INTERNAL MEDICINE

□ CASE REPORT □

Renal Intravascular Large B-cell Lymphoma: A Case Report and Review of the Literature

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Abstract

We herein report the case of a 52-year-old woman who consulted us because of a 2-month history of a fever, anorexia and weight loss. A physical examination was unremarkable. The blood count showed mild anemia and lymphopenia, and lactate dehydrogenase was elevated. Creatinine clearance was normal and proteinuria was undetectable. CT showed enlarged kidneys. A bone marrow biopsy was normal. PET-CT showed an intense uptake of ^{18}fluorodeoxyglucose in both kidneys. A kidney biopsy provided the diagnosis of intravascular large B-cell lymphoma (IVLBCL). Kidney-limited IVLBCL without an impairment in the renal function or proteinuria has not been described. We analyzed the 38 published cases of IVLBCL involving the kidney to describe the main features of this entity.

Key words: intravascular lymphoma, fever of unknown origin, PET-CT, kidney

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Introduction

As defined by Petersdorf, a fever of unknown origin (FUO) is "a fever of 38.3°C or more lasting for at least three weeks for which no cause can be identified after three days of investigation in hospital or after three or more outpatient visits" (1). Lymphomas remain an important cause of a FUO, primarily in forms with atypical clinical presentations because current imaging, pathology, microbiology and immunology resources typically provide a thorough work-up and rapid diagnosis.

Intravascular large B-cell lymphoma (IVLBCL) is a subtype of non-Hodgkin lymphoma characterized by preferential proliferation of malignant B cells within the lumina of small blood vessels (2). It is difficult to diagnose, however, most recent imaging techniques, such as ^{18}fluorodeoxyglucose positron emission tomography - computed tomography (FDG PET-CT) fusion images can render a simple and rapid diagnosis. Renal involvement is rarely reported in IVLBCL and, when it is present, it is usually associated with an impaired renal function. We herein describe a case of kidney-limited IVLBCL in a patient whose disease manifested as a FUO with a preserved renal function, highlighting the diagnostic usefulness of FDG PET-CT. Moreover, we performed a comprehensive review of published cases to characterize the renal involvement in IVLBCL.

Case Report

A previously healthy 52-year-old woman consulted us because of a 2-month history of a low-grade fever (38.5°C), weight loss, anorexia, fatigue and night sweats. Her recent history was unremarkable: no travel, dental procedure or unusual exposure to, for example, tick or animal bites. On examination, the patient's temperature was 38.2°C, blood pressure was 100/55 mmHg, pulse was 73 beats per minute, respiratory rate was 13 breaths per minute, and oxygen saturation 100% while breathing ambient air. The abdomen was soft and non-tender without palpable masses. No lymphadenopathy, pelvic tenderness, or skin rash was present. A neurological examination was normal. Laboratory analyses data (Table 1) showed aregenerative normocytic anemia and mild lymphopenia. There was no iron or vitamin deficiency. C-

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Table 1. Laboratory Data.

Variable	Reference range	On presentation
Hemoglobin (g/dL)	12 - 16	9.4
Hematocrit (%)	37 - 52	28
Mean corpuscular volume (μm^3)	80 - 100	84
White-cell count (per mm^3)	4 - 10	6,430
Differential count (%)		
Neutrophils	40 - 70	67
Lymphocytes	22 - 44	22
Monocytes	4 - 11	10
Eosinophils	0 - 8	0.5
Basophils	0 - 3	0.5
Platelet count (per mm^3)	150,000 - 400,000	256,000
Reticulocytes (per mm^3)	20,000 - 120,000	56,000
Sodium (mmol/L)	135 - 145	135
Potassium (mmol/L)	3.5 - 5	4.5
Chloride (mmol/L)	95 - 107	100
Glucose (mmol/L)	4 - 6.1	4.4
Urea nitrogen (mmol/L)	2.4 - 6.5	5.2
Creatinine (µmol/L)		72
Estimated glomerular filtration rate (mL/min/1.73 m^2)	> 60	77
Carbon dioxide (mmol/L)	23 - 29	27
Aspartate aminotransferase (U/L)	10 - 35	73
Alanine aminotransferase (U/L)	5 - 40	132
Lactate dehydrogenase (U/L)	5 - 248	533
Haptoglobin (g/L)	0.3 - 2	4.65
β2-microglobulin	1.2 - 2.5	4.56
C-reactive protein (mg/L)	< 5	137
Ferritin (µg/L)	11 - 306	528
Folate (ng/mL)	3.1 - 19.9	6.2
Vitamin B12 (ng/L)	180 - 914	783
TSH (µUI/mL)	0.34 - 5.6	0.37
Serum protein electrophoresis	Normal pattern	Inflammatory pattern
Albumin (g/L)	38 - 48	29
α 1 globulins (g/L)	1.8 - 3.2	5.6
α 2 globulins (g/L)	5 - 8.3	12.8
Gammaglobulins (g/L)	7.8 - 16	17 (polyclonal)
Urine		
Total protein (g/L)		0.08
Creatinine (mmol/L)		11.1
Protein/creatinin (mg/mmol)	< 15	7.2
Timed total protein (g/24 hours)	< 0.15	0.14
Blood	absence	absence

