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CASE REPORT

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Polycythemia secondary to bilaterally enlarged kidneys in T-Cell acute lymphoblastic leukemia: a case report and literature review

Koji Yoshimoto[1], Yujiro Maeoka[1*], Shiori Kubota[1], Ren Chishaki[2], Akira Takahashi[1], Yosuke Osaki[1], Tatsuji Mino[2], Tatsuo Ichinohe[2] and Takao Masaki[1*]

Abstract

Background Polycythemia is a rare disease that can cause hypertension. Secondary polycythemia with increased production of erythropoietin (EPO) is associated with several kidney diseases, including hydronephrosis and cystic disease. However, there have been no reports of a case presenting with polycythemia secondary to bilateral nephromegaly caused by renal infiltration of T-cell acute lymphoblastic leukemia (T-ALL).

Case presentation A 32-year-old Japanese man presented with marked hypertension (215/150 mmHg) with renal insufficiency (creatinine 3.7 mg/dL), proteinuria, hematuria, bilateral nephromegaly, polycythemia (hemoglobin 20.2 g/dL), and increased serum EPO (38.7 mIU/mL, range 4.2–23.7 mIU/mL). Based on renal and bone marrow biopsy findings, he was diagnosed with T-ALL and bilaterally enlarged kidneys caused by renal infiltration of leukemic cells. There was no evidence of endocrine hypertension or fluid retention. Remission induction chemotherapy led to a decrease in kidney size, hemoglobin levels, and serum EPO levels, and allowed dose reductions of most hypertensive drugs, suggesting that hypertension was secondary to polycythemia. The patient's renal function gradually improved and hemodialysis was discontinued after 1 month of chemotherapy.

Conclusions We report a case of marked hypertension and secondary polycythemia induced by severe renal infiltration of T-ALL at diagnosis, which were synchronically improved with induction chemotherapy. This case history suggests the importance of considering lymphoproliferative diseases in the differential diagnosis of secondary polycythemia, leading to severe hypertension.

Keywords T-cell acute lymphoblastic leukemia/Lymphoblastic lymphoma (T-ALL/LBL), Acute kidney injury, Enlarged kidneys, Polycythemia, Hypertension

*Correspondence:
Yujiro Maeoka
ymaeoka@hiroshima-u.ac.jp
Takao Masaki
masakit@hiroshima-u.ac.jp
[1]Department of Nephrology, Hiroshima University Hospital, 1-2-3 Kasumi, Minami-ku, Hiroshima 734-8551, Japan
[2]Department of Hematology and Oncology, Research Institute for Radiation Biology and Medicine, Hiroshima University, 1-2-3 Kasumi, Minami-ku, Hiroshima 734-8553, Japan

Background

T-cell acute lymphoblastic leukemia (T-ALL) and lymphoblastic lymphoma (LBL) are recognized to be the same disease, a malignant neoplasm of immature lymphoblasts, and are practically distinguished by an arbitrary cut-off of 25% lymphoblasts in the bone marrow, as defined in the World Health Organization classification of hematolymphoid tumors [1]. T-ALL, which accounts for 15% of pediatric and 25% of adult ALL cases [2], preferentially localizes in the bone marrow, central nervous system, and lymphoid organs, particularly in the mediastinal lymph nodes. Renal impairment has diverse causes, including mechanical compression of the kidney and ureter by extrarenal tumors, treatment-associated tumor lysis syndrome, and sepsis [3, 4]. Although rare, leukemic or lymphoma cells can directly infiltrate the interstitium and glomeruli of the kidney, leading to acute kidney injury (AKI) and bilateral kidney enlargement [5, 6]. Because renal involvement usually occurs late in the course of T-ALL, only four cases have been reported of adult T-ALL presenting at diagnosis with enlarged kidneys due to cell infiltration [7–10].

