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CASE REPORT

Primary renal lymphoma presenting as renal failure: A case report and review of literature from 1989

Seul-Bi Lee, Young-Min Yoon, Ran Hong

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Abstract

BACKGROUND

Primary renal lymphoma (PRL) is extremely rare with an incidence of 0.7% among extranodal lymphomas. Occult renal lymphoma, which mimics medical renal disease and bilateral renal involvement, presents a diagnostic challenge to nephrologists and radiologists as the clinical and radiological findings are mostly non-specific or inconclusive. Acute kidney injury (AKI) is not an uncommon finding in renal infiltration due to malignant lymphoma. However, only 14% of cases are detected before death, and the low diagnostic rate may be due to the non-specific clinical manifestations of renal involvement, with only 0.5% of these cases presenting with AKI. Moreover, PRL is difficult to diagnose based on clinical, biochemical, and radiologic features, especially, in the case of bilateral diffuse involvement.

CASE SUMMARY

Herein, we report a 74-year-old woman with primary diffuse large B-cell lymphoma who presented with AKI diagnosed by ultrasound-guided needle biopsy. We also report the clinicopathologic findings of 121 PRL cases reported since 1989, by conducting a literature review of published cases.

CONCLUSION

A timely renal biopsy provides the most expedient means of establishing the diagnosis. Thus, early identification of the disease by the clinician facilitates early diagnosis toward effective treatment.

Key Words: Lymphoma; Kidney; Acute kidney injury; Case report

Core Tip: We report a patient with bilateral primary renal lymphoma (B-PRL) presenting with acute kidney injury (AKI), and conducted a literature review of 121 cases of PRL since 1989. Among the 121 cases, 29.8% were bilateral. AKI occurred in all bilateral cases, and 71.4% of patients who died were diagnosed with B-PRL. There is a need to discuss more active treatment for B-PRL. In particular, differentiating diffuse involvement of lymphoma from other kidney diseases causing AKI is difficult clinically or radiologically; therefore, a kidney biopsy is essential for the diagnosis. Clinicians should endeavor to make a preoperative diagnosis, to avoid unnecessary surgery.

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INTRODUCTION

Primary renal lymphoma (PRL), defined as a lymphoma involving the kidneys in the absence of extrarenal lymphoma, is a rare disease. Additionally, PRL accounts for approximately 0.7% of extranodal lymphomas[1], as the kidney is an extranodal organ and the renal parenchyma does not contain lymphatic tissue[2]. Occasionally, patients present with nonspecific signs and symptoms including flank pain, weight loss, hematuria, a palpable mass, or symptoms of acute kidney injury (AKI). Evaluation of renal lymphoma is important and includes differentiating PRL from other renal neoplasms, making pathologic diagnoses, and preserving renal parenchyma and function[3]. Radiologically, the typical computed tomography (CT) pattern in renal lymphoma can be grouped approximately as multiple renal masses (approximately 60%, most common), solitary masses (< 6%, rarest), renal invasion from retroperitoneal disease, or diffuse renal infiltration[1,3-6]. The diffuse infiltration pattern is always bilateral, observed in approximately 25%-30% of renal lymphomas[6]. Moreover, the pattern is difficult to diagnose by imaging alone due to the non-specific manifestations. In particular, PRL with a diffuse growth pattern in the bilateral kidney may clinically mimic medical renal disease and even escape detection during the routine radiological work-up, including ultrasonography (US) and CT, preceding biopsy. Percutaneous renal biopsy is generally used in the diagnosis of medical renal diseases, although the indications for biopsy vary. Renal neoplasms, which are typical urological disorders, are not generally recommended for percutaneous biopsy [7], but the tumor may be detected incidentally during a biopsy. For a rapid and confirmative diagnosis, kidney biopsy remains the gold standard. Therefore, although rare, clinicians should consider lymphoma as a differential diagnosis during percutaneous renal biopsy for diagnosing the aforementioned lesions. According to a population-based analysis using the Surveillance, Epidemiology, and End Results Program, factors such as old age, primary diffuse large B-cell lymphoma (DLBCL) histologic type, and male patients are associated with short overall survival (OS)[8].

To the best of our knowledge, to date, 121 cases of PRL have been reported in the literatures[1,2,4,9-97]. Herein, we report the case of primary renal DLBCL of 74-year-old woman presenting with AKI, diagnosed by US-guided needle biopsy. We also conducted a literature review of the 121 cases reported since 1989 and described their clinicopathologic findings. This study was approved by the Institutional Review Board (IRB) of Chosun University Hospital, Gwangju, Korea, which waived the requirement for written informed consent due to the nature of the study (IRB No. 2023-02-020).

CASE PRESENTATION

Chief complaints

A 74-year-old woman who was treated outside our hospital due to complaints of general weakness, hematuria, dysuria, and decreased renal function was transferred to Chosun University Hospital, Gwangju, Korea, as her renal function did not improve despite treatment.

