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Case Report

Deep Cerebral Venous Thrombosis Mimicking Bilateral Thalamic Pathology: Diagnostic Insights from a Case Report

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Abstract: Deep cerebral venous thrombosis (CVT) is a rare cause of stroke causing a diagnostic dilemma on imaging. Its presentation can be variable and is often radiologically characterized by bilateral gangliothalamic involvement, which can mimic other pathologies. We present a case of a 15-year-old male who developed deep cerebral venous thrombosis, manifesting as a bithalamic hemorrhagic lesion on imaging. This case underscores the importance of considering venous thrombosis in the differential diagnosis of bilateral thalamic abnormalities, such as acute viral hemorrhagic fever, especially in young patients presenting with atypical stroke symptoms.

Keywords: Deep Cerebral Venous Thrombosis, Bithalamic Lesion, Stroke, Neuroimaging.

INTRODUCTION

Stroke remains a prevalent neurological emergency, with 80% attributed to ischemic causes and the remainder to hemorrhagic ones. Cerebral venous thrombosis is characterized by thrombosis within the superficial and deep dural venous sinuses or deep intracranial veins. Cerebral venous thrombosis (CVT) accounts for only 1–2% of all strokes, with thrombosis of the deep cerebral venous system, including the internal cerebral veins and straight sinus, comprising around 10.9% of all CVT cases [1]. Among young individuals, CVT contributes to approximately 5% of all stroke cases [2, 3]. The clinical manifestations of CVT are often nonspecific and may include headache, focal neurological deficits, seizures, or altered levels of consciousness [4]. Imaging in deep CVT can mimic other pathologies such as acute hemorrhagic necrotising encephalitis, Artery of Percheron infarct & bithalamic glioma. This variability can complicate imaging diagnosis, frequently leading to delays or missed cases. However, certain imaging features can be used to differentiate between them. Here we present a case of deep cerebral venous thrombosis & thalamic hemorrhages, with a close imaging differential kept as acute viral hemorrhagic encephalitis.

Early detection is crucial, as timely initiation of anticoagulation therapy can reverse the disease process and significantly lower the risk of acute complications and long-term neurological sequelae [5].

CASE PRESENTATION

A 15-year-old male presented to the emergency with an acute onset of multiple episodes of abnormal body movements and decorticate posturing for the past 24 hours, with loss of consciousness. The patient had a history of high-grade fever for the past 5-6 days. Headache, altered sensorium, and bilateral lower limb weakness were also observed a day before the patient's presentation to the emergency room. Routine blood investigation including Platelet counts were normal. Initial Non-Contrast CT of the head showed ill-defined hypodensities involving bilateral thalami and gangliocapsular regions, with thin linear hyperdensities around the thalami (Figure 1a). On sagittal MIP NCCT images (Figures 1b), the septal veins, thalamostriate veins, internal cerebral veins and the straight sinus appear hyperdense suggestive of thrombosis.

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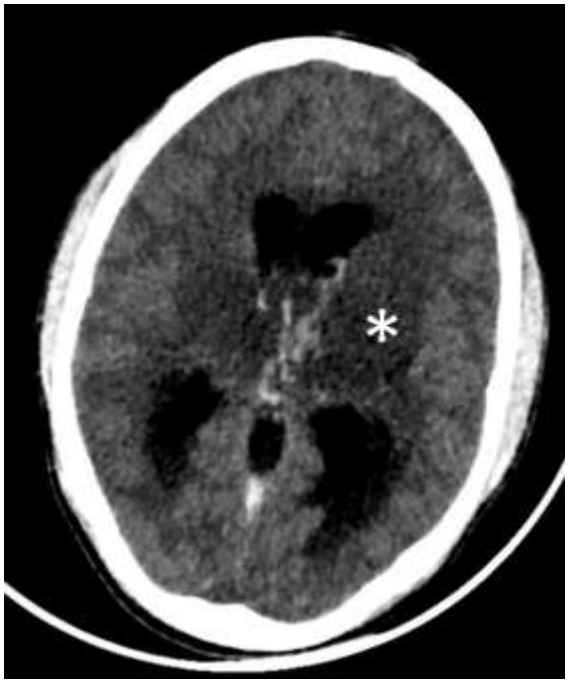


Figure 1 (a): NCCT axial images of the brain show ill-defined hypodensities involving bilateral thalami, basal ganglia and deep white matter of bilateral frontal, parietal and temporal lobes (White Asterisk). Thin linear hyperdensities are seen within bilateral thalami, suggestive of hemorrhages

