Case Report

Autoimmune-associated cerebral venous thrombosis in a young female: A clinical pharmacist's perspective case report

Samyuktha D V[1*], Maruthu pandi[2]

[1]Group Clinical Pharmacist, Kauvery Hospital, Trichy, Tamil Nadu
[2]Clinical Pharmacist- Intern, Kauvery Hospital, Trichy, Tamil Nadu
*Correspondence

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Abstract

Background: Cerebral venous thrombosis (CVT) is an uncommon but important cause of stroke in young adults, particularly women with prothrombotic risk factors. We report a case of a 27-year-old female who presented with acute transient left-sided visual disturbance and headache. Neuroimaging revealed cerebral venous thrombosis involving the right transverse and sigmoid sinuses with associated right occipital venous infarction, explaining her visual field defects. Her clinical course was complicated by hemorrhagic transformation of the venous infarct. A detailed evaluation identified lupus anticoagulant positivity, suggesting an acquired prothrombotic state, along with contributory factors including recent pregnancy loss, short-term estrogen exposure, diabetes mellitus, hypertension, dyslipidemia, and hypothyroidism. Anticoagulation was initiated and later modified due to hemorrhagic infarction, with close neurological and hematological monitoring. The patient showed gradual clinical improvement and was discharged in a stable condition with appropriate long-term management and follow-up. This case highlights the importance of early recognition of CVT, comprehensive thrombophilia workup, and individualized management in young women presenting with atypical neurological symptoms.

Keywords: Cerebral venous thrombosis; Occipital lobe infarction; Visual disturbance; Lupus anticoagulant; Young female; Prothrombotic state

1. Introduction

Cerebral venous thrombosis (CVT) is a relatively uncommon cause of stroke, accounting for approximately 0.5–3% of all stroke cases. The estimated annual incidence is 3–5 cases per 100,000 populations. CVT predominantly affects young adults between 20 and 50 years of age, with a higher prevalence among women of reproductive age, largely due to hormonal factors. CVT results from the occlusion of cerebral veins or dural venous sinuses, leading to impaired venous drainage. This causes venous hypertension, which may progress to cerebral edema, venous infarction, or intracranial hemorrhage. Clinically, CVT presents with a wide range of symptoms. Headache is the most common

manifestation, occurring in 80–90% of patients, followed by seizures in 30–40% of cases. Other presentations include focal neurological deficits, altered sensorium, and papilledema. The pathogenesis of CVT is best explained by Virchow's triad, which includes hypercoagulability, endothelial injury, and venous stasis. Hypercoagulable states may arise from inherited thrombophilias such as factor V Leiden mutation or acquired conditions like antiphospholipid antibody syndrome, including lupus anticoagulant positivity. Hormonal factors, particularly oral contraceptive use, increase the risk by 2–13 fold, while pregnancy and the postpartum period further contribute to thrombotic risk. Endothelial injury may result from infections, trauma, or malignancy, whereas venous stasis can occur in conditions such as dehydration or heart failure.Diagnosis of CVT is primarily established through magnetic resonance imaging (MRI) combined with magnetic resonance venography (MRV), which allows accurate visualization of venous occlusion. Anticoagulation remains the cornerstone of treatment, even in the presence of intracranial hemorrhage, as it prevents thrombus propagation and promotes recanalisation. With early diagnosis and prompt management, 80–90% of patients achieve favourable outcomes, highlighting the importance of timely intervention [1,2].

2. Case Presentation

A 27-year-old female presented to the emergency department on 02 December 2025 with an acute onset of visual disturbance characterized by blurring of vision, predominantly involving the left visual field, which lasted for approximately two hours. This was associated with a moderate-intensity headache. There was no history of trauma, loss of consciousness, seizures, giddiness, vomiting, chest pain, palpitations, or focal limb weakness at the time of presentation. The patient had multiple pre-existing medical comorbidities, including type 2 diabetes mellitus managed with oral antidiabetic agents, hypertension treated with nebivolol, and hypothyroidism on thyroxine replacement therapy. She also had a history of exotropia since childhood.

Her obstetric history was significant for a spontaneous conception approximately two months before presentation. During her first antenatal visit, she was identified to have diabetes mellitus and hypertension. Subsequent ultrasonography revealed the absence of fetal heart activity, following which she underwent dilatation and curettage. Post-procedure, she remained clinically stable. Notably, she had a history of short-term estrogen exposure in the form of combined oral contraceptive pills (Levonorgestrel with Ethinyl estradiol) for two months before conception. She also reported an episode of fever lasting two days following the obstetric event, which was treated symptomatically. Family history was significant for diabetes mellitus in her father. On admission, the patient was conscious, oriented, and hemodynamically stable, with a blood pressure of 130/90 mmHg, heart rate of 83 beats per minute, respiratory rate of 20 breaths per minute, oxygen saturation of 98% on room air, and a Glasgow Coma Scale score of 15/15. Neurological examination revealed no motor or sensory deficits, and she was able to move all four limbs without difficulty.

