Unilateral Internal Cerebral Vein Thrombosis: Imaging of an Uncommon Case and Literature Review

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AUTHORS' CONTRIBUTIONS

Dr. Shaun Previn Appaduray: Study concepts and design, Manuscript editing
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DISCLOSURES

No disclosures. The authors declare that there is no conflict of interest.

CONSENT

No written informed consent was required for manuscript publication. This report fulfills the requirements of our institution for ethics board waiver.

HUMAN AND ANIMAL RIGHTS

Not applicable.

ABSTRACT

Cerebral venous thrombosis can lead to detrimental outcomes such as cerebral edema, hemorrhagic infarcts, neurological deficits, and death if not diagnosed and treated promptly. Patients with deep cerebral venous thrombosis face a poorer prognosis, with 29.4% experiencing lifelong dependence or death. Unilateral internal cerebral vein thrombosis is an uncommon and potentially life-threatening entity not widely published in literature. It can mimic arterial infarcts from artery of Percheron/basilar tip thrombosis, infectious, neoplastic and metabolic disorders. Radiologists must recognize its imaging features to ensure early diagnosis and timely treatment. In this article, we present the imaging findings of an uncommon case of unilateral internal cerebral vein thrombosis and how it can be distinguished from other diagnoses. We also review relevant literature, explore the tendency for left internal cerebral vein thrombosis, and discuss the diagnostic accuracy and pitfalls in imaging.

CASE REPORT

CASE REPORT

A 91-year-old female presented with new onset right-sided upper and lower limb weakness. The patient had a background of mild dementia and known small vessel disease with chronic infarcts in the right hemipons, left thalamus and left striatocapsular region on previous imaging. She had no known risk factors for thrombosis. The initial clinical impression was a right pure motor syndrome and a magnetic resonance imaging (MRI) brain stroke protocol was performed to look for evidence of acute infarct. The MRI stroke protocol at our institution comprises T2-weighted (T2W) sequence, diffusion weighted imaging (DWI) with apparent diffusion coefficient (ADC) map, gradient echo sequence (GRE), and a time-of-flight magnetic resonance angiography (TOF MRA). Computed tomography (CT) venography was also performed.

On MRI brain (Figure 1), axial DWI image showed focal hyperintense DWI signal in the left thalamus with corresponding hypointense ADC signal, suggestive of restricted diffusion. Axial T2W image showed slight asymmetry of the thalami with swelling of the left thalamus. Hyperintense T2W signal was seen in bilateral caudate and lentiform nuclei as well as both thalami, worse on the left. Axial GRE image showed curvilinear susceptibility along the course of the left ICV. Faint ill-defined susceptibility was also seen in the left caudate head and left putamen, suggestive of petechial hemorrhage. Reformatted TOF MRA image showed linear hyperintensity along the straight sinus, raising suspicion of T1W hyperintense blood products.

The radiologist raised suspicion for acute left ICV thrombosis and recommended CT venogram (Figure 2) for

further evaluation. CT venogram was recommended as the MRI stroke protocol at our institution was not a dedicated study for evaluating the cerebral veins. CT venography was also faster and easier to acquire than MRI venography in the acute setting at our institution.

Unenhanced axial CT brain showed the left ICV appearing hyperdense relative to the right ICV with corresponding filling defect of the left ICV seen on axial CT venogram. There was preserved contrast opacification of the right ICV. A thin sliver of contrast was also seen in the straight sinus and vein of Galen. Hypodensities in bilateral basal ganglia and thalami on unenhanced CT were seen, representing edema detected on the prior MRI study.

Findings were correlated on unenhanced coronal CT brain and coronal CT venogram which again demonstrated a hyperdense left ICV with filling defect. Unenhanced axial CT brain showed a hyperdense straight sinus with corresponding filling defect seen on axial CT venogram. Overall, these findings confirmed thrombosis of the deep cerebral venous system with unilateral complete thrombosis of the left ICV and partial thrombosis of the straight sinus and vein of Galen.

