Dialysis Toolkit for Severe Thrombocytopenia in Patients with Acute Kidney Injury

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Abstract

Acute kidney injury (AKI) in the setting of severe thrombocytopenia represents a formidable challenge in nephrology, where both the underlying disease and therapeutic interventions carry a high risk of bleeding. Conditions such as lupus nephritis, thrombotic thrombocytopaenic purpura (TTP), haemolytic uraemic syndrome (HUS), haematological malignancies, chemotherapy-induced marrow suppression, and severe viral infections including dengue or sepsis-related disseminated intravascular coagulation often present with simultaneous kidney dysfunction and profound thrombocytopenia. In such renothrombocytopenic syndromes, initiation of kidney replacement therapy (KRT) is often lifesaving but complicated by the heightened risk of haemorrhage because of various factors including anticoagulation use, vascular access placement, and fragile haemostasis. This article reviews the unique challenges of dialysis in patients with thrombocytopenia and proposes a structured "Dialysis Toolkit" for clinicians.

Keywords: Acute kidney injury, bleeding diathesis, dialysis, haemodialysis, rapidly progressive renal failure, renal replacement therapy, thrombocytopenia,

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Introduction

Severe thrombocytopenia is defined as a platelet count below 50,000/µL and these patients have an increased risk of spontaneous or procedure-related bleeding.[1] Clinically, platelet counts below 10,000/µL are considered critical, often requiring urgent intervention.[2] Being an operational challenge in patients requiring kidney replacement therapy (KRT), it significantly increases the risk-benefit ratio of dialysis. Such renothrombocytopaenic syndromes are seen across a wide spectrum of diseases, ranging from haematological to autoimmune and systemic conditions. Haematological malignancies such as acute leukaemia, lymphomas, and aplastic anaemia often present with marrow suppression and severe thrombocytopenia, complicating the initiation of dialysis in patients with acute kidney injury (AKI).[3] Similarly, sepsis and septic shock can result in both AKI and thrombocytopaenia due to disseminated intravascular coagulation (DIC), haemophagocytic syndromes, and increased platelet consumption.[4] Such conditions create a high-risk environment for invasive procedures such as vascular access placement. Autoimmune diseases also contribute significantly to this overlap. Lupus nephritis, particularly in its severe proliferative forms, often progresses to rapidly progressive renal failure (RPRF) and may coexist with autoimmune thrombocytopaenia.[5] In such cases, patients require urgent initiation of KRT, but their bleeding risk is heightened because of ongoing systemic inflammation and coagulopathy.[6] Likewise, thrombotic microangiopathies such as thrombotic thrombocytopaenic purpura (TTP) and haemolytic uraemic syndrome (HUS) represent classic renothrombocytopaenic syndrome. While plasma exchange remains the cornerstone of treatment in TTP, dialysis is required in severe cases of HUS and atypical HUS.[7] Infections like dengue, malaria, and leptospirosis also present with renothrombocytopaenic syndrome, especially in tropical regions.[8] Chronic liver disease, with its associated hypersplenism and coagulopathy, is another common setting where both kidney dysfunction and thrombocytopaenia occur.[9,10]

There are many challenges in providing dialysis to such patients. Vascular access placement carries a high bleeding risk, with tendency for catastrophic complications like neck haematomas, haemothorax, or retroperitoneal bleeding.[11,12] Standard anticoagulation protocols during haemodialysis (HD) increase the bleeding risk while anticoagulation avoidance causes frequent circuit clotting and therapy interruption, reducing the dialysis adequacy.[13,14] Peritoneal dialysis (PD) may circumvent some of these risks but requires institutional availability and professional expertise in terms of catheter placement and PD nurses.[15] A systematic, structured approach is necessary to address the challenges for KRT in renothrombocytopaenic syndromes.

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Table: Dialysis Toolkit for Patients with Severe Thrombocytopaenia.