Polycythemia, which is known to be associated with increased prevalence of hypertension (Gaisböck syndrome) [11], is classified into primary and secondary based on its etiology. Primary polycythemia results from gene mutations that affect red blood cell (RBC) progenitor cells and typically presents with subnormal erythropoietin (EPO) levels. In contrast, secondary polycythemia is caused by increased production of EPO by interstitial fibroblasts in the kidney and other etiologies [12]. The primary stimulus for EPO production is reduced oxygen delivery to the renal tubulointerstitial space, typically as a result of systemic hypoxia or anemia. Several kidney disorders, such as hydronephrosis, renal cysts, and renal malignancies, are associated with secondary polycythemia, likely due to renal ischemia [13–15]. Although a limited number of cases have been reported of polycythemia secondary to bilateral renal enlargement caused by infiltration of lymphoma and leukemia [16–18], none have been due to T-ALL/LBL.

We report a rare case of T-ALL in which secondary polycythemia and reduced renal function with bilaterally enlarged kidneys were present at the time of diagnosis, leading to severe hypertension. EPO levels and kidney size were concurrently improved by induction therapy, suggesting polycythemia secondary to bilaterally enlarged kidneys caused by T-ALL.

Case presentation

A 32-year-old Japanese man with no medical history visited a local doctor because his blood pressure (BP) was alarmingly high (215/150 mmHg) at the time of a health check-up, and he was aware of a loss of appetite and headache lasting more than 2 months. Given the diagnosis of severe hypertension, the patient was administered doxazosin, an α-adrenergic antagonist, and he was referred to our hospital for further examination and treatment. He was a current smoker of up to 10 cigarettes per day.

At the referral visit, the patient's BP was still severely high at 170/102 mmHg, and his pulse rate was 74 beats per minute. Physical examination revealed no edema or signs of meningeal irritation. Laboratory data indicated polycythemia (RBC $703 \times 10^4/\mu L$, hemoglobin [Hb] 20.2 g/dL) and renal failure with proteinuria and hematuria (creatinine [Cr] 3.70 mg/dL, urine protein-to-Cr ratio 0.28 g/gCr, RBC 10–19/high-power field) (Table 1). His serum cortisol, plasma renin activity, plasma aldosterone concentration, and catecholamine levels were within normal ranges, and no autoantibodies associated with glomerulonephritis were detected (Table 1). Abdominal ultrasonography showed bilaterally enlarged kidneys (right: 14.2 cm × 8.4 cm, left: 14.0 cm× 8.5 cm) and no hydronephrosis. Although severe hypertension caused by polycythemia and renal failure was suspected, the precise cause of the polycythemia, renal failure, and renal enlargement was unclear.

To reduce BP, 40 mg/ day of nifedipine controlled-release (CR), a long-acting calcium channel blocker, and 20 mg/day of olmesartan, an angiotensin receptor blocker, were added. The patient's BP improved significantly to 135/95 mmHg on day 8 (7 days after the referral visit), and a renal biopsy was performed to determine the cause of renal failure and enlargement. Light microscopy revealed virtually normal structures in seven obtained glomeruli, but diffuse and extensive interstitial infiltration with medium- to large-sized atypical lymphoid cells. Immunohistochemistry showed the cells to be positive for cytoplasmic CD3, terminal deoxynucleotidyl transferase, and Ki-67, but negative for CD20 (Fig. 1). Immunofluorescence staining and electron microscopy revealed no evidence of immune deposits. Bone marrow biopsy revealed > 25% blasts, and myeloperoxidase staining was negative (Fig. 2). Based on these findings, the patient was diagnosed with T-ALL and bilaterally enlarged kidneys caused by renal infiltration of leukemic cells. Computed tomography (CT) showed moderate right pleural effusion; anterior mediastinum mass; slightly enlarged lymph nodes in the neck, mediastinal, right hilar, right axillary, and abdominal para-aortic areas; and enlarged kidneys (Fig. 3a). Consistent with these findings, [^{18}F]-fluorodeoxyglucose (^{18}FDG)-positron emission tomography (PET)/CT demonstrated an increase in metabolic uptake in these organs, with the largest increase observed in the kidneys (Fig. 3b). Serum EPO levels were high at 38.7 mIU/mL (normal range: 4.2–23.7 mIU/mL) despite high Hb levels; thus, he was diagnosed with secondary

