

Review

# An unusual case of extensive peritoneal calcification: A case report

Diogo Roriz\*, Inês Abreu, João F. Costa, Pedro Belo Soares, Filipe Caseiro-Alves

Medical Imaging Department and Faculty of Medicine, University Hospital of Coimbra, Praceta Mota Pinto/Av. Bissaya Barreto, 3000-075 Coimbra, Portugal

Received 30 September 2014; received in revised form 26 November 2014; accepted 2 December 2014

Available online 16 December 2014

## Abstract

The peritoneum is the largest serous membrane of the body and can be exposed to several injuries that may cause abnormal findings on imaging exams. Linear peritoneal calcification is remarkably rare, usually secondary to long duration peritoneal dialysis.

We report an uncommon case of extensive peritoneal calcification in a 39-year-old female without long exposure to peritoneal dialysis solutions, in which peritoneal calcification could be linked to Alport syndrome and previous adverse reaction to intraperitoneal iodinated contrast.

Radiologist should be aware of this and related imaging findings, know when to search for them as well as understand their clinical value.

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**Keywords:** Peritoneal calcification; Encapsulating Peritoneal Sclerosis; Alport syndrome; Iodinated contrast

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## 1. Introduction

Cases of linear peritoneal calcification have been reported in literature, though they occur rarely. In Encapsulating Peritoneal Sclerosis (EPS) a peritoneal membrane damage develops an inflammatory cascade that results in sclerosis and eventually calcification [1].

EPS has been can be either primary or secondary, being long exposure to peritoneal dialysis solutions [2,3] the most common cause of the secondary form. The incidence of EPS has only been studied in patients on peritoneal dialysis and is estimated to be 0.54–4.4% [4], although this number can rise considerably with the time on PD.

The diagnosis of EPS combines clinical symptoms with pathological and imaging findings [5]. The symptoms manifest disturbances in intestinal function such as abdominal pain, nausea, vomiting and ultimately anorexia and weight loss [6]. Among the imaging techniques available, CT is the modality of choice in the diagnosis of EPS, demonstrating peritoneal thickening, calcification, bowel wall thickening, bowel tethering, dilation and fluid loculation [5]. Final diagnosis requires direct observation of peritoneum and histology [7].

In a symptomatic patient, the mortality associated with EPS is high, reaching 60% 4 months after the diagnosis [6]

## 2. Case presentation

In August 2014 a 39-year-old female presented to the emergency department of our hospital. She had Alport disease, end-stage renal disease and secondary hyperparathyroidism.

\* Corresponding author. Tel.: +351 916351739; fax: +351 239482840.  
E-mail address: [djr.radiologia@gmail.com](mailto:djr.radiologia@gmail.com) (D. Roriz).



Fig. 1. An abdominal CT was obtained with iodinated contrast injected through the peritoneal catheter (CT peritoneography). The exam revealed a good diffusion of contrast, with no images of leak or abdominal collections.

She referred a past history of one cesarean followed by a laparoscopic tubal ligation. In October 2013 she initiated peritoneal dialysis. The catheter introduced was soon found dysfunctional, with no drain of dialysate. Laparoscopic removal of adhesions and catheter repositioning were performed, but complicated with hemoperitoneum. Peritoneal lavage by the peritoneal catheter was performed, but abdominal wall swelling was noticed and a leak within the laparoscopic port was suspected.

A CT peritoneography (Fig. 1) was obtained to search for an abdominal leak. Iodinated contrast was injected through the peritoneal catheter and the patient was encouraged to walk, allowing a good diffusion of contrast through the peritoneal cavity. Shortly after contrast injection the patient developed intense abdominal pain and hypotension that were attributed to contrast adverse reaction. The CT was obtained 30 min after contrast injection, revealing a good diffusion of contrast, with no images of leak or abdominal collections. The contrast was then drained through the catheter. Ten days later the patient was seen for abdominal pain and elevated inflammatory markers (C-reactive protein = 40) and the Tenckhoff catheter was removed for a suspected infection. Cultures of peritoneal fluid were negative. Antibiotics for twelve days were given and the patient improved clinically and analytically. The patient chose then to start hemodialysis.

On the day she visited our emergency department, she reported complaints of lumbar pain and dysuria for the previous few days. She had no nausea nor vomiting. She had no fever. On physical examination the abdomen was soft and nontender. The bowel sounds were normal. Laboratory data were normal, except for: hemoglobin = 8.9 (normal 12–16 g/dL), leukocytosis = 14,700 (4000–10,000), leukocyturia > 200/field (< 5/field); C-reactive protein = 23.5 (< 1.0), urea = 22.4 (2.4–6.4 mmol/L), creatinine = 316 (46–92  $\mu$ mol/L), PTH = 324 (16–87 pg/mL), calcium = 2.40 (2.10–2.55 mmol/L) and phosphorus 1.47 (0.41–1.45 mmol/L).



Fig. 2. A lumbar radiography showed diffuse peritoneal calcification, most evident in the lower abdomen.

A plain lumbar radiography (Fig. 2) showed diffuse peritoneal calcification, most evident in the lower abdomen. There were no abnormalities of the lumbar spine. Renal ultrasound was normal, with no signs of renal obstruction.

The abdominal CT (Fig. 3) revealed extensive visceral (arrows in a) and parietal peritoneal calcification (arrowheads in a) with areas of focal thickening in the pelvic peritoneum (arrowheads in c).

Antibiotics to the urinary infection were given and the patient was hospitalized. Blood cultures were negative, while *Escherichia coli* was isolated in the urine culture. She improved clinically, inflammatory markers decreased and she was then discharged from the hospital with no symptoms.

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