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Unilateral Basal Ganglion, Thalamus, and Splenium of Corpus Callosum Lesions in Deep Cerebral Venous Thrombosis: A Case Report

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Abstract

Introduction: Deep cerebral venous thrombosis primarily affects young individuals, women of reproductive age, and patients with a prothrombotic state. Unilateral thalamic lesions resulting from deep cerebral venous thrombosis are relatively unusual. We describe a rare case of unilateral basal ganglion, thalamus, and splenium of the corpus callosum lesions due to thrombus in the deep cerebral venous system.

Case Presentation: A 31-year-old Asian woman presented with acute-onset headache, somnolence, and left limb weakness after a miscarriage. Subsequent investigations, including computed tomography, magnetic resonance imaging, and magnetic resonance venography, confirmed deep cerebral venous thrombosis as the underlying cause of her symptoms. Because of progressive deterioration in symptoms and consciousness, she underwent emergent endovascular treatment and anticoagulation therapy, and her symptoms subsequently resolved without complications. At the 1-year follow-up, she remained asymptomatic.

Conclusions: Deep cerebral venous thrombosis can induce unilateral thalamic lesions and lesions in adjacent structures, requiring increased caution in diagnosis and invasive investigations. For patients with severe symptoms despite medical treatment, endovascular intervention may serve as a viable alternative therapeutic option.

Keywords: Deep Cerebral Venous Thrombosis, Arachnoid Granulation, Endovascular Intervention

1. Introduction

Deep cerebral venous thrombosis, which refers to the formation of blood clots within the internal cerebral veins, basal veins, and vein of Galen, is a rare type of cerebrovascular disease that often occurs in young people, women of reproductive age, and patients with a prothrombotic state. Various risk factors can create a prothrombotic state, resulting in clot formation that obstructs cerebral venous drainage. Major risk factors include oral contraception, pregnancy or puerperium, acquired thrombophilias (antiphospholipid antibody syndrome, JAK2 mutations, malignancy, and autoimmune disease), genetic thrombophilias (protein C and protein S deficiency, factor V Leiden, and prothrombin G20210A polymorphism), infections, dehydration, other medications (corticosteroids, L-asparaginase, and vaccine-induced thrombotic thrombocytopenia), and mechanical provoking factors (head trauma, neurosurgical procedures, and compressive lesions) (1). In most cases, patients present with headache. Other common signs and symptoms include nausea, transient visual obscurations or vision loss, papilledema, diplopia, seizure, focal neurological deficits according to brain lesions, encephalopathy, and somnolence or coma (1, 2). The mainstay of treatment is anticoagulation, typically with low-molecular-weight heparin or oral anticoagulants. In severe or refractory cases, thrombolysis or endovascular interventions may be considered. Deep cerebral venous thrombosis typically leads to bilateral thalamic lesions, along with damage to cerebral structures adjacent to the thalami,

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such as the basal ganglia, splenium of the corpus callosum, and mesencephalon. However, in rare cases, the damage is unilateral. We describe a rare case of unilateral basal ganglion, thalamus, and splenium of the corpus callosum lesions due to thrombus in the deep cerebral venous system.

2. Case Presentation

A 31-year-old Asian woman had a headache for 2 days after a miscarriage. She presented with left limb weakness and somnolence 10 hours after the headache and arrived at the emergency department 1 day after headache onset. She had no medical history and did not use contraceptives. Her National Institutes of Health Stroke Scale score was 8, and her Glasgow Coma Scale score was 14. Laboratory tests revealed elevated D-dimer (5.38 μg/mL) and fibrinogen degradation products (8.18 μg/mL). Noncontrast computed tomography indicated hypodensity in the right basal ganglion, thalamus, and splenium of the corpus callosum and hyperdensity of the right internal cerebral vein and straight sinus (Figure 1A). The patient underwent magnetic resonance imaging (MRI) and magnetic resonance venography (MRV). T2 fluid-attenuated inversion recovery imaging demonstrated a hyperintensity signal involving the right basal ganglion, thalamus, and splenium of the corpus callosum (Figure 1B). Diffusion-weighted imaging showed a heterogeneous hyperintensity signal in the right thalamus (Figure 1C). Hemorrhagic transformation in the right thalamus was confirmed by susceptibility-weighted angiography sequence. MRV revealed absence of the deep cerebral venous system (Figure 1D). Right thalamic infarction with hemorrhage, as well as right basal ganglion and splenium of the corpus callosum edema due to deep cerebral venous thrombosis, was diagnosed according to the clinical presentation and imaging features. The differential diagnosis initially included glioma and intracerebral abscess. However, these were not sufficiently supported by the MRV findings or by the absence of fever or mass effect.