reactive protein and haptoglobin levels were elevated. The serum creatinine level was 72 µmol/L [estimated glomerular filtration rate: 77 mL/min/1.73 m^2 using the Modification of Diet in Renal Disease (MDRD) formula] and proteinuria was undetectable. Liver enzyme levels were thrice the normal range, and lactate dehydrogenase and β_2-microglobulin levels were twice the normal range. Blood-protein electrophoresis showed hypergammaglobulinemia with a polyclonal profile. Blood, sputum and urine cultures were negative, as were *Brucella*, *Rickettsia*, *Chlamydophila*, *Mycoplasma pneumoniae*, *Borrelia* and syphilis serologies. Testing for antinuclear antibodies, rheumatoid factor, and antineutrophil cytoplasmic antibodies was negative. A whole-body CT scan was unremarkable, except for bilateral kidney hypertrophy (Fig. 1A). A microscopic analysis of urine sediment was normal.

A bone marrow biopsy showed no signs of a lymphoproliferative or neoplastic process.

FDG PET-CT showed a diffusely intense FDG uptake in both kidneys with a standard uptake value of 5.5. No other abnormality was found (Fig. 2A).

A histological examination of a renal biopsy (Fig. 3) showed infiltration of the renal parenchyma by abnormally large lymphoid cells with prominent nucleoli, consistent with lymphoma. These cells proliferated in the lumina of glomerular, peritubular and interstitial capillaries. Immunophenotyping showed the expression of CD20, BCL6, MUM1, CD5, and BCL2, while CD10 and CD30 were negative. Epstein-Barr virus was also negative. The Ki67 index was 95%, reflecting a high proliferative activity. The MYC expression was positive. According to these findings, Stage IV IVLBCL was diagnosed.

The patient received 8 cycles of anthracycline-containing chemotherapy associated with rituximab (R-CHOP) and complete remission was achieved, as assessed by kidney CT (Fig. 1B) and FDG PET-CT (Fig. 2B). Thirty months after