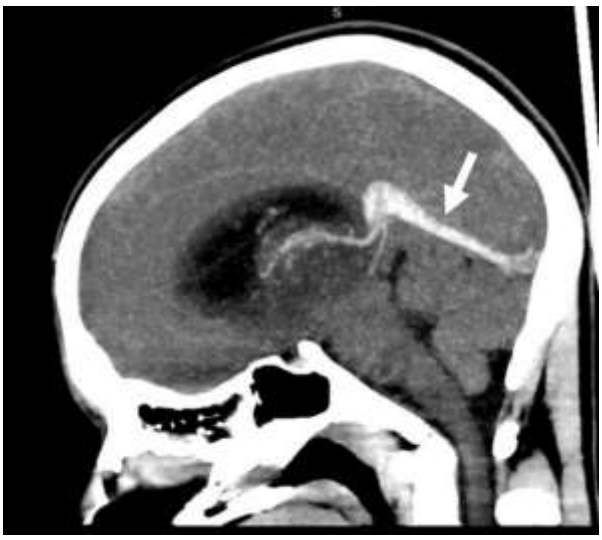


Figure 1 (b): Sagittal MIP NCCT images showing septal veins, thalamostriate veins, internal cerebral veins and straight sinuses with hyperdense contents within – "dense clot" sign (White Arrow)

On subsequent MRI imaging, FLAIR (Figure 1c) hyperintensities are seen in bilateral thalami, internal capsules, basal ganglia and hippocampi, extending to involve deep white matter regions of bilateral frontal lobes, parietal lobes and periventricular white matter of bilateral temporal lobes. On DWI (Figure 1d) and ADC (Figure 1e), therse areas show diffusion restriction. SWI images (Figure 1f) show blooming along the internal cerebral veins and bilateral thalami, more intensely hypointense on left side than on the right side. Findings were highly suggestive of deep cerebral venous thrombosis, with acute viral hemorrhagic encephalitis kept as a second possibility in view of history of high grade fever and thalamic hemorrhages.

Patient has progressive neurological and cardiovascular deterioration, ultimately leading to his demise in the next 24 hours.

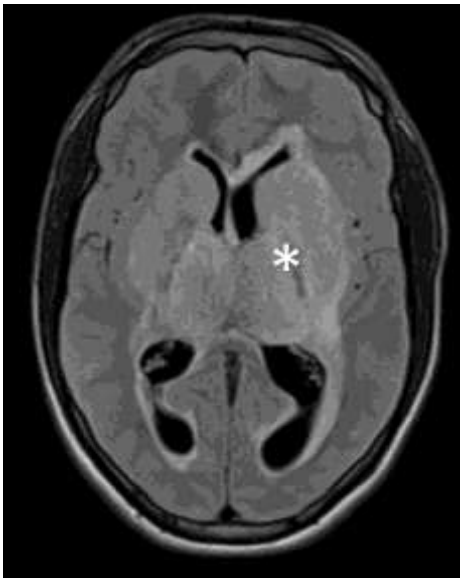
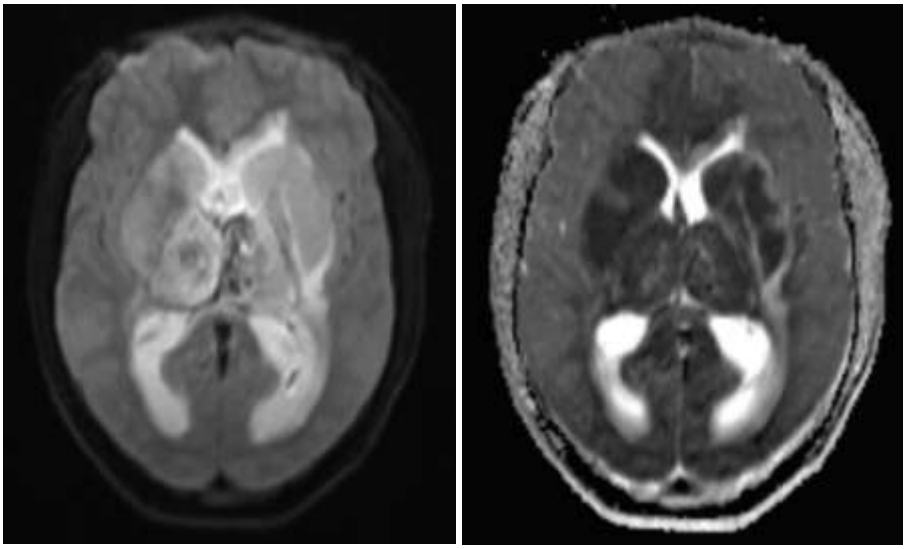


Figure 1 (c): Axial FLAIR image of the brain shows diffuse hyperintensities involving bilateral thalami, internal capsule, basal ganglia, deep white matter of bilateral frontal, parietal and temporal lobes (White Asterisk)



Figures 1 (d, e): Axial DWI images and ADC maps show diffusion restriction in these areas

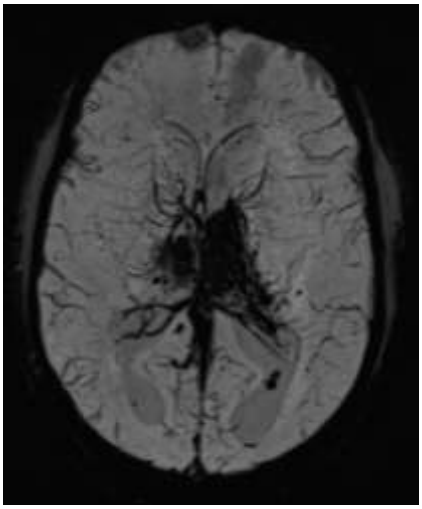


Figure 1(f): Axial MIP SWI images of the brain show blooming along the deep cerebral veins and bilateral thalami (left>right), suggestive of hemorrhages

DISCUSSION

The deep cerebral venous system is composed of paired internal cerebral veins that unite with the basal veins of Rosenthal to form the great cerebral vein (vein of Galen). This vein then converges with the inferior sagittal sinus to form the straight sinus [6,7].

