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Case Report

Early onset imminent eclampsia with antiphospholipid antibody syndrome and recurrent epilepsy with HELLP syndrome

Ruby Bhatia, Yeso Ashwini*, Mahak Singaal

Department of Gyneacology-Obstetrics, MMIMSR, Mullana, Ambala, Haryana

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***Correspondence:**
Dr. Yeso Ashwini,
E-mail: ashwininagaraj125@gmail.com

ABSTRACT

Early-onset pre-eclampsia and eclampsia superimposed on antiphospholipid antibody syndrome (APLA) and a pre-existing seizure disorder represent one of the most challenging constellations in contemporary obstetric practice. The convergence of these conditions demands coordinated multidisciplinary expertise and carries very high maternal and foetal morbidity and mortality with known case of epilepsy on treatment.We report a 28-year-old woman (G6P1L1A4) at 18–23 weeks of gestation who presented across three sequential hospital admissions between 18-23 weeks with recurrent eclamptic seizures, severe hypertension (180/100 mmHg), moderate-to-severe anaemia, bilateral uterine artery diastolic notching indicating uteroplacental insufficiency, and a prior caesarean scar. Despite optimisation of antihypertensive and antiepileptic therapy including intravenous levetiracetam loading, eclampsia remained refractory. Multidisciplinary evaluation encompassing obstetrics, neurology, nephrology, haematology, psychiatry, ophthalmology, and neonatology ultimately concluded that continuation of the pregnancy posed imminent and extreme risk to maternal life. Medical termination of pregnancy (MTP) was performed at 23 weeks.This case underscores the catastrophic potential of untreated APLA syndrome in a patient with recurrent pregnancy loss, the diagnostic challenge of eclampsia superimposed on known epilepsy, and the critical importance of multidisciplinary management, proactive social support, and comprehensive pre-conception counselling in high-risk obstetric populations.

Keywords: Early-onset pre-eclampsia, Eclampsia, Antiphospholipid antibody syndrome, Recurrent pregnancy loss, Seizure disorder, Levetiracetam, Medical termination of pregnancy, Uteroplacental insufficiency, Multidisciplinary obstetric care

INTRODUCTION

Pre-eclampsia and eclampsia remain among the leading causes of maternal mortality globally, accounting for approximately 10-15% of direct maternal deaths,[1] Early-onset pre-eclampsia (before 34 weeks) is pathobiologically distinct, driven by failed trophoblast invasion of spiral arteries, resulting in uteroplacental insufficiency and multi-organ endothelial injury.[2] Concurrent antiphospholipid antibody (APLA) syndrome further amplifies thrombotic and vascular risk , and appropriate anticoagulant prophylaxis has been shown to reduce adverse pregnancy outcomes in affected women.[3,4] Its co-occurrence with a pre-existing seizure disorder creates an exceptionally challenging clinical scenario both in differentiating eclampsia from breakthrough epilepsy and in selecting pharmacotherapy safe for mother and foetus.[5]

We report a 28-year-old G6P1L1A4 woman in whom APLA syndrome had remained unrecognised despite four prior consecutive pregnancy losses. She presented with three escalating hospital admissions over one week, recurrent refractory eclampsia, and ultimately required medical termination of pregnancy (MTP) at 23 weeks.[6] The case highlights critical gaps in thrombophilia screening, the complexity of polypharmacy in pregnancy,

and the systemic factors driving dangerous discharge against medical advice (DAMA) behaviour.[7]

CASE REPORT

A 28-year-old woman (G6P1L1A4, married 8 years) presented to our Obstetrics emergency unit of rural teritiary care hospital. Her last menstrual period was 23 September 2025 (EDD 30 June 2026); gestational age ranged 18–23 weeks across her admissions, concordant with a 15-week scan. Her only surviving child was delivered by lower segment caesarean section (LSCS) eight years prior for non-progress of labour. She had subsequently suffered four consecutive first-trimester abortions (~12 weeks each), all managed by suction and evacuation yet antiphospholipid antibody workup was never performed. She is a case of seizure disorder since 12 years and managed with levetiracetam (Tab Levepsy 500 mg BD). Blood group AB+; TSH 2.15 mIU/ml (normal); HIV, HBsAg, HCV, syphilis all non-reactive.

Table 1: Chronological summary of hospital admissions.

Adm.	Date	IP no.	POG	Presentation	BP	Key Management	Outcome
1st	29 Jan 2026	IP2601290002	18+2 wks	Headache, vomiting, fainting; referred post-MgSO4 loading	150/90	IV LEV 1g load; Enoxaparin; Labetalol; PRBC transfusion	DAMA 31 Jan
2nd	02 Feb 2026	IP2602020178	22+6 wks	Eclamptic seizures ×3 in 30 min	180/100	IV LEV 2g load; antihypertensive optimisation	DAMA 03 Feb
3rd	03 Feb 2026	IP2602030053	22+6 wks	Eclamptic seizures ×3 in 30 min (same day re-admission)	180/100	MDT decision → MTP; Clonazepam added; NCCT head	MTP 03 Feb; Discharged stable 06 Feb

DAMA = Discharged against medical advice; LEV = Levetiracetam; MDT = Multidisciplinary team; MTP = Medical termination of pregnancy; POG = Period of gestation.

