DOI: 10.21088/ijem.2395.311X.12226.1

Indian Journal of Emergency Medicine
Volume 12 Number 2, April–June 2026

CASE REPORT

When Immunity, Coagulopathy, and Kidneys Collide: A Rare Case of Spontaneous SAH

Girish Kulle[1], Anita Rawat[2], Kishalay Datta[3], Goonja Bhattachary[4]

HOW TO CITE THIS ARTICLE:

Girish Kulle, Anita Rawat, Kishalay Datta et. al, When Immunity, Coagulopathy, and Kidneys Collide: A Rare Case of Spontaneous SAH. Ind J Emerg Med. 2026; 12(2): 71-73.

ABSTRACT

Subarachnoid haemorrhage (SAH) is a life-threatening neurological emergency, most often caused by aneurysmal rupture. However, in patients with multiple systemic comorbidities, including autoimmune disease, coagulopathy, and end-stage renal disease, the presentation and outcome may be particularly devastating. We report the case of a 56-year-old male with a background of lupus nephritis, secondary antiphospholipid antibody syndrome, chronic kidney disease on dialysis, and prior cerebrovascular accident who presented with acute headache and subsequently developed spontaneous SAH. Despite timely diagnosis and supportive management, the patient's condition rapidly deteriorated and he succumbed. This case underscores the multifactorial risks of intracranial haemorrhage in complex autoimmune and renal disease patients, and emphasizes the need for vigilant monitoring and individualized risk-benefit decisions regarding antithrombotic therapy.

KEYWORDS

• Sub-arachnoid haemorrhage • APLA • CKD • Coagulopath

INTRODUCTION

Spontaneous subarachnoid haemorrhage accounts for approximately 5–10% of all strokes, most commonly due to rupture of intracranial aneurysms. Secondary causes, including vascular malformations, coagulopathies, and systemic disease, are less frequently described but often associated with poor outcomes. Autoimmune disorders such as systemic lupus erythematosus (SLE) and antiphospholipid

AUTHOR'S AFFILIATION:

[1] Resident, Department of Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, India.
[2] Principal Consultant, Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, India.
[3] HOD and Director, Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, India.
[4] Resident, Department of Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, India.

CORRESPONDING AUTHOR:

Girish Kulle, Resident, Department of Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, India.

E-mail: gkulle108@gmail.com

➢ **Received :** 04-02-2026 ➢ **Accepted :** 05-03-2026

antibody syndrome (APLA) increase the risk of both thrombosis and bleeding. The presence of chronic kidney disease (CKD) further amplifies vascular fragility and complicates the use of anticoagulation or antiplatelet therapy. Here, we describe a complex case of SAH in a patient with lupus nephritis, secondary APLA, and dialysis-dependent CKD, highlighting the diagnostic and therapeutic challenges encountered in the emergency setting.

CASE

A 56-year-old male presented to the emergency department with sudden severe headache, nausea, and altered sensorium. His medical history included lupus nephritis with secondary antiphospholipid antibody syndrome, chronic kidney disease on maintenance haemodialysis, and a cerebrovascular accident (ischemic) five years earlier, for which he was on long-term antiplatelet therapy.

On examination, the patient was drowsy but arousable (GCS 13/15). He had neck stiffness but no focal neurological deficits initially. Vitals revealed mild hypertension and tachycardia. Laboratory evaluation showed thrombocytopenia, elevated INR, and prolonged aPTT, suggestive of coagulopathy, likely due to antiplatelet therapy compounded by CKD-related platelet dysfunction.

