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Case report

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Hemoperitoneum in peritoneal dialysis, a red flag? Case report.

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Abstract

Hemoperitoneum is a complication of peritoneal dialysis. Its differential diagnosis is broad and the approach is based on its clinical manifestation and severity. It is important to evaluate all the causes of hemoperitoneum and to consider that it may be life risking. This is a case of a patient on long term peritoneal dialysis with hemoperitoneum, whose study showed calcifying peritonitis as the underlying condition.

Key words: hemoperitoneum, peritoneal dialysis, calcifying peritonitis, sclerosing encapsulating peritonitis.

¿Hemoperitoneo en diálisis peritoneal, un signo de alarma?

Resumen

El hemoperitoneo es una complicación de la diálisis peritoneal. Su diagnóstico diferencial es amplio y el enfoque se basa en el cuadro clínico y su severidad. Es necesario evaluar todas las causas del hemoperitoneo y tener en cuenta que tienen manifestaciones diferentes y que algunas arriesgan la vida del paciente. A continuación se describe un caso de un paciente con largo tiempo en diálisis peritoneal con hemoperitoneo, en quien el estudio sugiere peritonitis calcificante como enfermedad de base.

Palabras clave: hemoperitoneo, diálisis peritoneal, peritonitis calcificante, peritonitis esclerosante encapsulada.

Introduction

Hemoperitoneum is a common complication in peritoneal dialysis patients, it is more frequent in women and attributable to gynecological causes, but it may appear because of multiple etiologies. One of them is calcifying peritonitis, in which hemoperitoneum is caused by retraction of vessels or adhesions of intra-abdominal organs. Calcifying peritonitis may be associated with sclerosing encapsulating peritonitis, which, although uncommon, generates great morbidity and mortality in the patient on peritoneal dialysis[1,2].

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Case Description

A 55-year-old male patient with polycystic kidney disease on peritoneal dialysis for 18 years; with a history of coronary disease and hyperparathyroidism.

During the last 6 years on peritoneal dialysis, he was maintained as a high creatinine transporter and average for glucose, at the time of presentation of the described table, he presented average ultra-filtrationof more than 1 liter per day, without residual renal function and without signs of fluid overload. No history of gastrointestinal symptoms, weight loss or infections, no history of peritonitis associated with peritoneal dialysis. Table 1 describes the results of the laboratory tests taken at the last control in the renal unit (25 days before the consultation).

The patient comes to consultation because of the presence of blood-peritoneal fluid in the last 2 days, without abdominal pain. Three hours before admission to the emergency room, he presents syncope. Years ago, he had presented 2 self-limited episodes of hemoperitoneum. At physical examination, he had a blood pressure of 90/50 mmHg, heart rate of 105 beats per minute and 18 breaths per minute. Upon inspection of the abdomen, the peritoneal catheter and its orifice were in good condition, had no palpation pain, and no signs of peritoneal irritation. No other findings were found at physical examination. He was admitted with a diagnosis of hemoperitoneum. Diagnostic tests showed hemoglobin of 6.2 g / dL, which required transfusion of red blood cells, achieving hemodynamic stability. Due to the presence of anemia and hemodynamic compromise, vascular or intraabdominal organ injury was discarded, and abdominal angiotomography was performed. The images documented extensive mesenteric and peritoneal calcifications and multiple renal cysts with some calcifications, with no signs of rupture or bleeding (figure 1).

Table 1.

Patient's laboratory tests.

Tests	Results
Hemoglobin	10 gr/dL
Hematocrit	29,9%
Ferritin	121,3 pg/mL
TSAT	10%
Calcium	9,04 mg/dL
Phosphorus	5,4 mg/dL
PTH	1655 pg/mL
Albumin	2,6 gr/dL

TSAT: percentage of transferrin saturation.

PTH (parathyroid hormone).

CRP: C-reactive protein.

