

Peritoneal dialysis catheter dysfunction caused by fallopian tube entrapment[☆]

Disfunción del catéter de diálisis peritoneal por atrapamiento de la trompa de Falopio

Dear Editor,

Peritoneal dialysis catheter dysfunction is one of the main complications of peritoneal dialysis. The most common causes of infusion and/or drainage problems are caused by catheter migration or kinking, constipation, fibrin deposition, intraperitoneal adhesions or omentum entrapment.

We present a case of peritoneal catheter obstruction due to entrapment by the fallopian tube and solving the problem by laparoscopy.

This was a 62-year-old patient with CKD stage 5 of unknown cause. Laparoscopy was performed to implant a self-positioning catheter, without incident either during surgery or postoperatively. Follow-up abdomen X-ray showed the tip of the catheter optimally positioned in the lower pelvis. Infusions were started with progressively larger volumes seven days after the intervention. A very slow flow was observed during the infusion and difficulty in drainage, not recovering the infused volume. Repeat abdominal X-ray showed no changes compared to the previous one. Laxatives and enemas were prescribed, and mobilisation was advised. Despite the patient having abundant, daily bowel movements, the infusion and drainage problems persisted, preventing the technique from being started. Iodine contrast was infused through the catheter and by contrast fluoroscopy the contrast was observed at the distal end of the catheter with images suggesting omentum entrapment (Fig. 1). Diagnostic laparoscopy was performed, but showed no omentum involvement. Entrapment of the catheter was found by the fimbriae of the left fallopian tube, which had surrounded the distal end of the catheter, blocking the exit holes. The catheter was released, its lumen was cleared out using a guidewire and flushing with normal saline, with large amounts of fibrin being found inside the catheter. After seven days the technique was restarted, the catheter functioning correctly with adequate infusion and drainage volumes.

Mechanical obstruction or malpositioning of the catheter are complications that prevent the proper functioning of peritoneal dialysis. The incidence of catheter dysfunction due to mechanical problems varies from 2% to 36% depending on the series, but decreases with the implementation of laparoscopy as an insertion technique.^{1,2} Omentum entrapment is a common cause of catheter obstruction, but entrapment by intraperitoneal structures such as the appendix³ or the fallopian tube, although rare, are other possible mechanisms of blockage of the flow of fluid.^{4,5} Obstruction by the fallopian tube fimbriae is a very rare cause of mechanical catheter dysfunction. It may occur asymptotically as in our case, but sometimes it may manifest clinically with pain in the iliac area and vaginal fluid leakage.⁶ Laparoscopy is the technique of choice for diagnosis and treatment, enabling the viability of the catheter to be restored and have the dialysis procedure restarted.⁷ Despite being a rare cause of dysfunction, we should assess the possibility of catheter entrapment by the fallopian tube in women on the peritoneal dialysis programme.



Fig. 1 – Fluoroscopic peritoneogram with output of contrast from the distal end of the catheter.

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Metformin and diuretics[☆]

Metformina y diuréticos

Dear Editor,

Lactic acidosis associated with metformin is a rare complication (9/100,000 person years).¹⁻³ However, it is also serious, and usually occurs in association with certain predisposing conditions, all of which are absolute contraindications for the use of metformin: acute kidney injury; severe chronic kidney disease; liver disease; alcohol abuse; congestive heart failure; coronary heart disease; age >80; and tissue hypoxia states accompanied by haemodynamic instability.⁴ Lactic acidosis is defined as pH < 7.35, blood lactate > 2.0 mmol/l and PaCO₂ < 42 mmHg.

We studied 30 patients treated with metformin who attended the Accident and Emergency department (1 September 2016 to 30 September 2017) and were found to have lactic acid levels in blood above 2 mmol/l. Fourteen patients required haemodialysis (HD). Haemodialysis was indicated following the usual clinical and analytical criteria in acute kidney injury: oliguria/anuria; fluid overload; severe water/electrolyte or acid/base alterations; and elevation of nitrogen products. Metformin clearance could not be monitored because our hospital's laboratory does not measure metformin levels.

From a clinical point of view (Table 1), patients who required HD had a higher blood level of lactic acid (10.50 ± 6.61 vs 3.43 ± 1.91 mmol/l; $p=0.000$), lower pH (7.07 ± 0.21 vs 7.34 ± 0.048 ; $p=0.000$), a higher rate of associated acute kid-

ney injury (AKI) (100% vs 50%; $p=0.003$) and a higher rate of admission to ICU (64.2% vs 6.3%, $p=0.001$).

From a therapeutic point of view (Table 1), more patients requiring HD were taking diuretics (69.2 vs 31.3%; $p=0.048$), with no differences found regarding treatment with ACE inhibitors or ARBs or the dose of metformin.

Table 2 shows the characteristics of the group of patients who received dialysis.

As both metformin and loop diuretics are removed by active tubular secretion, we analysed whether or not the pharmacological interaction between them might be at this level. Active secretion of metformin is carried out through the hOCT2 (human organic cation transporter 2) (basolateral)/hMATE1/2-K (human multi-drug and toxin extrusion proteins 1 and 2-K) (apical) pathway,^{5,6} which is the cationic organic acid secretory pathway. However, the active tubular secretion of loop diuretics takes place through hOAT1 (human organic anion transporter 1) and hOAT3⁵ (basolateral)/hMATE2/4-K (apical), which is the secretory pathway for anionic organic acids. Thus, in principle and from a theoretical point of view, there would not appear to be any interaction between metformin and loop diuretics at the level of active tubular secretion of organic acids (hOCT or hOAT). It remains to be determined whether the interaction could occur at the level of the hMATE1 or hMATE2 transporters located at the apical level. In fact, furosemide has

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