CASE REPORT

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Acute phosphate kidney injury superimposed on diabetic nephropathy

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ABSTRACT

Diabetic nephropathy is an ever-increasing clinical entity which nonetheless goes frequently underdiagnosed and undertreated. We present the case of a long-term diabetic patient with poor glycaemic control treated at the Gastrointestinal Unit for rectorrhagia. A colonoscopy was recommended and the patient was prepared with high doses of a phosphate and sodium-based purgative (Fosfosoda^{R}). The patient then developed acute renal failure requiring haemodialysis. He has not recovered to date, and remains on dialysis. Renal biopsy confirmed a suspected phosphate nephropathy, superimposed on chronic diabetes nephropathy data meaning the patient is high risk for this kind of purgative, and more care should have been taken when prescribing it.

Key-Words:
Acute renal failure, diabetic nephropathy, phosphate nephropathy.

INTRODUCTION

Type 1 and type 2 diabetic nephropathy are leading causes of chronic renal disease worldwide. During the course of the disease, approximately 20-30% of patients will develop microalbuminuria after a mean disease duration of 15 years and circa half of these patients will develop diabetic nephropathy[1]. Microalbuminuria can be reduced or stabilised if there is adequate glycaemia and blood pressure control, both considered main risk factors for progression of nephropathy[1]. Diabetic nephropathy is a clinical entity with scarce initial symptomatology[1,2]. Stages I, II and III are practically asymptomatic and on many occasions this pathology goes unnoticed. For this reason, preventive measures should be taken when using diagnostic techniques as is the case with use of Fosfosoda^{R} prior to a colonoscopy. Phosphate nephropathy is a clinical-pathological entity that occurs after the oral ingestion of great quantities of the abovementioned substance prepping for colonoscopies. Average daily phosphate ingestion is approximately 1g and that used for a colonoscopy is about 11.6g of phosphorous in 24h[4]. The pathogenesis is not fully known, but believed to be due to renal tubular and interstitial deposits of calcium phosphate[4], associated also with volume depletion[5].

CASE REPORT

We present the case of a 57 year-old male with no known allergy to drugs and whose most outstanding personal medical details were long-term high blood pressure (BP) treated with 160 mg/day Valsartan and 20 mg/day Manidipine, to satisfactory control; dysli-

paemia treated with 20mg/day Atorvastatin; and diabetes mellitus type 2, diagnosed more than 15 years ago. The patient took Glargine insulin 6 UI/sc/day with a rapid insulin pattern according to glycaemias. Patient had poor controls and secondary diabetic retinopathy associated with 100% and 80% loss of vision in the right and left eyes, respectively. Until three years ago, the patient had been a smoker and used to drink a moderate amount of alcohol. Some years back the patient suffered a car accident, with fracture of arm and leg, needing surgery and blood transfusions. From the nephro-urological viewpoint, there were no special records of interest. The patient was being treated at the Gastrointestinal Unit for rectorrhagia. Given his symptoms, he underwent a colonoscopy and was given high doses of an oral phophate-sodium purgative (Fosfosoda^{R}). The colonoscopy revealed a polyp which was pending polypectomy. The day after the colonoscopy, the patient's general symptoms worsened: asthenia, anorexia, nausea and vomiting, and reduction in diuresis. A blood test revealed creatinine of 10mg/dl at 6 days post-colonoscopy, (patient's baseline creatinine values were 1.2 mg/dl). A second blood test some days later revealed creatinine 13.5 mg/dl and potassium 5.9 mEq/L. Given the patient's general deterioration and blood test results, the patient was referred to our hospital's emergency service. Other than taking the oral purgative, the patient said he had not taken anti-inflammatories or nephrotoxic antibiotics, but admitted he had not taken insulin for 6 days because of repeated hypoglycaemias. He presented no oedema or macroscopic alterations in urine. In the blood test that the patient brought with him, apart from kidney function, high levels of phosphorous were noted (6.15 mg/dl) despite the colonoscopy being performed 15 days before and the fact that the patient had eaten very little at the time. Given the patient's significant kidney function deterioration and his poor clinical status, the right femoral vein was cannulated as access for haemodialysis. Valsartan treatment ceased and the patient was hydrated. The initial clinical suspicion was phosphate nephropathy as the patient's clinical progress was satisfactory, but not his renal function. A renal biopsy was performed showing diabetic nephropathy (Figures 1 and 2) with positive Von Kossa staining revealing phosphate deposits (Figure 3). Ultrasound scan showed both kidneys were of normal location, size and ecomorphology, with good corticomedular differentiation, with no evidence of tract ectasia, kidney stones or expansive lesions. Proteinuria was detected in the urine (0.9 g/day). Laboratory data did not support any autoimmune or tumoural cause or any other aetiology other than diabetes and phosphate nephropathy to explain the patient's renal failure. Despite the patient maintaining good diuresis, his kidney function did not improve. The temporary access was changed to a semipermanent one (Tesio-type catheter with subcutaneous tunnel). He was discharged from hospital to continue haemodialysis in the Dialysis Unit assigned to him. When a viral serology for HIV, HBV and HCV was performed predialysis, he was found positive for HCV.

