Case Report

Diffuse Large B-Cell Lymphoma Presenting as Isolated Epitrochlear Lymphadenopathy with Renal Involvement: An Unusual Presentation

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Abstract

Although epitrochlear lymph nodes may be enlarged as a part of generalized lymphadenopathy, isolated enlargement of epitrochlear lymph nodes is rarely seen. Epitrochlear lymph node involvement, as a first presentation, is rarely seen in non-Hodgkin's lymphoma. We describe non-Hodgkin's lymphoma in a 60-year-old male who presented with isolated epitrochlear lymphadenopathy along with other organ involvement, including the kidneys.

Keywords: Non-Hodgkin's lymphoma, Diffuse large B-cell lymphoma, Epitrochlear lymph node

Introduction

Non-Hodgkin's lymphomas (NHL) are a heterogeneous group of malignancies of the lymphoid system. B-cell lymphoma accounts for approximately 90% of all lymphomas.1 Lymphomas often produce marked nodal enlargement that is almost always non-tender. Epitrochlear lymph node involvement is a rare occurrence in both Hodgkin's lymphoma and NHL, either as sole site of involvement or as a part of more widespread disease. Only very limited information is available on the association of epitrochlear nodes and lymphoma.2 We hereby report a case who presented solely with epitrochlear lymphadenopathy diagnosed as diffuse large B-cell NHL(DLBCL). Staging work up revealed involvement of the kidneys, and bilateral adrenals with multiple small lesions in the pancreas.

Case Report

A 60-year-old male, a chronic smoker, was admitted in our institution with complaints of swelling of his right elbow for three months. He also complained of loss of appetite and weight loss for the past one year along with right flank pain for the past 15 days. Pain was
insidious in onset, a dull ache and non-radiating. One episode of hematuria was also documented two weeks prior to his hospital visit. There was no history of animal bites or scratches, syphilis or HIV exposure. Physical examination revealed a painless enlarged, firm, non-tender and non-mobile epitrochlear lymphnode on his right side that measured 2x1 cm. There was no lymphadenopathy at any other site including the cervical, axillary and inguinal regions. On systemic examination there was fullness of the right flank and renal angle which was tender, extended below the costal margin and immobile with respiration. The remainder of the examination was within normal limits. Hematological findings revealed hemoglobin (Hb;8 g/dl);total leucocyte count(5600 cells/µl); differential leucocyte count with neutrophils (N; 76%), lymphocytes (L: 18%), monocytes (M:4%), and eosinophils (E; 2%); platelet count(3.5 lac/µl); and ESR (50mm in the first hour). Urine microscopy revealed 10-12 pus cell/high power field (hpf) and 6-8 RBC/hpf. Renal function tests were mildly deranged as evidenced by blood urea (70 mg/dl), serum creatinine (1.9 mg/dl), and blood uric acid (5.6 mg/dl) with elevated LDH (1813 U/l). Liver function tests of SGOT(70 U/l), SGPT (62 U/l), and ALK (109IU/l) had minor elevated levels and electrolyte levels were within normal limits. X-ray of the chest showed both lung fields and costophrenic angles clear with no radiological evidence of enlarged hilar or mediastinal lymph nodes. Ultrasound (USG) revealed an enlarged right kidney that measured 14.3x7.4 with loss of corticomedullary differentiation and an isolated hypodense renal mass that measured 2.7x1.8 cm at the lower pole. There was bilateral enlargement of the adrenal glands and a hypoechoic lesion in the pancreas. Liver, spleen and gall bladder were normal. Contrast enhanced computerized tomography (CECT) of the abdomen showed an enlarged right kidney with poorly defined hypodense lesions that obliterated corticomедullary differentiation with patchy areas of non-enhancement in the left kidney along with nodular and irregular areas in the bilateral adrenals with multiple small lesions in the pancreas (Figure 1). CECT thorax was within normal limits. Excision biopsy of the epitrochlear lymph node was performed and the histopathology revealed a diffuse growth pattern (Figure 2a and 2b) with large cells that had the appearance of medium-to-large-sized lymphocytes with scanty cytoplasm, oval-to-round nuclei that contained vesicular chromatin, with prominent nucleoli within each nucleus (Figure 2a and 2b). Immunohistochemical examination showed positivity for LCA, CD 19 (Figure 3a) and diffuse membrane staining for CD20 (Figure 3b). Histological diagnosis of diffuse large B cell lymphoma (DLBCL) was rendered. The patient was advised to undergo a renal biopsy but he did not give consent. He underwent a chemotherapy regimen for DLBCL that consisted of R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine and prednisolone) where each cycle was repeated every three weeks for eight cycles. Ultrasound of the abdomen was repeated after three completed cycles of chemotherapy which revealed regression of the renal mass.