Table 1 Summary of the patient's laboratory results

Parameter	Value	(Normal range)
(Urine)		
Urine specific gravity	1.010	
pH	5.5	
Urine protein/creatinine ratio (g/gCr)	0.28	(< 0.15)
Red blood cells (/HPF)	10–19	(< 5)
β2MG (mg/L)	5.69	(0.03–0.37)
NAG (IU/L)	17.1	(0–11.5)
(Blood)		
White blood cells (/μL)	8900	(3040–8540)
Red blood cells (10^4 /L)	703	(378–499)
Hemoglobin (g/dL)	20.2	(10.8–14.9)
Hematocrit (%)	61.6	(35.6–45.4)
Platelets (10^4 /L)	18.8	(15–36)
AST (U/L)	24	(13–33)
ALT (U/L)	19	(8–42)
LD (U/L)	670	(124–222)
Total protein (g/dL)	8.4	(6.7–8.3)
Serum albumin (g/dL)	4.5	(4–5)
Blood urea nitrogen (mg/dL)	24.5	(8–20)
Creatinine (mg/dL)	3.7	(0.4–0.7)
eGFR (mL/min/1.73m^2)	17	(> 90)
Sodium (mmol/L)	136	(138–146)
Potassium (mmol/L)	4.4	(3.6–4.9)
Chloride (mmol/L)	96	(99–109)
Calcium (mg/dL)	10.3	(8.6–10.4)
Phosphorus (mg/dL)	4.5	(2.5–4.7)
Uric acid (mg/dL)	10	(2.3–7)
Plasma glucose (mg/dL)	102	(70–109)
Hemoglobin A1c (NGSP) (%)	5.5	(4.6–6.2)
C-reactive protein (mg/dL)	0.14	(< 0.2)
PT-INR	0.98	
APTT (sec)	34.2	(24–34)
Fib (mg/dL)	272.1	(200–400)
Immunoglobulin G (mg/dL)	2021	(870–1700)
Immunoglobulin A (mg/dL)	251	(110–410)
Immunoglobulin M (mg/dL)	81	(46–260)
CH50 (U/mL)	45	(30–46)
C3 (mg/dL)	95	(86–160)
C4 (mg/dL)	32	(17–45)
Anti-nuclear antibody	Negative	Negative
Anti-neutrophilic cytoplasmic antibody	Negative	Negative
Anti-glomerular basement membrane antibody	Negative	Negative
sIL-2R (U/mL)	1426	(121–613)
HBs-Ag	Negative	Negative
HCV-Ab	Negative	Negative

Cr: creatinine, HPF: high-power field, β2MG: β2-microglobulin, NAG: N-acetyl-β-D-glucosaminidase, AST: aspartate transaminase, ALT: alanine transaminase, LD: lactase dehydrogenase, eGFR: estimated glomerular filtration rate, NGSP: National Glycohemoglobin Standardization Program, PT-INR: prothrombin time international normalized ratio, APTT: activated partial thromboplastin time, Fib: fibrinogen, CH50: 50% hemolytic complement activity, C3: Complement Component 3, C4: Complement Component 4, sIL-2R: soluble interleukin-2 receptor, HBs-Ag: hepatitis B virus antigen, HCV-Ab: hepatitis C virus antibody

polycythemia. No adrenal or liver tumors or hypoxic lung disease were detected, and oxygen saturation was 95% in room air; therefore, we considered that secondary polycythemia may have been caused by the bilaterally enlarged, infiltrated kidneys.

