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Renal oncocytoma and papillary microcarcinoma coexistint with cystic disease acquired before dialysis

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To the editor: We report the case of a 76-year old female patient with acquired cystic disease (ACD) who underwent nephrectomy due to the occurrence of images suggesting malignancy leading to a diagnosis of associated oncocytoma and papillary microcarcinoma.

She had been diagnosed in 1982 of HBP without renal involvement, and experienced a cerebrovascular episode leaving no sequelae. In 1999 the patient showed clinical signs of nephrosis, and a biopsy revealed membranous glomerulonephritis (GM) resistant to steroids and azathioprine. She had glomerular filtration rates of approximately 50 mL/min and nephrotic proteinuria. Treatment was started in 2002 with mycophenolate mofetil with an excellent response. Proteinuria decreased from 12.9 g to 1.03 g in 6 months, and eventually disappeared in 3 years, with Cr levels of 1.6 mg/dL.

In December 2005, she was admitted for anuria, oedema, and infraabdominal pain, showing acute impairment in creatinine (3.5 mg/dL), proteinuria of 4.2 g/24 h, and haematuria of 100 cells per high-power field. Early haemodialysis was required. A new biopsy was reported as sclerosis in 50% of glomeruli. A majority of the other glomeruli showed extracapillary GN with epithelial crescents associated to GM, with moderate interstitial fibrosis. Imaging studies found images suggesting ACD, particularly a 2.5-cm solid nodule in the upper pole of the right kidney, suspected to be malignant and for which nephrectomy was performed.

The specimen showed multiple cysts, and the 3-cm nodule was greyish green in colour. Microscopically, renal parenchyma was highly destructured, with sclerosis, crescents, and severe tubulointerstitial involvement, showing papillary and tubulopapillary adenomas, eventually forming a papillary microcarcinoma, and with abundant oncocytic changes, forming interstitial nodular aggregates. The biggest nodule was a conventional oncocytoma.

Coexistence of GM and extracapillary GN occurs in 3%-5% of biopsies with a main diagnosis of GM, either at the time of diagnosis or as transformation of a primary GM in cases of lymphoproliferative disease, vasculitis, collagen diseases…[1,2] In this case, tumour degeneration of an ACD provides the additional mechanism of an extracapillary GN related to neoplasm.

ACD has been considered virtually exclusive of patients on dialysis, but age, grade of CKD, and particularly time since disease onset are also impor-

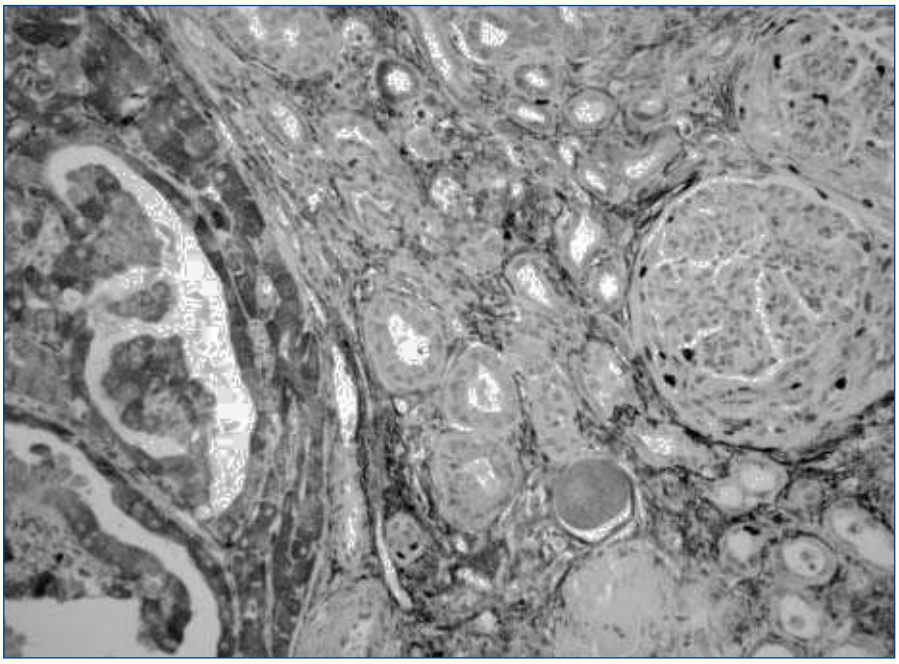

Figure 1. Epithelial crescents with significant proliferative activity close to oncoytic nodule (MIB 1).