However, she complained of intermittent left-sided visual field defects suggestive of left hemianopia. No signs of meningeal irritation were present. Initial laboratory investigations showed mild neutrophilic leukocytosis with a total white blood cell count of 13,050/µL and an elevated absolute neutrophil count, while haemoglobin and platelet levels were within normal limits. Given the acute neurological symptoms, the patient was initiated on emergency management, including subcutaneous enoxaparin for anticoagulation, intravenous levetiracetam for seizure prophylaxis, intravenous pantopra-

zole, ondansetron, and acetaminophen for symptomatic relief. Ophthalmological consultation revealed intermittent blurring of vision in the left eye lasting approximately ten minutes per episode. Bedside visual acuity was ≥3/60 in both eyes, pupils were equal and reactive to light, extraocular movements were full, and fundoscopic examination showed no evidence of papilledema or optic disc edema. Based on clinical findings, a provisional diagnosis of visual disturbance secondary to occipital lobe involvement was made. Magnetic resonance imaging (MRI) of the brain revealed acute cerebral venous thrombosis involving the right transverse and sigmoid sinuses.

Additionally, focal T2/FLAIR hyperintensity was noted in the right occipital lobe, consistent with venous infarction and associated vasogenic edema. There was no evidence of diffusion restriction, suggesting the absence of acute arterial ischemia. These findings correlated well with the patient's visual symptoms and supported the diagnosis of cerebral venous thrombosis with occipital lobe involvement.

A detailed thrombophilia workup was undertaken in view of the patient's young age and absence of major provoking factors. Lupus anticoagulant was detected, while anticardiolipin IgM and IgG antibodies and beta-2 glycoprotein IgG antibodies were negative. This suggested an underlying acquired prothrombotic state contributing to the development of cerebral venous thrombosis.

Peripheral smear examination revealed mild neutrophilic leukocytosis with toxic granules, consistent with an inflammatory response. During the hospital course, the patient experienced intermittent headaches, though her visual symptoms gradually improved. Thyroid function tests showed suboptimal control of hypothyroidism, and the dose of thyroxine was increased accordingly. Inflammatory markers, including C-reactive protein, were elevated.

Lipid profile demonstrated dyslipidemia, with elevated total cholesterol, triglycerides, LDL, and reduced HDL levels, prompting initiation of rosuvastatin therapy. On 07 December 2025, the patient reported worsening intermittent headaches, raising concern for raised intracranial pressure. She was started on intravenous mannitol. A subsequent CT scan of the brain performed on 08 December 2025 revealed hemorrhagic transformation of the right occipital venous infarct along with multiple calcified granulomas. In view of the hemorrhagic infarction, anticoagulation with enoxaparin was discontinued, and the patient was closely monitored. Following modification of therapy, the patient's headache significantly improved, and no further visual disturbances were reported. Neurological status remained stable, with no development of new focal deficits. Serial monitoring of coagulation parameters showed elevated INR values, necessitating careful adjustment of therapy. By 10 December 2025, the patient was conscious, oriented, afebrile, and obeying commands, with stable vital signs and satisfactory glycemic control. Genetic testing performed during hospitalization revealed an LMNB2 variant of uncertain clinical significance, which required correlation with clinical findings.

The patient remained neurologically stable, with no complaints of headache, giddiness, vomiting, or new focal deficits. Glycemic control was satisfactory, and coagulation parameters were closely monitored (Details of pharmacological management during hospitalization are provided in Table 1). On 12 December 2025, the patient was discharged in a stable condition with antiepileptic therapy, optimized antidiabetic and antihypertensive medications, thyroid hormone supplementation, statin therapy, and anticoagulation planning under close follow-up. She was advised to undergo regular neurological and hematological follow-up to monitor for recurrence and long-term complications.