Because of progressive deterioration in the patient's symptoms and consciousness, emergent endovascular treatment, including aspiration and thrombolysis, was performed after adequate discussion with the patient's family. After successful vascular access was obtained in the left femoral artery and right femoral vein using the Seldinger technique, digital subtraction angiography showed absence of the deep cerebral venous system and confirmed the thrombus. A 6F AXS Catalyst catheter (132 cm) was advanced to the torcular Herophili. Through this catheter, a Synchro-14 guidewire (300 cm) and an Echelon-10 microcatheter were navigated into the straight sinus, guided the Catalyst catheter to the straight sinus, and were withdrawn. Thrombus was aspirated through the Catalyst catheter. This process was repeated several times, and subsequent digital subtraction angiography showed partial recanalization of the straight sinus. The Synchro guidewire and Echelon microcatheter were reintroduced into the straight sinus, and after the Synchro guidewire was withdrawn, urokinase was rapidly infused through the microcatheter at a rate of 30,000 units per hour. Postinterventional MRI revealed mild hemorrhage in the right thalamus. Her symptoms resolved 3 days later without complications, and the microcatheter was withdrawn after discontinuation of urokinase infusion, with transition to low-molecular-weight heparin. Subsequent tests for thrombophilia and autoimmune antibodies were negative. Upon discharge, she was prescribed a vitamin K antagonist, with a target international normalized ratio of 2 - 3 for therapeutic anticoagulation.

2.1. Follow-up

At the 1-year follow-up, she was asymptomatic. Follow-up MRI showed a small patchy area with hypointensity distributed in the right thalamus, which suggested hemosiderin deposition (Figure 1E). MRV demonstrated a round filling defect located in the proximal segment of the straight sinus (Figure 1F). High-resolution magnetic resonance vessel wall imaging detected an arachnoid granulation protruding into the straight sinus (Figure 1G). Furthermore, 3-dimensional reconstruction based on high-resolution images showed symmetry of venous drainage in the bilateral deep venous system (Figure 1H).

3. Discussion

Cerebral venous thrombosis is an unusual cerebrovascular disease caused by obstruction of cerebral venous drainage. The most common affected sites are the superior sagittal sinus and transverse-sigmoid sinus. Thromboses also occur independently in deep venous structures, including the straight sinus, vein of Galen, inferior sagittal sinus, internal cerebral veins that drain the thalami, and basal veins of Rosenthal (1). Disease onset can be acute, subacute, or chronic. Headache occurs in most patients and is sometimes followed by a decreased level of consciousness, focal neurological deficits related to the affected vein, and seizure (1, 2, 3). Current practices recommend MRI and MRV as the preferred brain imaging modalities to differentiate deep cerebral

