CASE REPORT
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Effectivity of Sustained Low-Efficiency Dialysis (SLED) in a critically ill child with Rapidly Progressive Glomerulonephritis (RPGN): a case report

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ABSTRACT

Background: Rapidly Progressive Glomerulonephritis (RPGN) is a rare condition in children and the incidence remains unknown. Systemic lupus erythematosus (SLE) is one of the etiologies, usually presented as lupus nephritis. RPGN causes fast deterioration of renal function and requires timely intervention to preserve it. RPGN can lead to acute renal failure, which management requires emergency dialysis as Renal Replacement Therapy (RRT). This case study aims to evaluate the effectiveness of SLED in a critically ill child with RPGN.

Case Presentation: A 16-year-old girl with a 10-day history of worsening dyspnea with edema anasarca, frothy sputum, and oliguria. Laboratory findings revealed anemia, leukocytosis with lymphopenia, thrombocytopenia, high procalcitonin, decreased glomerular filtration rate with uremia, hyperkalemia, increased anion gap metabolic acidosis, low C3 complement, haematuria, and proteinuria. Chest X-Ray showed pulmonary edema, pneumonia, and pleural effusion, while a CT scan revealed hydronephrosis, proximal ureter ectasis, hepatomegaly, and ascites. According to the 2015 American College of Rheumatology (ACR)/ Systemic Lupus International Collaborating Clinics (SLICC) criteria, her score was 6. She was diagnosed with RPGN due to SLE, acute kidney injury stage failure, acute respiratory distress syndrome, community-acquired pneumonia, and septic shock. The patient received SLED as a mode of dialysis due to her unstable hemodynamics. Other management included high-dose methylprednisolone pulses, antibiotics, and epinephrine drip. Her condition improved significantly both clinically and metabolic after receiving SLED.

Conclusion: SLED can be the choice of dialysis mode as emergency renal replacement therapy in hemodynamically unstable children with acute renal failure due to RPGN.

Keywords: Acute Renal Injury, Children, Renal Failure, RPGN, SLED.
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INTRODUCTION

Dialysis is a type of Renal Replacement Therapy (RRT) in which the kidney's filtration function is supplemented by artificial equipment that eliminates excess water, solutes, and toxins from the blood.[1] It is a method of artificially replacing kidney function, particularly in renal insufficiency. Dialysis cannot replace lost kidney function fully, although it can help regulate it to some extent by diffusion and ultrafiltration.[2] Technological advancements have led to dialysis as important management for severe acute kidney injury (AKI).[3-5] AKI remains a significant cause of morbidity and mortality in hospitalized children, particularly critically ill patients. One of the causes of AKI including rapidly progressive glomerulonephritis (RPGN), which requires emergency dialysis to preserve renal function and prevent complications to end-stage renal disease (ESRD).

Dialysis procedures include peritoneal dialysis (PD) and hemodialysis (HD). The classic modalities of HD, including continuous renal replacement therapy (CRRT) and intermittent hemodialysis (IHD), while sustained low-efficiency dialysis (SLED), a hybrid technic, have been used lately, although their studies in children are still limited.[6-8] CRRT which permits gradual fluid and solute removal, is the preferred modality for managing AKI and fluid overload in critically ill children with unstable hemodynamics. However, CRRT frequently gives rise to complications and its Feasibility is often limited in some centers. Additionally, the logistic burdens of administering CRRT are quite a lot, including the need for anticoagulation and specialized pre-manufactured solutions with overall high costs.[9,10]

Rapidly Progressive Glomerulonephritis (RPGN) is quite uncommon anywhere. Compared to the United Kingdom, where 2 cases per million person-years have been

documented, the prevalence in the United States of America is approximately 7 cases per million person-years. Further reported global clusters to suggest a significant environmental impact on the etiology.[11] The advantages of periodic treatment with hemodynamic stabilization and metabolic management may be available with a more recent technique called Sustained low-efficiency dialysis (SLED), which uses prolonged hemodialysis treatment sessions with gradual fluid removal.[12,13]

