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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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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

remained within established limits throughout the pregnancy, with acceptable foetal progression until the eighth month. At that time, increased blood pressure (136/91), the presence of oedema, significant weight gain, and a mild increase in creatinine were noted. At 34+4 weeks, induction of labour was decided on for intrauterine growth restriction. During her admission she required treatment with labetalol due to increased BP. No haematologic or hepatic abnormalities were evident at any point. The neonate weighed 1,640g (3-10 percentile) and had respiratory distress consistent with hyaline membrane disease. Later, the neonate showed signs of persistent ductus arteriosus that required surgical closure. At 23 days of age, the infant was discharged with progressive weight gain and normal development to date. Three months after delivery, the mother was asymptomatic, with BP: 139/89, weight: 49.3 and Hb: 11.4, Cr: 5.5, urea: 137, and eGFR: 9, awaiting RRT initiation.

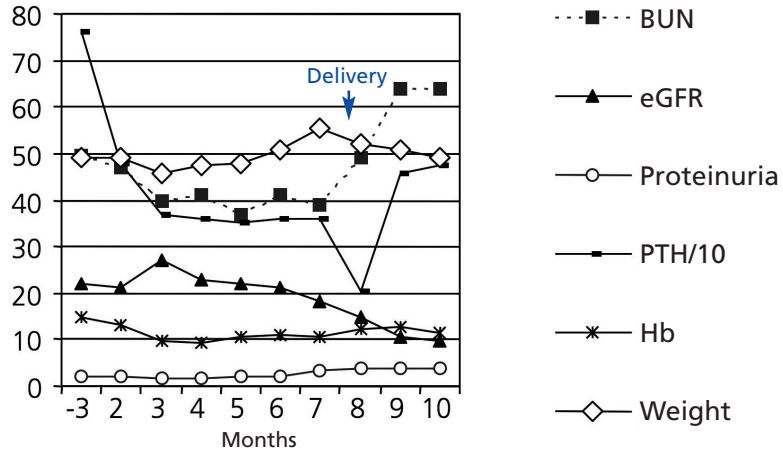
The association of advanced chronic kidney disease (CKD) and pregnancy is a rare event, with an incidence between 0.002 and 0.01% depending on the series.[3] Decreased fertility, and the general tendency to discourage pregnancy in these stages, result in this low incidence.[4] In turn, it is accepted that pregnancy at early stages, with eGFR greater than 60ml/min/1.73m^2, does not alter the course of the renal disease and foetal viability is similar to women without chronic kidney disease.[1] Outcomes in more advanced stages are not as clearly defined. In the largest collected series of 49 women with advanced CKD, stage 3-4, it was found that eGFR less than 40ml/min/1.73m^2 and proteinuria greater than 1g/day at the start of pregnancy led to greater reduction in renal function and increased foetal morbidity and mortality.[4] In another series, up to 10% of patients progressed to ESRD after pregnancy.[5]

On the other hand, estimation of renal function in pregnant women is not well defined. It is accepted that the formulas for estimating glomerular filtration rate are not adjusted for this cases[6] and, at the same time, there are no clear indications for starting RRT in these situations. Some authors have set serum creatinine values between 3.5 and 4mg/dl for starting RRT.[1] Other more recent studies in patients on haemodialysis have reported better results, with urea levels less than 100mg/dl.[7,8] Although there is no firm evidence to support this, those values were set as limits in our patient. Renal function deteriorated, but did not exceed the limits that had been set, and thus it was not necessary to start RRT.

At the same time, the presence of preeclampsia in these patients is also increased.[4] However, the increase in BP and proteinuria in pregnant women makes it difficult to differentiate it from an exacerbation of the baseline disease.[3] In our case we observed an increase in proteinuria and BP in the last trimester. The absence of hepatic or haematologic involvement could indicate a course related to the renal disease in the context of gestational changes.

