Renal Replacement Therapy

CASE REPORT

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Sustained high-efficiency daily diafiltration using a mediator-adsorbing membrane in Burkitt lymphoma with a very high risk of tumor lysis syndrome: a case series with literature review

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Abstract

Background Tumor lysis syndrome is an oncological emergency triggered by the rapid release of intracellular materials from lysed malignant cells. Intensive chemotherapy is challenging for patients with severe renal dysfunction and a high risk of tumor lysis syndrome. Sustained high-efficiency daily diafiltration using a mediator-adsorbing membrane (SHEDD-fA) could work not only as a renal replacement therapy, but also as a novel method to control intracellular materials, including cytokines and damage-associated molecular patterns. We aimed to describe two cases of patients with Burkitt's lymphoma with a very high risk of tumor lysis syndrome who were successfully treated with a combination of chemotherapy and SHEDD-fA.

Case presentation The first case was of a 67-year-old man who was admitted to the intensive care unit for respiratory failure and diagnosed with Burkitt's lymphoma. Extremely high lactate dehydrogenase levels and anuria, indicating severe acute kidney injury, are considered to be associated with a very high risk of tumor lysis syndrome. SHEDD-fA was initiated prophylactically to prevent exacerbation of tumor lysis syndrome. To ensure the blood concentration of antitumor drugs, SHEDD-fA was stopped temporarily and restarted 6 h after the completion of chemotherapy. No tumor lysis syndrome-related complications were observed. The second case involved a 68-year-old man who was admitted to the intensive care unit due to exacerbation of Burkitt's lymphoma complicated by severe pneumonia and disseminated intravascular coagulation. The patient exhibited metabolic acidosis, hyperkalemia, hyperuricemia, and anuria. SHEDD-fA was performed immediately. As in the first case, we temporarily discontinued SHEDD-fA before chemotherapy and restarted it 6 h after the completion of chemotherapy. No tumor lysis syndrome-associated complications were observed and renal function recovered. Interleukin-6, interleukin-8, and high-mobility group box-1 protein levels in the blood were lower on the outlet side than on the inlet side.

Conclusions SHEDD-fA allows safe and effective administration of chemotherapy in patients with severe renal dysfunction and a very high risk of tumor lysis syndrome. Our findings indicate that blood purification modality may need to be selected according to tumor lysis syndrome severity.

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Keywords Sustained high-efficiency daily diafiltration using a mediator-adsorbing membrane, SHEDD-fA, Chemotherapy, Tumor lysis syndrome, Burkitt lymphoma, Damage-associated molecular patterns, DAMPs

Background

Tumor lysis syndrome (TLS) is characterized by hyperuricemia, hyperkalemia, and hyperphosphatemia caused by the release of substantial amounts of cellular components owing to tumor cell lysis [1]. Short-duration intensive chemotherapy with minimal treatment delays might offer the highest efficacy in the treatment of Burkitt lymphoma [2], but intensive chemotherapy is challenging in patients with severe renal dysfunction. Recently, rasburicase, a recombinant urate oxidase, was shown to improve TLS mortality and was associated with remission of the underlying malignancy [3, 4]. However, a reduction of uric acid levels alone may not be sufficient to prevent and treat TLS.

Many cytokines and damage-associated molecular patterns (DAMPs), such as high-mobility group box-1 protein (HMGB-1) produced by tumor cells exposed to anticancer drugs, are recognized by pattern recognition receptors and are believed to induce further inflammation. DAMPs are not commonly targeted by TLS therapy.

In our intensive care unit (ICU), sustained high-efficiency daily diafiltration using a mediator-adsorbing membrane (SHEDD-fA) is routinely performed in patients with sepsis and septic shock [5]. SHEDD-fA is a highly efficient and intensive hemodiafiltration modality that encompasses diffusion, convection, and adsorption. Therefore, SHEDD-fA may be more suitable to remove the high levels of metabolites that occur in patients with TLS. We aimed to report two cases of Burkitt lymphoma with a very high risk for TLS that were successfully treated with a combination of initial chemotherapy and SHEDD-fA.