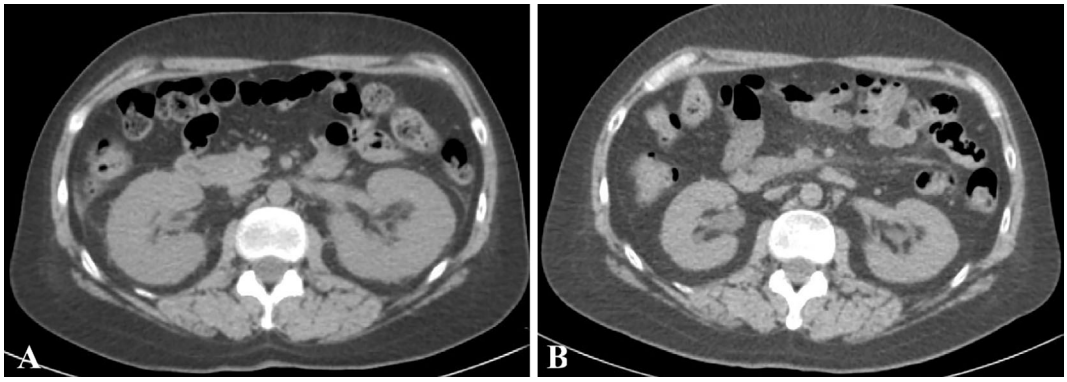

Figure 1. **Kidney CT before (A) and after (B) 8 cycles of chemotherapy. CT at diagnosis showed marked bilateral kidney hypertrophy (A) which subsided after 8 cycles of anthracycline-containing chemotherapy associated with rituximab (B).**

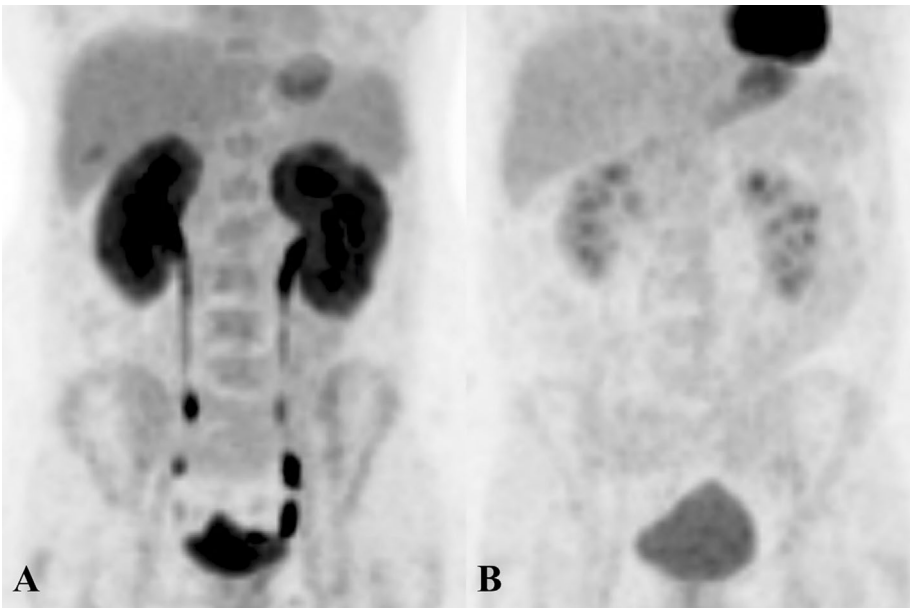

Figure 2. **Maximum intensity projection PET images before (A) and after (B) 8 cycles of chemotherapy. PET at diagnosis showed intense tracer uptake in both kidneys with a standard uptake value of 5.5 (A). After 8 cycles of treatment, complete remission was achieved (B). PET: positron emission tomography**

completing chemotherapy, the patient remains in remission.

Discussion

Renal involvement commonly occurs in lymphoma according to the largest case series of autopsies, which found lymphocytic infiltration in up to 34% of patients (3). Fewer patients diagnosed with lymphoma (3-8%) are found to have renal abnormalities on a CT scan during disease staging (4). A wide spectrum of non-Hodgkin lymphomas can be associated with renal involvement, and the pattern of kidney injury is diverse (e.g., glomerulonephritis, minimal change disease, intravascular lymphomatous infiltration, interstitial infiltrate, and intracapillary immunoglobulin deposits) (5). Proteinuria is present at diagnosis in nearly all cases and nephrotic syndrome is frequent. The renal function is often impaired. A retrospective study which analyzed 55 cases of large diffuse B-cell lymphomas involving the kidneys found that 36% had central nervous system relapse after first-line chemotherapy. The 5-year overall survival was 29% in this cohort (6).

