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Sclerosing Encapsulated Peritonitis: A devastating and infrequent disease complicating kidney transplantation, case report and literature review

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ABSTRACT

INTRODUCTION: Sclerosing Encapsulating Peritonitis (SEP) is a rare condition with an incidence of up to 3% and a mortality of up to 51% among peritoneal dialysis (PD) patients (Brown et al., Korte et al. and Kawanishi et al.). In the last ten years, the incidence of SEP in kidney transplant recipients has increased (Nakamoto, de Sousa et al. and Korte et al.).
PRESENTATION OF CASE: A 31-year old male with a 15 years history of PD and later kidney retransplantation was admitted to the emergency service after experiencing several weeks of diffuse abdominal pain which had escalated to include vomiting and diarrhea during the 24 h previous to admission.

The patient underwent an exploratory laparotomy where severe peritoneal thickening was found, in addition to signs of chronic inflammation and blocked intestinal loops. Histopathologic findings were suggestive of sclerosing peritonitis. After two months of treatment in hospital, the patient presented an obstructed intestine, with a rigid and thickened peritoneum compromising all the intestinal loops.
DISCUSSION: Despite being rare, SEP, represents a significant complication due to its high mortality and recurrence. It is insidious in its early stages and culminates in an intestinal obstruction (Fieren). Risk factors for its development in kidney transplant recipients include a history of prolonged treatment with PD and the use of calcineurin inhibitors as an immunosuppressive treatment (Korte et al.).
CONCLUSION: Given the increase in the incidence of SEP in kidney transplant recipients, the clinician should be alert to the presence of this complication. A greater number of multi-centre studies are required to identify the risk factors for SEP that are inherent in renal transplant recipients.

1. Introduction

SEP is an uncommon complication of PD with an incidence of 0.7-3.3% and a mortality approaching 51% in the first year [1–3]. It is characterized by a diffuse fibrosis of the peritoneal membrane with an encapsulation of the intestinal loops, causing intestinal obstruction [4]. Although its etiology continues to be non-specific, it is believed that it originates from an injured peritoneal membrane, which, after a second inflammatory stimulus, such as peritonitis, hemoperitoneum or surgery, SEP is triggered [5].

Few cases have been reported involving kidney transplanted patients [6,7], and given the rare nature of this pathology, we decided to publish this case report in line with the SCARE criteria [8] and with emphasis on a review of the literature. To our knowledge, this is the first case report of SEP in a kidney retransplanted patient.

2. Presentation of case

A 31-year-old male patient, with Alport Syndrome, diagnosed at the age of 9, with kidney disease and hypertension who received a kidney transplant at ten years old, from a related living donor. Ten years after the kidney transplant, the patient presented an episode of rejection, and lost the kidney graft; for which he received PD for

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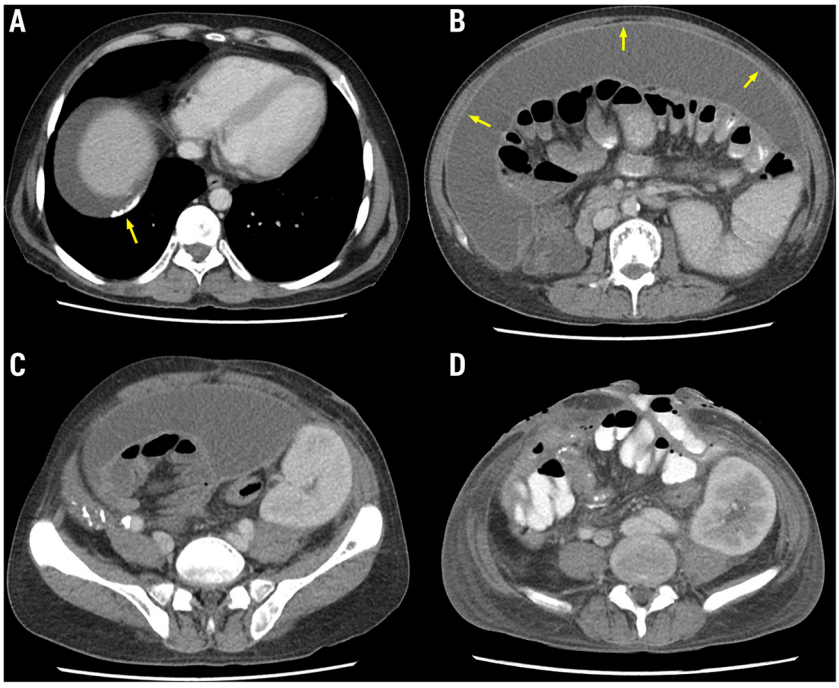