Table 2: Serial laboratory investigations.

Parameters	Admission 1	Admission 2	Reference range
Haemoglobin (g/dl)	7.3 → 8.1 (post-PRBC)	10.4	12–15
WBC (×10³/cumm)	9.75	22.35	4–10
Platelet (×10³/cumm)	158	317	150–400
MCV (fL)	72–75.7 ↓	75.2 ↓	83–98
LDH (IU/l)	462 ↑	—	98–192
Bilirubin Total (mg/dl)	1.007 ↑	1.848 ↑	0.2–1.0
Albumin (g/dl)	2.93 ↓	3.53	3.5–5.3
Uric Acid (mg/dl)	6.5 ↑	8.0 ↑	2.0–5.0
Creatinine (mg/dl)	0.60	0.72	0.7–1.2
Urine ACR (mg/g)	1007.3 ↑ (macro)	—	<30
INR	1.07	—	0.9–1.2
TSH (mIU/ml)	2.15	—	0.27–4.20
Lupus anticoagulamt	Positive	—	—
Anti cardiolipin IgG	44 U/ml (>99 PERCENTILE	—	—
Anti cardiolipin IgM	46 U/ml (>99 PERCENTILE	—	—
Anti b2 glycoprotein -1 IgG	18U/ml	—	—
Anti b2 glycoprotein -1 IgG	16U/ml	—	—

ACR = Albumin-creatinine ratio; LDH = Lactate dehydrogenase; MCV = Mean corpuscular volume; ↑ = elevated; ↓ = reduced.

Clinical course across three admissions

Table 1 summarises the chronology. On first admission (29 January 2026), she presented with headache, vomiting, and one episode of fainting; she had been given MgSO4 loading at a district hospital prior to referral. Vitals: BP 150/90 mmHg, PR 86 bpm, SpO2 100%. Pallor was noted; uterine size 20 weeks with caesarean scar. She was managed with intravenous levetiracetam (1 g loading), enoxaparin 0.4 mL SC daily (for APLA positive), Tab Labetalol titrated to 200 mg TDS, Tab Ecospirin 150 mg nocte, and one unit of packed red blood cells for severe anaemia (Hb 7.3 g/dl). Despite high-risk counselling, she discharged herself against medical advice (DAMA) on 31 January 2026.

Only two days later (02 February 2026), she returned in emergency with three generalised tonic-clonic seizures within thirty minutes and BP 180/100 mmHg. Intravenous levetiracetam was escalated to a 2 g loading dose. She

again elected DAMA on 03 February 2026 after documented counselling. On the same day, she was re-admitted with an identical presentation. Given refractory eclampsia unresponsive to maximal therapy, pre-viability (23 weeks), suspected APLA with four prior losses, prior caesarean, severe anaemia, and evidence of uteroplacental insufficiency, a multidisciplinary team (obstetrics, neurology, nephrology, psychiatry, medicine) concluded that continuation of pregnancy posed imminent maternal life-threat. Informed consent was obtained and MTP was performed on 03 February 2026 by medical method; the expelled foetus was male, 300 g. The patient was discharged in stable condition on 06 February 2026 on Tab Labetalol 200 mg TDS, Tab Levepsy 500 mg BD, Tab Telma AM 40/5 mg OD, and Tab Clonazepam 0.25 mg nocte, with follow-up for MRI brain, APLA profile to be repeated after 12 weeks, and psychiatric review.

Investigations

Key laboratory values across both admission episodes are summarised in Table 2. Haemoglobin was 7.3 g/dl at first admission (microcytic hypochromic anaemia; peripheral smear showing tear-drop cells and elliptocytes), recovering to 10.4 g/dl post-transfusion by the second. LDH was markedly elevated at 462 IU/l (reference 98–192), along with elevated bilirubin (1.007 → 1.848 mg/dl) and hypoalbuminaemia (2.93 g/dl) a biochemical pattern consistent with HELLP syndrome. Urine albumin-creatinine ratio (ACR) of 1007.3 mg/g confirmed macroalbuminuria. Uric acid rose from 6.5 to 8.0 mg/dl (normal 2–5), a recognised surrogate of pre-eclampsia severity. Renal function and coagulation (INR 1.07) remained normal. Obstetric ultrasonography (29 January) confirmed gestational age 20+4 weeks, foetal weight 366 g, adequate amniotic fluid, and critically bilateral uterine artery diastolic notching with elevated PI and RI, the Doppler hallmark of uteroplacental insufficiency. Ophthalmology confirmed hypertensive retinopathy without papilloedema.