According to the 2008 World Health Organization classification, IVLBCL is an extranodal B-cell lymphoma (BCL) characterized by the location of malignant cells within the lumina of small- to medium-sized blood vessels. It is a rare subset of large diffuse BCL without marked lymphadenopathy, which renders its diagnosis elusive (2). IVLBCL typically occurs in patients over 60 years of age. Clinical manifestations are mostly related to the organs involved. Two clinical variants have been described: a Western phenotype, characterized by a high frequency of central nervous system and skin involvement; and an Asian phenotype, frequently comprising hemophagocytic syndrome and bone marrow in-

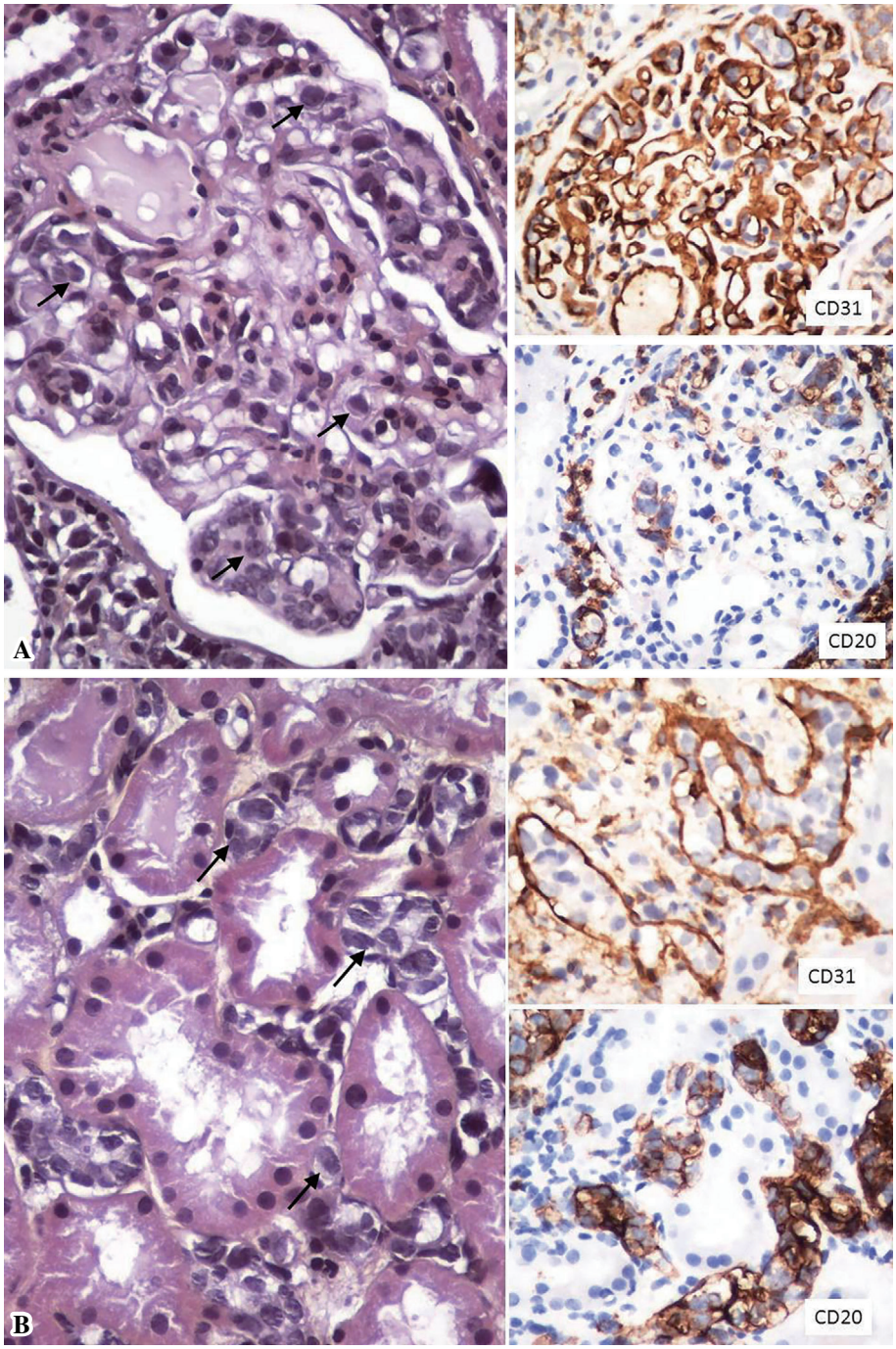

Figure 3. **Renal biopsy findings. A: Glomerular capillary lumens containing lymphomatous cells (arrows). Hematoxylin and Eosin (H&E) staining (left). CD31 staining marking the endothelium (upper right-hand corner). CD20 staining marking lymphomatous cells (lower right-hand corner). Magnification 40×. B: Lymphomatous cells occluding the peritubular capillaries (arrows). H&E staining (left). CD31 staining (upper right-hand corner). CD20 staining (lower right-hand corner). Magnification 60×.**

