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Case Report

Acute Renal Failure Due to Renal Lymphomatous Infiltration as the Initial Manifestation

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ABSTRACT

A male patient with acute renal failure (ARF) due to large B-cell non-Hodgkin lymphoma infiltration of kidney is presented. The diagnosis was suspected because of coincidence of ARF and tumor lysis syndrome non-responsive to conservative renal therapies. A renal biopsy confirmed diagnosis and appropriate chemotherapy led to complete improvement of renal function.

Key words: Acute renal failure, Non Hodgkin Lymphoma, Tumor lysis syndrome

Introduction

Renal involvement by large B-cell lymphoma is a very rare presentation of non-Hodgkin lymphoma (1). Acute renal failure (ARF) due to lymphoma infiltration of the kidney has been reported extremely rare in the literature (2). The patient who was hospitalized with upper GI-bleeding, tumor lysis syndrome, and oliguria, is a good example of such rare presentation of malignant lymphoma uncovered eventually by renal biopsy (3). Spontaneous tumor lysis is an extremely uncommon cause for ARF. ARF presenting with hyperkalemia, marked hyperuricemia, hyperphosphatemia, and hypocalcemia should lead to further workup for occult hematological malignancy (leukemia, Burkitt's lymphoma) and solid tumor (like small cell lung carcinoma and germ cell tumor) (4).

Case report

A 32-years old male was admitted to the emergency ward with acute GI bleeding, oliguria, edema, and hypotension. He also complained of nausea, vomiting, and loss of appetite. There was a history of cutaneous lesions in the upper chest and head 2 weeks ago. At that time, all lab tests were normal, except for the serum creatinine of 2.4 mg/dL and low phosphorus (2.1 mg/dL). However, his serum creatinine was normal 6 months ago.

On physical examination, there were exclusively axillary and inguinal lymph nodes enlargement and peripheral edema. Examination of other organs was normal. At the time of hospitalization, laboratory tests showed marked elevations for BUN (180 mg/dl), serum creatinine (11.2 mg/dl), LDH (2550 U/L), uric acid (41 mg/dl), phosphorus (19.6 mg/dl), and ESR (30 mm/hr). Urine sedimentation revealed hematuria, leukocyte casts, and many uric acid crystals.

Other lab tests including liver enzymes, serological and complement assays were within normal limits except for hemoglobin concentration which was lower due to GI bleeding. The ultrasound showed enlarged

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kidneys (right kidney 12.5 cm and left kidney 12.2 cm) without any urinary obstruction. Renal parenchyma was hypo-echoeic.

Because of elevated serum creatinine and uric acid and low creatinine clearance (below 5 ml/min), supportive and dose-adjusted pharmaceutical therapy was initiated. Fortunately, the serum creatinine and uric cid declined to 5.8 mg/dl and 10.2 mg/d respectively under medical and judicious hemodialysis therapy. Axillary and inguinal lymph nodes biopsy was performed, but they were reactive histologically.

Eventually, a renal biopsy was performed. Histologically, kidney specimen showed diffuse infiltration of malignant large B-cells of non-Hodgkin lymphoma (Figure 1). These cells were positive for CD20 and CD45 on immunohistochemistry staining. We could not find any sign of other organ involvement in staging including bone marrow and lymph node biopsies and the CT scan of head, chest, and abdomen. Thus, diagnosis of primary renal lymphoma was considered for the cause of ARF in this case.