History of present illness

Clinically, rapid progressive glomerulonephritis (RPGN) was suspected. Two days after admission, US-guided percutaneous renal biopsy was performed to confirm the pathologic diagnosis, before initiating steroid treatment. Contrary to expectations, the light microscopic examination identified diffuse infiltration of pleomorphic cells throughout the specimen. The pleomorphic cells were immunoreactive for CD20, bcl-2, bcl-6, and MUM-1, but negative for CD3, CD10, and Epstein-Barr encoding region in situ hybridization (Figure 1). No fluorescence deposit was identified during immunofluorescence examination. In electro-microscopic examination, no electron-dense deposit was observed, and the glomerular basement membrane appeared normal in thickness, contour, and texture. However, strikingly, diffuse prominent infiltration of atypical lymphocytes was observed in the interstitium. The cells displayed round to oval cleaved and non-cleaved nuclei with variable clumping of chromatin, and large prominent, marginated nucleoli (Figure 1). We

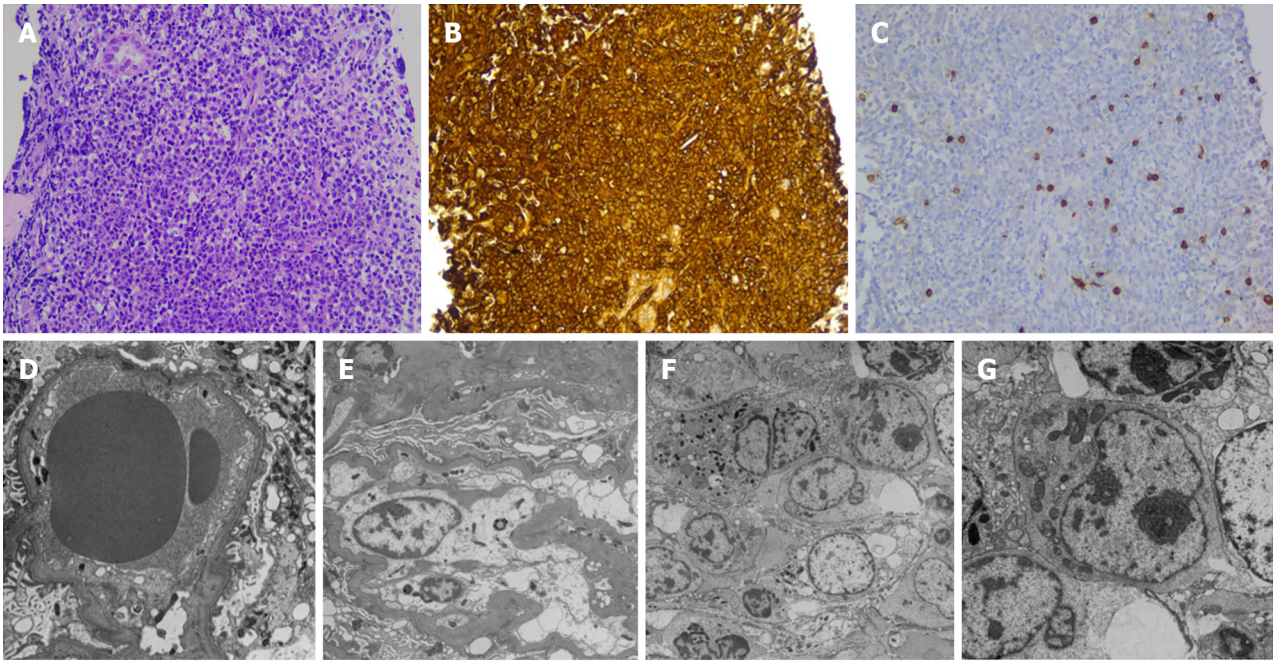

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Figure 1 Histologic, immunohistochemical examination and electron-microscopic findings. A: Diffuse infiltration of pleomorphic cells is identified throughout the specimen (HE × 5) (× 2.0 k); B: The cells were immunoreactive for CD20 (× 20) (× 1.5 k); C: Negative for CD3 (× 20) (× 1.0 k); D and E: No electron-dense deposit is recognized, and glomerular basement membrane appeared normal in thickness, contour, and texture; however, strikingly, diffuse prominent infiltration of atypical lymphocytes is seen in the interstitium (× 2.0 k); F and G: The cells exhibited round to oval cleaved and non-cleaved nuclei with variable clumping of chromatin and large prominent marginated nucleoli.

diagnosed the condition as DLBCL, not medical renal disease. After pathologic diagnosis, radiologic re-evaluation was performed. Abdominal CT examination (with contrast) revealed diffuse homogeneous enhancement in both kidneys without definite visible cortico-medullary differentiation, and lymphomatous involvement was diagnosed. A few mild enlargements of lymph nodes in the pericardial and periaortic chains were identified, and such nodes were considered to indicate secondary lymphomatous involvement. On fluorodeoxyglucose-positron emission tomography CT, intense hypermetabolism (19.6) was identified in both kidneys, and some lymph nodes exhibited mild hypermetabolism (Figure 2). In the laboratory tests, serum lactate dehydrogenase (LDH) level was elevated to 376 U/L (125-220 U/L). The international prognostic index (IPI) was reported as 3 when the following laboratory data and clinicopathologic factors were considered [old age, 1; Eastern Cooperative Oncology Group (ECOG) performance status (PS), 1; Ann Arbor stages III–IV, 0; serum level > 1 × normal, 1; and > 1 extranodal site, 0].

History of past illness
The patient had no previous renal problems.

