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Abdominal pain in peritoneal dialysis: peritonitis or pancreatitis, a report of three cases.

(Douleurs abdominales en dialyse péritonéale : péritonite ou pancréatite, à propos de trois cas.)

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Summary

Pancreatitis in peritoneal dialysis (PD) patients is a complex clinical challenge, often masked by symptoms that overlap with other conditions such as peritonitis. This article presents three clinical cases that illustrate the difficulty of diagnosing and managing pancreatitis in this setting.

The first case concerns a 40-year-old patient who presented with severe acute pancreatitis after two years of PD. The diagnosis, initially mistaken for peritonitis due to the presence of hematic drained dialysat, was rectified by a marked rise in pancreatic enzymes and a CT scan confirming pancreatic necrosis. Despite rapid treatment, including CT-guided drainage, the patient died of septic shock.

The second case involved a 64-year-old patient with chronic pancreatitis predisposed by familial hypertriglyceridemia. Although this patient survived several episodes of pancreatitis, the management of her condition required constant monitoring and ongoing adjustment of her PD therapy.

The third case describes a 58-year-old patient whose acute lithiasis-induced pancreatitis rapidly progressed to pancreatic necrosis, leading to fatal septic shock, despite CT-guided drainage.

These cases highlight the importance of increased vigilance in monitoring patients with PD, in whom pancreatitis can develop insidiously. Diagnosis must be based on rigorous clinical assessment, relevant biological markers such as lipase, and appropriate imaging to avoid potentially fatal delays in treatment. Early multidisciplinary management is essential to improve the prognosis of this serious complication, which is often underestimated in the peritoneal dialysis population.

Keywords: peritoneal dialysis, peritonitis, pancreatitis, absominal pain

Résumé

La pancréatite chez les patients en dialyse péritonéale (DP) représente un défi clinique complexe, souvent masqué par des symptômes qui se chevauchent avec ceux d'autres affections, comme la péritonite. Cet article présente trois cas cliniques illustrant la difficulté du diagnostic et de la prise en charge de la pancréatite dans ce contexte.

Le premier cas concerne un patient de 40 ans, présentant une pancréatite aiguë sévère après deux ans de DP. Le diagnostic, initialement confondu avec une péritonite en raison de la présence de liquide de dialysat hématique, a été rectifié grâce à une élévation marquée des enzymes pancréatiques et une tomodensitométrie confirmant une nécrose pancréatique. Malgré une prise en charge rapide, incluant un drainage scanno-guidé, le patient est décédé des suites d'un choc septique.

Le deuxième cas met en lumière une patiente de 64 ans, atteinte de pancréatite chronique, favorisée par une hypertriglycéridémie familiale. Bien que cette patiente ait survécu à plusieurs épisodes de pancréatite, la gestion de sa condition a nécessité une surveillance constante et une adaptation continue de son traitement sous DP.

Le troisième cas décrit un patient de 58 ans, dont la pancréatite aiguë lithiasique a rapidement évolué vers une nécrose pancréatique, entraînant un état de choc septique fatal, malgré un drainage scanno-guidé.

Ces cas soulignent l'importance d'une vigilance accrue dans la surveillance des patients en DP, où la pancréatite peut se manifester de manière insidieuse. Le diagnostic doit reposer sur une évaluation clinique rigoureuse, des marqueurs biologiques pertinents comme la lipase, et des imageries adaptées pour éviter des retards de traitement qui peuvent être fatals. La prise en charge précoce et multidisciplinaire est essentielle pour améliorer le pronostic de cette complication sévère, souvent sous-estimée dans la population sous dialyse péritonéale.

Mots-clés : dialyse péritonéale, péritonite, pancréatite, douleur abdominale.

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INTRODUCTION

Pancreatitis is an inflammatory process of the pancreas and takes different forms in terms of severity, ranging from moderate to severe forms requiring special intensive care.
Diagnosis of pancreatitis is based on clinical, biological, and radiological factors. Abdominal computed tomography (CT) scans are used to classify pancreatitis according to severity. Hypertriglyceridemia, the presence of gallstones, and alcoholism are the main causes [1].
The occurrence of pancreatitis in peritoneal dialysis (PD) patients can be serious and life-threatening, and its management is complicated, especially when it recurs. Abdominal pain is the most frequent clinical manifestation of pancreatitis, which can be confused with medical peritonitis [1-2].
Hence, early diagnosis and appropriate treatment are important.
Here, we report the observation of three PD patients who presented with pancreatitis.