According to the prior study, SLED offers consistent renal replacement therapy and favorable clinical results. The logistical aspects of SLED delivery by ICU nursing staff are acceptable. According to current standards, a small solute clearance is sufficient for intermittent hemodialysis and continuous renal replacement therapy (CRRT), and a bigger solute clearance is significant. SLED is an effective substitute for CRRT in this situation.[14]

Sustained Low-Efficiency Dialysis (SLED) has emerged as an alternative therapy in AKI. A previous study in the adult population with kidney injury and septic shock showed that SLED is a viable modality and has proven hemodynamically tolerable and efficacious in a patient with septic shock compared to CRRT. Therefore, SLED is gaining popularity due to its logistic advantages and apparent cost benefits. Unfortunately, the literature which discusses the benefits of SLED in pediatric patients is still limited.[8,9,15]

Based on those mentioned above, this case study aims to evaluate the effectiveness of SLED in a critically ill child with RPGN at Sanglah General Hospital, Bali, Indonesia.

CASE REPORT

A 16-year-old girl was referred from a private hospital with a diagnosis of pneumonia, hypertension, acute kidney injury stage failure, septic shock, and anemia. The patient complained of shortness of breath ten days before admission, preceded by a cough with frothy pink sputum. She had decreased urination ten days before admission, followed by swelling all over the body, starting in both eyelids and face, as well as spreading to the stomach and extremities. In the last twenty days before admission, she appeared more fatigued and lost her appetite. She became more drowsy four days before admission without a history of headaches, seizures, behavioral change, or trauma. About 3-4 times a day, vomiting is approximately 50-100 ml since eighteen days before admission. Each vomiting episode that contained water and the meal she had consumed was preceded by nausea with no projectile, no blood, no preceded by headache, no abdominal pain, no bloated stomach, no diarrhea, nor a history of eating unusual food, spicy or sour foods. Defecation was regular, once a day, with normal consistency. There was no headache, palpitation, purpura at the buttocks, photosensitivity, weight loss, or pain in the extremity. She had no history or close contact with a confirmed Coronavirus Disease 2019 (COVID-19) case. She had no history of previous illness, blood transfusion, and allergy. The history of taking drugs, in addition to medical advice, was denied. The family had no allergy, kidney disease, hypertension, lupus or diabetes history. The patient had hypotension from the physical examination, which responded to an epinephrine intravenous drip, worsened dyspnoea, anuria, edema anasarca, and stupor. Laboratory findings revealed leucocytosis 25.2 K/µL, neutrophilia 23.42 K/µL (92.95%), lymphopenia 0.24 x K/µL (0.97%), anemia normochromic normocytic (Hb) 10.55 g/dL, thrombocytopenia 57.52 x K/µL, high procalcitonin 41.49 ng/mL, hypoalbuminemia 2.4 g/dL, normal blood sugar 93 mg/dL. The renal function panel revealed increased blood urea 207 mg/dL and creatinine serum 14.8 mg/dL with estimated creatinine clearance (eCCI) of 5.9 ml/min/1.75 m^2. Arterial blood gas (ABG) analysis revealed metabolic acidosis with increased anion gap (pH 7.05, pCO2 44 mmHg, pO2 52 mmHg, Base excess (BE) -18 mmol/L, HCO3 12 mmol/L, total CO2 (tCO2) 13 mmol/L, SO2 95.5%), serum Sodium (Na) 134 mmol/L, Potassium (K) 5.2 mmol/L, Chloride (Cl) 94.3 mmol/L, Calcium (Ca) 8.3 mg/dL, phosphor (P) was 4.12 mg/dL, and lactic acid 2.15 mmol/L. Urinalysis revealed haematuria 8-12/HPF and proteinuria (3+). C3 Complement 74.7 mg/dL, Parathyroid hormone was 144.9 pg/mL and 25(OH) D was 7.1 ng/mL. The immuno-serology examination was performed for suspicion of SLE, which revealed Anti-double-stranded DNA nucleosome-complexed (Anti-dsDNA-NcX) <10 IU/mL and Anti-Nuclear Antibody (ANA) was negative. RT PCR SARS-COV-2 was negative and the Blood culture was negative. Chest X-ray (Figure 1a) revealed pneumonia, pulmonary edema, and minimal bilateral pleural effusion. CT scan abdomen without contrast revealed grade II hydronephrosis sinistra, proximal ureter ectasis, hepatomegaly, ascites, and bilateral pleural effusion.