Case presentation

Case 1

A 67-year-old man tentatively diagnosed with polymyositis was transferred to our hospital. Initial blood tests revealed a creatinine kinase (CK) level of 1,230 IU/L and lactate dehydrogenase (LDH) level of 5,114 IU/L. After admission, methylprednisolone (1 g/day) was administered for 3 days, followed by prednisolone (80 mg/day). However, the patient's condition deteriorated, and he was transferred to the ICU with respiratory failure on day 5. Blood tests on ICU admission revealed the following results: white blood cell (WBC) count, 20,900/μL (neutrophils: 94%, lymphocytes 2%, monocytes: 4%); hemoglobin, 14.2 g/dL; platelet count, 36.2×10^4/μL; prothrombin time-international normalized ratio (PT-INR), 0.98; activated partial thromboplastin time (APTT), 23.7 s; LDH, 8262 IU/L; blood urea nitrogen (BUN), 54.4 mg/dL; CK, 1,230 IU/L; creatinine, 1.65 mg/dL; potassium, 5.2 mEq/L, phosphate, 3.6 mg/dL; uric acid, 10.9 mg/dL; and lactate, 83.8 mg/dL. The patient's clinical course is shown in Fig. 1.

The patient had normal renal function before admission, but had anuria with a urine output of 72 mL/day on ICU admission. Blood purification was initiated to improve renal function and electrolyte correction. We changed the administration to methylprednisolone (1 g/day) on day 3 after ICU admission, but the patient developed a disturbance in consciousness, and his LDH levels further increased. On day 8 after ICU admission, computed tomography-guided biopsy of the enlarged iliopsoas muscle confirmed the diagnosis of Burkitt lymphoma. On day 11 after ICU admission, CHOP combination chemotherapy was administered (dose was reduced by 50%, cyclophosphamide 375 mg/m^2, doxorubicin 25 mg/m^2, and vincristine 1 mg/m^2). The dose of the anticancer agents was reduced because it was clinically determined that the patient could not tolerate the usual dose of chemotherapy. SHEDD-fA was initiated prophylactically to prevent TLS exacerbation under the following conditions: blood flow rate, 150 mL/min; filtration rate, 1,250 mL/h; and dialysate flow rate, 300 mL/min. A large polymethylmethacrylate (PMMA) membrane hemodialyzer (Filtrazer BG-PQ, 2.1 m^2: Toray Industries, Tokyo, Japan) was placed in the blood circuit. Because cyclophosphamide is a dialytic drug, SHEDD-fA was temporarily discontinued prior to chemotherapy.

Treatment was restarted 6 h after completion of chemotherapy and continued for 36 h. The LDH level was 7707 IU/L before chemotherapy, which increased to 9229 IU/L thereafter owing to the lysis of cancer cells. However, uric acid, potassium, phosphate, creatinine, and BUN levels did not increase above the reference values. In addition, lactate levels improved from 124 mg/dL before chemotherapy to 20 mg/dL after the completion of SHEDD-fA (Table 1). SHEDD-fA was used, followed by continuous hemofiltration. No complications related to TLS or blood purification were observed, and renal function recovered completely.

On day 21 after admission to the ICU, the patient was administered rituximab (375 mg/m^2), and no complications were observed. The patient recovered and was discharged from the ICU 23 days after admission.