Personal and family history
There was no specific personal or family history.

Physical examination
The patient looked ill.

Laboratory examinations
After admission, the blood urea nitrogen (normal range, 7.0-20.1 mg/dL)/creatinine (0.57-1.11 mg/dL) levels on June 30, July 9, and July 11, 2022 were as follows: 27.7/4.09; 41.0/6.61; and 48/7.62 mg/dL, respectively.

Imaging examinations
Radiologically, renal US exhibited heterogeneously increased parenchymal echogenicity and a 1.43 cm-sized hypoechoic cystic lesion in the right kidney. Thus, the radiologist suggested probable medical renal disease with a right cystic lesion. During abdominal CT (contrast-free CT while admitted to the emergency room), no neoplastic lesion was suspected.

FINAL DIAGNOSIS
The patient was diagnosed with DLBCL.

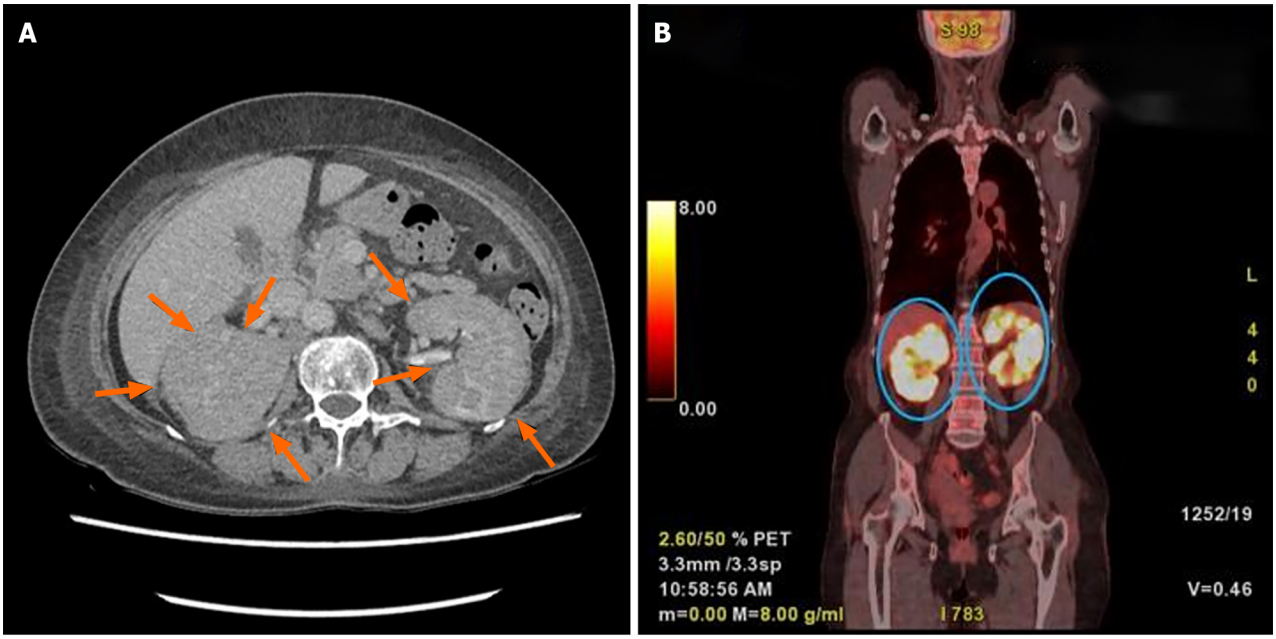

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Figure 2 Radiologic findings. A: Abdominal computed tomography (CT) examination revealed diffuse homogeneous enhancement in both kidneys without definite visible cortico-medullary differentiation and was diagnosed as lymphomatous involvement; B: On fluorodeoxyglucose-positron emission tomography CT, intense hypermetabolism was also identified in both kidneys.

TREATMENT

She was immediately started on chemotherapy (CTx), which was composed of rituximab-cyclophosphamide, vincristine, adriamycin, and prednisolone.

OUTCOME AND FOLLOW-UP

The patient succumbed to her illness 3 mo after diagnosis during her third round of CTx.

DISCUSSION

As demonstrated during the literature search, according to Stallone *et al*[5] who reported 29 cases in a literature review in 2000[66,76,98,99], PRL is very rare with an incidence of 0.7% in extranodal lymphomas[1]. Since 1989 when Paganelli *et al* [2] presented the first patient with PRL diagnosed by open kidney biopsy, to our knowledge, 121 cases have been reported in the medical literature, including our case (Table 1). The present study reviewed all 121 cases of PRL reported in the literature since 1989.

Of these 121 cases, the male-to-female ratio was 1.6:1 (72:45; unknown, 4 patients). The average age of the patients was 55 years, and the distribution is displayed in Table 2. A total of 36 (29.8%), 81 (66.9%), and 4 (3.3%) cases of bilateral renal involvement, unilateral lesion, and unknown, respectively, were observed. The bilateral involvement in PRL may be age-related. Patients aged < 40 years have higher bilateral renal involvement (17/23, 73.9%) compared with those aged ≥ 40 years (19/98, 19.4%) (Table 2).