CLINICAL CASES

Clinical case 1

This was a 40-year-old male patient, not known to be an alcoholic, on continuous ambulatory peritoneal dialysis (CAPD) for 2 years with a history of familial Mediterranean fever.
Two years after the start of exchanges, he presented with generalized abdominal pain with vomiting and a fever of 38.2 °C.

Clinical examination revealed a blood pressure of 123/62 mmHg with sinus tachycardia at 127 beats per minute, diffuse abdominal tenderness, and systemic inflammatory response syndrome (SIRS) with a score of 3.

Figure 1 : Dialysate fluid on patient admission (observation 1)

Biologically, we found an infectious syndrome with hyperleukocytosis at 14,800 elements/mm3, predominantly neutrophilic polynuclear, with an increase in C-reactive protein (CRP) to 550 mg/L and an increase in procalcitonin (PCT) to 8 ng/ml. In view of this picture, the diagnosis of peritonitis was evoked. The dialysate effluent was hematic (*Figure 1*), with a dialysate white blood cell (WBC) count of 59 cells/mm3 and a negative culture. The diagnosis of peritonitis was ruled out. Biological and radiological tests were carried out to rule out other diagnoses, such as an abdominal surgical emergency.

The diagnosis of acute pancreatitis was based on a plasma lipase level of 13 times normal (13 x N) and a plasma amylase level of 7 times normal (7 x N). The abdominal CT scan showed stage D acute pancreatitis.

In order to determine the cause of this pancreatitis, a complementary workup was requested, and it came back without any particularities, i.e., no hypercalcemia, no hypertriglyceridemia, and no vesicular lithiasis on the abdominal ultrasound and CT scans. The immunological workup for autoimmune pancreatitis included serum IgG4.

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The patient was placed on dietary restriction and parenteral nutrition with maintenance of CAPD (3 exchanges/day). The immediate evolution was favorable, with a decrease in pancreatitis-specific markers, under symptomatic treatment with imipenem-based antibiotics.

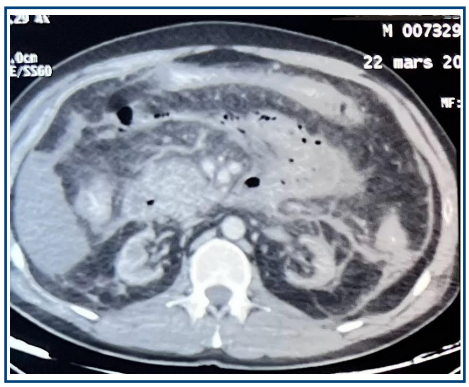

Figure 2: Abdominal CT scan showing necrotic flow (clinical case 1)

Two weeks later, his clinical condition worsened, and he developed severe sepsis. The abdominal CT scan (*Figure 2*) showed a progression to stage E pancreatitis, with increasing necrotic flow, requiring urgent scanno-guided drainage. Sadly, the patient died within 24 hours from septic shock secondary to the pancreatitis.

Clinical case 2

This was a 64-year-old female patient, type II diabetic and hypertensive for 15 years, with familial hypertriglyceridemia and dyslipidemia, put on automated peritoneal dialysis (APD) for 6 years for IgA nephropathy.

Three months after the start of exchanges, she presented with transfixing epigastric abdominal pain, with hematic dialysate fluid.

On clinical examination, the patient was hemodynamically stable, apyretic, and tachycardic at 100 beats per minute, and her abdomen was tender but not contracted.

Biological tests revealed lipasemia at 3.5 times normal. The abdominal CT scan showed stage B pancreatitis, and the workup showed hypertriglyceridemia at 11 g/L.
However, the patient presented six episodes of pancreatitis during her follow-up, which were resolved by symptomatic treatment and dietary restriction. The diagnosis of chronic pancreatitis of metabolic origin was retained. The patient is still on PD, at the moment of this report; with good purification and adequacy.

Clinical case 3

This male patient was 58 years old, suffering from chronic end-stage renal failure due to tubulointerstitial nephritis, and he had been on APD for 8 months. One month after the start of exchanges, the patient presented with transfixing epigastralgia-like abdominal pain radiating to the right hypochondrium without fever.