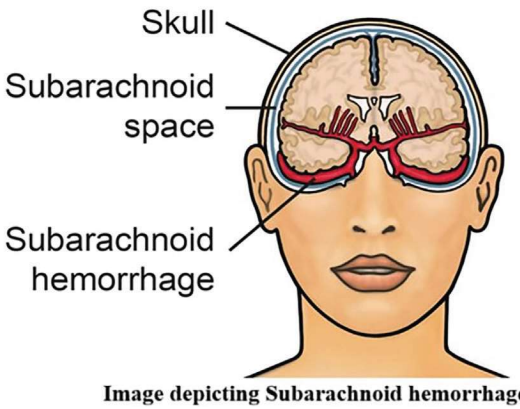

Figure 1: Depicting Subarachnoid hemorrhage (***Image Source:*** Google Search Engine)

A non-contrast CT brain revealed diffuse subarachnoid haemorrhage in the basal cisterns and sylvian fissures. Coagulopathy was promptly addressed with platelet transfusion, fresh frozen plasma, and vitamin K. Given worsening renal parameters and metabolic acidosis, continuous renal replacement therapy (CRRT) was initiated.

Over the next 48 hours, his neurological status declined with progressive fall in GCS, anisocoria, and ventilatory dependence. Neurosurgical consultation advised conservative management due to poor neurological reserve and multisystem disease. Despite intensive care with ventilatory support, hemodynamic stabilization, CRRT, and correction of coagulation parameters, his condition failed to improve. The patient succumbed on the fifth day of admission.

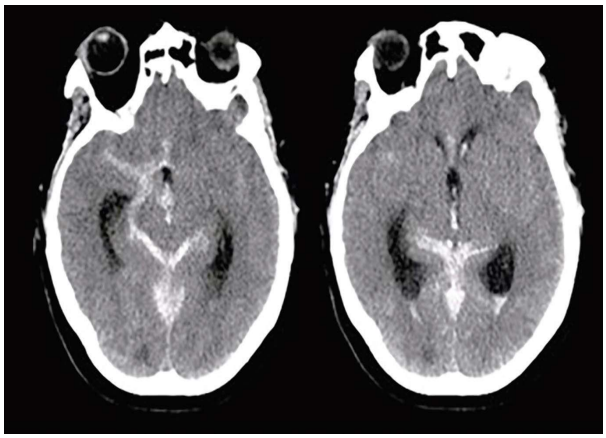

Figure 2: CT head of the patient depicting massive SAH (***Source:*** Department of Emergency Medicine and Department of Rdiology, Max Super Speciality Hospital, Shalimar Bagh, New Delhi)

DISCUSSION

This case illustrates the complex interplay of autoimmune disease, renal dysfunction, and antithrombotic therapy in predisposing to catastrophic intracranial bleeding. While SLE and APLA are classically associated with thromboembolic complications, vascular endothelial damage and long-term antithrombotic use increase bleeding risk. CKD adds further haemostatic derangements, including platelet dysfunction, impaired fibrinogen activity, and hypertension-related vessel fragility.

Several studies have documented increased risk of intracranial haemorrhage in lupus patients, especially those with concurrent APLA or on antiplatelets/anticoagulants.[1,2] Dialysis further raises the risk of haemorrhage, both due to uremic platelet dysfunction and exposure to anticoagulation during dialysis sessions.[3,4] The coexistence of these risk factors in our patient created a perfect storm for fatal SAH.

The management of such patients is particularly challenging:

- **Airway and hemodynamic stabilization** remain the first priorities.
- **Prompt reversal of coagulopathy** is essential.
- **CRRT** allows hemodynamic stability while correcting metabolic derangements.
- **Multidisciplinary approach** (emergency, nephrology, neurology/neurosurgery, critical care) is vital.