The patient was treated in the intensive care unit with an Intensivist consultant, Paediatric Nephrologist, Immunologist, and Pulmonologist. Scoring according to 2015 American College of Rheumatology (ACR)/Systemic Lupus International Collaborating Clinics (SLICC) criteria was counted by the Immunologist, 6 points in sum (pleurisy 1 point, nephritis 2 points, hematology involvement with lymphopenia and thrombocytopenia 2 points, and decreased C3 complement 1 point). Therefore, the patient was diagnosed as RPGN due to SLE, AKI stage failure, Acute Respiratory Distress Syndrome (ARDS), community-acquired pneumonia, and septic shock. Due to the ARDS, she required Intermittent Mandatory Ventilation (IMV) for breathing. The patient received hemodialysis with the SLED method with a duration of 6-8 hours, blood flow (Qb) of 200 ml/min, and ultrafiltration goal of 2.5-3 liters. SLED was chosen due to her unstable hemodynamics. During sled, invasive blood pressure monitoring was done with blood flow 200ml/min 4ml/Kg/min, Dyalisate flow (QD) 400ml/min, and UF 0.1ml/kg/min. She was also given high-dose intravenous (IV) methylprednisolone (30 mg/kg/day, given at a maximum dose of 1000 mg) for three consecutive days and an albumin 20% transfusion. Other medicines were given: levofloxacin (300 mg/24 hours), cefotaxime (2 g/12 hours), and epinephrine drip.

After receiving SLED, her clinical condition improved. Her consciousness began to improve, and she no longer required inotropic, as well as reduced generalized edema and improved

pulmonary edema (Figure 1b). Laboratory examination results showed improvement in metabolic acidosis and decreased serum urea and potassium levels (Table 1).

DISCUSSION

Rapidly progressive glomerulonephritis (RPGN) is manifested by the clinical feature of progressive deterioration of renal function and glomerulonephritis in a relatively short time, ranging from a few days to three months. Glomerulonephritis happens when the glomerular injury occurs due to inflammation, which is manifested by hypertension, edema, proteinuria, and haematuria.[16] In RPGN, the patient usually presents with the features of emergency clinical manifestation, including oliguria, fluid overload, uremia, acidosis, hyperkalemia, gross hematuria, and rapidly progressing acute renal failure (severe decrease in glomerular filtration rate characterized by anuria and increased serum level of BUN and creatinine). This disease is also characterized by histology findings of crescent lesions involving>50% of glomeruli.[15,16] RPGN is quite rare in children with unknown precise incidence.[14,16,17]

Several causes of RPGN have been discovered, including anti-GBM linear (IgG deposits), immune complex, infection (e.g., streptococcus, infective endocarditis, HIV, hepatitis B and C), systemic disease (e.g., SLE, HSP), and post-renal transplantation.[18] A previous study showed that only 19,6% cases of 36 cases of pediatric RPGN have a complete recovery, with 30% progressing to chronic kidney disease and 12,5% requiring dialysis.[19] RPGN is broadly classified based on the histopathology and immune complex deposition as follows: Linear antibody deposition; Granular immune complex deposition disorders; Pauci-immune (absence of deposition) disorders.[20]