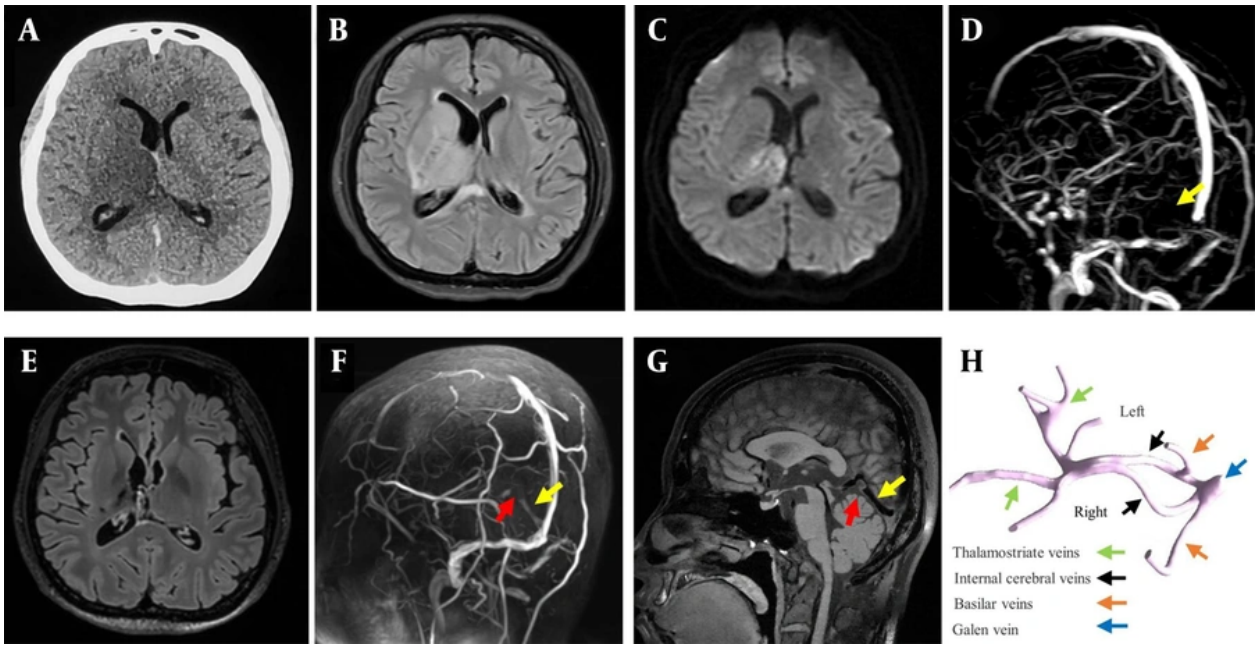

Figure 1. Images of a 31-year-old Asian woman who presented with headache, left limb weakness, and drowsiness. A, Noncontrast computed tomography showed hypodense lesions in the right basal ganglion, thalamus, and splenium of the corpus callosum and hyperdensity of the right internal cerebral vein and straight sinus; B, T2 fluid-attenuated inversion recovery imaging demonstrated a hyperintensity signal involving the right basal ganglion, thalamus, and splenium of the corpus callosum; C, diffusion-weighted imaging showed a heterogeneous hyperintensity signal in the right thalamus; D, magnetic resonance venography revealed absence of the deep cerebral venous system; E, 1-year follow-up T2 fluid-attenuated inversion recovery imaging showed a small patchy area with hemosiderin deposition in the right thalamus; F, 1-year follow-up magnetic resonance venography demonstrated a round filling defect located in the proximal segment of the straight sinus; G, 1-year follow-up high-resolution magnetic resonance vessel wall imaging detected an arachnoid granulation protruding into the straight sinus; H, 3-dimensional reconstruction based on high-resolution images indicated symmetry of venous drainage in the bilateral deep venous system.

venous thrombosis from other disorders, although spontaneously increased density of cerebral veins can sometimes be detected by noncontrast computed tomography in the acute phase of the disease (4).

The primary etiologies of the disease include oral contraceptives, hormone-replacement therapy, pregnancy or the postpartum period, thrombophilia, infection, autoimmune disorder, surgery, and trauma (1, 2). Regarding thrombus formation in the present case, based on Virchow's triad, hypercoagulability resulting from miscarriage heightened vulnerability to cerebral venous thrombosis. In addition, protrusion of an arachnoid granulation into the straight sinus was detected. Seventeen percent of arachnoid granulations are localized in the straight sinus, appearing as focal filling defects on vessel imaging with cerebrospinal fluid-like content (5). It is presumed that impaired outflow in the straight sinus potentially contributed to thrombosis in this case.

Due to the symmetry of venous drainage, lesions secondary to deep cerebral venous thrombosis are usually bilateral (6). Unilateral lesions secondary to deep cerebral venous thrombosis are rare. Reported cases in the literature are shown in Table 1. The presence of adequate collateral venous drainage on the contralateral side may explain unilateral lesions (14). Given the relative rarity of such cases, these unilateral lesions can closely resemble deep-seated gliomas, to the extent that clinicians may at times consider brain biopsy in some patients (9). This underscores the need for heightened caution when making diagnoses and conducting invasive examinations in these patients.

