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Primary Renal Lymphoma: An Unusual Finding Following Radical Nephrectomy. Case Report

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ABSTRACT

Primary renal lymphoma is a rare malignant disease. We present the case of a 48 -year-old woman who presented to our hospital with asthenia, anemia and intermittent pain in the left flank. An abdominopelvic CT scan revealed a mass in the left kidney that mimicked a renal cell tumor. The patient underwent open left radical nephrectomy because of the large tumor size. The histopathological finding was surprising, considering the diagnosis of renal lymphoma.

The patient underwent further investigations to confirm the diagnosis of primary renal lymphoma. Currently, the treatment for renal lymphoma is not surgical, but rather chemotherapy. We present this case because very few studies have been published, which contributes to the literature by understanding the presentation of this entity mimicking a clear cell tumor.

Keywords: Kidney neoplasms; Lymphoma; Nephrectomy; Open surgery

Introduction

Primary renal lymphoma (PRL) is defined as non-Hodgkin lymphoma (NHL) involving the kidney in the absence of primary extrarenal lymphatic disease¹. Since the first case was reported by Knoepp in 19563, the existence of this disease has remained controversial because the kidney lacks lymphoid tissue in its parenchyma. With the description of the diagnostic criteria for primary renal lymphoma postulated by Malbrain in 1994 no more than 70 cases of PRL have been reported in the literature and the majority are NHL large B-cell type².

In adult patients, PRL is often present on only one side of the kidney. Occasionally, patients with PRL present nonspecific symptoms and signs, flank pain, weight loss, fever, hematuria and a palpable mass³. The lack of precise knowledge of the disease has led to different therapeutic approaches and the reported prognosis in general has been very poor, with 75% mortality before one year after the diagnosis was established⁴.

We review this case of primary renal lymphoma that was diagnosed based on the histopathological report of the surgical specimen after performing radical nephrectomy as a diagnostic suspicion of renal cell carcinoma.

Case Presentation

A 48-year-old female presented with the following medical conditions Systemic arterial hypertension in treatment. She denied any allergies or history of surgery. She reported a history of asthenia and adynamia for three months, accompanied by intermittent pain in the left flank. She denied having hematuria, fever or weight loss. She underwent enhanced CT examination (**Figures 1,2**) at our hospital and a mass (10 x 9 cm in diameter) was noted in the left kidney.



Figure 1: Axial computed tomography revealing a mass at the left kidney without enhancement upon administration of contrast medium (red dots). With measures of 9x 8 cm with 8 HU



Figure 2: Computed tomography coronal section showing a left kidney mass located at the upper pole (red dots) with measures of 10x 9 cm that has contact with the spleen

On physical examination, the abdomen was distended due to adipose tissue, normal bowel sounds were auscultated and abdominal mass is palpated at the level of the left renal fossa. There was no sign of either lymphadenopathy or hepatosplenomegaly.

Her laboratory results were as follows: (Table 1)⁵.

Given the suspicion of left kidney cancer radical nephrectomy and lymph node dissection were performed. With the following surgical findings: (Figure 3) Kidney measured 7 x 6 x 5 cm, with a tumor located in the upper pole of approximately 12 cm, with abundant newly formed vessels, loose and firm adhesions tumor-spleen and tail of pancreas, one artery, one vein and one ureter (Figure 3).

An open approach was performed with a Chevrone-type incision, transperitoneal approach. The average operating time

was 2 h 30 min and the estimated blood loss was 1500 ml due to the abundant neo formed vessels that the tumor had.



Figure 3: Surgical specimen.

A) Surgical specimen resulting from radical nephrectomy with lymph node indicated by the red arrow.

B) Macroscopic section from the pathology service.

Table 1: Laboratory results.

