They release calcium from bone and promote the renal reabsorption of calcium in the distal nephron. They also produce hyperphosphaturia by decreasing the tubular reabsorption of phosphate that may result in hypophosphataemia. However, patients with hyperparathyroidism may present with hypercalciuria.[8] The elevated calcaemia in our patient is related to high levels of PTHr. Significantly, hypercalciuria persisted after excision of the tumour and normalisation of calcaemia levels.

Another remarkable finding was the presence of hypophosphataemia, which became significant. The hypophosphataemia coincided with a situation of renal phosphate loss, evidenced by low levels of TRP and TP/GFR, indicating that the hypophosphataemia was of renal origin.

In conclusion, we present a case of PTHr-mediated hypercalcaemia secondary to an ovarian tumour in an age group that is rarely affected by this condition. In addition to transient renal failure, the biochemical data show hypercalcaemia accompanied by hypercalciuria. The hypophosphataemia is secondary to an excessive loss of phosphate through the kidney.

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Diagnosis of obstruction peritoneal catheter by fluoroscopic peritoneography☆

Diagnóstico de obstrucción de catéter peritoneal mediante peritoneografía fluoroscópica

Dear Editor,

Infusion/drainage problems are defined as a slowed flow, hampered or prevented by causes related to the catheter itself and not to the functioning of the peritoneum as a dialysis membrane. The occurrence of these problems ranges between 5% and 20% and is often associated with the technique used fro the placement of the catheter. They are more common in cases of implantation by laparoscopy.[1]

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Let us look at the case of a patient suffering catheter flow problems who was diagnosed with partial catheter obstruction by fluoroscopic peritoneography and whose transfer to haemodialysis was avoided.

The patient was a 17-year-old female with CKD secondary to IgA nephropathy, on an automated peritoneal dialysis (APD) program since April 2015 through a double-cuff silicon catheter and with a pigtail end implanted by open surgery. She had experienced no mechanical or infectious problems related to the technique, and maintained a residual diuresis of 1500 ml per day. She came to the unit complaining that she could not perform dialysis due to multiple concerns. She did not report constipation, although she had only one small bowel movement a day. An exchange was performed at the unit, in which infusion was very slow and drainage heavily obstructed. A *push* and *suck* maneuver was applied, checking the permeability of the catheter. The most common causes of infusion problems are catheter buckling and blockage of the catheter lumen/hole due to fibrin. And the causes of drainage problems are either of the two previously mentioned, plus poor catheter placement, obstruction due to omentum entrapment and constipation.[2] An abdominal X-ray was performed showing the distal end of the catheter displaced in the left iliac fossa and abundant gases and stool remains. For diagnosis, the simple X-ray identifies the position of the catheter in the abdominal cavity. Enemas and laxatives were prescribed and walking was recommended. Patient showed no volume problems or uremic symptoms. 48 h later the patient returned without any resolution of the problem. The abdominal X-ray was repeated which showed no changes in terms of the positioning of the catheter, and so the alpha maneuver was performed. Poorly placed catheters can be repositioned using a vascular guide wire (alpha maneuver).[3] This can resolve some 50–80% of cases, although only 33% will achieve a permanent solution. The catheter was able to be moved down by around 3 cm, but the drainage problem persisted. Iodinated contrast medium (10 ml) was infused by catheter and fluoroscopy showed that it only came out through the proximal orifices of the catheter (Fig. 1).

In cases where diagnosis is difficult, performing a CT or MR peritoneography, or the less-used fluoroscopy, can diagnose virtually all of such types of complications, ruling out leakage.[4,5]

As for treatment, this depends on the case; constipation can be treated with a fiber-rich diet, laxatives or enemas. Nearly 50% of drainage difficulties are resolved using these methods. Where fibrin plugs or strands appear in the effluent, adding 200–500 U/l of heparin to the dialysis fluid is beneficial. Where the fibrin causes occlusion of the catheter lumen, as in the case in point, the instillation of 5000 U of urokinase may be adopted, which should be maintained for 1 h.[1] The catheter was sealed with urokinase, achieving acceptable infusion and drainage flows. Since this measure was effective, heparin was added to the subsequent changes at the dose mentioned, the prescription was changed to manual with CAPD and a regular use of laxatives was recommended. The patient was subsequently monitored as an outpatient by telephone. Since the flows improved over time, the patient resumed APD.