Domain	Strategies	Rationale/Considerations
Vascular Access	- Prefer temporary central venous catheter in acute settings (Femoral vein > Internal jugular vein) - Avoid subclavian vein - Use real-time ultrasound guidance to reduce trauma	High bleeding risk makes AVF/AVG creation unsafe in acute settings Minimize attempts and optimize site selection
Anticoagulation Strategy	- Avoid systemic anticoagulation where possible - Regional citrate anticoagulation (RCA) preferred in CKRT if available - Use heparin-free dialysis with saline flushes in intermittent HD - Consider nafamostat if available	Reduces bleeding risk while maintaining filter patency Choices depends on institutional availability
Dialysis Modality	- CKRT or SLED in haemodynamically unstable patients - Intermittent HD feasible with careful anticoagulation management - PD as alternative if peritoneal access and setting permit	Continuous therapies provide haemodynamic stability and more controlled anticoagulation PD avoids extracorporeal circuit but may not be feasible in emergencies
Platelet Support	- Maintain platelet count >20,000/µL for standard dialysis - Target >50,000/µL if invasive procedures anticipated - Use transfusions judiciously to avoid alloimmunization in patients awaiting transplantation	Platelet thresholds reduce spontaneous and procedure-related bleeding risk Transfusion strategies must balance benefit vs risks
Circuit and Filter Management	- High-flux membranes to reduce clotting risk - Frequent saline flushes during intermittent HD - Monitor circuit pressures closely for early clotting	Technical adjustments prolong circuit life without systemic anticoagulation
Adjunctive Measures	- Correct underlying coagulopathy (Multidisciplinary management) - Maintain optimal calcium levels in citrate anticoagulation - Use desmopressin in uremic platelet dysfunction	Addresses multifactorial contributors to bleeding risk in patients with uraemia and thrombocytopaenia
Monitoring & Safety	- Close monitoring of access site bleeding post-dialysis - Hourly checks of circuit function and patient vitals - Review platelet trends, coagulation profile	Early detection of bleeding or clotting complications improves patient safety and improve dialysis adequacy

Challenges

Various challenges in dialyzing patients with renothrombocytopenic syndromes are depicted in Table. Central venous catheter insertion and systemic anticoagulation for circuit patency stays the limiting factor in such cases.[11-14] Even with ultrasound guidance, platelet transfusion support and meticulous technique are required to minimize procedural risks while ensuring adequate dialysis delivery.[11,12] Balancing adequate circuit patency with patient safety is difficult. Heparin-free dialysis or regional citrate anticoagulation can be considered, though both approaches bring practical and logistical limitations in resource-limited settings.[13,14] Choosing between intermittent HD, sustained low-efficiency dialysis (SLED), or continuous kidney replacement therapy (CKRT) needs an individualized approach[16,17] Intermittent HD increases procedural risks because of higher anticoagulant dose and rapid hemodynamic shifts. CKRT requires prolonged vascular access patency and consistent anticoagulation, often impractical in patients with renothrombocytopenic syndrome.[17]

Many patients require prophylactic or therapeutic platelet transfusions to safely undergo dialysis procedure.[11,12] Repeated transfusions are not always feasible because of resource limitations and alloimmunization risk.[18] Use of central venous catheters, frequent transfusions, and compromised immunity increase susceptibility to infections.[16,17] Catheter-related bloodstream infections worsen both morbidity and mortality in these patients.[19] Patients with renothrombocytopenic syndrome often have underlying comorbidity or multi-organ dysfunction, which make them prone to haemodynamic instability during dialysis.[16] Tailoring ultrafiltration goals, using slower modalities, and providing haemodynamic support are essential in such cases.

Dialysis Toolkit for Severe Thrombocytopenia

We propose a dialysis toolkit for patients with renothrombocytopenic syndrome. The key dilemma lies in balancing dialysis adequacy with minimizing vascular and extracorporeal circuit-related trauma. Temporary femoral access may reduce bleeding complications but increase the infection risk if prolonged.[20] Thus, individualized vascular access decisions remain central. Moreover, anticoagulation strategies must be carefully adapted- ranging from heparin-free dialysis, saline flushes, and regional citrate anticoagulation, to low-dose heparin protocols tailored by close monitoring of bleeding and clotting tendencies. The dialysis toolkit presented in Table 1 covers various strategies for vascular access, anticoagulation, dialysis modality, transfusion thresholds, and peri-procedural care. Importantly, it emphasizes a multidisciplinary approach involving nephrologists, haematologists, intensivists, and nursing staff to individualize therapy.

Conclusion

Severe thrombocytopenia poses a formidable challenge in the management of AKI, where timely initiation of KRT may be life-saving. The dialysis toolkit for severe

thrombocytopaenia offers a structured framework encompassing patient selection, vascular access modifications, anticoagulation strategies, membrane and modality choice, and supportive transfusion practices. By systematically addressing these dimensions, it minimizes bleeding complications while ensuring adequacy of solute clearance and volume management. Future directions must emphasize evidence generation through collaborative registries and randomised controlled trials.