Histologically, DLBCL is the most common (62/121, 51.2%) lymphoma, followed by marginal zone lymphoma (23/121, 19.0%). Symptoms and signs include abdominal distension, fever, flank pain, nausea and vomiting, hematuria, frequency, urinary retention, hydronephrosis, and AKI. Some cases were identified incidentally without any symptoms. According to Coggins[100], AKI in renal lymphoma may occurs by several causes such as infections and obstructive urinary disease with leukemic infiltration contributing to the progression of renal failure. Bridoux *et al*[101] suggested that invasion of lymphoma to the renal interstitium induces compression of tubules and peritubular capillaries, which leads to tubular obstruction and increase of post-glomerular vascular resistance.

In the current review, AKI with symptoms was found in 11 cases (8.9% of all the cases and 30.6% of cases with bilateral lesions), and all exhibited bilateral diffuse renal involvement. However, the incidence of AKI did not correlate with age but occurred more consistently in those over 40 years old (< 40 years old, 1/23, 4.3%) (Table 3).

Renal lymphoma can present as a solitary mass (10%-25% of cases) or multiple parenchymal nodular masses of variable sizes, typically 1.0 to 4.5 cm in diameter, which is the most common pattern in approximately 50%-60% of cases. The second most common pattern is a retroperitoneal nodular lesion with continuous extension into the kidneys or

Table 1 Summary of 121 cases of primary renal lymphoma since 1989, *n* (%)

Factor		Factor	
Gender		Age (yr)	
Male	72 (58.5)	≤ 20	16 (13.2)
Female	45 (36.6)	21-40	7 (5.8)
Unknown	4 (3.3)	41-60	41 (33.8)
		61-80	48 (39.7)
		≥ 80	9 (7.4)
Site		Prognosis	
Unilateral	81 (66.9)	Died due to lymphoma	21 (17.4)
Bilateral	36 (29.8)	Free of disease	68 (56.2)
Unknown	4 (3.3)	Recurrence	5 (4.1)
		Unknown	27 (22.3)
Histologic diagnosis		Treatment	
DLBCL	62 (51.2)	CTx	67 (55.4)
BCL, other	22 (18.2)	CTx + surg	24 (19.8)
MZL	23 (19.0)	CTx + RTx	5 (4.1)
FL	3 (2.5)	CTx + stem cell	1 (0.8)
TCL	2 (1.7)	TPL	2 (1.7)
T-LBL	3 (2.5)	CTx + Surg + RTx	2 (1.7)
NHL	2 (1.7)	RTx Surg	9 (7.4)
Unknown	4 (3.2)	Steroid therapy	3 (2.5)
		Antibiotics	1 (0.8)
		No	3 (2.5)
		Unknown	4 (3.3)

BCL: B-cell lymphoma; MZL: Marginal zone lymphoma; FL: Follicular lymphoma; TCL: T cell lymphoma; T-LBL: T lymphoblastic lymphoma; NHL: Non-Hodgkin lymphoma; CTx: Chemotherapy; RTx: Radiotherapy; Surg: surgery; DLBCL: Diffuse large B-cell lymphoma.

perinephric space (observed in 25%-30% of cases). Diffuse renal enlargement without distortion of kidney shape or formation of any discrete masses was found in 6%-19%[102]. The unilateral masses and grossly nodular forms are relatively easily detected by US or CT; therefore, when necessary, the patients undergo open renal biopsy or nephrectomy. On the contrary, bilateral diffuse infiltrative lesions are difficult to detect by radiologic examination. Furthermore, due to various symptoms of lymphomatous infiltration, they are often mistaken for medical renal disease, and frequently percutaneous needle biopsy is performed for confirmative diagnosis. In our case, no definite mass-like lesion was identified on abdominal CT due to bilateral diffuse involvement of PRL, and a sono-guided percutaneous needle biopsy was performed to diagnose RPGN, clinically. In this case, based on the clinical and radiological background, renal involvement of malignant lymphoma could not be suspected.

Of all 121 patients, 96 had the following prognostic data: 68 (56.2%) patients survived, 21 (17.4%) died during/before/shortly after treatment, and 5 (4.1%) had relapsed disease. The mortality rate was especially high in patients younger than 10 years of age at 45.5%, and was 30.4% and 14.3% for those aged < 40 and ≥ 40 years, respectively. In addition, when the mortality rate was stratified by the location of the tumor, 41.7% (15/36) of patients demonstrated bilateral involvement whereas 7.2% (6/83) had unilateral lesions. Younger patients and those with bilateral PRL had a shorter survival time and more rapid disease progression compared to older individuals. Therefore, special procedures should be considered for the patients mentioned above, including a combination of surgery, CTx, or radiotherapy (RTx).

