



BMH Med. J. 2026;13(4):92-93. **Case Report**

Acute Cerebral Venous Thrombosis of the Vein of Trolard with Parietal Haemorrhage and Protein S Deficiency Following Viral Meningoencephalitis: A Case Report

Shamla O, Ummer K

Department of Neurology, Baby Memorial Hospital, Calicut, Kerala, India

Address for Correspondence: Dr. Ummer K, Chief & Senior Consultant, Department of Neurology, Baby Memorial Hospital, Calicut, Kerala, India. Email: ummerneuro1@gmail.com

Abstract

Cerebral venous thrombosis (CVT) involving cortical veins, such as the vein of Trolard, is a rare and diagnostically challenging entity. We report a young man who developed a first generalized seizure after two weeks of headache and behavioural change, occurring two weeks after hospitalisation for viral meningoencephalitis. MR venography confirmed acute thrombosis of the left vein of Trolard with an associated left parietal haematoma; thrombophilia screening revealed reduced Protein S activity (56%). He improved with anticoagulation and antiepileptic therapy despite haemorrhagic transformation, and was discharged with a plan to repeat thrombophilia testing after three months. This case highlights the role of MR venography and targeted thrombophilia screening in young patients with unexplained CVT, particularly after recent CNS infection.

Keywords: Cerebral venous thrombosis; vein of Trolard; Protein S deficiency; parietal haemorrhage; meningoencephalitis

Introduction

Cerebral venous thrombosis (CVT) accounts for 0.5-1% of strokes and predominantly affects young adults. Thrombosis of the vein of Trolard, a cortical vein, is a rare and diagnostically challenging subtype that may present with focal haemorrhage and seizures. Protein S deficiency is a recognised predisposing thrombophilia, and recent central nervous system infection is an established acquired prothrombotic trigger. We report a young man with cortical venous thrombosis and parietal haemorrhage following viral meningoencephalitis, in the setting of Protein S deficiency.

Case Report

A young previously healthy male presented with a first generalised tonic seizure following two weeks of progressive headache and behavioural change. He had been hospitalised two weeks earlier for viral meningoencephalitis (CSF protein 43.8 g/L, white cell count 288/ μ L). On examination he was irritable with tachycardia (113 bpm) and hypertension (160/90 mmHg); tongue-bite marks and dysarthria were noted, with no focal weakness. MRI brain showed a left parietal intraparenchymal haematoma; MR angiography was normal. MR venography confirmed acute thrombosis of the left vein of Trolard. EEG was normal. Rheumatoid factor was elevated (235) with negative ANA and

ANCA. Thrombophilia screening revealed reduced Protein S activity (56%; reference >70%), with normal serum homocysteine level. He was treated with therapeutic anticoagulation despite haemorrhagic transformation, plus antiepileptic therapy, with good clinical improvement, and was discharged with instructions to repeat Protein S testing after three months to exclude an acute-phase reduction which was also reduced (50%).

Discussion

Isolated cortical vein thrombosis is easily missed on standard imaging without dedicated venographic sequences and should be considered in young patients with new headache, behavioural change, and seizures, particularly after recent CNS infection, a recognised prothrombotic trigger via endothelial activation and cytokine-mediated coagulation. Protein S levels can be transiently suppressed during acute inflammatory illness, so confirmatory retesting after recovery is essential before diagnosing hereditary deficiency. Anticoagulation remains indicated in CVT even with haemorrhagic transformation, given the net benefit of recanalisation over thrombus propagation. This case underscores the value of MR venography and a targeted thrombophilia workup in young patients with otherwise unexplained CVT.

References

1. Ferro JM, Canhao P, Stam J, Boussier MG, Barinagarrementeria F; ISCVT Investigators. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). *Stroke*. 2004;35(3):664-670.
2. Coutinho JM, Ferro JM, Canhao P, et al. Cerebral venous and sinus thrombosis in women. *Stroke*. 2009;40(7):2356-2361.
3. Saposnik G, Barinagarrementeria F, Brown RD Jr, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2011;42(4):1158-1192.
4. Martinelli I, Sacchi E, Landi G, Taioli E, Duca F, Mannucci PM. High risk of cerebral-vein thrombosis in carriers of a prothrombin-gene mutation and in users of oral contraceptives. *N Engl J Med*. 1998;338(25):1793-1797.
5. Dlamini N, Billinghamurst L, Kirkham FJ. Cerebral venous sinus (sinovenous) thrombosis in children. *Neurosurg Clin N Am*. 2010;21(3):511-527.