Table 1: Medications administered during the hospital stay (3/12–11/12)

S. No	Drug	Dose	Freq	Route	3/12	4/12	5/12	6/12	7/12	8/12	9/12	10/12	11/12
1	Inj. Enoxaparin	40 mg	BD	SC	✓	↑60 mg	✓	✓	Stopped	×	×	×	×
2	Inj. Levetiracetam	500 mg	TID	IV	✓	✓	✓	✓	✓	✓	✓	✓	✓
3	Inj./Tab. Pantoprazole	40 mg	BID	IV/PO	✓	Changed to Tab	✓	✓	✓	✓	✓	✓	✓
4	Inj. Ondansetron	4 mg	TID/SOS/ BD	IV	✓	SOS	BD	✓	Stopped	×	×	×	×
5	Tab. Acetaminophen	650 mg	TID	PO	✓	✓	✓	✓	✓	✓	✓	✓	✓
6	Inj. Heparin	1 cc	OD	IV	✓	✓	✓	W/H	×	×	×	×	×
7	Tab. Thyroxine	75 → 88 mcg	OD	PO	✓	✓	88 mcg	✓	✓	✓	✓	✓	✓
8	Tab. Nebivolol	5 mg	OD	PO	✓	✓	✓	✓	✓	✓	✓	✓	✓
9	Tab. Clonazepam	0.25 mg	HS	PO	✓	✓	✓	✓	✓	✓	✓	✓	✓
10	Tab. Sitagliptin + Metformin	50/500 mg	BID	PO	—	✓	✓	✓	✓	✓	✓	✓	✓
11	Tab. Rosuvastatin	10 mg	HS	PO	—	—	✓	✓	✓	✓	✓	✓	✓
12	Tab. Dapagliflozin	5 mg	OD	PO	—	—	✓	✓	✓	✓	✓	✓	✓
13	Tab. Nicoumalone	2 mg	HS	PO	—	—	✓	✓	✓	✓	✓	✓	✓
14	Tab. Livogen	—	OD	PO	—	—	✓	✓	✓	✓	✓	✓	✓
15	Inj. Mannitol	100 mL	BID/TID	IV	—	—	—	—	✓	✓	✓	✓	✓

✓ – Drug administered; X – Not administered; ↑ – Dose escalation; W/H – Withheld; BD – Twice daily; TID – Three times daily; OD – Once daily; HS – At bedtime; IV – Intravenous; PO – Oral; SC – Subcutaneous.

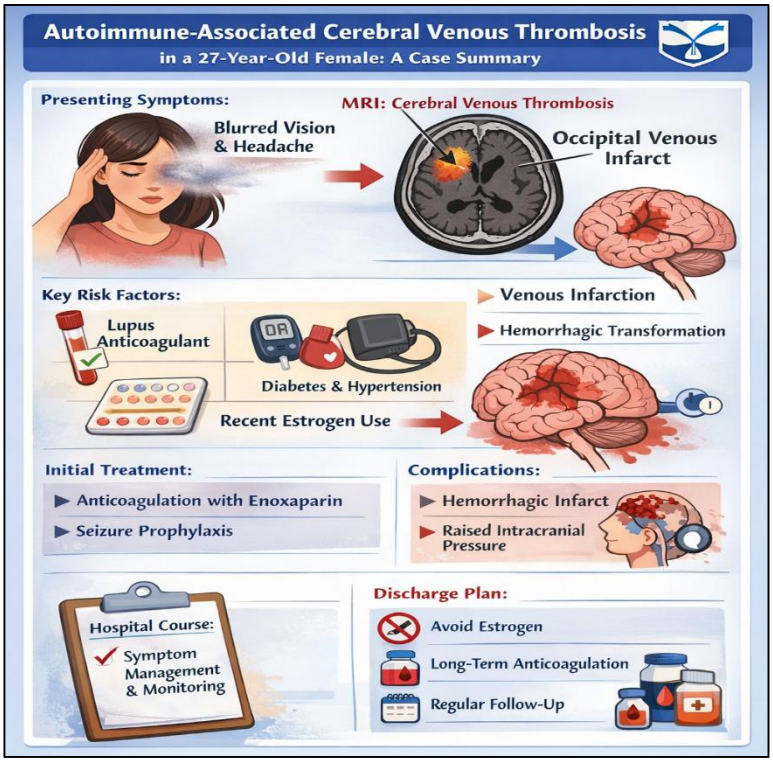

Fig (1): Case Overview: Cerebral Venous Thrombosis in a Young Female – Key Highlights – AI Illustrated image*

3. Discussion

The cerebral venous thrombosis (CVT) in this patient, affecting the right transverse and sigmoid sinuses, developed due to the combined effect of several risk factors acting together. This can be explained using Virchow's triad, which describes three conditions that promote clot formation: increased tendency of the blood to clot, damage to blood vessel walls, and slowing of blood flow [3].

One of the important contributing factors in this patient was her recent use of combined oral contraceptive pills containing estrogen. Even short-term use of estrogen-containing contraceptives is known to increase the risk of blood clot formation by altering the balance of clotting and anti-clotting factors in the blood. This likely played a triggering role in the development of CVT in this young woman [4].