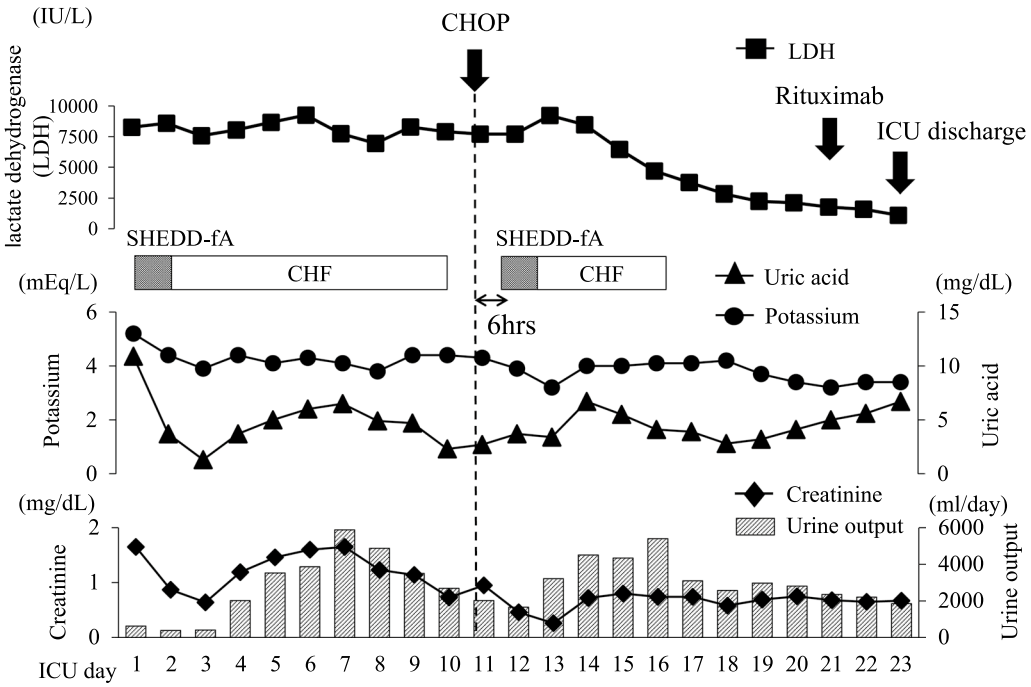

Fig. 1 Clinical course of case 1 after admission to the intensive care unit (ICU). The patient had severe renal dysfunction; therefore, blood purification was initiated immediately. On day 11 after ICU admission, the patient received CHOP therapy. Sustained high-efficiency daily diafiltration using a mediator-adsorbing membrane (SHEDD-fA) was initiated prophylactically to prevent the exacerbation of tumor lysis syndrome (TLS). To maintain therapeutic blood concentrations of anticancer agents, we initiated SHEDD-fA 6 h after administration of the agents, followed by continuous hemofiltration (CHF). There were no complications related to TLS. On day 21 after ICU admission, the patient was administered rituximab with no complications. Renal function recovered fully, and he was discharged from the ICU on day 23 after ICU admission

Table 1 Clinical data at admission to the ICU, before chemotherapy, and after SHEDD-fA

Variable	Case 1			Case 2		
	At ICU admission	Before chemotherapy	After SHEDD-fA	At ICU admission	Before chemotherapy	After SHEDD-fA
PT-INR	0.98	0.99	1.02	1.74	1.35	1.28
APTT (sec)	23.7	31.1	33.9	93.2	33.2	35.0
BUN (mg/dL)	54.4	44	11.9	37.7	23.6	16.4
Creatinine (mg/dL)	1.65	0.95	0.29	2.23	1.34	0.83
Uric acid (mg/dL)	10.9	2.7	3.4	5.3	5.3	2
Potassium (mEq/L)	5.2	4.3	3.8	7.29	3.8	4.2
Phosphate (mg/dL)	3.6	3.9	3.3	12.2	2.3	2.7
LDH (IU/L)	8262	7707	9229	52,645	18,490	16,990
Lactate (mg/dL)	112	124	20	97	18	15
NAd (μg/kg/min)	0.5	0.3	0.1	0.25	Free	Free

PT-INR prothrombin time-international normalized ratio, *APTT* activated partial thromboplastin time, *BUN* blood urea nitrogen, *LDH* lactate dehydrogenase, *NAd* noradrenalin

Case 2

The patient was a 68-year-old man with chronic obstructive pulmonary disease. He presented to our hospital with a history of fatigue and dyspnea for several days. Chest radiography revealed infiltration in the right lung, suggesting pneumonia. LDH levels were extremely elevated at 40,100 IU/L. A bone marrow examination confirmed the diagnosis of Burkitt lymphoma. The patient was admitted to the ICU with severe respiratory failure secondary to pneumonia. Blood test findings after

admission to the ICU were as follows: WBC count, 25,700/μL (neutrophils, 7%; myelocytes, 2%; metamyelocytes, 5%; band cells, 3%; segmented cells, 21%; lymphocytes, 31%; monocytes, 3%); hemoglobin, 8.6 g/dL; reticulocytes, 2500/μL; platelet count, 6.1×10^4/μL; PT-INR, 1.74; APTT, 93.2 s; LDH, 52,645 IU/L; BUN, 37.7 mg/dL; creatinine, 2.23 mg/dL; potassium, 7.3 mEq/L; phosphate, 12.2 mg/dL; uric acid, 5.3 mg/dL; and lactate, 97 mg/dL. Arterial blood gas analysis revealed severe metabolic acidosis, with a pH of 7.18. The patient's clinical course is shown in Fig. 2.

The patient had sepsis, acute kidney injury (AKI), and disseminated intravascular coagulation; therefore, he was unable to receive chemotherapy immediately. He also had anuria, and SHEDD-fA was rapidly initiated to provide renal function support, electrolyte and acid–base correction, and cytokine modulation. His general status improved after intensive therapy.

