Minimal change glomerulonephritis associated with cancer of the pancreas

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Abstract

The authors describe a patient with nephrotic syndrome and a pancreatic carcinoma. Renal biopsy revealed minimal change nephropathy. Two months later, the patient presented a bile duct obstruction secondary to a pancreatic adenocarcinoma. There was regression of the proteinuria, despite progression of the tumor. This fact suggested a cell-mediated immunity that was modified by the tumor, and not a factor liberated by the carcinoma that was directly damaging the kidney.

Key words: minimal change glomerulopathy, nephritic syndrome, carcinoma.

Introduction

The association between neoplasia and nephritic syndrome that may be due to amyloidosis, renal vein thrombosis, or neoplastic infiltration is well--documented in the literature.[1,2] Nephrotic syndrome can occur concomitantly with neoplasia, or precede it by an interval of several months to one year.[3] Minimal lesion Glomerulonephritis may be of idiopathic etiology, or it may be associated with lymphoproliferative diseases (generally Hodgkin's disease, and occasionally, non-Hodgkin's lymphomas and leukaemias),[4] while carcinomas are associated with membranous nephropathy.[5,6] The association of solid tumors and minimal lesion glomerulonephritis is extremely rare, and only a dozen cases have been identified recently in the literature.[1,4,7]

The authors present a case of nephrotic syndrome with minimal lesion nephropathy associated with pancreatic neoplasia.

Clinical case

J.J.C., 63 years of age, female, Caucasian, a housewife, born and residing in Tomar. She visited the emergency service of the Tomar District Hospital with back pain, fatigue, muscle weakness, anorexia and generalized edema with four weeks of evolution. She also reported pyrosis and postprandial fullness, without other digestive, urinary or other organ or systemic complaints, and remained apyretic. Patient denied taking any medications. The personal history included pulmonary tuberculosis twenty-five years earlier.

Objective examination revealed reasonable general condition, accentuated edema of the periorbital areas, hands and lower third of the legs, AT 110/70 mmHg, pulse 70/min. Cardiac auscultation – rhythmic, regular and without murmurs. Lung auscultation – occasional crackles in the lung bases. Abdomen painless, without hepatosplenomegaly or renal Murphy's sign. Absence of adrenomegaly.

For diagnostic clarification, the following complementary exams were performed:

Hemoglobin 150 g/dL, hematocrit 44%, leukocytes 8.6 x 10^3/mm3 (N 64%, L 30%), platelets 283 x 10^3/mm3, ESR 104mm in the 1st hour, glycemia 78 mg/dL, urine nitrogen 21.5 mg/dL, creatinine 0.74 mg/dL, Na 141 mEq/L, K 4.1 mEq/L, uric acid 3.2 mg/dL, total bilirubin 0.30 mg/dL, direct bilirubin 0.10 mg/dl, oxaloacetic transaminase 24 U/L and pyruvic transaminase 26 U/L, alkaline phosphatase 287 U/L. Total proteins 4.10 g/dl, albumin 1.9 g/dl, cholesterol 545 mg/dl, triglycerides 361 mg/dl. Proteinuria 4g/24H. Serum electrophoresis and serum and urinary immunoelectrophoresis did not reveal paraproteins. C3 and C4 normal and antinuclear antibodies absent. Serology for hepatitis B, HIV 1 and HIV 2 all negative.

The clinical and laboratory evaluation led to a

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diagnosis of nephrotic syndrome. Given the patients age, and in the belief that it would be possible to treat the paraneoplastic syndrome, and due to the presence of dyspeptic symptomology, upper digestive endoscopy with biopsy was carried out, which showed abundant biliary reflux and marked chronic arthritis with rigidity of the antrum, while histology revealed inflammatory infiltrate of the lamina propria and phenomena of moderate dysplasia of some glands, aspects of lining papillary polyps (regenerative?)

Abdominal ultrasound showed a liver with heterogeneous echostructure, vesicular lithiasis, and dilation of the intrahepatic bile ducts, evidencing a hyperechogenic nodular image of 2 cm in diameter. The study of the pancreatic area was inconclusive, due to the interposition of gas.

Abdominal CAT scan should an enlarged head of pancreas, with heterogeneous structure and calcifications; dilation of the intrahepatic bile ducts and proximal segment of the main bile duct; and hypodense nodular image of 2cm in diameter next to the hepatic artery, possibly corresponding to adenopathy.

Renal biopsy revealed minimal lesion glomerulonephritis.

Therapy was initiated with corticoids (1 mg/kg/day) and captopril (12.5 mg/3x/day) revealing progressive regression of the edemas and proteinuria up to the 15th day of hospitalization, but with worsening of the general state, and an increase in bilirubin (total bilirubin 11.9 mg/dl, direct bilirubin (99 mg/dl) GOT (289 U/L) and alkaline phosphatase (1904 U/L).

Endoscopic retrograde cholangiopancreatography was carried out, which showed deformity of the main bile duct as far as the hilum. A Pigtail prosthesis was placed in the direct bile duct, and papillotomy performed, as it was considered an unresectable tumor. Cytology revealed that it was an adenocarcinoma. There was a regression of cholestasis, but the clinical evolution was conditioned by the evolution of the tumor, and the patient died at the end of five months.

Discussion

Minimal lesion glomerulonephritis is the main cause of nephrotic syndrome in Hodgkin's disease. The most probably hypothesis is that dysfunction of the T cells, results in the release of cytokines which leads to lesion of the glomerular membrane. The association of minimal lesion glomerulonephritis and solid neoplasia is rare.

This case demonstrates the association of minimal lesion glomerulonephritis and pancreatic carcinoma, and that renal lesion can respond to immunosuppressive therapy, like the idiopathic form. There is only one reported case in the literature of a temporal association between minimal lesion glomerulonephritis and pancreatic carcinoma.[4]

The sequence of signs and symptoms in this patient suggested that the neoplastic syndrome began with the nephrotic syndrome, until the appearance of the bile duct obstruction by the tumor. The interval between diagnosis of the nephrotic syndrome and *exitus letalis* is comparable with what is described for other patients with neoplasia and nephrotic syndrome.[4,8] The regression of the proteinuria in response to corticoid therapy suggests that the renal lesion is mediated through the T cells and not by the production of a substance induced by the Tumor that directly affects the kidney. [9,10]

In this case the favorable response of the nephrotic syndrome to immunosuppressive therapy did not alter the evolution of the neoplasia, as has been reported by some authors. [3]

We conclude that particularly in elderly patients with a nephrotic syndrome, the presence of a neoplastic disease should always be ruled out, and that minimum lesion glomerulonephritis may be a secondary nephropathy to solid tumors, including pancreatic carcinoma.

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