–Pugh score 5), and no fever. In the case series published, the mean Child–Pugh score was 10, and the main initial symptom was fever.² Moreover, it is the only case reported to date that coincided with treatment of HCV infection with the new DAAs and occurred in the context of a coinfection with HIV. The role that DAA therapy may have played in this case is unknown; it was probably just a circumstantial fact. Regarding the contribution of HIV infection, although the patient had good immunovirological management, this infection did add a deficit in cell-mediated immune response to the deficit in humoral immune response characteristic of cirrhosis.²

In these cases it is unclear whether the duration of targeted antibiotic therapy should exceed the 5 days usually indicated for this microorganism.² There is also uncertainty about the possibility that secondary prophylaxis with conventional quinolones increases the risk of recurrence of SBP due to Gram-positive bacteria, or at least fails to prevent it.^{2,4} Therefore, alternatives such as cotrimoxazole could be considered.⁵

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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