There are some mixed, as well as idiopathic variants are also reported. Granular immune complex deposition disorders can be idiopathic or secondary to other diseases; one of them is lupus nephritis.[20] Kidney injury is one of the possible complications in glomerulonephritis when the kidney loses the filtering function of the nephron, which can lead to AKI.[21] Acute kidney injury or failure is one of the possible complications of glomerulonephritis when the kidney loses the filtering function of the nephron. Acute kidney injury (AKI) is characterized by an abrupt deterioration of kidney function and is common in critically ill children and adults. Paediatric AKI has been associated with higher morbidity and mortality after adjustment for other risk factors and is a risk factor for hypertension

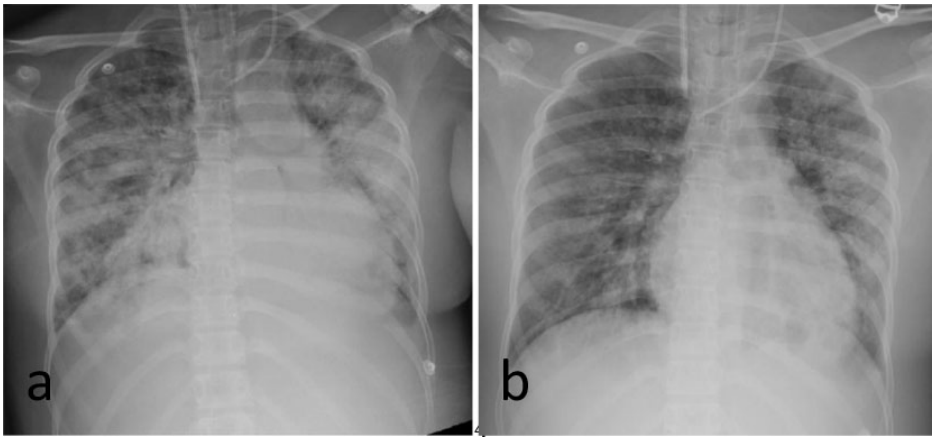

Figure 1. Chest x-ray posteroanterior view. (**a**) Before SLED, showing pleural effusion, pneumonia, and pulmonary edema. (**b**) After SLED, showing improvement compared with the initial result.

Table 1. Laboratory data following SLED

Test	Before SLED	After SLED	Unit	Reference range
Renal Function				
Serum urea	207	36.9	mg/dL	8.00 - 23.00
Serum creatinine	14.8	2.90	mg/dL	0.50 - 0.90
Estimated-creatinine clearance (eCCI)	5.9	30.3	mL/min/1.73m^2	
Electrolyte				
Potassium (K)	5.20	3.51	mmol/L	3.50 - 5.10
Sodium (Na)	134	140	mmol/L	136 - 145
Chloride (Cl)	94.3	107	mmol/L	94 - 110
Calcium (Ca)	8.3	8.0	mg/dL	9.20 - 11.00
Phosphor (P)	4.12	3.47	mg/dL	2.5-4.5
Magnesium (Mg)	1.36	1.8	mg/dL	1.6-2.6
Arterial Blood Gas				
pH	7.05	7.32		7.35 - 7.45
pCO2	44	54.4	mmHg	35.00 - 45.00
pO2	52.0	143.5	mmHg	80.00 - 100.00
BEecf	-18	1.5	mmol/L	-2 - 2
HCO3-	12	27.5	mmol/L	22.00 - 26.00
SO2c	95.5	98.6	%	95 % - 100 %
TCO2	13.00	29.2	mmol/L	24.00 - 30.00