tant, in our patient even with a kidney function only slightly impaired.[3-5]

ACD is in turn determinant in the occurrence of renal cancer, appearing most commonly as a papillary microcarcinoma, unlike in the general population, in which clear cell carcinoma predominates.[5,6] We have found 5 cases of oncocytoma reported as occurring in association with ACD, 4 of them in patients on dialysis and one in the native kidney of a transplanted patient. Our case is the first reported occurring without prior replacement therapy.[7-9]

Association of extracapillary GN to renal neoplasms is well known,[6,10] and its relationship to oncocytoma,[11] a rare tumour that is usually associated to other tumour cell lines, is doubtful. Here, it would more probably be related to the papillary microcarcinoma.

As a practical suggestion, we recommend that in patients with chronic glomerular disease on immunosuppressants, screening for renal tumours should be the same as in transplanted patients.

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Acute renal failure after venography

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To the editor: Venography is a procedure intended to ascertain the location of veins in the arm in order to select the most adequate for performing the arteriovenous fistula required for haemodialysis. The case of a 73-year old female patient with a history of diabetes mellitus and high blood pressure, both starting 20 years before, atrial fibrillation, mitral and aortic stenosis, chronic anaemia, and chronic kidney disease diagnosed five years before and monitored at our department of nephrology, with creatinine levels of 3 mg/dL and a creatinine clearance of 15 mL/min, is reported here. She was being treated with insulin, acenocoumarol, furosemide, oral iron, doxazosin, atenolol, isosorbide dinitrate patches, folic acid, and omeprazole. The patient attended the hospital reporting oliguria (150 mL/24 h) for the past 24 hours after a venography. There was no other potential triggering factor of oliguria. Physical examination found no fever and blood pressure values of 130/60 mmHg. CA: Rhythmic heart sounds. PA: Preserved vesicular murmur. Lower limbs: No oedema or signs of DVT. Laboratory tests showed a normal WBC differential, haemoglobin 8.9, platelet count 159,000. Urea 104, creatinine 7.2, sodium 128, potassium 4.6, LDH 564, elemental urine analysis: pH 5, specific gravity 1005, positive protein (+++), sodium 13 mEq/L, and potassium 53 mEq/L. The ECG showed atrial fibrillation with controlled ventricular response at approximately 80 bpm. Chest and abdominal X-rays revealed no radiographic changes. During admission, patient received intravenous fluid therapy, diuretics, and N-acetyl cysteine, showing basal creatinine levels of 4 mg/dL at three days of admission.

Renal failure triggered by intravenous contrast after a venography is very uncommon, but has been reported as one of its complications.[1,2] Acute renal failure caused by a contrast agent is defined in absolute form as a 0.5 mg/dL increase and in relative form as a 25% increase in creatinine levels 48-72 h after administration.[3]

Contrast-induced renal failure is more common in patients who previously have some grade of renal insufficiency, those with a prediabetic state, those with diabetes mellitus starting some years before,[4-6] or patients with hyperuricemia[7] (values higher than 7 mg/dL in males and 5.9 mg/dL in females). The most common clinical sign is oliguria from renal function impairment, occurring as a consequence of renal vasoconstriction and medullary hypoperfusion.[8] There is no defined treatment for contrast toxicity, and there are different theories about the most adequate treatment. Effective treatment with fluid therapy and N-acetyl cysteine has been reported in the literature,[9] but there are also articles reporting no benefits from use of these treatments.[10]

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