A. Guitián Pena[1], A. Alonso Hernández[2]
[1] Nephrology Department. [2] Nephrology Department and Renal Transplant Unit. University Hospital Commplex of A Coruña. La Coruña, Spain.
Correspondence: Ana Guitián Pena
Servicio de Nefrología. Complejo Hospitalario Universitario de A Coruña, Spain.
Canceliña, 1, 4.º A. 15011 La Coruña, Spain.
ana.guitian.pena@sergas.es

Intestinal pseudo-obstruction due to lanthanum carbonate

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Dear Editor,
Currently, we have a wide range of options regarding the treatment of bone metabolism in haemodialysis.[1] Recently, even with limited time that sevelamer has been used to control hyperphosphataemia, many nephrologists have opted for lanthanum carbonate based largely on the fewer number of tablets required for this therapy.[2] The most frequent side effect of these drugs is manifested in the gastrointestinal tract, primarily affecting gastrointestinal transit, since they are binding compounds.

In our unit, after one year of treating hyperphosphataemia with lanthanum carbonate, our level of control may be considered acceptable in terms of the percentage of patients with phosphate levels less than 5.5. Tolerance has been good, in general, and we have radiologically confirmed the residual presence of this substance in the colon on more than one occasion, which is to be routinely expected and has been previously described.[3]

Here we present the case of a patient who presented with severe abdominal pain with intestinal paralysis, in whom lanthanum carbonate could not be excluded as a causal or contributing agent.

A 75-year-old man, diagnosed with ischaemic nephropathy on haemodialysis for the past 5 years, was admitted to the emergency room with pain in the right iliac fossa. The patient was afebrile, without vomiting, but did have constipation. On physical examination, there was absence of peristalsis and tenderness to palpation in the right iliac fossa. The laboratories were unremarkable (no leukocytosis, amylase, or lipase within the ranges adjusted to the degree of uraemia, etc.). Plain abdominal radiography showed remains of lanthanum carbonate in the colon, dilated loops of bowel, and, overall, a pseudobstructive pattern. Surgical intervention was decided upon for suspicion of an obstructed bowel loop, with a preoperative diagnosis of intestinal ischaemia. The patient underwent surgery during which no signs of mesenteric thrombosis were seen, bowel loops appeared normal, as well as the appendix and the abdominal environment.

Clearly, although we are unable to state anything conclusively, we must suggest a possible iatrogenic aetiology related to lanthanum ingestion, as previously reported, and the importance of remaining alert to the occurrence of processes similar to those described.

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A. Pobes Martínez de Salinas[1], I. Blanco Rodríguez[2], L. Quiñones Ortiz[1], A. Suárez Laurés[1]
[1] Nefrology and [2] Anaesthesia departments. Cabueñes Hospital. Gijón. Asturias, Spain.
Correspondence: Ana Suárez Laurés
Servicio de Nefrologia. Hospital Cabueñes. Gijón. Asturias, Spain.
luysquio@hotmail.com

Pregnancy and advanced chronic kidney disease

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Dear Editor,
The ability to become and remain pregnant in patients with chronic kidney disease depends on its stage. In early stages of the disease, there are practically no differences from a normal pregnancy.[1] On the other hand, the difficulties that pregnancy poses to renal replacement therapy (RRT) are well known, and better outcomes have been described in patients who have undergone renal transplantation.[2] However, the presence of advanced chronic kidney disease (stage 3-4) and pregnancy is an uncommon occurrence. Here we present the progression and treatment of a pregnant woman with stage 4 chronic kidney disease, which is especially unusual.

The patient is a 23-year-old female with epilepsy and chronic renal failure secondary to interstitial nephropathy. She was not hypertensive and presented, at one month of gestation, with the following laboratory findings: Hb: 13.1g/dl, Cr: 2.7mg/dl, urea: 101mg/dl, Ca: 9.1, P: 3.8mg/dl, HCO3: 19mmol/l, PTH: 480pg/ml, estimated glomerular filtration rate (eGFR) (MDRD-4): 21ml/min/1.73m^2, proteinuria: 2.23g/day; other tests without significant abnormalities. Weight 45.8kg and blood pressure (BP) 113/75mmHg. The progression of laboratory values can be seen in Figure 1. Clinical progression, BP control, presence of urea less than 100mg/dl or serum creatinine less than 4mg/dl, and ultrasound follow-up were established as the parametres to be assessed at the beginning of the RRT. These values