On day 20, the patient was treated with prednisolone to reduce the tumor mass. After 1 week, remission induction chemotherapy (vincristine, daunorubicin, dexamethasone, L-asparaginase, methotrexate, cytarabine, prednisolone) was administered according to the Japan Adult Leukemia Study Group (JALSG) ALL202-O protocol. On day 21, hemodialysis was initiated due to severe tumor lysis syndrome. After 1 month of chemotherapy, the patient's renal function improved, and he was taken off hemodialysis. His kidneys were smaller on day 24 (right: 11.9 cm × 7.2 cm, left: 13.0 cm× 7.5 cm) and their size had normalized on day 60 (right: 9.4 cm × 5.5 cm, left: 10.8 cm× 5.0 cm). His Hb levels normalized on day 23 but gradually decreased from day 45 to day 62, at which time the Hb level was 7.3 g/dL. EPO level was low at 12.0 mIU/mL on day 59; therefore, 30 μg of darbepoetin alfa was started for the treatment of renal anemia (Figs. 3 and 4). Consistent with the improvements of the bilaterally enlarged kidneys and polycythemia, his BP improved, and doxazosin and olmesartan were discontinued on day 22. His systolic BP improved to 110–120 mmHg at a dose of only 20 mg/day of nifedipine CR. Future plans for the patient include a hematopoietic stem cell transplantation after hyper-CVAD therapy (cyclophosphamide, vincristine, doxorubicin, dexamethasone) and MA therapy (methotrexate, cytarabine).

Discussion and conclusions

We report a rare case of AKI associated with bilateral nephromegaly secondary to T-ALL infiltration of the bilateral tubulointerstitium at the time of diagnosis. The patient's kidney dysfunction and nephromegaly were improved with chemotherapy. Renal infiltration of leukemia or lymphoma is usually observed in the late stages of the disease [19], and autopsy studies show that it is present in 60–90% of patients. Only a few cases have been reported in which AKI caused by lymphomatous infiltration was present at diagnosis [20, 21]. Although leukemic or lymphoma cells can directly infiltrate the interstitium and glomeruli of the kidney, the incidence of acute kidney failure and bilaterally enlarged kidneys is higher for the interstitial type (87% and 91%, respectively) compared with the intraglomerular type (45% and 10%, respectively) [6]. Interstitial infiltration is thought to cause AKI by obstruction of the urinary tubules by leukemic or lymphoma cells, resulting in glomerular collapse [22]. This hypothesis is supported by our previous case report of diffuse large B-cell lymphoma with bilateral nephromegaly and AKI, in which chemotherapy

