Primary Lymphoma of the Kidney: Complete Remission After Systemic Chemotherapy

Renal involvement by lymphoma typically is manifested by parenchymal infiltration, ureteral obstruction due to lymph node enlargement, stone formation as a result of hypercalcemia or uricemia, or nephrotic syndrome (1,2). Extranodal lymphoma limited to the kidney is extremely uncommon as a presenting symptom or sign of lymphoma (3,6,8-10). We report here an unusual case of lymphomatous infiltration of the kidney presenting as uremia of unknown etiology. To our knowledge, this correspondence is the first documentation of chemotherapy-induced complete remission of primary lymphoma of the kidney.

A 52-year-old woman was hospitalized in August 1991 because of deteriorating renal function in conjunction with hypertension. The hospitalization was preceded by 2 months of weakness, intermittent nausea, myalgias, arthralgias, and generalized weakness. An outpatient evaluation revealed a blood pressure of 200/110 mm Hg. A 24-hour urine collection to determine levels of metanephrines, vanillylmandelic acid, and catecholamines revealed normal levels. The patient had no history of fevers, sweats, or unexplained weight loss. Her hypertension was controlled with clonidine and nifedipine.

Physical examination revealed no peripheral lymphadenopathy and no hepatosplenomegaly. A complete blood cell count disclosed a total leukocyte count of 6400/mm³ with a differential count of 66% granulocytes, 5% monocytes, and 26% lymphocytes. Flow cytometric analysis of the patient's peripheral blood mononuclear cells showed a normal ratio of T to B cells and no evidence of clonal excess. The hemoglobin level was 9.8 g/dL, the hematocrit was 27.9%, and the platelet count was 277000/mm³. Laboratory studies disclosed the following values in serum: blood urea nitrogen of 43 mg/dL with a creatinine level of 5 mg/dL, serum lactate dehydrogenase elevated to 461.0 U/L (normal, 220-240 U/L), and normal liver transaminases and alkaline phosphatase. A serologic test for human immunodeficiency virus (HIV) was negative. The reticulocyte count was 0.8%, the level of serum iron was 50.0 µg/dL, and the level of serum ferritin was 217.0 ng/dL. A serum protein electrophoresis gave normal results. A 24-hour urine collection revealed 686.0 mg protein; immunoelectrophoresis of the urine revealed no paraprotein.

Bilateral bone marrow biopsy specimens revealed normal cellular marrow without evidence of lymphoma. A computerized axial tomogram (CT) scan of the abdomen demonstrated lobulation and slight enlargement of both kidneys. A percutaneous needle biopsy of the left kidney disclosed an atypical lymphocytic infiltrate. An open biopsy of the left kidney revealed a diffuse infiltrate of large, cleaved lymphocytes consistent with large-cell lymphoma. Immunophenotypic stains confirmed expression of B-cell markers. No other sites of disease were identified by further systemic staging, including CT scans of the chest and abdomen. Systemic chemotherapy of prednisone, methotrexate, doxorubicin, cyclophosphamide, etoposide–cytarabine, bleomycin, oncovin, methotrexate (ProMACE–CytaBOM) was started (7). After the first two cycles of chemotherapy, renal function normalized, with levels of blood urea nitrogen at 20.0 mg/dL and creatinine at 0.9 mg/dL. After four cycles, a follow-up CT scan showed a normalization of kidney size. After six cycles of ProMACE–CytaBOM, a repeat CT scan revealed no additional changes, and a percutaneous needle biopsy of the kidney demonstrated no evidence of residual lymphoma.

As a presenting symptom or sign, uremia due to lymphomatous infiltration of the kidney without other nodal sites of disease is uncommon, having been noted in only a few individual case reports (3,5,6,8-10). In all but one of these reports, flank pain and/or an abdominal mass was present to suggest malignancy involving the kidney. In two cases in which the patients presented with uremia and had no evidence of flank pain or abdominal mass, the diagnosis was established only at autopsy (3,10).

Ellman et al. (5) was the first to report a patient with uremia due to lymphomatous infiltration of the kidney in which the diagnosis was established by biopsy. Their patient was treated with systemic chemotherapy but died of complications and never achieved remission. Thus, this correspondence is the first report of a patient who had uremia due to occult lymphomatous infiltration of the kidney and was treated successfully with chemotherapy.

This patient has now been followed for approximately 14 months since completion of chemotherapy and shows no evidence of lymphadenopathy or other abnormalities that suggest recurrent lymphoma. Renal function remains normal, and her hypertension is controlled with low doses of clonidine. Our experience in this patient certainly suggests that primary lymphoma of the kidney can be effectively treated with systemic chemotherapy and that renal function will return to normal.

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adjusted incidence rates of U.S. Black men have been estimated at nearly twice those of U.S. White men at all ages in Los Angeles (2). Therefore, it is important to assess whether this difference in testosterone levels has a genetic or an environmental basis. Preventive measures may be taken more readily against some environmental factors. The following considerations fail to support the notion that the racial difference in testosterone levels has a genetic basis.

Iványi et al. (3) noted that genes on the H-2 system (the murine equivalent of the human HLA system) control steroid hormone metabolism in mice, and they predicted a similar situation in humans. The prediction was confirmed by GerenCer et al. (4) and Ollier et al. (5). The inequality above holds for neither the HLA-A nor the HLA-B system of antigens. Therefore, it seems that the racial variation in testosterone levels may exist despite, not because of, these genetic factors. This judgment may later have to be modified if other genes are shown to be associated with testosterone levels. However, at present, it appears that racial variation in testosterone levels may depend, on sociocultural factors, e.g., diet or stress.

Because of the apparent associations of testosterone levels with both prostate cancer and the HLA gene system, it would be interesting to know whether prostate cancer has any associations with the relevant HLA genes.

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References