and chronic kidney disease (CKD) in the long term. Pediatric Risk, Injury, Failure, Loss, End Stage Renal Disease (pRIFLE) provided a standardized definition of AKI in children.[21] It was characterized by estimating creatinine clearance and/or a decrease in urine output. The severity of AKI is staged according to the amplitude of serum creatinine elevation from baseline value or the duration of compromised urine output.[21] In this case, the patient presented with clinical kidney function deterioration, which showed anuria, fluid overload (edema anasarca with pulmonary edema), and uremic syndrome (high urea serum level with decreased consciousness, nausea, and vomiting). The blood laboratory examination revealed decreased e-CCI according to stage failure (F) (5.9 mL/min/1.73m^2); urine laboratory examination confirmed haematuria and proteinuria along with hypertension and edema. Those conditions were known in acute onset (approximately 1 month) and which family denied a history of the same complaints before. This condition led to the diagnosis of acute kidney injury stage failure caused by the RPGN. The patient didn't do a biopsy to diagnose the RPGN because the patient had already been diagnosed with nephritis lupus with rapid deterioration of kidney function.

Of all the various etiologies of RPGN, Systemic Lupus Erythematous (SLE) is usually presented as lupus nephritis. This condition is considered severe and prominent.[22] Childhood-onset SLE, defined as onset before 18 years of age, is a rare disease, with an incidence of 0.3 to 0.9 per 100,000 person-years and a prevalence of 1.89 to 25.7 per 100,000 children worldwide.[23] SLE is called the great mimicker, as the disease shares characteristics with many other autoimmune diseases. Especially when the classic malar rash is absent, diagnosing SLE can be a challenge. Most patients who are diagnosed with SLE fulfill ACR classification criteria for SLE. Although not rigorously studied in SLE, the criteria have a greater than 95% sensitivity and specificity for the diagnosis of SLE.[24-26]

Patients are classified as having SLE if they satisfy four of the criteria such as at least one clinical criterion and one immunologic criterion or if the patient has biopsy-proven nephritis compatible with SLE and with ANA or anti-dsDNA antibodies. SLE can affect any organ system and leads to glomerulonephritis and central nervous system involvement arguably more often in SLE than in adults with SLE.[25] In this case, the patient was diagnosed with SLE. Based on 2015 ACR/SLICC criteria, our patient had a total score of 6, which included pleurisy (1 point), nephritis (2 points), hematology involvement with lymphopenia and thrombocytopenia (2 points), and decreased C3 complement (1 point).[26]

In RPGN, the decision for aggressive intervention should be prompted due to the rapid progression to end-stage renal disease (ESRD). Dialysis should be considered to stabilize renal function as the essential measure for managing RPGN, especially in the etiology of SLE.[18] Immediate dialysis in the setting of acute and emergency events is indicated in the condition of Acute kidney injury; Uremic encephalopathy; Pericarditis; Life-threatening hyperkalemia; Refractory acidosis; Hypervolemia causing end-organ complications (e.g., pulmonary edema); Failure to thrive and malnutrition; Peripheral neuropathy; Intractable gastrointestinal symptoms; Asymptomatic patients with a GFR of 5-9 mL/min/1.73 m^2; Any toxic ingestion.[1]

These conditions result in cytokine (immune response modulator) dysregulation and poor clearance, resulting in end-organ injury, hemodynamic instability, or immunosuppression caused by vasodilation, cardiac depression, and immunosuppression, putting off renal recovery.[1] Acidosis (due to intercellular shift) and reduced renal excretion cause potassium anomalies in individuals with chronic kidney disease or renal failure. Elevated urea levels in renal failure patients can cause uremic pericarditis. Fluid retention occurs in patients with CKD and heart failure, worsening heart failure and pulmonary edema. Arrhythmias caused by electrolyte imbalances, uremic pericarditis, and fluid overload caused by severe congestive heart failure with poor kidney function require dialysis.[1]

