Fibrillary glomerulonephritis in a patient with adenocarcinoma of stomach

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Introduction

Lee and associates were the first to emphasize the apparently high prevalence of carcinoma in adult patients with nephrotic syndrome [1]. In the report linking nephrotic syndrome with underlying cancer compiled by Eagen et al. [2], 67 of 171 patients with malignancy had a carcinoma. Forty-eight of these 67 patients had a renal biopsy performed; 33 had membranous nephropathy with classical subepithelial deposits on electron-microscopy, two had amyloidosis and the rest had either minimal-change or proliferative glomerulonephritis (GN). Fibrillary deposits have much less frequently been reported in malignancy-associated GN [3, 4]; the deposits are usually mesangial in distribution although Rosenmann et al. have reported fibrillar subepithelial deposits in a patient with malignant lymphoma [5]. Fibrillary GN has however usually been associated with other clinical conditions such as cryoglobulinaemia, SLE, and paraproteinaemia [4–6]. We report a case of fibrillary GN in a young patient who was subsequently found to have adenocarcinoma of stomach.

Case report

A 43-year-old white male patient was referred by his GP, having been found to have renal impairment and nephrotic syndrome; plasma creatinine 222 mmol/l, urea 10.5 mmol/l, total protein 47 g/l, and albumin 22 g/l. Plasma creatinine and an IVU were normal 5 years prior to presentation. Previous medical history included an orchidopexy and uretherotomy for a urethral stricture of unknown aetiology which had required repeated cystoscopy and dilatation. At presentation the patient was found to have a BP of 148/94 mmHg, a creatinine clearance of 53 ml/min, and a 24-h urinary protein of 15 g. Autoantibodies including antineutrophil cytoplasmic antibodies (ANCA) were negative and complements C3 and C4, immunoglobulins and serum protein electrophoresis were all normal. A renal biopsy was performed and light-microscopy showed non-uniform glomerular enlargement with no increase in cellularity. There was a diffuse and irregular lobular widening of the mesangia due to the deposition of a finely granular eosinophilic material. In many glomeruli, the capillary walls were thickened due to the deposition of a similar eosinophilic granular material (Figure 1) and areas of mild to moderate interstitial fibrosis were present. Congo red staining for amyloid protein was negative. Immunofluorescence was positive for IgG, IgM, C3, C1q, and kappa and lambda light chains. The ultrastructural studies demonstrated the presence of randomly oriented fibrils in the mesangium, which were larger than amyloid fibrils and which did not have a microtubular structure (Figure 2). This confirmed the diagnosis of a fibrillary glomerulonephritis.

Four months after presentation the patient developed epigastric discomfort responding to H2

Fig. 1. Light-micrograph showing granular material in glomerular mesangium (H&E × 300).

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blocker and an upper GI endoscopy demonstrated macroscopic evidence of gastritis but no other abnormality. Renal function deteriorated and 9 months after his presentation he required renal replacement therapy, which was initially haemodialysis. Whilst on dialysis he developed ascites, and investigations including a repeat upper GI endoscopy revealed a polypoid mass with central ulceration on the anterior wall of the stomach. Histology demonstrated this to be an adenocarcinoma.

He was treated with chemotherapy consisting of epirubicin, carboplatin, and 5FU, but this was abandoned when he was shown to have lung secondaries. He died a few months later.

Discussion

This patient presented with nephrotic syndrome, and a renal biopsy revealed a fibrillary glomerulonephritis with a predominantly mesangial deposition of the fibrillar protein. As frequently occurs in this condition [9], renal function deteriorated rapidly and dialysis was started 9 months after presentation. Two months later he developed malignant ascites due to a gastric adenocarcinoma from which he subsequently died. This is the first report linking fibrillary GN with a gastric adenocarcinoma.

Fibrillary GN has been described in association with a number of conditions including, rarely, lymphomas and carcinomas. The presence of a gastric adenocarcinoma in our relatively young patient suggests a pathogenic link with the fibrillary GN, although this is difficult to prove. The distribution of the fibrillar deposits does not appear to correlate with the underlying medical condition as most cases demonstrate a mesangial deposition of the protein, yet subepithelial and subendothelial deposits occur in patients with both underlying malignancies and other aetiologies [6,7]. Several studies have unsuccessfully attempted to demonstrate tumour antigens in the fibrillary protein [5], yet the presence of polyclonal immunoglobulins and complement components in the fibrillary deposits in most patients, including ours, indicates an immunological process. The pathogenesis, however, remains unclear.

References


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