DISCUSSION

Four consecutive unexplained first-trimester losses fulfil the obstetric clinical criteria for APLA syndrome under the revised Sapporo (Sydney) criteria, as defined by Miyakis et al even without confirmed laboratory seropositivity at the time of this report.[3] APLA-mediated mechanisms placental thrombosis, complement activation, and direct trophoblast dysfunction both cause recurrent miscarriage and predispose to early-onset severe pre-eclampsia, precisely as seen here, as described by Miyakis et al and Mak et al.[3,4] Low-dose aspirin combined with LMWH reduces adverse pregnancy outcomes in confirmed APLA by approximately 50-54%, per Mak et al.[4] Had this workup been performed after the second or third loss, prophylactic therapy may have altered the obstetric trajectory substantially. Clinicians must adopt a zero-tolerance approach to unexplained recurrent pregnancy loss without antiphospholipid antibody screening, in line with Miyakis et al and Mak et al.[3,4]

In a hypertensive pregnant patient with known seizure disorder, differentiation of eclampsia from breakthrough epilepsy is critical yet often not straightforward, as noted by Sibai and Meador et al.[2,5] In this case, the coexistence of severe hypertension, macroalbuminuria Thangaratinam et al.[8] Elevated LDH and uric acid (Sibai²), bilateral uterine artery diastolic notching, and early gestational age unambiguously established eclampsia, necessitating concurrent management of both conditions. Intravenous levetiracetam (LEV) loading at 2 g as an adjunct to magnesium sulphate is supported by emerging evidence in patients with breakthrough seizures or co-existing epilepsy, where MgSO4 alone may be insufficient, as reported by Meador et al.[5] LEV carries a favourable teratogenicity profile compared to valproate and does not induce hepatic enzymes, making it preferable in the pregnant patient Meador et al.[5]

Under the MTP (Amendment) Act, 2021 (India), termination up to 24 weeks is permissible when the pregnancy poses serious risk to maternal life, per the Government of India.[6] The clinical threshold was clearly exceeded here: refractory eclampsia unresponsive to maximum antihypertensive and antiepileptic therapy, early HELLP biochemistry, a pre-viable foetus with uteroplacental insufficiency, and haemodynamic instability Sibai²; Government of India.[6] The multidisciplinary consent process spanning obstetrics, neurology, psychiatry, and medicine ensured that the decision was clinically sound, legally compliant, and compassionately communicated to the patient and her family.

The patient's two DAMA episodes each followed by immediate re-presentation in a worse clinical state reflect well-documented barriers in high-risk obstetric populations: financial constraints, childcare responsibilities, low health literacy, and inadequate perceived severity of illness, as described by Rangel-Frausto.[7] Documentation of informed refusal is a medicolegal necessity but is insufficient to prevent harm. High-risk obstetric units should implement structured DAMA protocols that include social work assessment, community health worker follow-up, and a clearly defined re-admission pathway activated at first DAMA, not as a reactive afterthought, per Rangel-Frausto.[7]

CONCLUSION

This case illustrates the catastrophic convergence of undiagnosed APLA syndrome, early-onset severe pre-eclampsia with eclampsia, co-existing epilepsy, severe anaemia, and repeated DAMA behaviour in a young woman whose pregnancy was deeply desired. Systematic APLA screening after consecutive unexplained pregnancy losses, multidisciplinary management protocols for complex obstetric co-morbidities, and proactive structural

responses of patient and her family to DAMA in high-risk patients are the three core lessons this case demands. Pre-conception counselling, APLA-directed prophylaxis, and neurological optimisation prior to future conception are essential steps for this patient's ongoing care.

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REFERENCES

1. Magee LA, Khalil A, von Dadelszen P. FIGO Initiative on pre-eclampsia: a pragmatic guide for first-trimester screening and prevention. Int J Gynaecol Obstet. 2019;145(Suppl 1):1–33.
2. Sibai BM. Diagnosis, prevention, and management of eclampsia. Obstet Gynecol. 2005;105(2):402-10.
3. Miyakis S, Lockshin MD, Atsumi T. International consensus statement on classification criteria for antiphospholipid syndrome. J Thromb Haemost. 2006;4(2):295-306.
4. Mak A, Cheung MWL, Cheak AAC, Ho RCM. Heparin and aspirin vs. aspirin alone in recurrent pregnancy loss with antiphospholipid antibodies: a meta-analysis. Rheumatology (Oxford). 2010;49(2):281-8.
5. Meador K, Reynolds MW, Crean S. Pregnancy outcomes in women with epilepsy: a systematic review. Epilepsy Res. 2008;81(1):1–13.
6. Government of India. The Medical Termination of Pregnancy (Amendment) Act, 2021. Gazette of India Extraordinary Part II – Section 1. 25 March 2021.
7. Rangel-Frausto MS. Discharge against medical advice: challenges and opportunities. Arch Med Res. 2015;46(4):322-5.
8. Thangaratinam S, Coomarasamy A, O'Mahony F, et al. Proteinuria as a predictor of complications of pre-eclampsia: systematic review. BMC Med. 2009;7:10.

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