To date, CTx remains the main treatment for PRL. Among these 121 cases, 99 (81.8%) were treated with CTx (CTx alone or in combination), 67 (55.4%) with single CTx, and the remainder received various combination therapies including RTx, surgery, stem cell transplantation, and surgery. Regardless of the treatment, the overall mortality rate was 17.1%. When classified according to the treatment, the mortality rate of patients on single CTx was 21.2% (14/66), whereas, with combined therapy, a much lower mortality rate was observed [surgery with CTx, 12.5% (3/24); no treatment, 1 case; steroid treatment, 1 case; surgery, 1 case; surgery n with concurrent chemoradiation therapy and CTx with stem cell transplantation, no death]. Apart from single CTx, the number of patients on other therapies was small; therefore, this

Table 2 Comparison of clinical pathological factors according to age distribution, *n* (%)

Age (yr)		Bilateral	AKI	Prognosis-dead
≤ 10	11 (9.1)	9 (81.8)	0	5 (45.5)
11-20	5 (4.1)	3 (60.0)	0	0
21-30	3 (2.5)	3 (100)	1 (33.3)	0
31-40	4 (3.3)	2 (50.0)	0	2 (50.0)
41-50	17 (14.0)	3 (17.6)	3 (17.6)	2 (11.8)
51-60	24 (19.8)	6 (25.0)	2 (8.3)	2 (8.3)
61-70	22 (18.2)	6 (27.3)	1 (4.5)	5 (22.7)
71-80	26 (21.5)	4 (15.4)	4 (15.4)	5 (19.2)
≥ 80	9 (7.4)	0	0	0
	123	36 (29.3)	11 (8.9)	21 (17.4)

AKI: Acute kidney injury.

result should be interpreted with caution. To date, CTx remains the most-preferred treatment; however, a combination of CTx with RTx, surgery, and other methods should be considered in young patients or those with bilateral PRL.

Using the IPI, 4 independent patient risk groups with any combination of the following five clinical variables were identified, including age, LDH level, tumor stage, ECOG-PS, and extranodal sites of disease[103]. Moreover, the IPI has been widely used in clinical applications and is the standard practical prognostic tool for patients with DLBCL. In our case, the IPI was 3 (old age, 1; ECOG-PS, 1; Ann Arbor stages III–IV, 0; LDH > 1 × normal, 1; and > 1 extranodal site, 0). In addition to the IPI, because the correlation between cancer and inflammation has received attention in recent years, the prognostic significance of platelet/lymphocyte ratio (PLR) and neutrophil/lymphocyte ratio (NLR) in DLBCL has been reported in several studies[104]. For nearly all oncology records, PLR and NLR are calculated and routinely investigated from platelets, lymphocytes, and neutrophil counts. They are easily available measures in daily clinical practice, are inexpensive, and can provide useful prognostic information for the management of DLBCL[104]. Wang *et al*[104] reported markedly short OS and progression-free survival in patients with higher NLR and PLR compared with those with low NLR and PLR. Patients with a high NLR exhibit significantly low ECOG-PS, a high disease stage, and B symptoms, more extranodal sites of disease, and high IPI and LDH levels at diagnosis. Patients with a high PLR more frequently displayed significantly low ECOG-PS and B symptoms and a high LDH level at diagnosis. This revealed that patients with a PLR < 150 or NLR < 2.32 at diagnosis experienced better relapse-free survival and OS than those with a PLR ≥ 150 or NLR ≥ 2.32. In their study, NLR displayed no significant difference in multivariate analysis; however, univariate and multivariate analysis confirmed the predictive ability of PLR, indicating that PLR may be superior to NLR as a prognostic factor for DLBCL. In the present case, the pretreatment PLR was 186 (≥ 150) and NLR was 8.23 (≥ 2.32), implying a poor prognosis [platelets, 279000/μL; lymphocytes, 1500/μL; lymphocytes (%), 18.2%; and neutrophils (%), 67.2%].

This literature review had several limitations. Most importantly, all follow-up data were obtained from different article references and therefore have different follow-up periods. Thus, further studies are required regarding the prognosis of the disease.

Pathological diagnosis is important for the early diagnosis of PRL. When PRL shows bilateral and diffuse patterns, as in our case, predicting the diagnosis based on clinical and biochemical characteristics may be difficult, as PRL may clinically mimic refractory medical renal disease. Thus, a kidney biopsy would be required for confirmation of a radiologic or clinically suspected lymphoma. A biopsy is essential following a diagnosis of renal lymphoma, especially PRL, to institute early treatment, to achieve a cure in patients. Additionally, kidney biopsy helps confirm the exact subtype of lymphoma to apply appropriate treatment.

CONCLUSION

In conclusion, we report a 74-year-old patient with PRL presenting with AKI, and we conducted a literature review of 121 cases of PRL since 1989, to report their clinicopathologic findings. The literature search demonstrated that bilateral PRL is rare and has a poor prognosis. Among the 121 cases, only 36 were bilateral PRL, similar to our case. Additionally, AKI occurred in all bilateral PRL cases, and 71.4% of patients who died were diagnosed with bilateral PRL. Therefore, a need to discuss more active treatment for bilateral PRL is necessary. Moreover, bilateral renal involvement in malignant lymphoma can cause AKI. In particular, differentiating diffuse involvement (rather than the nodular form) clinically or radiologically from other kidney diseases that causes AKI is difficult; therefore, a kidney biopsy is essential for the diagnosis of renal lymphoma. Thus, clinicians should endeavor to make a preoperative diagnosis, to avoid unnecessary surgery.