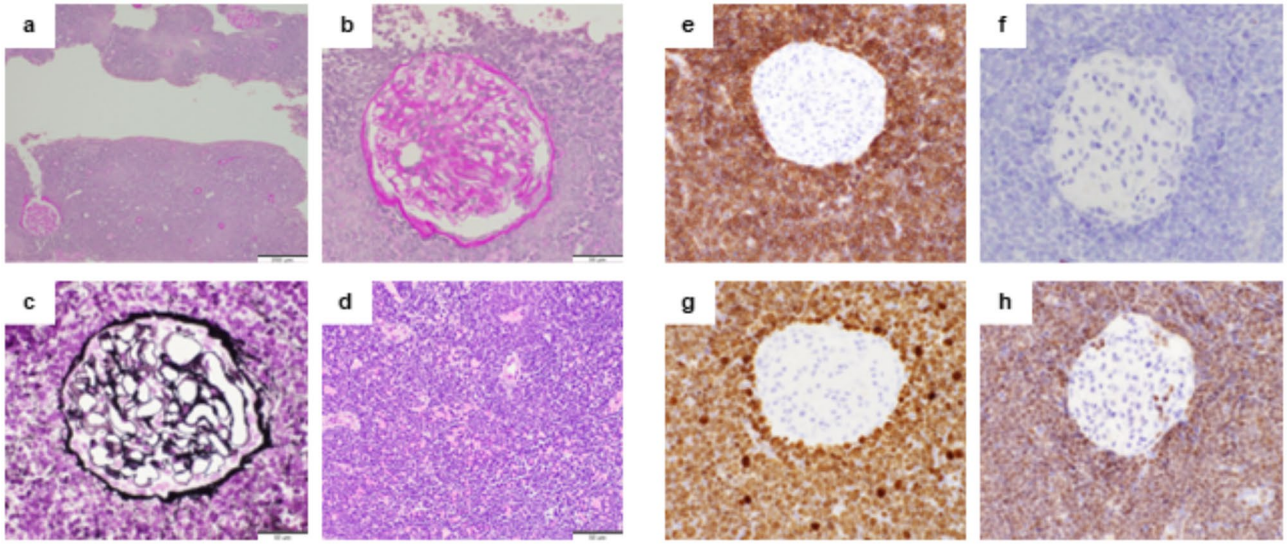

Fig. 1 Light microscopic images of the renal biopsy
(**a**–**d**) Normal glomeruli but diffuse and extensive interstitial infiltration with medium- to large-sized atypical lymphoid cells were observed in periodic acid–Schiff staining (**a**: × 100, **b**: × 400), periodic acid–methenamine–silver staining (**c**: × 400), and hematoxylin and eosin staining (**d**: × 400). (**e**–**h**) Immunostaining showed that lymphocytic cells were positive for CD3 (**e**), Ki-67 (**g**), and terminal deoxynucleotidyl transferase (**h**), but negative for CD20 (**f**)

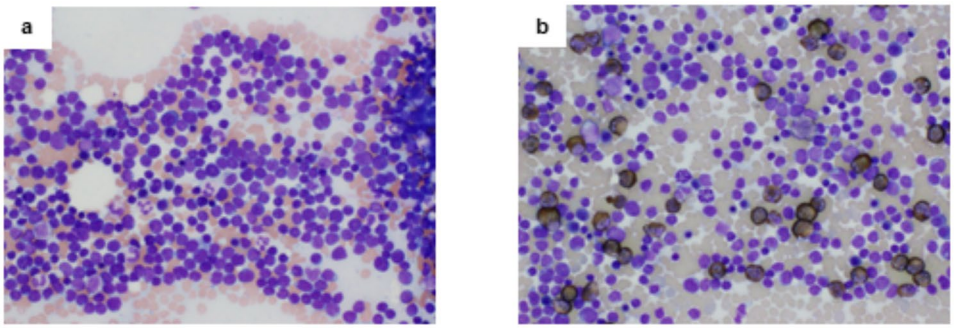

Fig. 2 Images of hematopoietic cells in the bone marrow biopsy
(**a**) May–Giemsa staining showed > 25% blasts. (**b**) Myeloperoxidase staining was negative for blasts