Dialysis maintains homeostasis (a steady internal environment) in persons with a fast loss of kidney function, such as AKI, or a progressive loss, such as chronic kidney disease. Dialysis incorporates two processes for removing solutes via a semipermeable membrane along a concentration gradient: Random molecule motion causes diffuse clearance. Small molecules go across the membrane faster than larger ones. A convective clearance occurs when water's osmotic force drives solutes through the membrane (solvent drag).[1] Dialysate is a mixture of sodium, potassium, magnesium, calcium, bicarbonate, chloride, and dextrose in highly filtered water. It is deficient in uremic blood's low-molecular-weight waste products. When a semipermeable membrane separates uremic blood and dialysate, the flux rate of waste solutes from blood to dialysate surpasses the back-flux from dialysate to blood. The concentrations of permeable waste products in the dialysate and blood eventually equalize, with no further net waste product removal.[1,2] A concentration equilibrium is avoided during dialysis, and the gradient is maintained by continually refilling the dialyzer with fresh dialysis fluid and replacing dialyzed with undialyzed blood.

The concentration differential of waste products between blood and dialysate is maximized with "countercurrent" flow. The amplitude of the concentration gradient, the membrane's mass transfer coefficient, and the membrane surface area all influence the rate of solute diffusion. Membrane thickness, solute size, and flow conditions on both sides of the membrane all influence the transfer coefficient. Popular dialysis modes for pediatric patients are Intermittent Hemodialysis (IHD) and Continuous Renal Replacement Therapy (CRRT). Patients with unstable hemodynamics often use CRRT as a modality of dialysis. While Sustained Low-Efficiency Dialysis (SLED) is the hybrid mode, its application in children is still debatable.[1,6,7]

In the last two decades, SLED has been used as a substitute for CRRT in hemodynamically unstable critically ill patients with renal failure. Several factors have influenced the advantage of performing SLED over CRRT; they are: (1) the nonavailability of CRRT machines, (2) the drive to reduce costs by avoiding

expensive CRRT solutions, also (3) the ability to provide adequate RRT in hemodynamically unstable patients while allowing downtime for procedures as well as reduced need for anticoagulation.[27,28]

Moreover, unlike CRRT, SLED can utilize standard IHD technology while providing slow solute and fluid removal, ensuring hemodynamic stability. By the time the article by Schlaeper C et al., was published in 1999, the role of SLED in Acute Kidney Injury (AKI) had gained more attention.[29] SLED has shown a good effect in treating Multiple Organ Dysfunction Syndrome (MODS) and acute poisonings in adults.[30–33] Unfortunately, despite the spurt of data on the utility of SLED in adults, a lack of pediatric reports impeded the widespread acceptance of this cost-effective therapy in pediatric practice.

The duration of SLED can be adjusted for each patient. Durations range from 6-18 hours each day. Dialysate flow (Qd) ranges from 100 to 300 mL/min, according to the dialysis equipment, treatment duration, and ultrafiltration tolerance. A Higher Qd of 300 ml/min is often utilized for a duration of <8 hours, whereas a lower Qd is used for a longer duration. The ultrafiltration (UF) rate is adjusted based on the patient's clinical necessity and hemodynamic stability.[34] The dialysate's composition changes depending on the clinical demands. 3.0–4.0 mEq/l potassium, 1.5–2.5 mEq/l calcium, and 24–35 mmol/l bicarbonate are typical dialysate baths. Phosphate depletion can be accomplished intravenously or by mixing 45 ml Fleets Phosphasoda with 9.5 liters of bicarbonate concentrate to reach the final concentration of 0.81 mmol/l. SLED circuit clotting without anticoagulation has been observed to occur in 26–46% of single-pass machines and significantly less in batch systems.[34]

Feasibility and tolerability of sustained low-efficiency dialysis in critically sick pediatric patients had been shown in a multicentric retrospective study that stated that SLED appears to be a feasible and well-tolerated method of providing renal replacement in critically ill pediatric patients. A previous study found that there was a significant decrease in urea and creatinine after SLED therapy.[8] In addition, there was a significant improvement in bicarbonate level.[8] In this case, the patient received SLED with a duration of 6-8 hours, Qb 200 ml/min, and an ultrafiltration goal of 2.5-3 liters. SLED was chosen due to her unstable hemodynamics. After undergoing SLED, she showed favorable clinical outcomes.