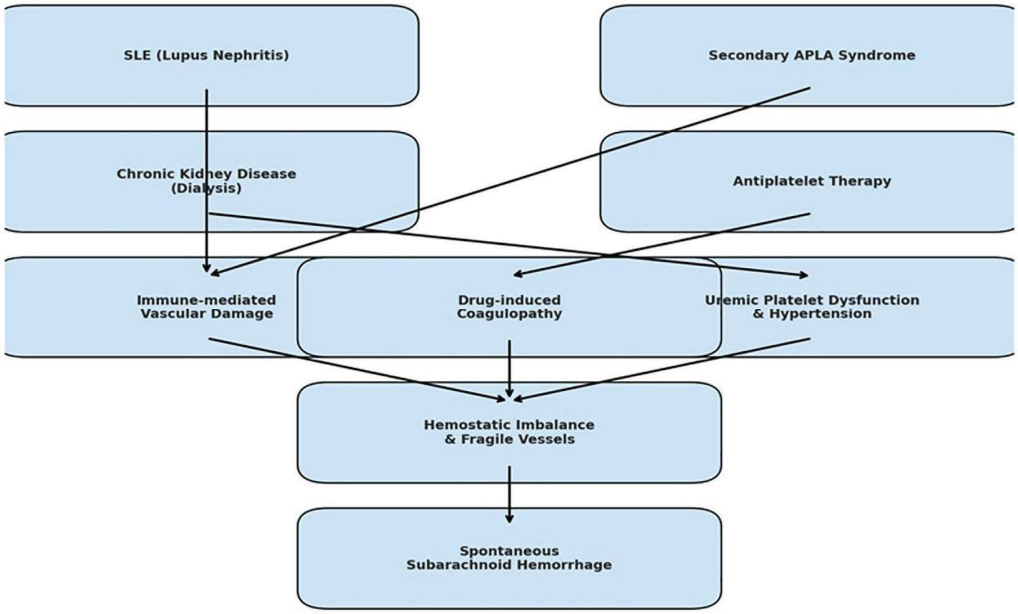

Figure 3: Possible Pathophysiology of SAH with underlying Lupus Nephritis, APLA, CKD

Unfortunately, despite aggressive management, prognosis remains poor when multiple systemic comorbidities coexist. This case adds to limited literature describing fatal SAH in lupus nephritis with APLA and CKD, underscoring the importance of individualized anticoagulant/antiplatelet decisions.

CONCLUSION

Spontaneous SAH in patients with SLE, APLA, and CKD represents a rare but devastating event. Emergency physicians must maintain high suspicion in patients presenting with acute headache and neurological decline. Early imaging, prompt reversal of coagulopathy, and initiation of renal support can stabilize patients, but outcomes are often unfavourable. This case highlights the need for careful balancing of thrombosis and bleeding risks in multimorbid patients and the role of vigilant follow-up to anticipate catastrophic complications.

REFERENCES

1. Kamel H., Navi B.B., Sriram N., Hovsepian D.A., Devereux R.B., Elkind M.S. Risk of a thrombotic event after the 6-week postpartum period. *N Engl J Med*. 2014; 370(14): 1307–1315.
2. Sultan S.M., Begum S., Isenberg D.A. Prevalence, patterns of disease and outcome in patients with systemic lupus erythematosus and intracranial haemorrhage. *Ann Rheum Dis*. 2003; 62(4): 362–365.
3. Toyoda K., Fujii K., Fujimi S., Kumai Y., Tsuchimochi H., Ibayashi S., Iida M. Stroke in patients on maintenance hemodialysis: A 22-year single-center study. *Am J Kidney Dis*. 2005; 45(6): 1058–1066.
4. Sood M.M., Larkina M., Thumma J.R., Tentori F., Bieber B.A., Gillespie B.W., *et al*. Major bleeding events and risk stratification of antithrombotic agents in hemodialysis: Results from the DOPPS. *Kidney Int*. 2013; 84(3): 600–608.
5. Razvi S.S., Weaver J.U., Markey J., Grundy D., Hall R. Subarachnoid haemorrhage in systemic lupus erythematosus: Case Report and literature review. *Clin Neurol Neurosurg*. 1995; 97(3): 235–238.
6. Baizabal-Carvallo J.F., Barragán-Campos H.M., Padilla-Aranda H., Alonso-Juárez M., Estanol B., Cantu C. Intracerebral hemorrhage in patients with systemic lupus erythematosus. *Eur J Neurol*. 2007; 14(4): 373–379.