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Thalamic stroke from deep cerebral venous thrombosis in a young woman on oral contraceptives

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Abstract

Background: Deep cerebral venous thrombosis (DCVT) is an infrequent subtype of cerebral venous thrombosis (CVT), often presenting with nonspecific neurological symptoms. Its imaging hallmark—bilateral thalamic involvement—necessitates rapid recognition to guide prompt anticoagulation.

Case summary: A 45-year-old woman presented with suddenonset headache, vomiting, aphasia, and left-sided hemiparesis. Non-enhanced CT demonstrated bilateral thalamic edema with intraparenchymal hemorrhage and hyperdense deep cerebral veins. CT venography confirmed thrombosis of the internal cerebral veins, basal veins of Rosenthal, and vein of Galen. The patient was taking oral contraceptive pills (OCPs), a known prothrombotic risk factor.

Conclusion: This case underlines the classic radiological features of DCVT and highlights oral contraceptive use as a common and modifiable risk factor in female patients. Recognition of hyperdense deep veins on non-contrast CT may be the first diagnostic clue.

Keywords: Deep cerebral venous thrombosis; Internal cerebral vein; Vein of galen; Oral contraceptives; Thalamic edema; Hyperdense vein sign.

Introduction

Cerebral venous thrombosis (CVT) is a rare cerebrovascular condition accounting for <1% of all strokes, disproportionately affecting young females, especially those on oral contraceptives. While most cases involve the dural sinuses, thrombosis of the deep venous system—comprising the internal cerebral veins (ICVs), basal veins of Rosenthal, vein of Galen, and straight sinus—can result in profound bilateral thalamic infarction, often misdiagnosed as arterial infarct, encephalitis, or metabolic encephalopathy. Early identification of this pattern on imaging is critical for timely intervention.

Case report

A 45-year-old woman, with no known history of thrombophilia but taking combined oral contraceptive pills (OCPs), presented with acute headache, vomiting, and progressive aphasia and left hemiparesis. The patient was alert but somnolent, with a Glasgow Coma Scale of 12. Non-contrast CT scan revealed symmetrical hypodensities in the bilateral thalami, right lentiform nucleus, and posterior limb of the internal capsule. A punctate hyperdensity within the right thalamus suggested venous hemorrhagic transformation. Spontaneous hyperdensity was noted in the region of the internal cerebral veins and the vein of Galen, consistent with acute thrombus ("hyperdense vein sign"). CT venography confirmed the absence of contrast opacification

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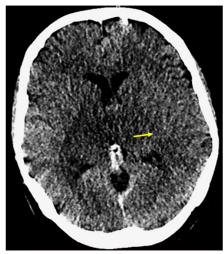


Figure 1: Axial non-contrast CT scan revealed symmetrical hypodensities in the bilateral thalami, right lentiform nucleus, and posterior limb of the internal capsule.



Figure 2: Axial non enhanced CT showing hyperdensity in internal cerebral veins suggestive of acute thrombosis.



Figure 3: Enlargement and spontaneous hyperdensity of basal veins of Rosenthal.

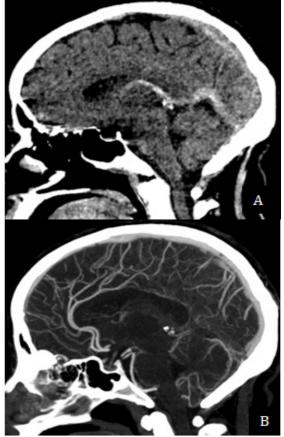


Figure 4: Sagittal non contrast CT **(A)** showing spontaneous hyperdensity in the deep veinous system contrasting with the lack of enhancement of these structures after contrast administration **(B)**.

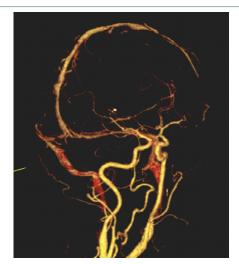


Figure 5: 3D reconstruction illustrating absent deep venous drainage pathways.

in the right internal cerebral vein, basal vein of Rosenthal, and vein of Galen, consistent with deep cerebral venous thrombosis. The superior sagittal and transverse sinuses were patent.

Discussion

DCVT, although rare, carries a high risk of morbidity due to the critical vascular territories it affects. Thrombosis of the deep venous system causes congestion in the thalamus, basal ganglia, and brainstem, resulting in characteristic imaging pat-

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terns. In our patient, oral contraceptive use likely contributed to a transient prothrombotic state, consistent with findings from multiple case series. Imaging plays a pivotal role, especially non-contrast CT, which may demonstrate subtle clues such as:

- Bilateral thalamic hypodensities
- Venous hemorrhage
- Hyperdense vein sign in the internal cerebral veins

CT venography confirms the diagnosis, demonstrating filling defects and non-opacified venous segments. Early diagnosis allows for prompt anticoagulation, even in the presence of hemorrhage, as supported by multiple guidelines.

Conclusion

This case illustrates the classical radiologic and clinical presentation of DCVT. In young females with acute neurological deficits, deep venous thrombosis should be considered, especially in the presence of oral contraceptive use. Early CT signs—such as hyperdense deep veins and bilateral thalamic involvement—should prompt emergent venography and anticoagulation.

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