A final diagnosis of DLBCL with isolated epitrochlear lymphadenopathy along with renal, adrenal and pancreas involvement was made.

Discussion

Non-Hodgkin’s lymphoma exhibits vast diversity in the cause and location of disease
manifestation in the body and the nature of its progression. Non-Hodgkin's lymphoma comprises many subtypes, each with distinct epidemiology, etiology, and morphologic, immunophenotypic, and clinical features. Most NHL are of B-cell origin. The most common NHL subtypes include diffuse large B-cell lymphoma and follicular lymphoma. Epitrochlear nodal involvement is a rare occurrence in NHL, either as the sole site of involvement or as part of more widespread disease. Only very limited information is available on the association of epitrochlear nodes and lymphomas. Renal infiltration is also an uncommon finding in NHL.

Diffuse large B-cell NHL is the largest subtype of NHL and is characterized by relatively frequent extranodal presentation. The most common extranodal sites are the stomach, CNS, bone, testes and liver. Renal involvement is a relatively uncommon clinical presentation of NHL. When it does occur, it is usually due to secondary involvement rather than primary lymphoma. As the kidneys lack lymphoid tissue, the origin of primary renal lymphoma (PRL) is controversial. The various possible pathologies for the development of lymphoma in these cases involve the presence of a chronic infection in the kidney that recruits lymphoid cells into the renal parenchyma during which an oncogenic event takes place. Another explanation is that lymphomas arise in the renal capsule, which is rich in lymphatics, and secondarily invade the renal parenchyma. Another possible origin of primary renal lymphoma is a lymphomatous process in the peri-renal adipose tissue with secondary involvement of the kidneys.

Among cases of renal lymphoma, between 37% and 47% occur due to dissemination of an advanced systemic disease, while 0.1% are due to primary involvement of the kidney. Primary renal lymphoma is a rapidly progressive disease (from renal to lymph node), so that it is not possible to determine whether renal affliction is primary or secondary from the onset of the disease. Either primary or secondary can make solid or bilateral involvement and their treatment modality for both presentations is chemotherapy. Diagnostic percutaneous biopsy of a renal mass is generally unnecessary because common non-malignant lesions, such as a simple cyst, angiomyolipoma, abscess, and xanthogran-

Figure 2. Microphotograph showing diffuse effacement of lymph node architecture (2A; H&E; 40×, 2B; Retic;40×) by medium-to-large sized lymphoid cells with scanty cytoplasm, round-to-oval nuclei that contain vesicular chromatin and prominent nucleoli (2C&D; H&E; 100×and 200×).
ulomatosus pyelonephritis have characteristic radiographic findings and/or clinical histories. Renal lymphoma, however, is important to include in the differential diagnosis of renal masses because generally it is a systemic disease and treatment is non-surgical\textsuperscript{10}.

Our case is unique because of the unusual presentation of DLBCL with involvement of epitrochlear lymph node, along with combination of B symptoms (weight loss and night sweats) and renal symptoms (flank pain, hematuria and deranged renal function).

**Conclusion**

Solitary epitrochlear lymphadenopathy as presentation is uncommon in NHL in association with renal involvement. Renal lymphoma should be kept in the differential diagnosis of a renal mass since high cure rates are achieved by chemotherapy and the avoidance of an unnecessary radical nephrectomy.

**Conflict of Interest**

No conflict of interest is declared.

**References**