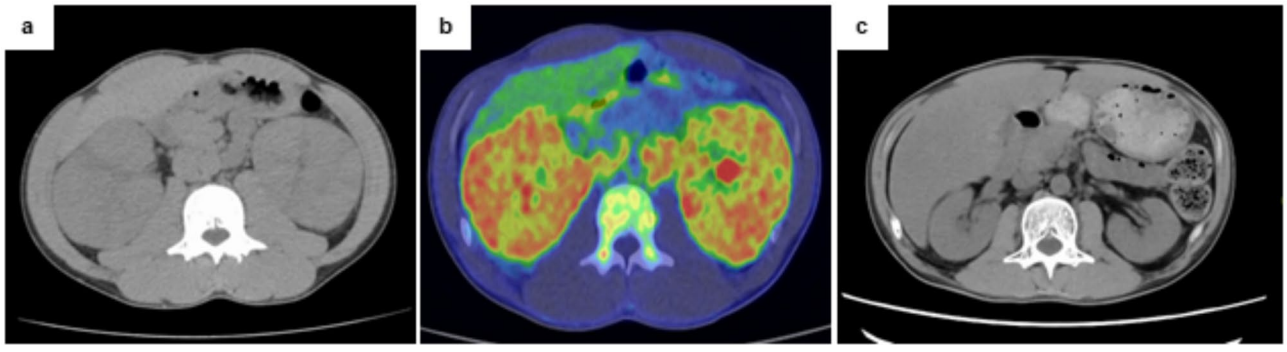

Fig. 3 Computed tomography (CT) and [^{18}F]-fluorodeoxyglucose (^{18}FDG)-positron emission tomography (PET)/CT images of the abdomen
(**a, b**) CT and PET/CT revealed bilateral nephromegaly and strong uptake of ^{18}FDG in the kidneys at diagnosis. (**c**) Bilateral nephromegaly was normalized after induction chemotherapy

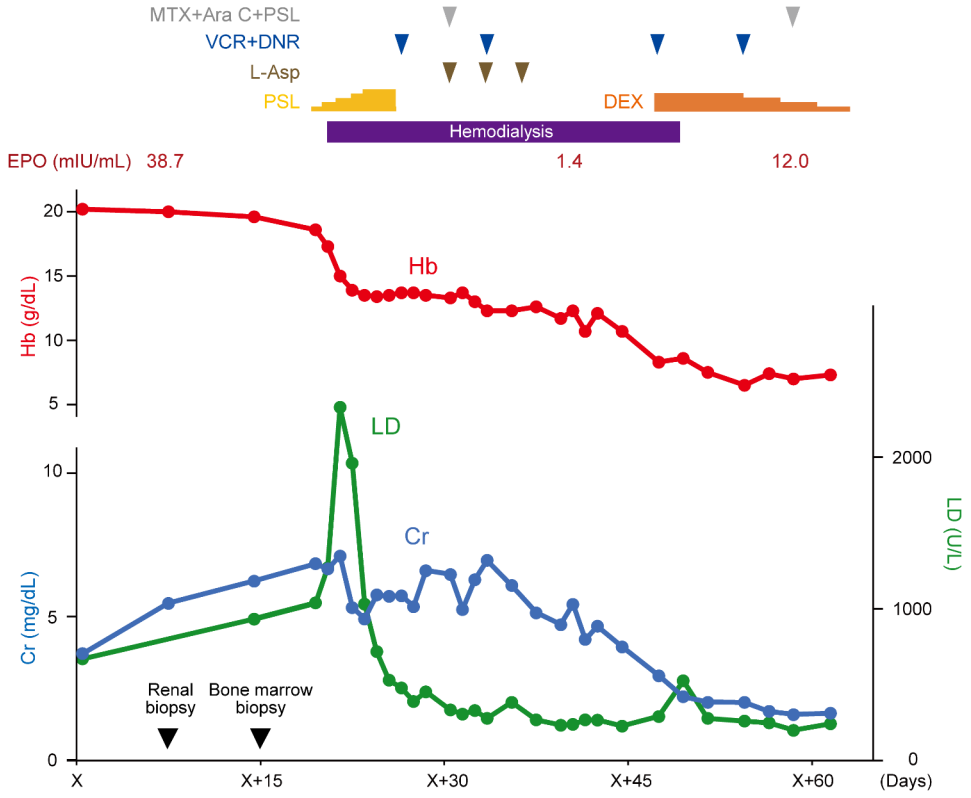

Fig. 4 Clinical progression of the patient
Changes in serum levels of Cr (blue line), LD (green line), and Hb (red line). X indicates the day of the referral visit. Cr: creatinine, LD: lactase dehydrogenase, Hb: hemoglobin, EPO: serum erythropoietin, PSL: prednisolone, VCR: vincristine, DNR: daunorubicin, DEX: dexamethasone, L-Asp: L-asparaginase, MTX: methotrexate, Ara-C: cytarabine

Table 2 Adult cases of T-LBL/ALL presenting with bilateral nephromegaly at diagnosis