Most complications that cause catheter infusion and drainage problems which cannot be resolved with conservative methods can be approached using laparoscopy techniques: repositioning of poorly placed catheters and suture fixation, fibrin occlusion cleaning, clearing obstructions caused by omentum entrapment and omentectomy, or replacement with a self-locating catheter.[6]

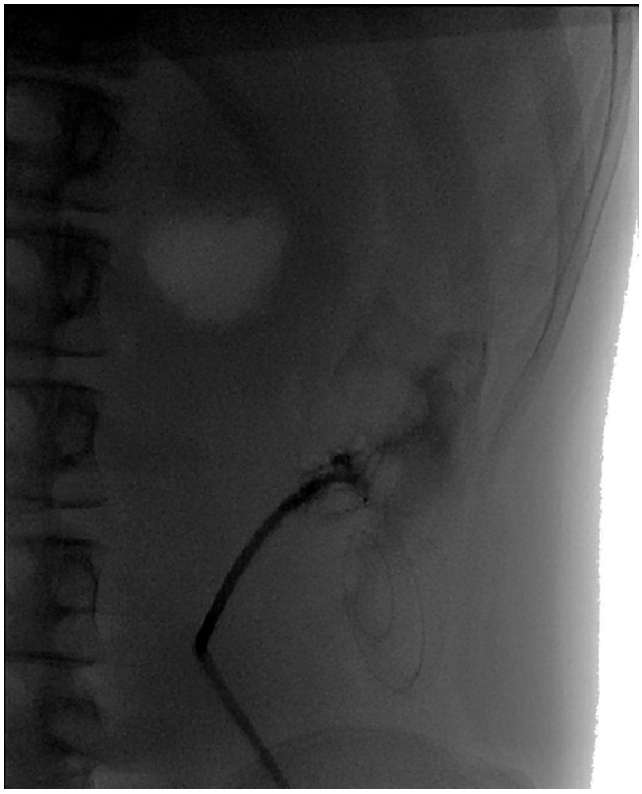

Fig. 1 – Fluoroscopic peritoneography with contrast outflow through upper orifices and tail obstruction.

Prevention of these types of conditions requires proper catheter placement and avoiding causes of constipation.

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Educating your patient is cost-effective: It reduces serum phosphate and saves €200 per patient per month☆

Las estrategias de educación a pacientes son coste efectivas: reduzca el fósforo ahorrando 200 € al mes por paciente

Dear Editor,

In dialysis patients, there are only 3 mechanisms of phosphate control: (1) elimination of phosphate by dialysis, (2) chelating therapy, and (3) dietary restrictions. Phosphate elimination by dialysis has been shown to be useful but insufficient, while the other two mechanisms require patients to follow the prescribed treatment.[1]

Failure to follow treatment in chronic patients is very common, multifactorial and involves factors related to the patient, the disease, the health system and the medication itself.[2] Non-compliance with medication is even more striking with chelators, and is possibly the leading cause of lack of phosphate control.[3,4] Various studies show that phosphate control improves with educational strategies, improving adherence to dietary and medication recommendations.[5–7] Despite the high prices of new, more effective chelators, very few studies have addressed the economic aspects deriving from potencial saving associated with improved compliance.

We want to determine whether educational activities improved compliance with medication and phosphate control and evaluate whether this is associated with a lower need for binders and savings in pharmaceutical expenditure.

In a population of haemodialysis and peritoneal dialysis patients, we measured the degree of phosphate control, the level of adherence to medication on 2 different scales (8-item Morisky and SMAQ),[8,9] knowledge about diet and treatment through a 6-item survey, the need for binders in each patient measured as the number of binders and dose prescribed of each binder (calcium carbonate/acetate, aluminium hydroxide, sevelamer hydrochloride/carbonate and lanthanum carbonate). The adherence using scales previously validated in chronic and dialysis patients. The scales classify patients as compliant and non-compliant, and also provide values that allows to be used as a continuous variable, assigning a score for each of the items including questions asked in the form of the Likert scale. The Morisky scale assigns a higher score a better adherence, while the SMAQ scale assigns a lower score to a better the compliance.

We conducted an educational activity as workshop in which we addressed the clinical consequences of uncontrolled phosphate, identification of chelating medication, appropriate methods of administration, identification of foods with a high phosphate content and how to reduced their intake.[10]

Three months after, we re-measured all of the parameters mentioned. We collected data from 35 patients with a mean age of 59.37 ± 14.9, 18 on haemodialysis (HD) and 17 on peritoneal dialysis (PD), 43% female, 47% diabetic, with a time on dialysis of 54.37 ± 82.9 months. The results of the study are shown in Table 1. Phosphate levels were significantly reduced (5.0 ± 1.5 vs. 4.4 ± 1.4). The percentage of patients with phosphate controlled rose from 40 to 71%. We observed no change in any other biochemical parameters except a negligible reduction in protein and albumin levels. The compliance with pharmacological treatment improved as measured by Morisky, as did the compliance of chelators measured by SMAQ. The number of errors in the knowledge test also improved significantly.

This improved control of phosphate led to a reduction in the number of binders and in the dose prescribed. The change in chelator dose and monthly expenditure is shown in Table 2. The number of binders an the dose prescribed per patient was reduced especially in calcium and sevelamer binders. The monthly savings in the treatment of 35 patients was over 6000 euros, and with a better control of serum phosphate.

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