Table 3 121 cases of primary renal lymphoma reported in the literature since 1989

No	Age (yr)	Sex	Side	Presentation	Diagnosis	Tx	Prognosis	Year	Ref.
1	53	F	B	Poor appetite, wt loss	NHL	CTx	Died at 3 d	1989	Paganelli *et al*[2]
2	58	M	B	Anorexia, wt loss, night sweat, malaise	B-cell NHL	CTx	NR	1992	Taneja *et al*[8]
3	51	F	U	Pain	SNCL	CTx	NR	1993	Van Gelder *et al*[9]
4	4	M	B	Fever, N/V	NR	CTx	Died after 16 mo	1994	Salem *et al*[10]
5	5	F	B	Fever, wt loss sweat	B-LBL	CTx	Died after 20 mo	1994	Salem *et al*[10]
6	49	F	B	Renal impairment, fever, wt loss, LBP	Centroblastic lymphoma	CTx	Died after 10 wk	1994	Salem *et al*[10]
7	52	F	U	Pain	DLBCL	CTx	Remission-relapse	1995	Arranz *et al*[11]
8	59	M	U	Pain	SNCL	CTx	Remission	1995	Arranz *et al*[11]
9	60	F	U	Pain	DLBCL	CTx	Remission	1995	Arranz *et al*[11]
10	60	M	U	Pain	DLBCL	CTx	Remission-relapse	1995	Arranz *et al*[11]
11	63	F	B	Pain	DLBCL	CTx	Died after 4.5 mo	1995	Arranz *et al*[11]
12	73	F	B	N/V, AKI	DLBCL	PDS	Died after 3 mo	1999	Okuno *et al*[12]
13	50	M	U	NR	MZL	No	Remission	1999	Chin *et al*[13]
14	76	F	U	Flank pain	MALT	Surg	NR	2000	Stallone *et a*[5]
15	45	M	U	Pain, fever	MZL	CTx	Remission-relapse	2001	Mhawech *et al*[15]
16	51	M	B	Flank pain	TCL	CTx	Died after 15 mo	2001	Jindal *et al*[16]
17	45	M	U	Incidental	BCL, Burkitt	Surg CTx	Remission	2002	O'Riordan *et al*[17]
18	62	M	B	Hematuria, acute urinary retention,	BCL, follicular	CTx	Died after 2 mo	2002	O'Riordan *et al*[17]
19	14	M	B	Headache, HTN, flank pain, Wt loss,	DLBCL	CTx	Alive at 2 wk	2002	Gellrich *et al*[18]
20	78	M	U	Hematuria	DLBCL	Surg, CTx	Alive at 2 yr	2002	Levendoglu *et al*[19]
21	77	M	U	Not determined	MZL	No	Remission	2002	Mansouri *et al*[20]
22	68	F	NR	NR	MALT	PDS	NR	2002	Mita *et al*[21]
23	72	F	NR	NR	MALT	PDS	NR	2002	Mita *et al*[21]
24	57	F	ND	Pain, fever	BCL	CTx	NR	2003	Stokes *et al*[22]
25	79	M	U	Pain, weakness, U/O↓	MZL	Surg, CTx	Alive at 2 mo	2003	O'Sullivan *et al*[23]
26	82	M	U	Pain	DLBCL	CTx	D's free after 1 yr	2003	Olusanya *et al*[24]
27	43	M	U	Lt. flank pain	MALT	Surg, CTx	Alive at 28 mo	2003	Pervez *et al*[25]
28	46	M	B	RF	DLBCL	Surg, CTx	Alive at 67 mo	2004	Tuzel *et al*[26]
29	17	M	U	Flank pain	DLBCL	CTx	Remission	2004	Cupisti *et al*[27]
30	70	F	U	Anorexia, malaise, fever	DLBCL	Surg, CTx	Alive at 8 mo	2004	Ozaltin *et al*[28]
31	65	F	U	Unknown	DLBCL	Surg, CTx, RTx	Alive at 18 mo	2005	Zomas *et al*[29]
32	68	M	B	Pain	DLBCL	No	Died after 10 d	2006	Ahmad *et al*[30]
33	70	M	U	LBP	DLBCL	CTx	D's free after 1 yr	2006	Kaya *et al*[31]