Fig. 1. Axial view of an abdominal enhanced CT. Panel A, right subdiaphragmatic calcified peritoneal plaque. Panel B, progression in the thickening of the peritoneum with contrast enhancement, which englobes the intestinal loops and conditions a discrete small bowel distention with the presence of air-fluid levels, compatible with an initial obstructive process. Panel C, inferior view of the pelvis, with left kidney graft without alteration and atrophic changes of the first right kidney graft. Panel D, postsurgical changes of parietal peritoneum resection with an abdominal wall defect and oral contrast leak compatible with enterocutaneous fistula.

Table 1
Laboratory test results taken in admission.

Glycemia (mg/dL)	92.8
Blood Urea Nitrogen (mg/dL)	26.7
Serum Creatinine (mg/dL)	1,28
Alanine Aminotransferase	6
Aspartate Aminotransferase	9,8
Serum Sodium (mEq/L)	138
Serum Potassium (mEq/L)	4,9
Serum Chloride (mEq/L)	97,4
Serum Calcium (mEq/L)	10
Serum Phosphorus (mEq/L)	3,83
Serum Lipase	28,7
Blood Count	
Leukocytes	12910
Neutrophils (%)	84%
Lymphocytes (%)	3,40%
Hemoglobin (g/dL)	6,9
Hematocrit (%)	24
Platelets	589000

15 years until a deceased kidney transplant donor was found, with 1dr-1a compatibility.

One year after the kidney re-transplant, the patient was admitted after experiencing several days of crampy abdominal pain localized in the epigastrium and mesogastrium, associated with vomiting bile and diarrheic bowel movements. At the moment he was receiving immunosuppressive management with Tacrolimus 7 mg/day, Mycophenolate Sodium 1080 mg/day and prednisolone 5 mg/day.

Physical examination revealed a globose abdomen, painful upon deep palpation, with no signs of peritoneal irritation and with no pain in the area of the transplant. Paraclinicals were taken upon admission (Table 1) and due to suspicion of an intra-abdominal infection, an enhanced abdominal Computed Tomography (CT)) (Fig. 1A and B) was taken which showed evidence of intra-abdominal collections in the right paracolic gutter. These findings

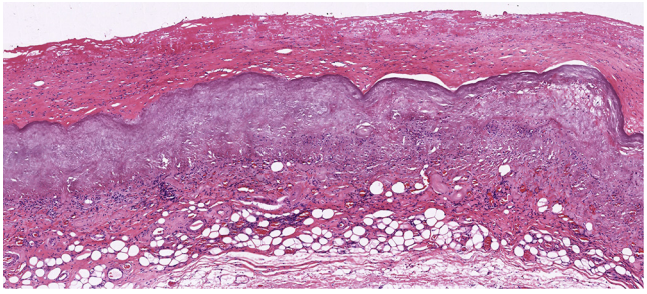

Fig. 2. Colouring of H&E at 200X. In the histological section, there is mesothelial replacement due to a proliferation of fibroblasts and capillaries, accompanied by a chronic and acute inflammatory response with exudation. Subsequently, an acellular layer with calcium deposits is formed and ends with a layer of fibrin.

were suggestive of peritonitis, with indirect signs of intestinal obstruction due to adhesions. It was therefore decided that an exploratory laparotomy should be conducted, which found three liters of cloudy brown liquid, severe thickening of the parietal peritoneum with chronic inflammation, abundant membranes and blocked loops; drainage of the peritonitis was performed, enabling partial release of the adhesions, and a biopsy was taken off the peritoneum. Subsequently, the pathology report revealed chronic peritonitis with histologic findings suggestive of a sclerosing peritonitis (Fig. 2).

Two months later, while still hospitalised, the patient developed a clinical picture of intestinal obstruction and underwent a laparotomy which showed a 100% blocked abdomen, a thickened and stoned peritoneum, compromising the coating of all the intraabdominal organs, with multiple collections. Associated with fibrinopurulent membranes and intestinal obstruction at the level of the distal ileum, involving multiple segments (Figs. 3 and 4). He required an Ileostomy but due to its high output, compromising the patient's hydroelectric balance and nutrition, it was later closed