References

1. Gernsheimer T, James AH, Stasi R. How I treat thrombocytopenia in pregnancy. Blood. 2013;121:38–47.
2. Stasi R. How to approach thrombocytopenia. Hematology Am Soc Hematol Educ Program. 2012;2012(1):191–197.
3. Shahrabi S, Behzad MM, Jaseb K, Saki N. Thrombocytopenia in leukemia: Pathogenesis and prognosis. Histol Histopathol. 2018;33:895-908.
4. Gonzalez DA, Kumar R, Asif S, Bali A, Dang AK. Sepsis and Thrombocytopenia: A Nowadays Problem. Cureus. 2022;14: e25421.
5. Pamuk ON. Thrombocytopenia in Patients with Systemic Lupus Erythematosus. Eur J Rheumatol. 2023;10:159-162.
6. Favaloro EJ, Pasalic L, Lippi G. Autoimmune Diseases Affecting Hemostasis: A Narrative Review. Int J Mol Sci. 2022;23:14715.
7. Winters JL. Plasma exchange in thrombotic microangiopathies (TMAs) other than thrombotic thrombocytopenic purpura (TTP). Hematology Am Soc Hematol Educ Program. 2017;2017:632-638.
8. Jha VK, Mahapatra D, Subbiah A. Tropical Infections and Acute Kidney Injury. J Assoc Physicians India. 2025;73(1):35-40.
9. Peck-Radosavljevic M. Thrombocytopenia in chronic liver disease. Liver Int. 2017;37:778-793.
10. Chancharoenthana W, Leelahavanichkul A. Acute kidney injury spectrum in patients with chronic liver disease: Where do we stand? World J Gastroenterol. 2019;25:3684-3703.
11. van Baarle FLF, van de Weerdt EK, van der Velden WJFM, Ruiterkamp RA, Tuinman PR, Ypma PF, et al. Platelet Transfusion before CVC Placement in Patients with Thrombocytopenia. N Engl J Med. 2023;388:1956-1965.
12. Zeidler K, Arn K, Senn O, Schanz U, Stussi G. Optimal preprocedural platelet transfusion threshold for central venous catheter insertions in patients with thrombocytopenia. Transfusion. 2011;51:2269-76.
13. Singh S. Anticoagulation during Renal Replacement Therapy. Indian J Crit Care Med. 2020 ;24(Suppl 3): S112-S116.
14. Gois PHF, McIntyre D, Ratanjee S, Pelecanos A, Scuderi C, Janoschka CL, et al. Hemodialysis without Systemic Anticoagulation: A Randomized Controlled Trial to Evaluate Five Strategies in Patients at a High Risk of Bleeding. Med Sci (Basel). 2024;12:38.
15. Cieszyński K, Grzegorzewska AE. Peritoneal Dialysis in a Patient with Acute Kidney Injury, Thrombocytopenia, Urosepsis, and Liver Cirrhosis: A Case Report. Am J Case Rep. 2020;21: e926226.
16. Griffin BR, Jovanovich A, You Z, Palevsky P, Faubel S, Jalal D. Effects of Baseline Thrombocytopenia and Platelet Decrease Following Renal Replacement Therapy Initiation in Patients with Severe Acute Kidney Injury. Crit Care Med. 2019 ;47: e325-e331.
17. Griffin BR, Wu C, O'Horo JC, Faubel S, Jalal D, Kashani K. The Association of Platelet Decrease Following Continuous Renal Replacement Therapy Initiation and Increased Rates of Secondary Infections. Crit Care Med. 2021;49: e130-e139.
18. Reckhaus J, Jutzi M, Fontana S, Bacher VU, Vogt M, Daslakis M, et al. Platelet Transfusion Induces Alloimmunization to D and Non-D Rhesus Antigens. Transfus Med Hemother. 2018 ;45:167-172.
19. Siempos II, Kopterides P, Tsangaris I, Dimopoulou I, Armaganidis AE. Impact of catheter-related bloodstream infections on the mortality of critically ill patients: a meta-analysis. Crit Care Med. 2009;37:2283-9.
20. Kirkpatrick WG, Culpepper RM, Sirmon MD. Frequency of complications with prolonged femoral vein catheterization for hemodialysis access. Nephron. 1996;73:58-62.