34	62	M	B	Oliguria, ARF	DLBCL	CTx	Died after 3rd CTx	2006	Bozas *et al*[32]
35	68	F	B	Flank pain, dysuria	LCL	Unknown	Died after 10 d	2006	Ahmad *et al*[30]
36	53	M	U	Not determined	MZL	CTx	Remission	2006	Guilpain *et al*[33]
37	72	M	U	Fever, chill	MZL	Surg	NR	2006	Guilpain *et al*[33]
38	83	F	U	Flank pain	MZL	CTx	D's free after 8 mo	2006	Guilpain *et al*[33]
39	83	F	U	Pain, fever	MZL	CTx, RTx	Remission	2006	Guilpain *et al*[33]
40	2	M	B	Abd. Distension, U/O↓	TCL	CTx	F/U loss	2006	Qiu *et al*[34]
41	71	F	U	wt loss, fever	BCL	Surg, CTx	Died after 4 mo	2006	Tefekli *et al*[1]
42	78	M	U	Pain	DLBCL	CTx	Remission	2006	Sharma *et al*[35]
43	5	M	B	HTN	TCL	CTx	Died after 2 mo	2007	Valarmathi *et al*[36]
44	77	ND	B	ARF	DLBCL	CTx, RTx	Remission	2007	Becker *et al*[37]
45	50	M	U	Abd pain	DLBCL	Surg, CTx	Alive at 1 mo	2007	Diskin *et al*[38]
46	62	M	U	Hematuria	DLBCL	Surg, CTx, IFN	Alive at 5 yr	2007	Diskin *et al*[38]
47	84	M	U	Unknown	BCL	Surg, CTx, IFN	Alive at 5 yr	2007	Diskin *et al*[38]
48	54	M	U	ND	MZL (MALT)	CTx	Remission	2007	Fang *et al*[39]
49	65	ND	U	ND	MZL (MALT)	Antibiotics	Remission	2007	Fang *et al*[39]
50	66	F	U	ND	MZL	Surg	Remission	2007	Fang *et al*[39]
51	75	M	U	ND	MZL (MALT)	CTx	Remission	2007	Fang *et al*[39]
52	77	F	ND	ND	MZL (MALT)	NR	Remission	2007	Fang *et al*[39]
53	83	M	U	ND	MZL (MALT)	CTx	Remission	2007	Fang *et al*[39]
54	49	M	U	Abd pain, fever	DLBCL	CTx	NR	2007	Bokhari *et al*[102]
55	21	M	B	Fever, wt loss, pain,	DLBCL	CTx	NR	2007	Garcia *et al*[40]
56	58	M	U	Headache, memory loss	DLBCL	Surg, CTx	Well on CTx	2007	Omer *et al*[41]
57	57	M	B	Dyspnea, RF, anemia	NR	CTx, Stem, cell TPL	NR (regression)	2008	Rajappa *et al*[42]
58	55	F	U	Fever	DLBCL	CTx	D's free after 1 mo	2008	James *et al*[43]
59	62	F	U	Fever, flank pain	DLBCL	Surg, CTx	Alive at 1 yr	2008	Ladha *et al*[4]
60	62	M	U	Pain	DLBCL	CTx, RTx	Remission-relapse	2008	Ladha *et al*[4]
61	3	M	B	Abd. distension, abd. pain, fever	BCL	CTx	Died after 5th CTx	2009	Kuo *et al*[44]
62	71	M	U	Unknown	DLBCL	CTx	Alive 2 yr	2009	Jindal *et al*[45]
63	74	M	U	Unknown	DLBCL	Surg, CTx	D's after 2nd CTx	2009	Jindal *et al*[45]
64	75	F	U	Unknown	DLBCL	Surg, CTx	Alive at 1 yr	2009	Jindal *et al*[45]
65	81	M	U	Hematuria	SBL	Surg, CTx	NR	2009	Jindal *et al*[45]
66	60	M	U	Dyspnea, fatigue	FL	Surg, CTx	NR	2009	Kose *et al*[46]
67	82	M	U	Mass	NR	RTx	NR	2009	Pinggera *et al*[47]
68	52	F	B	BP, headache, dysuria, hematuria, ARF, HTN	DLBCL	CTx	Alive 2 yr NR s	2009	Renaud *et al*[48]
69	46	M	U	Wt loss, fever, pain	DLBCL	CTx	Alive 7 mo	2009	Reuter *et al*[49]
70	47	M	U	Chronic graft dysfunction	BCL	Surg	Alive at 6.5 yr	2009	Reuter *et al*[49]