Cerebral venous thrombosis (CVT) results from a dynamic imbalance between prothrombotic and thrombolytic mechanisms, leading to the slow and progressive development of a thrombus. This gradual progression, along with the brain's extensive venous collateral network, often causes CVT to present with subacute symptoms developing over days to weeks—distinct from the abrupt onset typical of arterial strokes [8].

The underlying pathophysiology is explained by Virchow's triad: blood stasis, endothelial injury, and hypercoagulability. Obstruction in the venous outflow elevates venous pressure, reduces capillary perfusion, and increases cerebral blood volume. Initially, cerebral veins compensate through dilation and collateral formation; however, sustained venous hypertension disrupts the blood-brain barrier, causing vasogenic edema, and eventually leads to decreased cerebral perfusion pressure, resulting in infarction with both cytotoxic and vasogenic edema [8].

MRI is the most sensitive modality for detecting parenchymal changes, edema, and differentiating CVT from other pathologies. Characteristic imaging findings of deep CVT include bilateral thalamic edema as an early imaging finding, with possible venous infarction and hemorrhage which are late findings, although the presentation may occasionally be asymmetric or even unilateral. CVT should be suspected when infarcts cross arterial territories or appear in atypical locations. Digital subtraction angiography (DSA) may be considered when parenchymal involvement is evident but venographic studies are non-diagnostic [9]. Our case shows early appearance of thalamic hemorrhages, a feature of acute viral hemorrhagic encephalitis, hence creating a diagnostic dilemma [10].

The prognosis of CVT is highly variable, but involvement of the deep cerebral venous system is considered a poor prognostic factor due to the critical brain regions affected [11].

A companion case of a child with acute onset neurological symptoms, history of high grade fever and altered bithalamic signal is shown (Figures 2A & 2B), where the imaging findings can be suggestive of acute viral hemorrhagic fever, as the deep cerebral venous system was normal in this patient.

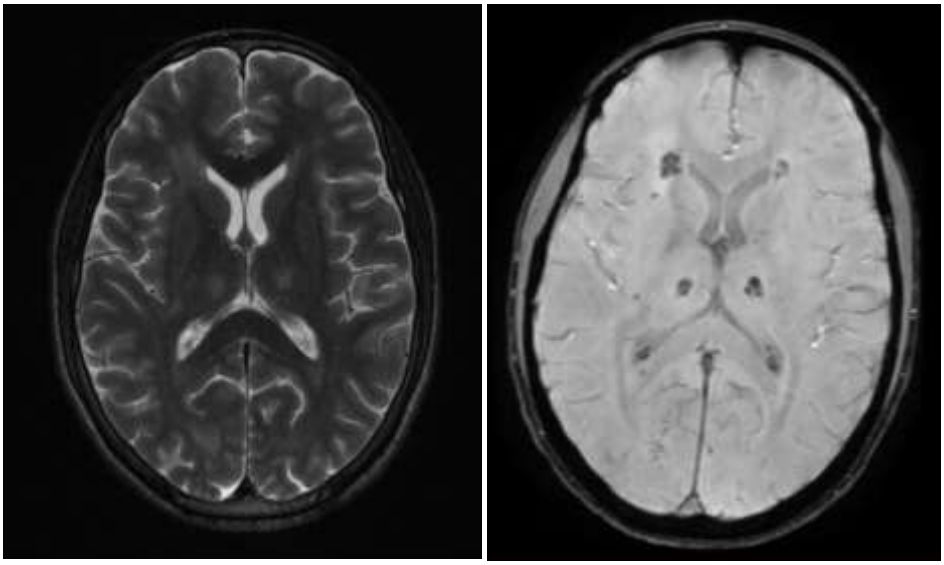


Figure 2a & 2b: Axial T2 and SWI images of the brain show bithalamic T2 hyperintensities (White Arrow) with blooming in both thalami and periventricular white matter of frontal and parietal lobes (Black Arrow), suggestive of hemorrhages

CONCLUSION

Deep cerebral venous thrombosis is a rare and potentially life-threatening condition that should be considered in patients—particularly younger individuals—presenting with unexplained bilateral thalamic lesions, altered mental status, or other atypical neurological symptoms. Early recognition through appropriate neuroimaging, particularly MRI, is essential for timely diagnosis. Prompt initiation of anticoagulation therapy can lead to significant clinical improvement and may prevent long-term neurological complications. This case highlights the importance of maintaining a high index of suspicion for deep CVT to facilitate early intervention and improve patient outcomes.

ABBREVIATIONS

CVT: Cerebral Venous Thrombosis
MIP: Maximum Intensity Projection
DWI: Diffusion Weighted Imaging
ADC: Apparent Diffusion Coefficient

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