volvement (7). The definitive diagnosis requires a biopsy of the affected organ(s).

IVLBCL renal involvement is rarely described. Since the first description by Jothy et al. in 1981 (8), only 39 cases have been published (the present case included) (8-41) (Table 2). Among them, 52% were limited to the kidney at the initial diagnosis. A fever was a prominent feature in 73% of the patients. Renal failure was present in 66%, proteinuria in 92%, and nephrotic syndrome in one-third of the patient. All previous cases had an impaired renal function or proteinuria at diagnosis, unlike our patient. The main histological finding of nephrotic-range proteinuria was minimal change disease, which is characterized by the absence of light microscopy abnormalities and glomerular immune deposits and a diffuse loss of podocyte foot processes on electron microscopy. Lymphomatous cells proliferated in the glomerular

Table 2. **Reported Cases of Kidney-proven IVLBCL.**

Reference	Age (years) / Sex	Renal failure	Proteinuria	Enlarged kidneys	Fever	Extra-renal involvement	Lymphoma cells location in kidney	Outcome
8	NR/NR	+	+	NR	NR	NR	G	NR
8	NR/NR	+	+	NR	NR	NR	G	NR
9	62/F	−	+ (N)	+	+	+	G / I	Dead 1 month after diagnosis
10	60/F	−	+ (N)	NR	+	−	G	Alive 8 months after diagnosis
11	52/M	+	+ (N)	NR	+	−	G	Alive 6 months after diagnosis
12	60/M	+	+	NR	NR	−	G / P	NR
13	61/M	+	+ (N)	−	+	+	G	Dead 1 month after diagnosis
14	35/F	NR	NR	−	+	+	G	NR
15	38/F	+	+	+	+	+	G / P	Alive 3 months after diagnosis
16	71/M	NR	+	NR	NR	NR	G	NR
17	64/F	+	+	−	+	+	G / P / I	Post-mortem diagnosis
17	65/M	+	+	+	+	+	G	Post-mortem diagnosis
17	82/M	+	−	−	+	+	G / P / I	Post-mortem diagnosis
18	85/F	+	+ (N)	−	−	−	G / I	Alive 3 months after diagnosis
19	49/F	NR	+	NR	NR	−	G	NR
20	69/M	+	+	−	+	−	G / P	Dead 1 month after diagnosis
20	63/M	+	+	−	−	+	G	Dead 21 months after diagnosis
21	53/F	−	+ (N)	NR	+	+	G	Dead 6 months after diagnosis
22	58/M	−	+ (N)	−	+	+	G / I	Alive 1.5 months after diagnosis
23	72/M	−	+	−	−	−	G	Alive 3 months after diagnosis
24	56/F	−	+ (N)	−	+	+	G / I	Dead 6 months after diagnosis
25	48/M	+	+	−	−	−	G	Alive 24 months after diagnosis
26	56/M	−	+	−	+	+	NR	Alive 8 months after diagnosis
27	67/M	+	+	−	+	+	G	Dead 9 months after diagnosis
28	35/F	+	+	+	+	−	P	Alive 6 months after diagnosis
29	NR/F	NR	NR	+	+	+	NR	NR
30	74/F	+	+	−	+	−	G / I	NR
31	52/M	+	−	+	−	−	G / I	Alive 26 months after diagnosis
32	40/F	−	+	−	−	−	G	Alive 24 months after diagnosis
33	76/M	+	+	+	+	+	NR	NR
34	47/M	+	+	−	+	+	G / P	Alive 6 months after diagnosis
35	41/F	−	+ (N)	+	+	+	I / P	Alive 9 months after diagnosis
36	72/F	+	NR	+	+	−	P	Alive 4 months after diagnosis
37	78/F	−	+ (N)	−	+	+	G / P	Dead 3 months after diagnosis
38	55/M	−	+	NR	−	−	G	NR
39	77/F	+	+	−	−	−	G	Alive 48 months after diagnosis
40	65/F	+	+	−	−	−	G	Alive 109 months after diagnosis
41	45/M	+	+	−	NR	−	P	NR
Present case	**52/F**	**−**	**−**	**+**	**+**	**−**	**G / P / I**	**Alive 30 months after diagnosis**