Test	Observed Value	Reference Range
hemoglobin level	9.5g/dL	12.1-15.1 g/dL
leukocyte count	3.4 x10 ⁹	4.0-11.0 x 10 ³ /µL
platelets	155x 10 ³	$150-450 \text{ x } 10^{3/}\mu\text{L}$
erythrocyte sedimentation rate	34,00 mm/h	0–15 mm/h
glucose	77 mg/dL	70-130 mg/dL
sodium	127 mmol/L	135-145 mmol/L
potassium	4.70 mmol/L.	3.5-5.0 mmol/L
lactate dehydrogenase	896 U/L	140–271 U/L
serum creatinine	0.7 mg/dL	0.6-1.3 mg/dL
albumin	3.40 g/dL	3.5-5.5 g/dL
alanine transaminase (ALT)	15 U/L	7-56 U/L
aspartate transaminase (AST)	34 U/L.	5-40 U/L

The tumor was sent to the pathology department and histological examination revealed the following: (Figure 4)

(A) An infiltrate of atypical lymphoid cells was observed with a diffuse growth pattern that erased the architecture of the renal parenchyma (10x).

(B) In another section, we can identify the same infiltrate with respect to two glomeruli of the residual kidney (asterisk)(10x).

(C) At higher magnification, the atypical lymphoid cells are medium to large with vesicular chromatin and evident nucleoli alternating with areas of necrosis (arrowheads)(40x).

(D) Abundant mitotic figures are observed at the center (arrows). Comparison of the size of the lymphoid cell defined as large (arrowhead), which is larger than the size of the nucleus of a histiocyte (circle) (40x). Postoperative pathology and immunohistochemistry indicated diffuse large B-cell lymphoma localized only to the kidney⁶.

Postoperative immunohistochemical staining (Figure 5) revealed Diffuse large B- cell lymphoma (DLBCL) with the following results:CD20 (+), MUM1 (+), CD10 (-), Bcl2 (+), Bcl6 (+), CD3 (-) and Ki-67 (>80% +).



Figure 4: Microscopic section of the pathology service

A) An infiltrate of atypical lymphoid cells is observed with a diffuse growth pattern that erases the architecture of the renal parenchyma (10x).

B) In another section we can identify the same infiltrate respecting two glomeruli of the residual kidney (asterisk)(10x).

C) At higher magnification, the atypical lymphoid cells are medium to large with vesicular chromatin and evident nucleoli that alternate with areas of necrosis (arrowheads)(40x).

D) Abundant mitotic figures are observed in the center (arrows).



Figure 5: Immunohistochemistry staining of the resected left renal kidney

The antibodies used were as listed in A, B, C, D, E, F, G each representative graph ($\times 200$).

A) CD20: (+) 100% B) Mum 1 (+) 100% C) CD10 (-) D) BCL2 (+) 100% E) BVL6 (+) 90% F) CD3 (-) G) KI-67 (+) 80% Bone marrow biopsy and CT of the chest region did not reveal any evidence of lymphoma invasion. Based to these findings, the patient was diagnosed with PRL. After the operation, the patient was admitted to the intensive care unit because of hypovolemic shock. She remained in intensive care for 3 days; two packs of blood were transfused, with a control hemoglobin of 9.2 g/dL. The patient remained hospitalized for 7 days and was discharged with a referral to the hematology service to assess adjuvant chemotherapy based on the histopathological results.

She was evaluated by the hematology department in the outpatient clinic, who decided to prescribe the RCHOP regimen: 6 cycles of a combination of 1 g cyclophosphamide on day 1,80mg epirubicin on day 1,3mg vindesine on day 1 and 10 mg dexamethasone on days 1-5 (1 cycle, 28 days). The patient is currently being monitored and has been disease-free for two years.

Follow-up included CT scans of the chest, abdomen and pelvis every 6 months, as there was no established follow-up regimen for these patients.

Epidemiology

Primary renal lymphoma (PRL) is an infrequent but clinically important entity. While the existence of true PRL (originating solely within the kidney) remains debated, it represents a rare manifestation of lymphoma, distinct from secondary renal involvement observed in 30-60% of lymphoproliferative disorders⁶. Diffuse large B-cell lymphoma constitutes the most common histological subtype^{7,10}.