He was neurologically and hemodynamically stable, with a blood pressure of 130/90 mmHg, a heart rate of 80 beats per minute, and a fever of 38.5 °C, with a SIRS score of 2.

Biologically, we found an inflammatory syndrome with predominantly neutrophilic hyperleukocytosis at 12,600 elements/mm3 and a C-reactive protein (CRP) level of 23 mg/l. The dialysate effluent was turbid, with a dialysate WBC of 68 elements/mm3.
The diagnosis of acute lithiasis pancreatitis was made in the presence of 6 times normal hyperlipasemia and Balthazar stage D acute pancreatitis with a multi-lithiasis gallbladder on the

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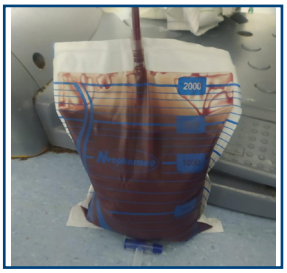

Figure 3:drained fluid from pancreas (case 3).

ultrasound and abdominal CT scans. The immediate evolution was favorable, with a decrease in pancreatitis-specific markers, under dietary restriction and symptomatic treatment.

A few days later, the patient's clinical condition worsened, with the appearance of subicterus, cholestasis, hepatic cytolysis, elevated CRP, and lipasemia at 3,299 elements/mm3 (20 x N). The abdominal CT scan showed a progression to stage E pancreatitis, with a progression of necrotic flow and a 14 mm collection, requiring urgent scanno-guided drainage (*figure 3*). Unfortunately, the patient died 2 weeks later from septic shock secondary to the pancreatitis.

Table I: summary of the three cases of pancreatitis on peritoneal dialysis

	Nephropathy	Clinical presentation	Abdominal scan	Cause of pancreatitis	Treatement and evolution
Cas 1 Case 1 40 years old, male	Familial Mediterranean fever	pain + vomiting + fever 38,2°C + hematic dialysate fluid Biological check-up : blood WC 14800/mm3 CRP 550 dialysate WC 59 Lipasemia 13xN	Stage E pancreatitis	Idiopathic pancreatitis	Scanno-guided drainage + food restriction Evolution : Death after severe sepsis with a digestive onset
Cas 2 64 years old, female	IgA nephropathy	Epigastralgia + hematic dialysate fluid Biology : Lipasemia at 3.5xN	Stage B pancreatitis	Metabolic pancreatitis (familial hypertriglyceridemia)	Diet restriction, symptomatic treatment, continuation of APD Favorable chronic pancreatitis, on PD for 6 years En DP depuis 15 ans sympotomatiqe Continuer DPA
Cas 3 58 years old male.	58 years old, male	Epigastralgia + fever + cloudy dialysate fluid biology : WC12600, CRP 23, WC dialysate 45, Lipasemia 13xN	Stage E pancreatitis	Multi-lithiasal BD	Scanno-guided drainage Unfavorable septic shock

Abbreviations: CRP: C-reactive protein; PD: peritoneal dialysis; WC: white blood cells; N: normal; BD: bile ducts. Normal values: WBC 4500 - 9500 / mm3; CRP < 5 mg/L; Lipasemia < 190 IU /L; dialysate WC < 100/ mm3.

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DISCUSSION

Table I summarizes our three observations. Acute pancreatitis (AP) is an inflammatory disease of the pancreatic parenchyma. AP has been reported to occur more frequently in PD patients than in the general population or in hemodialysis patients [3-5]. A recent German study found an incidence of AP of 266/100,000/year in PD patients [5]. This incidence is significantly higher than in the general population, with a value of 19.7/100,000/year, or in hemodialysis patients, with a value of 67/100,000/year [5].

A positive diagnosis of AP is based on a combination of two of the following three criteria: typical epigastric pain, elevation of pancreatic enzymes (lipase and amylase) above 3 times normal in the first 48 hours after the onset of symptoms, and imaging by CT, magnetic resonance imaging (MRI), or ultrasound [2].

Abdominal pain is virtually constant, progressing rapidly and reaching peak intensity within a few hours. Pain often persists beyond 24 hours. Other clinical signs are non-specific. General signs, such as hypotension, fever, tachycardia, and organ failure, are also indications of severity. The special feature of PD is that the dialysate effluent becomes reddish in color, as in our three clinical cases.