From the neonatal perspective, although improvements in the paediatric intensive care have improved prognosis, the described mortality rate is between 4 and 4.9%, higher than in the normal population.[4] The most common foetal complications are intrauterine growth restriction, low birth weight and preterm delivery.[5] The association of proteinuria exceeding 1g/day and eGFR of less than 40 are risk factors for development.[4] In our case there was intrauterine growth restriction in the final phase of pregnancy and low birth weight, complications associated with high morbidity. The presence of hyaline membrane syndrome and persistent ductus arteriosus is associated with premature birth, but we are unable to establish that role that renal disease plays in their development.

In short, pregnancy is uncommon at stages 3-4; the association of proteinuria and an advanced renal stage implies a greater likelihood of renal disease and foetal morbidity. The approach that should be followed is based on recommendations and, although no guidelines exist regarding this, it seems reasonable to set the described parameters for monitoring purposes.



BUN: Blood Urea Nitrogen, mg/dl, eGFR; Estimated Glomerular Filtration Rate, ml/min/1,73 m^2, Proteinuria, gr/24 h, PTH: Parathormone pg/ml, Hb: hemoglobin , gr/dl, Weight in Kg.

Figure 1. Laboratory parameters progression over the follow-up period. Follow-up at 3 months after the beginning of pregnancy and then monthly.

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Decrease in renal function associated with hypothyroidism

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Dear Editor,

The functional relationship between thyroid and kidneys has been described since the mid-twentieth century[1] and has been the subject of many publications with differing pathophysiologic approaches.[2-5]

Here we present the case of a patient with a striking recovery of glomerular filtration rate (GFR) after correction of a diagnosed hypothyroidism.

This was an 89-year-old woman with hypertension, hypertensive cardiomyopathy, atrial fibrillation (AF), mitral insufficiency, and moderate aortic insufficiency, cerebral small vessel disease, and obesity-hypoventilation syndrome. She had undergone a cholecystectomy in April 2008, with plasma creatinine in the normal range at that time.

From April 2008 and March 2009, she required several hospitalisations for episodes of rapid AF, lacunar infarct, heart failure, and digoxin toxicity. Progressive deterioration of renal function (RF) was seen until May 2009, at which point evaluation by the nephrology service was requested (Table 1).

Ultrasound showed left renal atrophy (76mm diameter with corticomedullary disruption) and 118mm right kidney with moderate cortical atrophy.

At that time, the patient was diagnosed with stage 4 chronic kidney disease (CKD) secondary to reduction of functional renal mass and severe cardiovascular comorbidity, with therapeutic changes consistent with the degree of CKD.

After laboratory detection of primary hypothyroidism and the subsequent diagnosis of multinodular goitre due to autoimmune thyroiditis, hormonal treatment was started with levothyroxine.

In the subsequent 6 months, adequate control of hypothyroidism was seen with progressive recovery of GFR and increased haemoglobin levels, exceeding expectations of reversibility of renal impairment (Table 1 and Figure 1).

Thyroid disorders cause abnormalities in many locations with the heart and kidneys being the main targets of action of thyroid hormones.[2-5]

Primary hypothyroidism, which develops after intrauterine growth, is

Table 1. Laboratory progression of plasma creatinine, TSH, T4, sodium, pH, and haemoglobin

Date	Creatinine (mg/dl)	TSH (µU/ml)	T4 (ng/dl)	Na (mEq/l)	pH	Hb (g/dl)
April/2008	0.9			142	7.5	9.6
Sept/2008	1.0			143	7.46	11.9
Oct/2008	1.1			136	7.47	12.1
Dec/2008	1.8			139		11.9
January/2009	1.3			134		
March/2009	2.2			141	7.32	
Start of nephrology follow-up						
May/2009[2,5]					7.35	11.5
June/2009	3.0	70.29	0.63	138	7.37	11.7
Start of levothyroxine treatment						
July/2009	3.2	52.93	0,88	141	7.36	11.6
August/2009	2.8	12.32		143	7.34	12.4
September/2009		12.06	1.16			
October/2009	1.9	8.76	1.21	137	7.34	12.7
February/2010	1.6	3.22	1.73	145	7.37	13.9