Epidemiologically, PRL demonstrates a male predominance (male-to-female ratio of 1.6:1) and typically presents in older adults, with a mean age of approximately 72 years^{6,11}. Bilateral involvement is uncommon, reported in only 2% of cases⁶. While generally considered an aggressive malignancy with a median survival of 15 months, this figure likely reflects diagnostic delays and historical treatment approaches⁶.

Pathophysiology

The pathogenesis of PRL is incompletely understood. The paucity of lymphoid tissue within the renal parenchyma suggests that the disease originates in the renal capsule, subsequently infiltrating the kidney⁸. Alternatively, chronic inflammatory kidney conditions may predispose to PRL development by attracting lymphoid cell infiltration, ultimately resulting in lymphomatous transformation⁹. Tumor proliferation commences in the interstitium, with the underlying nephrons and vasculature providing a scaffold for tumor growth, thereby preserving renal contour³. As the tumor enlarges, compression of the surrounding renal parenchyma ensues, potentially leading to architectural distortion¹.

Clinical presentation

Clinical presentations are variable, ranging from asymptomatic discovery as an incidental renal mass (in approximately 50% of patients) to non-specific renal symptoms (flank pain, hematuria, altered general condition) or systemic B-cell symptoms in advanced stages¹¹. Differential diagnosis should include other renal tumors, particularly in the presence of atypical renal mass characteristics or unexplained renal symptoms, warranting consideration of PRL, particularly given its potential for rapid systemic dissemination¹². Currently defined diagnostic criteria include: (I) renal mass presence, (II) absence of extrarenal lymphomatous involvement (viscera or lymph nodes) and (III) exclusion of leukemic hematoma and bone marrow involvement¹.

Imaging

Radiological findings on contrast-enhanced computed tomography (CT) scans typically reveal a solitary mass exhibiting minimal or absent enhancement. Isolated perinephric lymphoma, while infrequent (<10% of cases), is highly suggestive of PRL. [3, 9] The absence of calcifications, homogenous post-contrast attenuation and lack of renal vein thrombus or mass effect are also differentiating features in solitary renal tumors⁴.

Definitive diagnosis requires pathological confirmation via needle biopsy or, frequently, post-nephrectomy examination of the surgical specimen due to the similarity of imaging characteristics to renal cell carcinoma¹³.

Treatment

Treatment strategies have evolved since PRL's initial description in 1956, encompassing surgery combined with adjuvant radiotherapy, surgery alone and systemic chemotherapy¹⁴. Current treatment typically involves 6-8 cycles of cyclophosphamide, doxorubicin, vincristine and prednisone (CHOP) or, for CD20-positive non-Hodgkin lymphoma, in combination with rituximab (R-CHOP) to improve 5-year survival^{14,15}.

Preoperative biopsy confirmation is crucial to avoid unnecessary radical nephrectomy, enabling standardized routine chemotherapy with potentially favorable prognosis¹⁵.

The patient in this report presented with an incidentally discovered left renal mass. Initial suspicion of renal cell carcinoma prompted open nephrectomy, with subsequent histological evaluation revealing diffuse large B-cell PRL. The patient received six cycles of R-CHOP, with follow-up examinations revealing no local recurrence.

Conclusions

This case highlights the importance of considering PRL in the differential diagnosis of renal masses. While histological evaluation remains paramount, increased awareness of characteristic clinical and radiological features can guide appropriate management, potentially precluding unnecessary surgical intervention. Further research is needed to optimize diagnostic and therapeutic strategies for this rare malignancy.

Declarations

Contributors

All authors contributed to planning, literature review and conduct of the review article. All authors have reviewed and agreed on the final manuscript.

Competing interests

None.

Patient consent for publication

Informed consent was obtained from the patient, consent form available upon request.

Ethics approval and consent to participate

Consent for treatment and open access publication was

obtained or waived by all participants in this study. Clinical Trials issued approval NCT06525467. Consent for treatment and open access publication was obtained or waived by all participants in this study. Mexican Institute of Social Security issued approval 98337030. Written informed consent was obtained from the patient for the publication of this case report and accompanying images. The study was registered in ClinicalTrials.gov (identifier: NCT06525467).

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