+ : present, − : absent, M: male, F: female, N: nephrotic, NR: not reported, G: glomerular, P: peritubular, I: interstitial

IVLBCL: intravascular large B-cell lymphoma

capillaries in 89% of the cases, with peritubular and interstitial vessels affected in 30.5% and 27.8% of the cases, respectively. Clinical and biological manifestations were not predictive of parenchymal structure involvement.

Routine imaging studies, e.g., renal ultrasound and CT, visualized marked bilateral nephromegaly in 33.3% of the cases.

FDG PET-CT is a powerful tool for the diagnosis of lymphoma, however, physiologic FDG excretion in the kidneys makes the interpretation of the tracer uptake in this organ difficult. Single or multiple masses, renal invasion from the retroperitoneum, and diffuse renal infiltration constitute classical patterns of involvement (42).

Information on the ability of FDG PET-CT to diagnose renal IVLBCL is scarce, as it has been used for only 10 reported cases, including ours, with four showing a diffusely increased uptake of radiolabeled glucose in both kidneys. Miura and Tsudo obtained PET-CT images of four consecutive patients diagnosed with IVLBCL, which showed a bilateral FDG accumulation in the renal cortex; however, it is unknown if kidney involvement was biopsy-proven (43).

The differential diagnosis of isolated diffuse renal hypermetabolism comprises a short list of malignant and inflammatory/autoimmune diseases. Renal metastases of solid neoplasms can present as diffusely infiltrating hypodense lesions associated with nephromegaly that are intensely FDG-avid (42). Renal lymphoma, mainly in the context of widespread high-grade disease, and leukemic involvement of the kidney can also be observed. ANCA-associated vasculitis, IgG4-related disease and sarcoidosis have been described in case reports as the etiology of this imaging pattern.

Our patient consulted us because of a FUO associated with elevated lactate dehydrogenase and β_2-microglobulin levels, without any evidence of lymphadenopathy or splenomegaly on a CT scan. In this setting, PET-CT proved to be a highly valuable tool to guide a diagnostic biopsy by showing abnormally high metabolic activity in the affected organ.

Several case reports have illustrated the added input of PET-CT to diagnose IVLBCL in the early stage (44, 45). However, its low sensitivity makes it unfit for the evaluation of organ involvement, especially for central nervous system-localized disease (25% of all IVLBCL) (46, 47).

The outcome of IVLBCL is hampered by difficulty in obtaining a timely diagnosis. In 1994, DiGiuseppe et al. reported that the median survival without treatment was 3 months for six patients diagnosed with IVLBCL (48). In 2008, Shimada et al. described the largest retrospective series of IVLBCL patients treated with chemotherapy and rituximab, and demonstrated an overall 2-year survival of 66% (49).

Of the 39 cases of kidney biopsy-proven IVLBCL reported in the literature (ours included), 28 provided information on the patient's outcome. The mean follow-up for those patients was 6 months (ranging, 0 to 109 months). At 6 months, 50% of those patients were alive, 32% had died, and the follow-up was too short in the remaining 18%.

This report clearly characterized the classical presentation of a rare disease. A FUO associated with elevated lactate dehydrogenase and β_2-microglobulin levels may evoke IVLBCL. Renal involvement in IVLBCL should be suspected when renal failure, proteinuria and/or nephromegaly are present. In the absence of these signs, PET-CT fusion images can strongly contribute to an early diagnosis and prompt treatment.

The authors state that they have no Conflict of Interest (COI).

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