The extracorporeal hemodialysis (HD) circuit is frequently anticoagulated with intra-dialytic heparin. Intra-dialytic heparin's putative advantage is to reduce HD circuit clotting, decreasing medication delay and blood loss while improving HD adequacy.[35] The most often used anticoagulant for hemodialysis is unfractionated heparin (UFH). However, because UFH comprises various molecules with varying biological activity, it can cause both under and excess anticoagulation. Due to cheaper expenses, LMWHs seem to be the anticoagulant of preference for hemodialysis in several European nations. UFH is still widely used as an anticoagulant. Unlike UFH, which may require one or two bolus injections or a bolus preceded by an infusion, LMWHs only require a single bolus injection to begin dialysis.[36] Heparin, on the other hand, can pose a risk of bleeding. Numerous caregivers on HD use anticoagulants and platelet inhibitors owing to the increased rate of occlusive vascular events such as vascular access failure, myocardial infarction, and ischemic stroke. Patients with ESRD who require dialysis are already at an increased risk of bleeding because uremia can cause platelet dysfunction. Furthermore, patients with HD are more likely to have invasive surgical operations, which might be compounded by hemorrhage. All of these characteristics play a role in the elevated rate of serious bleeding episodes in HD.[37]

However, in a hospitalized population with a high level of disease, the risk of heparin-related adverse events such as bleeding and heparin-induced thrombocytopenia is an issue. As a result, using a heparin-free HD regimen may be a better option than using regular heparin during HD.[35]

Feasibility and tolerability of heparin-free dialysis using sustained low-efficiency dialysis in critically sick pediatric patients have proven that SLEDD-f is a feasible and well-tolerated method of renal replacement in critically ill pediatric patients. Post-SLEDD-f complications were relatively small such as premature session termination was 9.5% (with reasons hypotension was 8.6% and due to circuit clotting was 0.8%), post-SLED hypocalcemia was 6.2%, hypophosphatemia was 0.4%, and hypokalemia was 7.0%.[38] The patient was diagnosed with acute kidney injury stage failure. As the patient had thrombocytopenia and hemodynamic instability, we performed heparin free-SLED on her every two days with ultrafiltration target 2,3-3 liters. Every session took 6-8 hours. The patient reached significant clinical improvement, which resolved consciousness, anasarca edema, and pleural effusion. There were no severe complications of the SLEDD-f recorded.

The absence of a confirmed diagnosis of RPGN on histologic examination is a limitation of this case report. SLED in children and adolescents occurs relatively seldom. Therefore, it might be challenging to locate data in publications. To learn further, it is necessary to conduct more extensive research on RPGN, particularly concerning SLED therapy.

Previously, informed consent was obtained from patients whose data gathering had been authorized by the patients and their parents, who were standing as the patients' custodians in drafting this publication. All information the authors have gleaned from patients is protected by their legal rights, and they can only share the information if consent is provided.

CONCLUSION

For children with hemodynamically unstable acute renal failure brought on by RPGN, SLED may be the preferred dialysis mode for emergency renal replacement management. Due to her unstable hemodynamic and thrombocytopenia which may induce bleeding, the risk was managed with heparin-free SLED. The condition improved significantly after receiving the procedure. Based on this case and previous literature on SLED, this dialysis method can be the choice for renal replacement therapy in hemodynamically unstable children.

CONFLICT OF INTEREST

Regarding the manuscript, no conflicting interests exist.

ETHICS CONSIDERATION

Ethics approval and informed consent have been following COPE and ICMJE protocols for the case study prior to the study being conducted.

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None.

AUTHOR CONTRIBUTIONS

The study's conceptual framework, literature and data synthesis, data analysis, and subsequent scientific publishing are all the responsibility of the authors.

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