Anticoagulation is the standard management for deep cerebral venous thrombosis, and outcomes are usually favorable. Nevertheless, for patients who exhibit severe and progressive symptoms despite medical treatment, endovascular intervention, offering faster recanalization, may be a viable alternative option (1, 17). In the present case, unilateral lesions may have progressed to bilateral involvement without timely treatment.

4. Conclusions

Table 1. Clinical Features, Duration, and Outcomes of Reported Cases of Unilateral Infarct with Deep Cerebral Venous Thrombosis in the Literature

Author and Age/Sex	Presentation	Duration	Site	Vessel Involved	Outcome
Küker et al. (2001) (7)					
49/female	Headache, somnolence, aphasia	3 days	Left thalamus	ICV, straight sinus, left TS and SS	Without disability
58/female	Headache, drowsiness	-	Left thalamus	Left ICV, straight sinus, left TS and SS	Without new neurologic deficits
31/female	Headache, nausea	-	Left thalamus, medial part of left temporal lobe	ICV, straight sinus	-
Herrmann et al. (2004) (8)					
47/female	Headache, hemiparesis, retrograde amnesia	2 weeks	Left thalamus	Left ICV	Complete recovery
van den Bergh et al. (2005) (6)					
30/female	Headache, hemianopia, drowsiness, seizure	2 weeks	Left thalamus	ICV, vein of Galen, left TS and SS	Residual headache, fatigue
28/female	Right hemiparesis	1 day	Left thalamus	Left ICV, vein of Galen, straight sinus, left TS and SS	Mild hemiparesis, dysphasia
53/female	Headache, aphasia, right hemiparesis	3 days	Left thalamus	Partial thrombosis of straight sinus and left ICV	-
Wieshmann et al. (2009) (9)					
14/female	Headache, vomiting, drowsiness, aphasia, right-sided weakness	3 days	Left thalamus	ICV, vein of Galen, straight sinus	Mild weakness, memory impairment
Desai (2010) (10)					
30/female	Headache, vomiting	2 days	Left thalamus, caudate and lentiform nuclei	Vein of Galen, straight sinus	Improved
Chung et al. (2012) (11)					
36/female	Headache, dysarthria, right hemiparesis	1 week	Left thalamus	Left ICV and thalamostriate vein	Mild headache
Deshpande et al. (2014) (12)					
31/male	Headache, vomiting, status epilepticus	4 days	Right thalamus	ICV, vein of Galen, SSS	Memory impairment
Chung et al. (2018) (13)					
1/male	Right hemiparesis	3 hours	Left thalamus	ICV, vein of Galen, straight sinus	Complete recovery
Menon et al. (2019) (14)					
43/female	Headache, apathy, right hemiparesis	3 days	Left thalamus, caudate and lentiform nucleus; left superior frontal gyrus	Left ICV, vein of Galen and cortical vein	Complete recovery
23/female	Headache	2 days	Left thalamus	ICV, vein of Galen, straight sinus, SSS	Complete recovery
Linley-Adams et al. (2022) (15)					
40/male	Fatigue, unsteadiness, fine motor control difficulty of left hand	6 weeks	Right thalamus	ICV, straight sinus, vein of Galen	Occasional fine motor control difficulty of left hand
Ivaturi and Gopinath (2022) (16)					
32/male	Headache	6 weeks	Right thalamus	ICV	Complete recovery
Current case (2026)					
31/female	Headache, somnolence, hemiparesis	2 days	Right thalamus, basal ganglion, splenium of the corpus callosum	ICV, vein of Galen, straight sinus	Complete recovery

Abbreviations: ICV, internal cerebral vein; SS, sigmoid sinus; SSS, superior sagittal sinus; TS, transverse sinus.

Deep cerebral venous thrombosis can manifest as unilateral thalamic lesions with involvement of adjacent structures, demanding careful diagnosis and cautious invasive testing. Endovascular intervention may serve as a viable therapeutic alternative for patients

who exhibit persistent and worsening symptoms despite optimal medical management.

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Footnotes

Authors' Contribution S. D., Z. H., B. J., and N. M. were responsible for the concepts and design. S. D. and N. M. helped with acquisition of data. All authors contributed to the analysis of data. S. D., Z. H., and N. M. helped with the literature search. S. D., Z. H., and N. M. revised the article language, and all authors participated in writing and revisions. All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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