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Case Report

Acute kidney injury requiring dialysis: a very unusual presentation of non-Hodgkin's lymphoma

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Abstract

Acute kidney injury due to lymphomatous infiltration of the kidneys is uncommon, and it is rarely the initial manifestation of the lymphoma. Here, we present a case of lymphomatous infiltration of the kidneys resulting in acute kidney injury requiring dialysis, as the initial presentation of non-Hodgkin's lymphoma. Renal biopsy established the diagnosis, and renal function completely recovered after chemotherapy.

Keywords: acute kidney injury; non-Hodgkin's lymphoma

Introduction

Renal involvement in patients with diffuse lymphoma is rare. When present, it occurs late in the course of the disease and is generally silent [1–4]. Acute kidney injury (AKI) due to lymphomatous infiltration of the kidneys (LIK) is uncommon, and it is rarely the initial manifestation of the lymphoma [5,6]. Here, we present a case of AKI requiring dialysis secondary to LIK, as the initial presentation of non-Hodgkin's lymphoma (NHL).

Case report

A 51-year-old Caucasian female was admitted at our hospital for anaemia and renal failure. She presented a 3-month history of weakness, anorexia and weight loss (8%). Her past medical history was irrelevant. Physical examination revealed pale skin, blood pressure of 116/62 mmHg, heart rate of 82 beats per minute, respiratory rate of 23 cycles per minute, body temperature of 36 °C, oliguria (300 mL/day), and bilateral supraclavicular and axillary lymphadenopathies. Neither splenomegaly nor hepatomegaly was evident. Laboratory disclosed normocytic normochromic anaemia (haemoglobin, 7.5 g/dL; mean globular volume, 82.5 fl; mean globular haemoglobin, 28.4 pg), low platelets (130 000/mm^3), normal leucocyte formula (leucocytes, 9410/mm^3; neutrophils, 58%; lymphocytes, 12%), elevation of lactate dehydrogenase (825 UI/L) and of erythrocyte sedimentation rate (60 mm/h), and renal failure (uraemia, 189 mg/dL; creatinaemia, 8 mg/dL). Urinalysis showed proteinuria of 30 mg/dL, 80 erythrocytes/μL, and few hyaline and granulous casts; proteinuria was 1.2 g/day. Metabolic acidaemia (pH, 7.17; bicarbonate, 12 mmol; PaCO2, 15 mmHg) was detected. There was also an elevation of β2-microglobulin (14.8 mg/L). Hepatic function tests, haptoglobin and ionogram (sodium: 135 mmol; potassium: 4.5 mmol; chloride: 111 mmol) were on the normal range, and serum protein electrophoresis revealed no changes. There was mild hyperuricaemia (8 mg/dL), mild hyperphosphataemia (7 mg/dL) and mild hypocalcaemia (8 mg/dL). There were no schizocytes in the peripheral blood smear. Serology for human immunodeficiency virus types 1 and 2, hepatitis B, hepatitis C and human T lymphotropic virus type 1 was negative. Renal ultrasound revealed enlarged (right, 15 cm; left, 16 cm) and hyperechoic kidneys with normal differentiation and no hydronephrosis. Computed tomography of the chest and abdomen disclosed bilateral supraclavicular, axillary, mediastinal and para-aortic lymphadenopathies, and enlarged kidneys. There were no morphologic changes in the bone marrow aspirate and bone biopsy; however, abnormal chromosomal translocations [(14,18) and (11,14)] were detected by the fluorescence *in situ* hybridization technique. A renal biopsy was performed and revealed an extensive and diffuse interstitial infiltration by atypical lymphoid cells (Figure 1). Immunohistochemistry demonstrated a positive staining of the neoplastic cells for leucocyte common antigen (CD 45) and CD20 (Figure 2). According to these, a malignant diffuse large B-cell-type NHL was diagnosed. Positron emission tomography showed cervical, supraclavicular, mediastinal, abdominal and inguinal, as well renal involvement.

She underwent intermittent haemodialysis, and chemotherapy with R-CHOP (rituximab, 375 mg/m^2 IV; cyclophosphamide, 750 mg/m^2 IV; adriamycin, 50 mg/m^2 IV; vincristine, 1.4 mg/m^2 IV; and prednisone, 100 mg orally, 5 days) was instituted. Anaemia was corrected with epoetin.

