Case Report

Crescentic glomerulonephritis and centrocytic lymphoma

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Introduction

Glomerular injury has occasionally been observed in patients with Hodgkin’s disease (HD) and less frequently in non-Hodgkin’s lymphoma (NHL) [1–4]. We observed a case of crescentic GN, presenting as renal failure, in a patient with centrocytic lymphoma.

Case

In July 1997, a 77-year-old man was admitted to hospital for recent weight loss and weakness. The patient had been well until 1 month previously when high blood pressure was discovered and treated with angiotensin-converting enzyme inhibitors.

Physical examination was unremarkable except for enlarged liver and spleen. Blood pressure was 140/80 mmHg and rectal temperature, 37.9°C. Initial laboratory tests gave the following results: erythrocyte sedimentation rate, 172 in the first hour, C-reactive protein, 65 mg/l, haemoglobin, 9.7 g/dl, leucocyte count, 12 800/mm³ (10 860 lymphocytes), platelet count, 112 000/mm³ and LDH 506 IU/l. Renal function was normal (BUN 10.8 mmoles/l and serum creatinine 97 µmol/l). Serum total protein was 7.7 g/dl with normal electrophoresis. Urinalysis showed no leukocyturia or red blood cells. The urinary protein excretion rate was 1.57 g/24 h. Total body CT scan revealed multiple lymphadenopathies at various sites. The liver was shown to be enlarged and homogenous splenomegaly was diagnosed. Bone-marrow aspirate and trephine biopsy revealed global infiltration of B lymphocytes (CD20+ cells). The patient was thought to have a low-grade centrocytic lymphoma. He was put on a chemotherapy regimen combining cyclophosphamide, adriamycin, vincristine, and prednisolone. After treatment started, serum creatinine rose to 572 µmol/l. The patient was then referred to our department.

Apart from splenomegaly, physical examination was unremarkable. Blood pressure was 140/80 and temperature, 37.6°C. Urinary volume was 2200 ml/day. Renal failure was confirmed. Serum electrolytes and liver function tests were normal. Laboratory tests produced the following findings: calcium 1.93 mmol/l, phosphorus 1.78 mmol/l, and uric acid 520 µmol/l. C3 and C4 values were normal, but CH₅₀ was slightly elevated at 95. ANCA, anti-GBM antibody, and cryoglobulinaemia were negative. Urinary protein excretion was 1.5 g/24 h and the sediment showed microscopic haematuria (10³/mm³) but no leukocyturia.

A transjugular renal biopsy was performed. The light microscopy specimen contained eight glomeruli, two of which were sclerosed; two exhibited segmental necrosis, with fibrin formation and adjacent crescentic formation in one. The interstitium was oedematous and contained a few inflammatory cells without atypical lymphoid cells. Many tubules exhibited flattening or necrosis of epithelial cells with the presence of red cells in lumina. Some basement membranes were thickened. Significant arteriolosclerosis was observed but with no evidence of vasculitis. On immunofluorescence, no staining was seen except for segmental fibrin deposition in one of the four glomeruli. C3 deposits were seen in the arteriolar walls.

The patient was given 1 mg/kg/day of prednisolone and five cycles of the CHOP regimen. Because of acute electrolyte disorders, haemodialysis was initiated and in all, four sessions were necessary. During the following weeks, renal function recovered and dialysis was discontinued. One month later, serum creatinine had fallen to 210 µmol/l. Urinalysis showed persistent proteinuria and haematuria.

Because of the absence of improvement on a second CT scan, treatment with araclycin was started. After two cycles, diffuse polyradiculoneuritis, considered to be a paraneoplastic syndrome, appeared, and the patient died from respiratory failure.

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Renal involvement in lymphoid malignancy is rare, especially in NHL but it may be underestimated because renal lesions are not the major manifestation of the disease [2]. Many potential causes of renal complications have been described in NHL, including obstructed urinary outflow, organ infiltration [5], effects of monoclonal immunoglobulins, infections, and metabolic disorders [1]. Such complications may also be related to treatment [1].

Glomerulonephritis is rarely reported in lymphoid malignancy, even though the first case was published in 1936 by Cornig [6]. It is mainly observed in association with Hodgkin’s disease, the most common form being minimal-change disease [1,6,7]. In a review of the literature concerning 100 cases, Moulin et al. reported a prevalence of 42 and 37% for minimal-change disease and amyloidosis respectively [1]. Fewer cases of glomerular lesions are associated with NHL than in HD. In such cases, the spectrum of these lesions includes minimal-change disease, membranoproliferative GN, membranous GN, and crescentic GN [1,3,4,8,9]. In two reviews [1,3], crescentic GN was reported in nine and 13 cases of NHL respectively. Nearly all of these cases were associated with low-grade lymphomas. To our knowledge, the patient described here is the first reported case of crescentic GN associated with intermediate lymphocytic lymphoma, also known as centrocytic or mantle zone lymphoma. In NHL, the nephropathy is considered to be of paraneoplastic origin and is usually diagnosed concomitantly with or after the diagnosis of lymphoma. In rare cases, GN preceded and revealed the lymphoma [3,8]. The most common clinical renal presentations are proteinuria, nephrotic syndrome and/or renal failure.

In our patient, proteinuria was detected at the first admission, when lymphoma was diagnosed. GN may have been present at this time and renal failure may have been secondary to the acute tubular necrosis found on renal biopsy. The presence of GN is not considered to indicate a poor prognosis in NHL patients, and renal manifestations may disappear after treatment of the underlying disease. Remissions of renal manifestations have been reported after monotherapy with chlorambucil [1] or after radiotherapy distant from the kidneys [3,4]. These findings argue for the paraneoplastic origin of NHL-related GN. Moreover, the evolution of GN can be used as a marker of haemopathy progression, because a relapse of GN can precede the diagnosis of lymphoid malignancy’s recurrence. However, in the case of our patient, it is difficult to reach any conclusion about glomerular lesions healing, because proteinuria persisted and the haematological disease was not controlled. Improved renal function is probably secondary to the reversal of acute tubular necrosis.

References

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