71	77	F	U	Anorexia, asthenia, malaise	DLBCL	Surg, CTx	Alive at 15 mo	2009	Reuter *et al*[49]
72	70	M	U	Hematuria	DLBCL	Surg	NR	2010	Vázquez *et al*[50]
73	66	F	U	Incidental	MALT	CTx	D's free after 3 yr	2010	Chatzipantelis *et al*[51]
74	52	F	U	Flank pain, hematuria	DLBCL	CTx	D's free after Tx	2010	Contreras-Ibáñez *et al*[52]
75	32	M	U	Heaviness, wt loss,	BCL	Surg, CTx	Died after 2 mo	2010	Cyriac *et al*[53]
76	12	M	U	Lumbar mass	DLBCL	CTx	D's free after 3 mo	2010	Gupta *et al*[54]
77	48	M	U	Abd. distension	DLBCL	CTx	D's free after Tx	2010	Kumar *et al*[55]
78	2.5	F	B	Abd. distension	T-LBL	CTx	D's free after Tx	2010	Moslemi *et al*[56]
79	75	F	B	ARF	DLBCL	CTx	NR	2010	Paladugu *et al*[57]
80	67	F	B	Epigastric pain, N/V	Large BCL	CTx	Alive at 4 wk	2011	Weng *et al*[58]
81	72	M	U	Flank pain, wt loss,	DLBCL	CTx	Alive at 15 mo	2011	Al-salam *et al*[59]
82	7	F	B	Fever, joint pain, anemia	NR	CTx	NR	2011	Al-salam *et al*[59]
83	23	M	B	ARF	TLBL	CTx	NR	2011	Dash *et al*[61]
84	73	M	U	Unknown	Large BCL	Surg	Unknown	2012	Kwakernaak *et al*[62]
85	82	F	U	Dizziness, palpitation, loss of consciousness	BCL	CTx	Unknown	2012	Brancato *et al*[63]
86	46	M	U	Wt loss, flank pain	DLBCL	Surg, CTx, RTx	Alive at 5 yr	2012	Hart *et al*[64]
87	77	F	U	Anorexia, malaise	DLBCL	Surg, CTx	Alive at 5.5 yr	2012	Hart *et al*[64]
88	61	M	U	ND	DLBCL	CTx	NR	2013	Vázquez-Alonso *et al*[65]
89	77	M	U	Hematuria	MZL	RTx	Alive at 3 yr	2013	Chen *et al*[66]
90	12	F	U	Hematuria	DLBCL	Surg, CTx	Alive at 3.2 yr	2013	Dedekam *et al*[67]
91	27	F	B	N/V, fever	DLBCL	CTx	Recurrence	2013	Hayakawa *et al*[68]
92	39	M	U	Pain	NHL	CTx	Remission	2013	Hu *et al*[69]
93	64	F	U	Wt loss	DLBCL	Surg, CTx	D's free	2013	Pahwa *et al*[3]
94	42	M	U	Abd pain, abd. mass	DLBCL	CTx	Alive at 28 mo	2014	Patel *et al*[70]
95	49	M	U	Pain, abd. mass	BCL	Surg	Alive at 1 yr	2014	Geetha *et al*[71]
96	82	F	U	HTN	MALT	Surg, CTx	D's free after 10 mo	2014	Naveen *et al*[72]
97	44	F	B	ARF	DLBCL	CTx	NR	2015	Vedovo *et al*[73]
98	8	ND	B	Fever, joint pain, anemia,	BCL	CTx	Alive at 1 yr	2015	Bahure *et al*[74]
99	56	M	U	Flank pain, fever	DLBCL	CTx, RTx	Remission	2015	Dhull *et al*[75]
100	8	M	U	Wt loss	DLBCL	CTx	Recurrence	2015	Hagihara *et al*[76]
101	70	F	U	Anorexia, malaise, fever	DLBCL	CTx	Alive at 8 mo	2016	Wang *et al*[77]
102	19	M	B	Hematuria	DLBCL	CTx	Alive after Tx	2016	Chen *et al*[78]
103	72	M	U	Incidental	FL	Surg, CTx	NR	2016	Erdoğmuş *et al*[79]
104	50	M	U	Dull aching pain	DLBCL	CTx	Died before 3rd CTx	2016	Jipp *et al*[80]
105	12	M	B	Fatigue	DLBCL	CTx	D's free after 5 yr	2017	Shetty *et al*[81]

106	10	M	U	Flank mass	DLBCL	CTx	Died at 14 mo	2017	Butani *et al*[82]
107	54	F	U	Headache	DLBCL	CTx	NR	2017	Coca *et al*[83]
108	38	M	B	Flank pain	DLBCL	NR	NR	2017	Rissman *et al*[84]
109	64	F	U	Abd. pain	DLBCL	CTx	NR	2017	Saddadi *et al*[85]
110	51	M	U	Flank pain	DLBCL	Surg	D's free after Tx	2018	Thawani *et al*[86]
111	37	M	B	Hematuria	DLBCL	CTx	Died prior to CTx	2018	Agochukwu *et al*[87]
112	4	M	B	Fatigue	DLBCL	CTx	D's free after 4 yr	2018	Mustafar *et al*[88]
113	64	F	U	Frequency	DLBCL	Surg, CTx	NR	2019	South *et al*[89]
114	78	M	U	Abd. pain	DLBCL	Surg	Died during Tx	2019	Cheng *et al*[90]
115	79	M	U	Myalgia	DLBCL	NR	NR	2019	Li *et al*[91]
116	50	M	U	Flank pain	DLBCL	CTx, RTx	CR	2022	Silverman *et al*[92]
117	56	F	U	Back pain, hematuria	DLBCL	CTx	D's free after 1 yr	2022	Nasrollahi *et al*[93]
118	59	M	U	Back pain	BCL	CTx	CR	2022	He *et al*[94]
119	53	ND	B	Back pain, ARF	MZL	CTx	Under Tx	2023	Abdi *et al*[95]
120	56	F	U	Incidental	HG BCL	CTx	D's free	2023	Benmoussa *et al*[96]
121	74	F	B	ARF	DLBCL	CTx	Died at 3rd CTx	Present case	

Tx: Treatment; ND: Not defined; B: Bilateral; U: Unilateral; wt: Weight; N/V: Nausea/Vomiting; LBP: Lower back pain; AKI: Acute kidney injury; NR: Not reported; HTN: Hypertension; U/O: Urine output; ARF: Acute renal failure; NHL: Non-Hodgkin lymphoma; SNCL: Small non-cleaved cell lymphoma; B-LBL: B-lymphoblastic lymphoma; DLBCL: Diffuse large B-cell lymphoma; MZL: Marginal zone lymphoma; MALT: Mucosa-associated lymphoid tissue lymphoma; TCL: T-cell lymphoma; LCL: Large cell lymphoma; SABL: Small B-cell lymphoma; FL: Follicular lymphoma; T-LBL: T-lymphoblastic lymphoma; HG BCL: High-grade BCL; CTx: Chemotherapy; PDS: Prednisolone; Surg: surgery; RTx: Radiotherapy; IFN: Interferon; TPL: Transplantation.

FOOTNOTES

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