On day 3 of ICU admission, the patient was administered 70 mg/day prednisolone. On day 4, he received CHOP therapy (cyclophosphamide, 375 mg/m^2 doxorubicin, 25 mg/m^2 vincristine, 1 mg/body). The dose of the anticancer agents was reduced because it was clinically determined that the patient could not tolerate the usual dose of chemotherapy. As renal dysfunction persisted, continuation of blood purification was considered necessary to prevent TLS exacerbation. As in the first case, we temporarily discontinued SHEDD-fA before chemotherapy and restarted it 6 h after the completion of chemotherapy. SHEDD-fA treatment was continued for 48 h, followed by continuous hemofiltration. No abnormalities related to TLS were observed after chemotherapy (Table 1).

We further collected blood samples of case 2 patient from the blood removal side port and blood return side port of the blood purification circuit. We sampled three times from the specific time points of at the start of SHEDD-fA, 3 h later, and 12 h later. We compared the inlet and outlet concentrations of cytokines and HMGB-1 in the blood and calculated these clearances. Cytokine and HMGB-1 concentrations were lower at the outlet than at the inlet (Table 2). The mean clearances of interleukin 6 (IL-6), interleukin 8 (IL-8), and HMGB-1 by SHEDD-fA were 40.3, 70.0, and 36.5 mL/minute, respectively. The renal function recovered, and blood purification was stopped. The patient was discharged from the

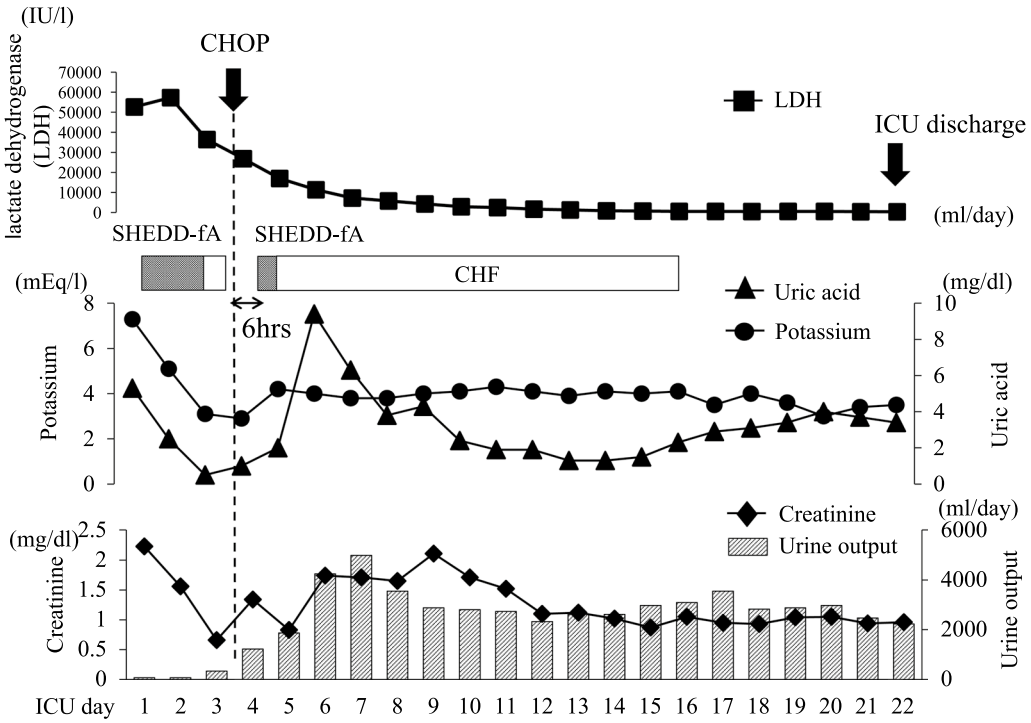

Fig. 2 Clinical course of case 2 after admission to the intensive care unit (ICU). The patient had sepsis, acute kidney injury, and disseminated intravascular coagulation. Intensive therapy improved his general health status. On day 4 after ICU admission, the patient received CHOP therapy. Blood purification was initiated to prevent exacerbation of tumor lysis syndrome (TLS). To maintain the therapeutic blood concentrations of the anticancer agents, we discontinued the use of the sustained high-efficiency daily diafiltration mediator-adsorbing membrane (SHEDD-fA) temporarily before chemotherapy, restarted it 6 h after completion of chemotherapy, and followed it up with continuous hemofiltration (CHF). After chemotherapy, no abnormalities related to TLS were observed. The patient was discharged from the ICU on day 21 after ICU admission