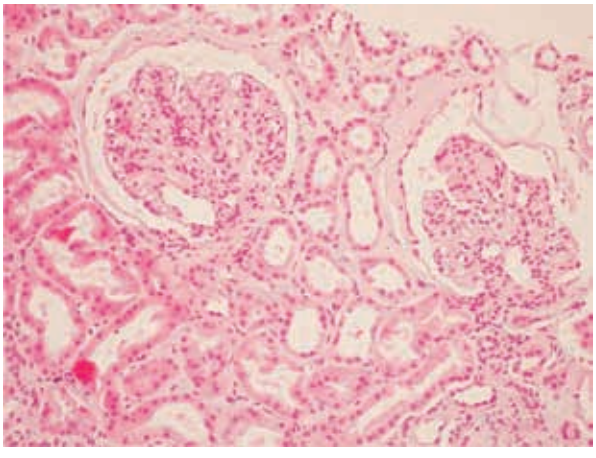

Figure 1

Changes compatible with diabetic nephropathy. Hematoxilina-eosina

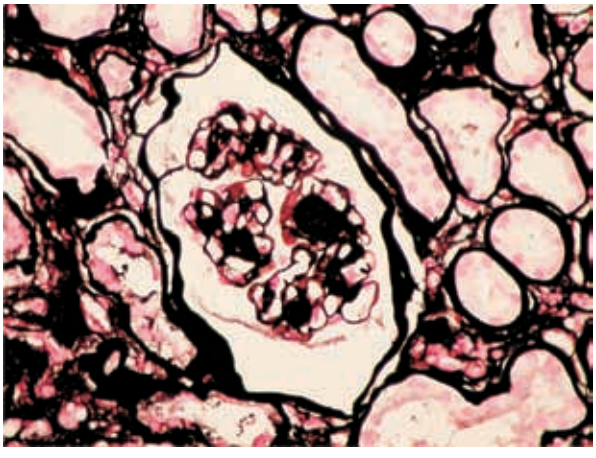

Figure 2

Kimmelstiel-Wilson's nodular formation characteristic of diabetic nephropathy. Silver stain

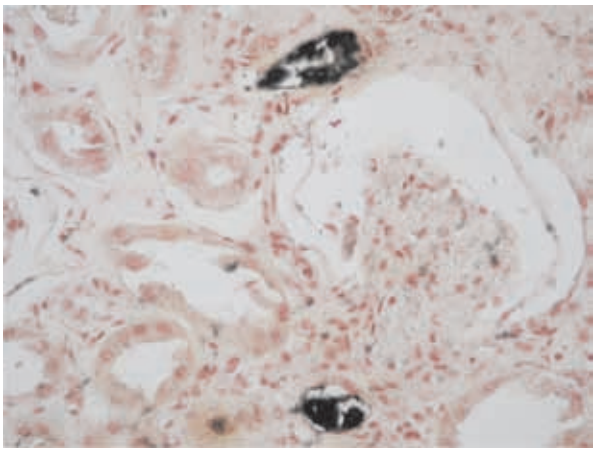

Figure 3

Von Kossa positive staining revealed phosphate deposits

DISCUSSION

While diabetic nephropathy is the most common aetiology of chronic renal failure in Western countries, this entity presents scarce initial symptomatology[1,2]. Stages I, II and III (Mogensen *et al.*) appear to be practically asymptomatic and go unnoticed in many cases, especially for physicians who are not familiar with nephrology[1-3]. Therefore despite normal renal function in routine blood tests, patients can in fact have reduced renal function[4]. This could also be true of elderly patients[4,5], hypertensive patients with poor control or long-term evolution[5]. These patients are more sensitive to injuries caused by nephrotoxic drugs, iodinated contrasts or sodium-phosphate purgatives. For this reason, we must always be aware with these patients to prevent kidney injury and to take timely steps to avoid or reduce renal damage. In our case, that of preparing a patient for a colonoscopy with high doses of a sodium phosphate-based purgative in which potential subclinical renal damage can be suspected, special attention should be paid following preventive measures applied. These include correct hydration of the patient avoiding, any possible added prerenal component[6], suspension of potentially nephrotoxic drugs such as IECAs, ARA II, AINEs, IDR, diuretics, prior to the preparation[5] and use of polyethylene glycol and magnesium citrate purgatives instead of sodium phosphate-based purgatives based[7]. However, if the use of Fosfosoda^{R} is necessary, use doses as low as possible and increase interval between doses[8].

The risk of developing acute renal failure after preparation with Fosfosoda^{R} in the general population is 1-4%[9]. Hence, it is important to detect the population with concomitant risk factors and apply the preventive measures previously described to minimise renal injury. Therefore, at least one control blood test must be run[3,9] in patients having a colonoscopy with Fosfosoda^{R} to obtain levels of glycaemia, urea, creatinine (with this we should be able to estimate glomerular filtration rate), sodium and potassium, calcium and phosphorus, as well as a urine sample to measure microalbumin[3,9].

Conflict of interest statement. None declared.

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