Author [ref]	Year	Age	Sex	Diagnosis	Cr (mg/dL)	Hb (g/dL)	Renal Biopsy	Kidney size (cm)	BP (mmHg)
Cho HS [9]	2008	18	M	T-ALL	1.1	8.9	-	13.1, 13.4	125/82
Kwakernaak AJ [8]	2011	23	M	T-LBL	6.2	7.9	+	15, 14.6	150/80
Bhuiyan MMU [7]	2020	27	M	T-LBL/ALL	1.8	N/A	-	N/A	130/80
Tamura Y [10]	2021	54	M	T-ALL	1.59	8.1	+	12, 13	120/74
Present case	2024	32	M	T-ALL	3.7	**20.2**	+	14.2, 14	**212/150**

ref: References, Cr: creatinine, Hb: Hemoglobin, BP: Blood Pressure, T-ALL: T-cell acute lymphoblastic leukemia, T-LBL: T-cell lymphoblastic lymphoma, N/A: Not Available

improved nephromegaly and kidney dysfunction and eliminated diffuse interstitial infiltration with lymphocytic cells [23]. To date, only four adult cases of T-ALL have been reported in which bilateral nephromegaly due to leukemia infiltration was present at diagnosis, and all four patients developed AKI [7–10]. Similar to the present case, both nephromegaly and renal function were improved by chemotherapy in those four cases [7–10]. Taken together, these findings suggest that nephromegaly caused by leukemic interstitial infiltration, rarely observed at the time of diagnosis in patients with T-ALL, often develops into AKI, but both renal function and kidney size can be improved by induction therapy.

To the best of our knowledge, this is the first reported case of secondary polycythemia in a patient with T-ALL/LBL (Table 2). The patient had a high EPO level of 38.7 mIU/mL at diagnosis, despite polycythemia with Hb 20.2 g/dL. After initiating induction therapy, the EPO level decreased to 1.4 mIU/mL, Hb was reduced to 12.3 g/dL, and improvement of nephromegaly was observed. Therefore, we speculate that the secondary polycythemia was caused by renal ischemia, similar to other kidney disorders such as hydronephrosis, renal cysts, and renal malignancies, but in this case was due to T-ALL infiltration [13–15]. This hypothesis is supported by two cases of secondary polycythemia in patients with lymphoproliferative diseases exhibiting bilateral

Table 3 Adult cases of lymphoma and leukemia presenting with secondary polycythemia

Author [ref]	Year	Age	Sex	Diagnosis	Cr (mg/dL)	Hb (g/dL)	EPO (mIU/mL)	Bilateral Nephromegaly	Kidney size (cm)	Renal biopsy	BP (mmHg)
Al-Tourah AJ [16]	2006	64	M	Small Lymphocytic Lymphoma	N/A	19.7	93 (3.3–16.6)	-	N/A	-	N/A
Osumi T [17]	2013	18	F	B-ALL	1.16	16.6	52.1 (9.1–32.8)	+	18.5/16.8	+	140/100
Bhat RA [18]	2014	14	M	non-Hodgkin's lymphoma	3.2	18.8	150 (30–110)	+	N/A	+	122/72
Present case	2024	32	M	T-ALL	3.7	20.2	38.7 (4.2–23.7)	+	14.2/14	+	**212/150**

ref: References, Cr: creatinine, Hb: Hemoglobin, EPO: erythropoietin, BP: Blood Pressure, B-ALL: B-cell acute lymphoblastic leukemia, T-ALL: T-cell acute lymphoblastic leukemia, N/A: Not Available

nephromegaly due to renal infiltration (Table 3) [17, 18]. Indeed, in the case of B-ALL reported by Osumi et al., expression of hypoxia-inducible factor-1α, a marker of hypoxia that is not detected in normal kidney, was observed in the renal tubule epithelium compressed by lymphoblastic cells [17]. In our case, severe infiltration of the renal tubulointerstitium by leukemic cells resulted in the renal tubule epithelium being almost undetectable, suggesting the possibility that renal ischemia may have stimulated EPO production. Although induction therapy rapidly improved our patient's secondary polycythemia, treatment for renal anemia was required later. Therefore, it is important to carefully monitor Hb levels after initiating treatment, particularly in cases with severe kidney dysfunction.