The patient persisted with anemia and blood peritoneal fluid, and scintigraphy with marked erythrocytes was performed, which was negative. Once the hemoglobin has stabilized and the characteristics of the peritoneal fluid have improved, the patient is discharged. The vital signs of discharge were blood pressure of 140/90 mmHg, heart rate of 84 beats per minute and respiratory rate of 18 breaths per minute. On the following day to hospital discharge, he consulted the renal unit for hemoperitoneum and had a blood pressure of 80/40 mmHg and heart rate of 112 beats per minute. He is admitted again and abdominal laparoscopy is performed. This shows a large self-contained hematoma (2 liters) on the major omentum of the splenic angle of the colon, gastroesplenic ligament and left subphrenic space. In addition, active bleeding of the left inferior phrenic artery branch near the esophageal hiatus and severe thickening of the fine intestinal loops by fibrous peritoneum is evidenced; The vessel is tied off and the peritoneal dialysis catheter is withdrawn.

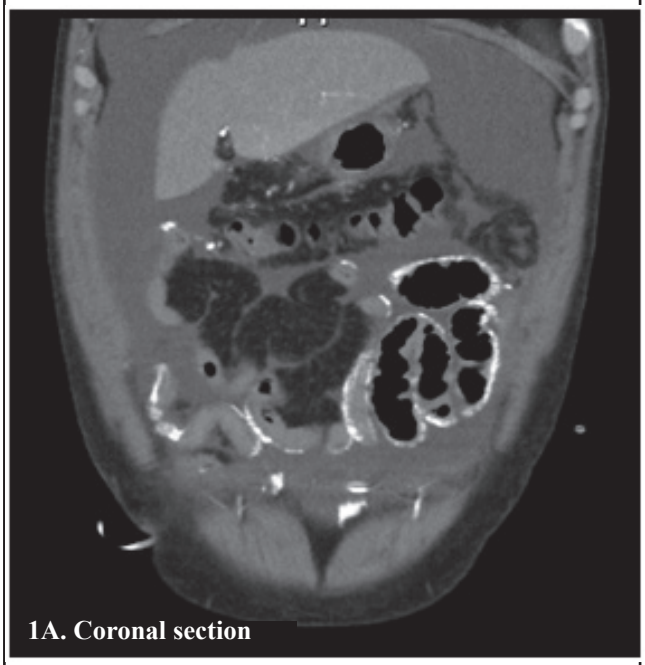
The clinical picture is resolved and the patient is transferred to hemodialysis therapy and is discharged from hospital. The evolution of the patient, 11 months after this picture, is adequate, continues on hemodialysis, and has not presented gastrointestinal symptoms.

Literature review

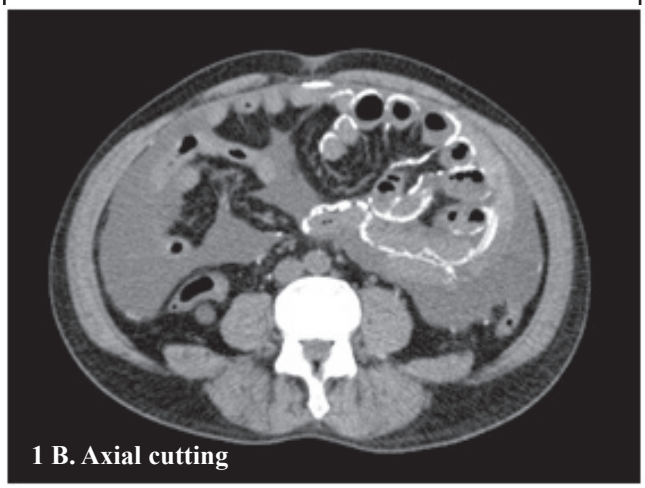
Hemoperitoneum is a symptom that patients can present on peritoneal dialysis. Only 2 mL of blood are required in a 1-liter peritoneal fluid drainage bag

Figure 1.

corresponds to angio-tomography images of the abdomen in the coronal and axial section where there is evidence of important calcification around the intestinal loops.



1A. Coronal section



1 B. Axial cutting

to change the color of the peritoneal fluid. There are multiple causes of hemoperitoneum and among the most frequent are those associated with the insertion of peritoneal dialysis catheter and menstruation in women. In the approach of a patient with hemoperitoneum, it should be taken into account if it is self-limited or persistent and the severity of the clinical picture. It is necessary to discard the different etiologies of hemoperitoneum such as those related to the catheter or other procedures, infectious processes, vascular or intra-abdominal organs, obstetric or gynecological causes, among others[1,3-5].