Table 2 Blood inlet and outlet concentrations during SHEDD-fA

	Blood inlet concentration (CBi)	Blood outlet concentration (CBo)	Mean clearance* (mL/min)
IL-6 (10^3 pg/mL)	11.2 ± 7.9	9.9 ± 7.7	40.3
IL-8 (10^3 pg/mL)	112.4 ± 5.6	69.3 ± 6.5	70.0
IL-10 (10^3 pg/mL)	7.5 ± 2.6	5.0 ± 3.0	70.6
MCP-1 (10^3 pg/mL)	218.6 ± 71.3	146.9 ± 53.0	63.8
HMGB-1 (10^3 pg/mL)	18.5 ± 1.9	16.3 ± 2.7	36.5

*Clearance = (CBi − CBo)/CBi × (Q_B − Q_F) + Q_F

Results are expressed as mean and standard deviation

IL interleukin, *MCP-1* monocyte chemotactic protein 1 *HMGB-1*: high-mobility group box-1 protein, *CBi* blood inlet concentration, *CBo* blood outlet concentration, Q_B blood flow rate, Q_F filtration flow rate

ICU 21 days after admission. Two days after being transferred from the ICU, the patient was administered rituximab (375 mg/m^2), with no complications.

Discussion and conclusions

We describe the safe and effective administration of chemotherapy in patients with Burkitt lymphoma at a very high risk of TLS and AKI. The TLS risk classification of patients with Burkitt's lymphoma is based on disease stage and LDH levels [6]. LDH levels at ICU admission were remarkably high in both our cases: 8262 IU/L in case 1 and 52,645 IU/L in case 2. Risk assessment and management of TLS should involve the preservation of renal function and prevention of AKI. However, both patients developed severe AKI prior to chemotherapy and met the diagnostic criteria for TLS on ICU admission. We were concerned that chemotherapy could not be safely administered; therefore, we initiated blood purification to prevent exacerbation of TLS. As the clearance provided by continuous kidney replacement therapy (CKRT) was not sufficient to remove the high levels of metabolites associated with TLS, SHEDD-fA was selected as the high-efficiency hemodiafiltration modality. The clearance of small molecules during SHEDD-fA (approximately 130 mL/min [7]) exceeds that during normal renal function. SHEDD-fA was started 6 h after chemotherapy because the risk of hyperkalemia is high 6 h thereafter [8]. SHEDD-fA is generally performed in 12 h. If potassium increases during SHEDD-fA, blood purification time will be extended accordingly. SHEDD-fA was followed by continuous hemofiltration to prevent the rebounding of electrolyte abnormalities. Neither of the patients developed fatal metabolic complications associated with TLS. Moreover, there were no complications related to blood purification, and renal function recovered in both cases.

AKI remains a common complication in cancer patients, occurring in 11–22% of patients [9]. Furthermore, 80% of critically ill patients with TLS in the ICU develop AKI [4]. AKI is associated with high morbidity and mortality. Although the indications for kidney replacement therapy (KRT) in patients with TLS are similar to those in patients with other causes of AKI, lower thresholds are used in patients with TLS because of potentially rapid potassium release and accumulation, particularly in patients with oliguria [10]. Currently, no data are available from a study comparing conservative treatment with preemptive dialysis for TLS [11]. CKRT is often the preferred modality in critically ill patients over KRT [12, 13]. Our literature search on KRT modalities for TLS revealed that there have been only four studies excluding case reports and no randomized controlled trials (Table 3). CKRT was chosen as the modality of blood purification in four studies [14–17]. Although potentially allowing for less hemodynamic instability, no study has compared the outcomes of CKRT with those of intermittent hemodialysis. In severe cases such as those described

Table 3 Review of the literature on modality of KRT for TLS

Author/year reference No	No of cases	Age (years)	Tumor type	Maximum LDH in cases (IU/L)	Modality of KRT (membrane)	KRT target
Saccente SL/1995 [14]	5	1–12	BL (*N* = 3), ALL (*N* = 2)	10,056	CVVH (Polysulfone)	Metabolic derangement
Choi KA/2009 [15]	11	34–63	BL (*N* = 11)	25,900	CVVH (Not available)	Metabolic derangement
Nakamura M/2009 [16]	4	19–68	ML (*N* = 2), ALL (*N* = 1), AML (*N* = 1)	32,820	CVVHDF (Polymethyl methacrylate)	Cytokines
Wang Y/2018 [17]	8	5–14	ALL (*N* = 5), NHL (*N* = 2), BL (*N* = 1)	Not available	CVVH daytime (Acrylonitrile)	Metabolic derangement

