Internal and Emergency Medicine (2018) 13:957–958
https://doi.org/10.1007/s11739-018-1851-9

CE - MEDICAL ILLUSTRATION

A rare and threatening complication in a cirrhotic patient

Joana Carvalho e Branco[1] · Vera Anapaz[1] · Liliana Santos[1] · Jorge Reis[1]

Received: 24 March 2018 / Accepted: 4 April 2018 / Published online: 26 April 2018

Case description

We present the case of a 72-year-old man with alcoholic cirrhosis Child–Pugh B (9 points) complicated by esophageal variceal bleeding in the past, hepatic encephalopathy (HE), and ascites. He was admitted to our department for HE. Laboratory analysis showed pancytopenia, INR 1.4, normal liver function tests, serum creatinine 3.56 mg/dL, urea 171 mg/dL, C-reactive protein of 8.39 mg/dL, bilirubin of 3.2 mg/dL and albumin of 2.74 mg/dL. On urinalysis, a leukocyturia was found; and on urine culture, *Escherichia coli* was isolated. Spontaneous bacterial peritonitis was excluded. Facing a decompensated liver cirrhosis Child–Pugh C (10 points) with hepatic encephalopathy in the context of urinary tract infection and acute kidney injury stage 3, he was started on ceftriaxone 2 g/day and albumin (60 g/day). After excluding other etiologies, hepatorenal syndrome type 1 was presumed. Despite initial improvement (creatinine 3.02 mg/dL) with the administration of albumin, 10 days later his creatinine reached a level of 4.9 mg/dL. Terlipressin was instituted in a dose of 1 mg every 6 h, which was optimized to 2 mg every 6 h due to lack of improvement. The day after this dose escalation, he developed skin necrosis on the tip of the first digit of the left foot (Fig. 1a), and cyanosis of all of the fingers of the right foot with initial signs of necrosis of the third, fourth and fifth digits (Fig. 1b). These changes were most likely due to terlipressin-induced skin necrosis; therefore, this medication was immediately stopped. Ischemic features improved in a few days with the complement of surgical debridement.

Image of the feet of the patient where it is visible necrosis of the skin on the tip of the first finger of the left foot (**a**) and cyanosis of all of the fingers of the right with initial signs of necrosis of the third, fourth and fifth fingers (**b**). This necrosis was attributed to terlipressin

Terlipressin is a synthetic analogue of the hormone vasopressin [1, 2]. It promotes vasoconstriction, and has a preferential action on the splanchnic circulation where it lowers portal venous pressure [1]. It has two main indications in patients with portal hypertension: treatment of bleeding esophageal varices and hepatorenal syndrome [1]. Terlipressin is the most studied pharmacological treatment for type 1 hepatorenal syndrome, and is effective in 40–50% of cases [3]. It is generally started at a dose of 1 mg/4–6 h, and increased to a maximum of 2 mg/4–6 h if there is no reduction in serum creatinine of at least 25% compared to the baseline value at day 3 of therapy [3]. It is considered a safe drug, and the most common adverse event, although relatively rare (< 5%), is diarrhea [1]. Ischemic complications are very rare, with a prevalence of less than 2% [1], and there are only 22 reported cases of skin necrosis related to terlipressin administration [2]. The occurrence of skin necrosis has been explained due to the presence of the vasopressin receptor type 1 in the skin and adipocytes besides the splanchnic circulation, kidney and bladder [2]. For treatment, weaning of terlipressin is usually sufficient although sildenafil, a vasodilatator, has been used with success [4].

✉ Joana Carvalho e Branco
cbranco.joana@gmail.com

[1] Serviço de Gastrenterologia, Hospital Professor Doutor Fernando Fonseca, Lisboa, Portugal

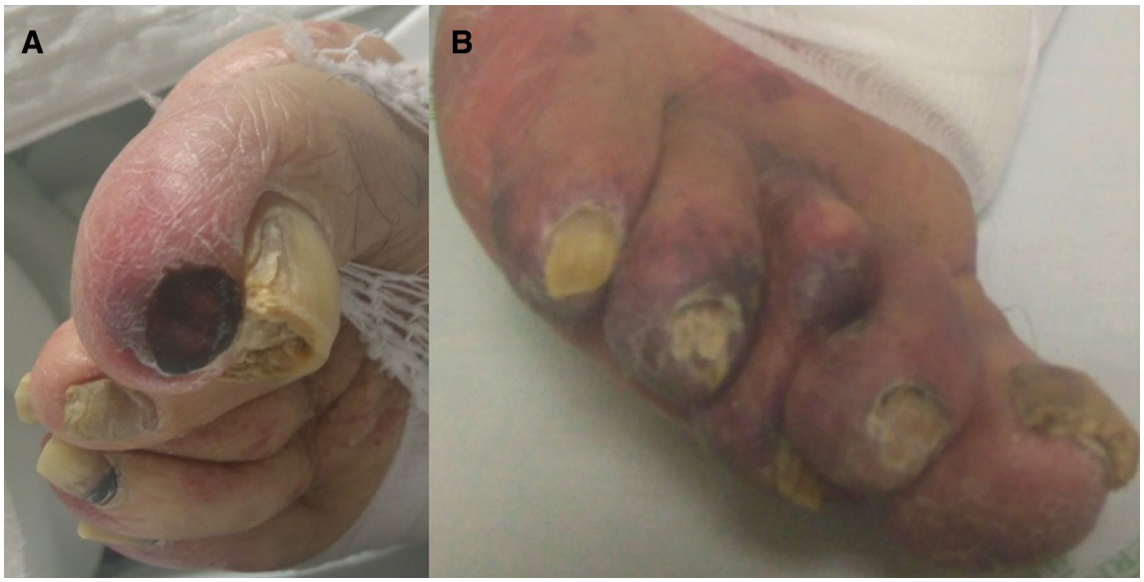

Fig. 1 Image of the feet of the patient where it is visible necrosis of the skin on the tip of the first finger of the left foot (**a**) and cyanosis of all of the fingers of the right with initial signs of necrosis of the third, fourth and fifth fingers (**b**). This necrosis was attributed to terlipressin

Funding This manuscript has no funding sources.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Statements on human and animal rights This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent Informed consent was obtained.

References

1. Moreau R, Durand F, Poynard T et al (2002) Terlipressin in patients with cirrhosis and type 1 hepatorenal syndrome: a retrospective multicenter study. Gastroenterology 122:923–930
2. Coskun B, Karaman A, Gorkem H, Bugday I, Poyrazoglu OK, Senel F (2014) Terlipressin-induced ischemic skin necrosis: a rare association. Am J Case Rep 15:476–479
3. European Association for the Study of the Liver (2010) EASL clinical practice guidelines on the management of ascites, spontaneous bacterial peritonitis, and hepatorenal syndrome in cirrhosis. J Hepatol 53(3):397–417
4. Ramírez DD, Alonso SS, Palma MM (2011) Sildenafil in severe peripheral ischemia induced by terlipressin: a case report. Reumatol Clin 7:59–60