The amylase assay is increasingly being abandoned in favor of serum lipase alone (at a threshold of 3 times normal), which is more sensitive (94%) and more specific (96%) than amylase (sensitivity: 83%; specificity: 88%) [6], and this was the case in our clinical cases. It should be noted, however, that the lipase assay is less sensitive in patients with chronic pancreatitis. Therefore, the diagnosis should not be excluded on the basis of a slightly elevated lipase level in patients with a history of pancreatitis flare-ups [6].

After an initial negative workup, the examination required for a causal assessment is pancreatic MRI to look for ductal anomalies (in particular, tumoral obstructions), followed by echo-endoscopy. The latter enables a diagnosis to be made in 32% to 88% of cases not seen on ultrasound [7]. Abdominal and pelvic CT scans may be repeated at a later stage, especially in cases of recurrent AP, as in our second case.

The patient's age, sex, digestive history, alcohol consumption, hyperlipidemia, hypercalcemia, drug intake, and any history of abdominal trauma must be ascertained. A physical examination is of little help at this stage, but it can look for signs of cholangitis or chronic alcoholism.

Icodextrin causes chemical irritation in the peritoneum and pancreatic glands, which may be the cause of pancreatitis. High intra-abdominal pressure caused by peritoneal fluid and non-physiological composition can promote premature activation of proteolytic pancreatic enzymes, local toxicity of the peritoneal dialysate, and high levels of uremic toxins, and local hypercalcemia is a frequent cause of pancreatitis in PD [8-9].

The classification of acute pancreatitis is based on distinct pathological and clinical features that reflect the severity of pancreatic inflammation. The condition is triggered by a variety of aetiological factors and often manifests itself suddenly with abdominal pain, vomiting and a va-

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riety of other symptoms. Pathologically, acute pancreatitis is classified into two main categories: mild and severe. Mild acute pancreatitis is characterised by interstitial oedema and infiltration of white blood cells, mainly neutrophils, with no major pancreatic necrosis visible to the naked eye. In contrast, severe acute pancreatitis results in extensive peripancreatic fat necrosis and more dramatic changes in pancreatic tissue, such as patchy or confluent areas of acinar necrosis and signs of systemic failure. A quantification system such as the Ranson or APACHE II criteria is often used to assess the severity of the initial attack and guide clinical management. Radiological changes, visualised by techniques such as computed tomography (CT), can also be used to assess associated complications and guide appropriate therapeutic interventions [10].
Pancreatic dysfunction can be caused by elevated gastrointestinal enzymes, such as cholecystokinin, glucagon, and gastric inhibitory polypeptide, which stimulate excessive pancreatic enzyme secretion, as well as histological lesions similar to chronic pancreatitis [11].
As observed in our case 1, the presence of hematic dialysate fluid and a negative culture initially suggested peritonitis, but a CT scan confirmed severe acute pancreatitis, requiring intensive treatment and unfortunately associated with a guarded prognosis. Similarly, our case 3 illustrates how rapid progression to severe pancreatic necrosis can lead to serious complications despite appropriate initial management.

Acute pancreatitis can be diagnosed by the sudden onset of epigastric pain radiating to the back, an increase in serum amylase or lipase enzymes to 3 times normal, and evidence of pancreatitis on imaging tests such as abdominal CT or MRI [12].

Case 2 illustrates the favorable evolution of chronic pancreatitis in a patient on PD, controlled by symptomatic treatment and careful management, underlining the importance of continuous monitoring to optimize clinical outcomes.

In PD patients, it is necessary to assess the clinical and radiological status to diagnose pancreatitis because of the association with elevated amylase and lipase levels without evidence of pancreatitis due to changes in renal clearance. Icodextrin may decrease amylase activity, so it is necessary to assess lipase rather than amylase levels in PD patients [13].

CONCLUSION

Pancreatitis in PD patients remains a severe complication, often diagnosed late due to masked symptoms and misinterpretation. Reported cases underline the crucial importance of heightened clinical vigilance and a rapid diagnostic approach to differentiate pancreatitis from peritonitis, especially in the presence of suggestive clinical and laboratory signs. Early, multidisciplinary management, including symptomatic treatment and possibly image-guided drainage, is essential to improve clinical outcomes and avoid the serious complications associated with this condition.

AUTHORS' PARTICIPATION

FB wrote the article; SEM,NHand NA reviewed the article, providing feedback and suggesting corrections; LB suggested the work and guided the author in writing it.

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Declaration of interest

The authors declare that they have no conflicts of interest.

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