A 50-year-old man was admitted for septic shock secondary to fecal peritonitis after perforation of a strangulated ileum loop. The background included morbid obesity and obstructive sleep apnea syndrome. The patient developed multiorgan failure with a SOFA score of 10. He required noradrenaline up to 1 μg/kg/min and dobutamine 20 μg/kg/min for a mean arterial pressure goal of 75 mmHg. Microbiological isolates from peritoneal exudate were identified as Escherichia coli and Streptococcus galalyticus ssp. pasteurianus. After 24 h he developed cutaneous lesions secondary to severe hypoperfusion (Figure A).

Cutaneous necrosis consists of tissue death due to lack of blood supply. The most frequent cause is vascular occlusion. Risk factors for this include the use of vasopressors, sepsis, acute renal failure, obesity, disseminated intravascular coagulation and peripheral arterial occlusive disease.1

In the present case a high dose of vasopressors was required to maintain adequate mean arterial pressure.

In general, these alterations improve after the progressive withdrawal of vasopressors, although in many cases treatment will require surgery for debridement, amputation of the affected areas or plastic surgery for the reconstruction of the skin defects. The present patient required debridement of the buttocks, while the rest of the lesions improved progressively without intervention (Figure B).

Figure. Necrotic areas in the buttocks, distal ischemia of the right lower extremity and cutaneous necrosis of the abdominal wall. (A) Lesions developed 24 h after admission to the intensive care unit (ICU). (B) Evolution of the lesions upon discharge from the ICU after 35 days.

Disclosures
The authors declare no conflicts of interest.

Reference