Her underlying medical conditions further increased the risk. Type 2 diabetes mellitus can damage the inner lining of blood vessels over time and promote inflammation, making blood more prone to clotting. Hypertension adds additional stress to blood vessel walls and can interfere with normal blood flow, particularly when present along with diabetes. Poorly controlled hypothyroidism may also contribute indirectly by increasing cholesterol levels and inflammatory markers, thereby worsening blood vessel function. Improvement in thyroid control during hospitalization likely helped reduce this risk [3,4].

The patient's history of exotropia since childhood was considered an incidental finding and was not responsible for the thrombotic event. Normal ophthalmological examination supported a brain-related cause for her visual symptoms. The visual disturbance

was explained by involvement of the right occipital lobe, which controls vision on the left side, resulting in left-sided visual field defects [5].

The presence of lupus anticoagulant indicated an acquired tendency for blood clot formation. However, this alone does not confirm antiphospholipid syndrome, which requires repeat testing after 12 weeks. This finding highlights the importance of long-term follow-up to guide future anticoagulation decisions and assess the risk of recurrence [6].The patient's management followed standard treatment guidelines for CVT. Anticoagulation was initiated early to prevent clot extension, and seizure prophylaxis and supportive care were provided.

When hemorrhagic transformation of the venous infarct was detected, anticoagulation was temporarily withheld, and treatment was adjusted to control raised intracranial pressure. The patient demonstrated a good clinical recovery with this individualized approach. Long-term prevention focuses on avoiding estrogen-containing contraceptives, maintaining good control of diabetes, blood pressure, and thyroid function, and ensuring regular neurological and hematological follow-up [7].This case emphasizes the importance of early recognition of cerebral venous thrombosis in young women presenting with atypical neurological symptoms, comprehensive evaluation of reversible and acquired prothrombotic factors, and individualized therapeutic decision-making, particularly in the setting of hemorrhagic complications.

4. Conclusion

Cerebral venous thrombosis should be considered in young women presenting with acute-onset headache or transient visual disturbances, even in the absence of focal neurological deficits. This case underscores the contribution of short-term estrogen exposure, metabolic comorbidities, and lupus anticoagulant positivity to the development of CVT. The detection of lupus anticoagulant suggests an underlying autoimmune-mediated hypercoagulable state and highlights the importance of long-term surveillance for antiphospholipid antibody–related disorders and the possible evolution of systemic lupus erythematosus (SLE). Early diagnosis using MRI with magnetic resonance venography and individualized anticoagulation strategies, particularly in the setting of hemorrhagic transformation, resulted in a favourable clinical outcome. Preventive measures, including avoidance of estrogen-containing contraceptives, strict control of comorbid conditions, and regular neurological, hematological, and rheumatological follow-up, are essential to reduce recurrence risk and enable early detection of autoimmune disease progression.

References

[1] Payne AB, Adamski A, Abe K, Reyes NL, Richardson LC, Hooper WC, Schieve LA. Epidemiology of cerebral venous sinus thrombosis and cerebral venous sinus thrombosis with thrombocytopenia in the United States, 2018 and 2019. Research and practice in thrombosis and haemostasis. 2022 Feb 1;6(2):e12682.

[2] Kalita J, Singh VK, Jain N, Misra UK, Kumar S. Cerebral venous sinus thrombosis score and its correlation with clinical and MRI findings. Journal of Stroke and Cerebrovascular Diseases. 2019 Nov 1;28(11):104324.

[3] Amri SF, Alsudairy NM, Kadhem F. Oral Contraceptive-Induced Cerebral Venous Thrombosis: A Case report. Cureus. 2025 Feb 3;17(2).

[4] Xu F, Liu C, Huang X. Oral contraceptives caused venous sinus thrombosis complicated with cerebral artery infarction and secondary epileptic seizures: a case report and literature review. Medicine. 2017 Dec 1;96(51):e9383.

[5] Aaron S, Arthur A, Prabakhar AT, Mannam P, Shyamkumar NK, Mani S, Mathew V, Peter J, Sivadasan A, Alexander A, Karthik M. Spectrum of visual impairment in cerebral venous thrombosis: importance of tailoring therapies based on pathophysiology. Annals of Indian Academy of Neurology. 2017 Jul 1;20(3):294-301.

[6] Amoozegar F, Ronksley PE, Sauve R, Menon BK. Hormonal contraceptives and cerebral venous thrombosis risk: a systematic review and meta-analysis. Frontiers in neurology. 2015 Feb 2;6:7.

[7] Moeindarbari S, Beheshtian N, Hashemi S. Cerebral vein thrombosis in a woman using oral contraceptive pills for a short period of time: a case report. Journal of Medical Case Reports. 2022 Jul 4;16(1):260.