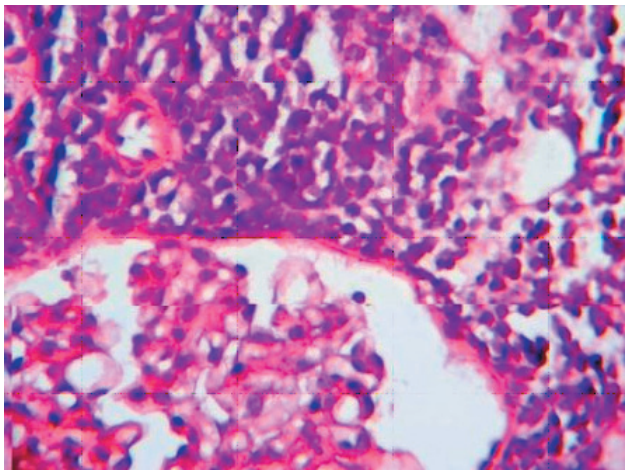

Figure 1: Diffuse infiltration of bizarre and malignant lymphocytes in the renal parenchyma (H&E × 400)

Chemotherapy (CHOP protocol) with adjusted dose was started with careful monitoring about aggravation of tumor lysis syndrome and judicious hemodialysis prescription. Fortunately, the dose-adjusted chemotherapy led to recovery of the renal function with a serum creatinine of 1.7 mg/dl and diminished renal size (RK 12 cm and LK 11.5 cm). The patient was discharged but unfortunately 2 months later, he died because of bone marrow involvement of B-cell lymphoma and complications due to chemotherapy (Figure 2).

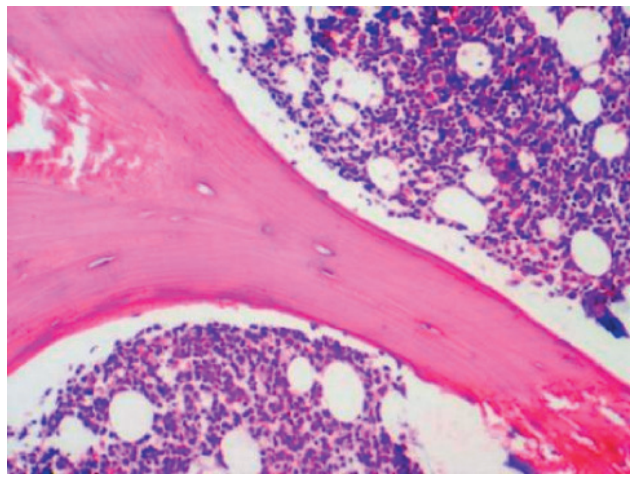

Figure 2: Bone marrow specimen shows diffuse infiltration of atypical lymphocytes (H&E × 400)

Discussion

ARF, as the initial manifestation, due to renal lymphomatous infiltration is very rare, although involvement of kidney by lymphomatous process occurs in 30-40% of cases of lymphoma, if the disease is left untreated (2). Incidence of renal involvement at initial presentation is 2.7%-6%. Mostly, it is in the form of insidious renal failure (1). In malignant disease, there are many causes of ARF including low perfusion due to renal vein thrombosis (5), urinary tract obstruction due to lymph nodes and tumor growth (6), and more commonly tumor lysis syndrome (7). As an example, acute tubular necrosis was identified as the cause of ARF in 90% of cases due to sepsis (96%) and nephrotoxic drugs (88%), in a single unit study over 10 years (8). A retrospective study in this setting showed 66% of patients recovered from ARF, but only 22% of cases survived and were discharged from the hospital and 78% died of ARF or from other complications (9). CHOP chemotherapy improves patients' survival from 6 to 8.6 months (10) and rituximab is associated with superior survival rates (11).

In literature, ARF due to lymphoma infiltration of the kidneys is reported in progressive disease (12). Patients with oliguria have worse outcomes than those without oliguria (9). In this case, despite conservative renal therapy with hydration, blood transfusion, and hemodialysis for lowering serum uric acid concentration, renal function did not improve completely. On other hand, leukocyte casts in the urinary sediment necessitated a renal biopsy to rule out various causes of ARF including, interstitial nephritis and proliferative glomerulonephritis.

In summary, kidney biopsy provided definite diagnosis of ARF cause and in this way to select the best treatment modality in this case.

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