Calcifying peritonitis is another less common cause of hemoperitoneum. This entity was described by Marichal et al.[6], who reported 2 patients on peritoneal dialysis with abdominal pain, symptoms of incomplete intestinal obstruction and calcifications in the peritoneum. Histologically, the parietal peritoneum of these patients showed fibrous thickening, ossification bands, calcium deposits and few cells. These patients were being dialyzed with solutions with acetate, and this was considered an etiological agent of calcification of the peritoneum. One patient had hyperparathyroidism and had presented hemoperitoneum. Patients progressed favorably after switching to hemodialysis therapy. These 2 cases and another reported in the literature, suggested that this pathology was of good prognosis[6,7].

The cause of calcifying peritonitis is uncertain, but different etiologies have been postulated, such as peritoneal dialysis solution components like acetate, repeated episodes of bacterial peritonitis, the same hemoperitoneum that can accelerate calcification and bone mineral alterations on patients on dialysis due to alteration of the phosphate-calcium axis-parathyroid hormone[6,8,9]. It has been postulated that hemoperitoneum associated with calcifying peritonitis may be caused by lacerations of adhesions in the intra-abdominal compartment (e.g., adhesions of the peritoneum to organs or organs to the diaphragm), by retraction of vessels because of surrounding calcified plaques or by the same vascular calcification that weakens the vessel wall[10]. Arterial causes of hemoperitoneum also include lesion of the vessels by the peritoneal dialysis catheter or spontaneous rupture of abnormal vessels with aneurysms or pseudoaneurysms[11]. The vessels involved are usually the splenic and renal arteries. However, there are reported cases of the gastroepiploic artery[12], hepatic[13], gastric[14] and superior mesenteric[11].

Calcifying peritonitis is occasionally associated with sclerosing encapsulating peritonitis (SEP). This association, reported several years ago in the litera-

ture, makes us doubt about the good prognosis of the first entity. SEP is one of the most serious complications of peritoneal dialysis and is characterized by symptoms of intestinal obstruction, peritoneal fibrosis and high morbidity and mortality. The intestinal loops are encapsulated and adhere within the sclerosed peritoneum. The diagnosis of SEP requires the presence of symptoms of intestinal obstruction with or without systemic inflammation and calcifications or encapsulated intestine. According to the clinical and pathological findings, SEP is divided into 4 periods (Table 2) [2].

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The diagnosis and early intervention of the SEP impacts the prognosis, so it is important to recognize the patients in the pre-SEP phase. However, there is difficulty in this because there is no universally accepted definition of the pre-SEP phase, the signs of the pre-SEP phase are not pathognomonic of this phase and not all patients in this phase progress to the clinical picture of SEP[15]. In addition, there is no availability of clinical or imaging markers that predict the progression to SEP of patients on peritoneal dialysis[16].

Therapeutic behaviors of calcifying peritonitis are not clearly established. Considering the possible etiologies, we should minimize the occurrence of peritonitis, using solutions with low concentration glucose and perform intervention of mineral bone disease[5,15]. Some suggest switching from dialysis therapy to hemodialysis[5]. Although the patients in the original reports had improved symptoms when they stopped peritoneal dialysis, there were data from patients who progressed to SEP and presented intestinal obstruction after the change to hemodialysis10.This has been explained by the removal of fibrin by dialysis and the absence of the peritoneal fluid that favors contact between the intestinal loops and, therefore, their adhesion[15].

The longer theperiods on peritoneal dialysis, the higher the risk of SEP. However, there are long-term patients on peritoneal dialysis with stability of membrane function and without developing this picture[2]. The precise time at which peritoneal dialysis should be discontinued is not established. This decision should be individualized and consideration should be given to the change in peritoneal transport, the failure of ultra filtration and the need for hypertonic solutions to control the volume, nutritional status of the patient, and signs of inflammation and peritonitis pictures[15].

In 2008, 3 clinical cases were published in the journal Therapeutic Aphaeresis and Dialysis[10], in which peritoneal calcification was documented in patients who were on peritoneal dialysis for a long

Table 2.