ALL acute lymphoblastic leukemia, *AML* acute myeloid leukemia, *BL* Burkitt lymphoma, *ML* malignant lymphoma, *NHL* non Hodgkin lymphoma, *CVVH* continuous venovenous hemofiltration, *CVVHDF* continuous venovenous hemodiafiltration

in this report, we recommend SHEDD-fA for the prevention and treatment of TLS, immediately followed by continuous hemofiltration to block rebound electrolyte abnormalities. Our findings indicate that blood purification modalities should be personalized and selected according to the severity of TLS. In our cases, SHEDD-fA is performed before chemotherapy to control organ dysfunction and general condition caused by TLS, and after chemotherapy, it is performed to prevent further exacerbation of TLS. We believe that SHEDD-fA is a good indication for TLS patients with extremely high counts of tumor cell (LDH levels of greater than three times the upper limit of normal) or organ dysfunction (severe AKI and shock, DIC).

Previous studies on KRT for TLS have focused on modulation of severe metabolic derangement. Interestingly, Nakamura et al. used PMMA, which has cytokine adsorption properties, for the treatment of TLS with hypercytokinemia [16]. On the other hand, ours is the first report of PMMA-KRT for the removal of DAMPs released from decaying cells. In addition to metabolic products, damaged or stressed tumor cells can release mediators called DAMPs during chemotherapy, leading to a vicious cycle of deterioration through further stimulation of immune cells [18]. This dysregulated immune response to infection is associated with the failure to return to homeostasis, which harms the host, resulting in cellular dysfunction and organ failure [19, 20]. CKRT is a type of renal replacement therapy that also functions in cytokine regulation and is effective against TLS [16]. However, to the best of our knowledge, there are no reports on the control of DAMPs such as HMGB1 released from cancer cells by blood purification. We previously reported that PMMA membranes adsorb cytokines [21] and HMGB1 [22, 23] in an in vitro closed-loop system. In case 2, the clearance of cytokines and HMGB1 by SHEDD-fA cells with the PMMA membrane was measured. SHEDD-fA shows high adsorptive clearance of these cytokines, even in patients with TLS. There are no reports comparing the adsorption capacity of various membranes for cytokines and HMGB1 in TLS. However, our previous in vitro report compared PMMA and polysulfone(PS) membranes. The PMMA membrane showed overwhelmingly higher clearance of cytokines [21] and HMGB1 [22, 23] than the PS membrane. Based on these results, we expected that PMMA membranes could be selected for TLS patients to effectively remove cytokines and DAMPs such as HMGB1.

However, several challenges remain regarding the use of blood purification therapies to prevent TLS. When initiating blood purification after chemotherapy, the possibility of removing the anticancer agents must be considered. In the present cases, we determined the optimal time for the initiation of blood purification based on the half-life, molecular weight, and protein binding ratios of the anticancer agents, and the time when the tumor started to collapse. In future studies, the therapeutic monitoring of anticancer drugs is recommended to ensure efficacious dosing in patients who require blood purification to prevent or manage TLS.

We describe the successful use of SHEDD-fA to prevent and treat TLS in two Burkitt lymphoma patients with AKI and a very high risk for TLS who were treated with chemotherapy. SHEDD-fA may allow safe and effective administration of chemotherapy in patients with severe renal dysfunction.

Abbreviations

AKI	Acute kidney injury
APTT	Activated partial thromboplastin time
BUN	Blood urea nitrogen
CK	Creatine kinase
CKRT	Continuous kidney replacement therapy
DAMPs	Damage-associated molecular patterns
HMGB-1	High-mobility group box-1 protein
ICU	Intensive care unit
IL	Interleukin
LDH	Lactate dehydrogenase
PMMA	Polymethylmethacrylate
PT-INR	Prothrombin time-international normalized ratio
SHEDD-fA	Sustained high-efficiency daily diafiltration using a mediator-adsorbing membrane
TLS	Tumor lysis syndrome
WBC	White blood cell

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TK wrote the original draft, prepared the tables, and edited the later versions. AO and KM reviewed and edited the manuscript for submission. All authors read and approved the final manuscript.

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Declarations

Ethics approval and consent to participate
Not applicable.

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

Competing interests
KM has consulting contracts with Toray Industries Inc. The other authors declare that they have no competing interest.

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