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Lanthanum carbonate and peritoneal catheter dysfunction

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To the Editor,

Clinicians are frequently faced with relatively banal issues that become factors of diagnostic confusion or can even trigger more severe complications.

In patients treated with peritoneal dialysis, constipation can become a very difficult problem, and can even reach the point of completely impeding the drainage function of the peritoneal catheter.[1] This is the result of displacement of the catheter towards the upper abdomen and the fact that, even with a properly positioned catheter, a rigid intestine hinders the recovery of infused peritoneal fluid. Over 50% of catheter dysfunctions are related to constipation, and at times this necessitates intensive laxative treatment.[2] Constipation is also an issue in the development of hernias and complications from pressure on the abdominal wall, and can even facilitate the passage of bacteria from the intestinal lumen, leading to peritonitis.[3]

Constipation can be associated with several different factors, such as a certain degree of intestinal paresis, insufficient mobility, and a diet low in fibre, which is often the result of diets that restrict the intake of fruits, and frequently is a result of the medications administered for concomitant problems. Several treatments administered to dialysis patients can generate or aggravate this situation, such as the resins used for hypercalcaemia and phosphate binders. Lanthanum carbonate is a phosphate binder, without calcium or aluminium, which is effective in controlling hyperphosphataemia, and being a radiopaque compound, results in very characteristic radiological images.[4] However, as is the case in other chelating agents, it can produce constipation that is difficult to treat using conventional measures.[5] A peritoneography can aid in the diagnosis of this type of mechanical issue.

We present the case of a patient in which the administration of lanthanum carbonate produced severe constipation and displacement of the catheter to the point where peritoneal dialysis treatment became impossible.

Ours was a 47 year-old patient with chronic renal failure from interstitial nephropathy secondary to reflux, who had been on haemodialysis since 1990. He underwent his first kidney transplant in 1991, which was then removed due to chronic dysfunction, and underwent a second transplant in 1999, which was again lost to the same reasons. He returned to haemodialysis in 2010. Due to intolerance to the second kidney, the patient underwent graft embolisation. He was receiving lanthanum carbonate at 750mg/8 hours due to secondary hyperparathyroidism and hyperphosphataemia. Due to several failed vascular accesses, it was suggested that the patient be transferred to peritoneal dialysis, and a straight, double-cuff Tenckhoff catheter was implanted. During training, we detected catheter malfunction with incomplete drainage, so we performed abdominal x-rays (Figure 1) and peritoneography (Figure 2). In addition to the remnants of the radio-opaque material from the graft embolisation, we observed a large quantity of faecal matter throughout the large intestine, with radiolucent images indicating the presence of lanthanum carbonate. The peritoneal catheter was poorly positioned towards the hepatic flexure of the colon, and the peritoneography dye was completely restricted between the transverse colon and the lower edge of the liver, which was clearly outlined, without disseminating into the rest of the abdominal cavity. Suspension of the lanthanum and intensive laxative treatment progressively resolved the constipation and dye restriction, although it did

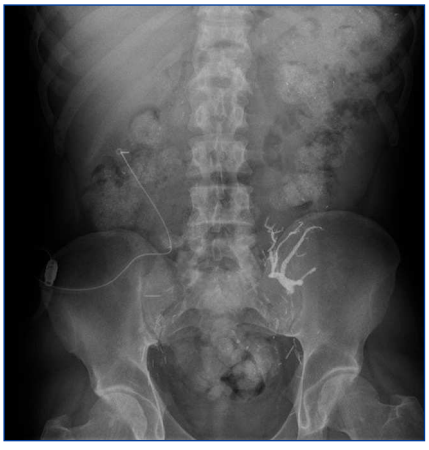

Figure 1. Simple abdominal x-ray
Remnants of graft embolisation. Poorly positioned catheter and severe constipation caused by lanthanum carbonate use in radiolucent images.

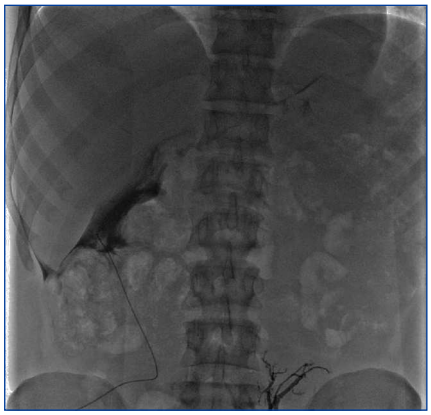

Figure 2.. Peritoneography.
The dye is encapsulated and contained between the intestine and lower edge of the liver, trapped by a large volume of faeces with residual lanthanum chelating agent.

not resolve the poor positioning of the catheter, which had to be relocated.

The radiological images presented show the mechanism of action and the mechanical consequences.

Conflicts of interest

The authors affirm that they have no conflicts of interest related to the content of this article.

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Bilateral renal infarctions

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To the Editor,

Here we present the case of a 64 year-old male with a history of obesity, arterial hypertension, diabetes mellitus, and chronic atrial fibrillation, under treatment with oral anti-platelet drugs, who had had pain in the right lumbar fossa radiating to the groin for more than 24 hours, nausea and vomiting.

The patient was without fever and had a blood pressure of 140/90mm Hg. Heart auscultation revealed systolic murmur. The patient's abdomen was soft and depressible, with pain in the left flank and hypochondrium and no succussion splash. The rest of the physical examination did not reveal any relevant findings.

Complementary tests also produced notable results including atrial fibrillation in the electrocardiogram, leukocytosis, elevated plasma creatinine, a marked increase in lactate-dehydrogenase (LDH) with normal transaminase levels,[1-3] and microhaematuria. The urine culture test was negative, as well as parameters for autoimmune disease, immunoglobulins, and complement.

Due to the persistent abdominal pain and lack of concordance with digestive diseases, we performed an abdominal axial computed tomography that revealed segmental bilateral hypodense areas (Figure 1) with no lithiasis or dilation of the urinary tract. Together with the rest of the findings from examining the patient, this was suggestive of multiple renal infarctions, probably of an embolic origin.[1]

We then performed an echocardiogram that revealed dilated cardiomyopathy of an unknown cause and aortic stenosis.

After the evaluation, we started the patient on conservative treatment, maintaining therapeutic anti-coagulation,[1-3] statins, and blood pressure control.

The patient's clinical and biochemical progression was favourable.

Our final diagnosis was of cardio-embolic renal ischaemia in a patient with previous anti-coagulation treatment.

Conflicts of interest

The authors affirm that they have no conflicts of interest related to the content of this article.

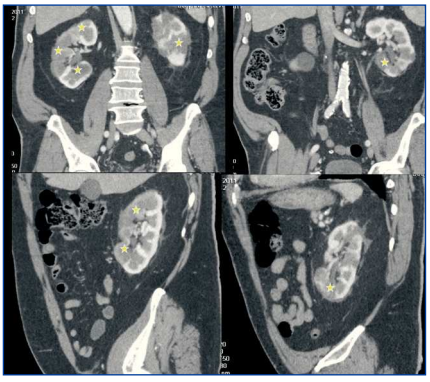

Figure 1. Abdominal axial computed tomography.
Abdominal axial computed tomography with intravenous contrast showing coronal (upper images) and sagittal (lower images) reconstructions. Observe several hypodense areas in both kidneys, a larger area in the right kidney and more focal area in the left one, with slightly altered peri-renal fat, indicating bilateral renal infarctions.