Stages of sclerosing encapsulating peritonitis (SEP) 2[2]

Stages	Findings
1. Asymptomatic (pre-SEPperiod)	Ultrafiltration failure, rapid transport, hypoproteinemia, hemoperitoneum, ascites, peritoneal calcifications.
2.Inflammatory period Hemoperitoneum	Fever, ascites, weight loss, hyporexia, diarrhea, C-reactive protein elevation.
3.Progressive orencapsulatingperiod	Signs and symptoms of ileus (nausea, vomiting, abdominal pain, constipation, abdominal mass, ascites)
4. Obstructiveperiod	Anorexia, complete bowelobstruction, abdominal mass

time (more than 7 years). In the first case, the patient had secondary hyperparathyroidism, required solutions with high glucose concentration to achieve ultra filtration and referred to abdominal symptoms and intermittent hemoperitoneum. The images showed calcification of the parietal peritoneum and of the hepatic surface without intestinal obstruction. The patient needed to be transferred to hemodialysis and at 5 years of being in this therapy he had no symptoms of intestinal obstruction or changes in the images regarding calcification. The other 2 cases described corresponded to patients who also had secondary hyperparathyroidism, a history of peritonitis and had required change from peritoneal dialysis to hemodialysis due to membrane failure to achieve ultra filtration. After this change, they presented gastrointestinal symptoms compatible with intestinal obstruction. Calcification of the intestine and parietal peritoneum were detected in the images, suggesting SEP diagnosis.

The authors of this article emphasize that although peritoneal calcification is considered an early sign of SEP, it does not always present concomitantly. However, they conclude that peritoneal calcification should be monitored even after discontinuation of peritoneal dialysis and that it is necessary to be alert to the presence of other signs due to the possibility of progressing to SEP[10].

Discussion

The case presented above corresponds to a patient with 18 years on peritoneal dialysis, with persistent and severe hemoperitoneum due to hemodynamic compromise and a significant decrease in hemoglobin. Due to the patient's history, the rupture of a renal cyst should be considered as a possible cause of hemoperitoneum. However, this was of vascular origin due to bleeding of the inferior phrenic artery. The relevance of the clinical case corresponds to the findings of extensive calcifications in the mesentery and peritoneumwhich, added to the long time in peritoneal dialysis, hemoperitoneum and hyperparathyroidism, could correspond to a picture of calcifying peritonitis.

When analyzing the case described and considering the association between calcifying peritonitis and SEP, the patient could be in the pre-SEP phase, in which even the clinical manifestations of the picture have not appeared. The patient had no symptoms of intestinal obstruction or failure of ultra filtration. However, he had signs of hemoperitoneum, peritoneal calcifications and rapid peritoneal transport. Rapid peritoneal transport may be due to long-term peritoneal dialysis and histological changes of the peritoneum exposed to dialysis solutions over a significant period; however, it should be noted that the shift from peritoneal to rapid transport may be an early marker of SEP.

In this case, the modality was changed to hemodialysis. At the moment, the patient has been 11 months on this therapy, without presenting gastrointestinal symptoms, signs of inflammation or malnutrition. The hemoperitoneum was the main symptom of our patient, which can be considered a warning sign of calcifying peritonitis, since its study allowed us to document the findings concerning this pathology. Taking into account the previously annotated discussion, it is important to be alert to the symptoms of progression to SEP in the patient and to make timely intervention to impact the prognosis.

Conclusion

Hemoperitoneum is a complication of patients on peritoneal dialysis. There are causes of hemoperitoneum that threaten life and require immediate intervention such as vascular lesions, of intra-abdominal organs, among others. Therefore, it is important to carry out the diagnostic approach based on the persistence and severity of this.

However, it should be kept in mind that hemoperitoneum may be a symptom of an underlying condition, such as calcifying peritonitis, which, even if it is not present, may contribute to the presentation of SEP, which worsens the prognosis of patients on peritoneal dialysis in terms of quality of life, morbidity and mortality. In patients on peritoneal dialysis for prolonged periods, we must be vigilant about the presence of hemoperitoneum and change in the behavior of the peritoneal membrane, since they could be considered early markers of SEP.

Conflict of interests and financing

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