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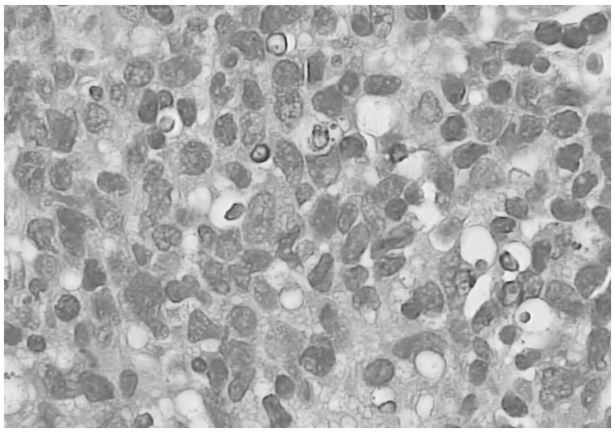

Fig. 1. Kidney biopsy showing an extensive and diffuse lymphocytic infiltration (haematoxylin–eosin, ×1000).

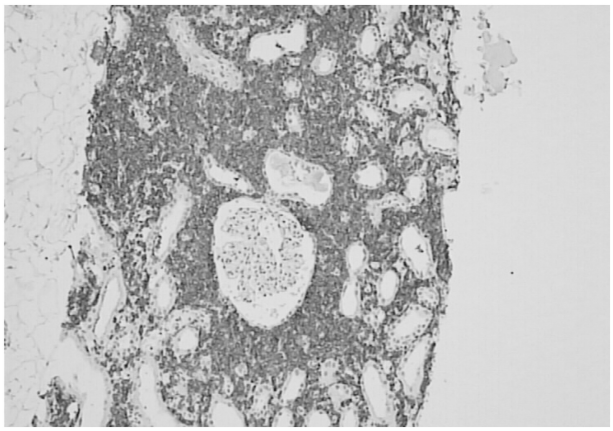

Fig. 2. Immunohistochemistry demonstrated a positive staining of the neoplastic cells for CD20 (×100).

Two weeks later, she recovered diuresis and improved renal function. At the time of hospital discharge (Day 45), uraemia and creatinine levels were 52 mg/dL and 1.4 mg/dL, respectively, and haemoglobin was 11.5 g/dL.

Discussion

This case illustrates several interesting points. The patient presented with diffuse LIK without involvement of other extranodal sites. This was accompanied by the uncommon occurrence of AKI requiring dialysis. The renal failure was completely reversed with chemotherapy.

Approximately 50% of patients with NHL will develop extranodal disease, most commonly involving the gastrointestinal tract, but the disease can arise in virtually any tissue [7]. Renal involvement has been reported in 6–60% of cases at autopsy [8,9]. When present, renal manifestations are non-specific and may include flank pain, haematuria, abdominal distension, or a palpable mass. Hypertension, presumably resulting from renal ischaemia from compression by the tumour, may also be found. Urinalysis usually reveals mild proteinuria, few red and white blood cells, and occasional hyaline and granular casts. Acute kidney injury due to LIK is uncommon, and it is rarely the initial manifestation of the lymphoma [1–6]. In one large autopsy series, only 0.5% of those with renal involvement had developed AKI [5]. The clinical picture of AKI and enlarged kidneys in our patient was suggestive of a renal infiltrative process, most likely a lymphoma. Acute kidney injury secondary to LIK, however, is a diagnosis of exclusion because kidneys may be massively infiltrated and still maintain relatively normal function. Only rapid reversibility of the renal failure with appropriate chemotherapy can convincingly establish the diagnosis [8].

In our patient, obstructive nephropathy was promptly excluded by ultrasonography. The absence of hypercalcaemia, severe hyperuricaemia and of uric acid crystals in the urine sediment made the diagnosis of hypercalcaemic or hyperuricaemic AKI unlikely. Compression of the renal arteries by lymphoma was ruled out by the absence of hypertension, and there were no signs suggestive of rupture of the renal pelvis or ureter, such as ascites or anuria. The presence of only mild proteinuria argued against a glomerular disease, and the renal biopsy did not show the typical glomerular lesions associated with lymphoma [10]. In addition, serum protein electrophoresis did not reveal monoclonal gammopathy, and there were no light chains on renal biopsy. Both the massively enlarged kidneys and the rapid improvement of renal function with chemotherapy strongly pointed towards the diagnosis of AKI due to LIK, and renal biopsy confirmed the extensive and diffuse infiltration of the kidneys by atypical lymphoid B-cells.

Although renal failure as a result of LIK is uncommon, it should be suspected in any patient presenting with an unexplained AKI and enlarged kidneys. Renal biopsy is an important tool to confirm the diagnosis. Prompt initiation of chemotherapy can result in a complete recovery of renal function.

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Conflict of interest statement. None declared.

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