Another potential cause of secondary polycythemia is paraneoplastic polycythemia, resulting from ectopic EPO production. Major EPO-producing tumors include hepatocellular carcinoma, renal cell carcinoma, cerebellar hemangioblastoma, pheochromocytoma, and uterine fibromyoma [24, 25]. However, to the best of our knowledge, there is no evidence of EPO production by leukemic cells, making it unlikely that T-ALL caused paraneoplastic polycythemia in this case.

Our patient exhibited extremely high BP before and at the referral visit, but there was no evidence of endocrine hypertension and fluid retention. Because severe hypertension in T-ALL patients with bilateral nephromegaly and AKI has not previously been reported (Table 2), we presume that the severe hypertension was mainly caused by polycythemia. This is supported by epidemiological evidence indicating a relationship between hematocrit and BP levels in both normotensive and hypertensive subjects [26, 27], and by clinical evidence showing an increased prevalence of hypertension in subjects with polycythemia, also known as Gaisböck syndrome [11]. Polycythemia can lead to hypertension through several mechanisms, including increased blood viscosity [26], elevated intravascular plasma volume, and chronic vascular inflammation, resulting in vascular remodeling and atherosclerosis [28]. It is possible that blood viscosity may be more sensitive than intravascular plasma volume to changes in Hb levels, given that Bertinieri et al. demonstrated concurrent reductions in Hb levels, BP, and blood viscosity at 7 to 10 days after isovolumic hemodilution in polycythemic patients, whereas there was no significant change in plasma renin activity levels, a marker of intravascular plasma volume [29]. Consistent with this possibility, induction therapy improved Hb levels and BP control in our patient, suggesting that the BP reduction was caused by decreased blood viscosity. Our patient exhibited higher BP and Hb levels than the other three adult cases of secondary polycythemia with lymphoproliferative diseases reported in the literature (Table 3). Despite this, his BP was subsequently well controlled by anti-hypertensive therapy, and the decrease in Hb levels following induction chemotherapy also reduced the need for anti-hypertensive drugs. Therefore, the possibility of lymphoproliferative diseases should be carefully considered in patients with severe hypertension and secondary polycythemia, and both BP and Hb levels should be monitored after initiating induction therapy.

In conclusion, we report a rare case of T-ALL/LBL diagnosed by kidney and bone marrow biopsy, presenting with secondary polycythemia, reduced renal function, and bilaterally enlarged kidneys at the time of diagnosis, leading to severe hypertension. Leukemia and malignant lymphoma should be kept in mind as differential diseases for patients with severe hypertension and secondary polycythemia.

Abbreviations

AKI	Acute kidney injury
BP	Blood pressure
Cr	Creatinine
CT	Computed tomography
EPO	Erythropoietin
Hb	Hemoglobin
LD	Lactase dehydrogenase
PET	Positron emission tomography
RBC	Red blood cells
T-ALL/LBL	T-cell acute lymphoblastic leukemia/lymphoblastic lymphoma
18FDG	[18 F]-fluorodeoxyglucose

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Author contributions
KY, RC, YO and TM1 treated the patient. KY and YO examined the kidney biopsy. KY, YM, SK, RC, AT, YO, TM1, TI and TM2 interpreted the pathological findings. KY drafted the manuscript. KY and SK created figures. YM, RC, AT, YO, TM1, TI and TM2 critically revised the manuscript. All authors read and approved the final manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This study was conducted according to the guidelines in the Declaration of Helsinki. Informed consent was obtained from the patient; the consent allowed their data to be stored, as required by Hiroshima University Hospital.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Competing interests
The authors declare no competing interests.

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