Table 1 summarizes the imaging features of unilateral left ICV thrombosis.

A thorough thrombotic workup for the patient yielded negative results. A CT thorax, abdomen and pelvis also did not detect any malignancy. The patient was treated with subcutaneous clexane and oral apixaban. At a follow-up clinic visit, she was well and her family reported symptomatic improvement despite residual mild right-sided weakness.

DISCUSSION

Etiology & demographics

Cerebral venous thrombosis (CVT) is responsible for 0.5% of all strokes [1]. It can result in serious complications, including intracranial hemorrhage, raised intracranial pressure, cerebral edema and death. CVT can be divided into thrombosis of the superficial and deep venous systems. The deep venous system comprises the basal veins of Rosenthal, deep medullary veins, internal cerebral veins, vein of Galen and straight sinus [2]. The deep grey matter and periventricular white matter are drained by the deep venous system. Deep cerebral venous thrombosis is uncommon, consisting 10.9% of patients diagnosed with CVT [3]. Unilateral thrombosis of the internal cerebral veins (ICV) is even rarer, with literature review only yielding 16 reported cases of unilateral ICV thrombosis. Deep cerebral venous thrombosis often affects bilateral thalami and may involve the basal ganglia and deep white matter [4].

Common risk factors for cerebral venous thrombosis include thrombophilia (34.1%), hematological disorders (12%) and women less than 50 years of age on oral contraceptives or in the puerperium period [3].

With regards to the demographics of unilateral internal cerebral vein thrombosis, not enough data is available to demonstrate any age or gender predilection due to the small number of reported cases (Table 2).

Clinical & imaging findings

A study of 624 cases of CVT in adults reported headache and seizures as the most common symptoms (88% and 88.9% respectively), followed by paresis (78%) [3].

Complications of CVT can be reversible with timely treatment, thus early diagnosis is critical. As clinical symptoms can be non-specific, multimodality imaging including CT and MRI allows for a definite diagnosis of CVT. It is thus imperative for the radiologist to recognize the imaging features and have a high index of suspicion for CVT, even more so in rare cases of unilateral ICV thrombosis not commonly seen in daily practice.

The general imaging features of cerebral venous thrombosis have been widely described in literature. However, the detailed imaging features of unilateral ICV thrombosis are less commonly described due to the paucity of cases. We will discuss the imaging features of both CVT and ICV thrombosis in this section (Table 3).

Direct signs of CVT refer to direct visualization of the thrombus on imaging. Unenhanced CT demonstrates hyperdense thrombosed veins. Contrast-enhanced CT venogram shows filling defects in the thrombosed vein. Thrombus of varying ages have varied signal intensities on unenhanced MRI (Table 4) [5]. Unenhanced MRI venogram such as phase-contrast and time-of-flight imaging can also demonstrate loss of flow signal in the thrombosed vein. The thrombus can appear as filling defects on contrast-enhanced MRI venogram.

Indirect signs of cerebral venous thrombosis include parenchymal changes such as hemorrhagic infarcts that do not follow major arterial territorial distributions and cerebral edema. Acute hemorrhage appears hyperdense on CT and demonstrates susceptibility on MRI GRE sequence. Cerebral edema appears hypodense on CT and has hyperintense T2W signal on MRI.

A study in 2006 by Idbaih et al. found that GRE sequence had an approximate sensitivity of 90% in detecting CVT in its early stage of within a week as the thrombus would demonstrate increased susceptibility [6]. GRE sequence is helpful in detecting acute thrombus where it may not be evident on other sequences, such as the T2W sequence where it is hypointense, mimicking the normal flow void of a patent vessel, and on T1-weighted (T1W) sequence where it is not yet hyperintense [6].

CVT can result in vasogenic and cytotoxic edema due to cerebral venous congestion and reduced perfusion. Both types of edema appear T2W hyperintense on MRI. On DWI/ADC, cytotoxic edema demonstrates restricted diffusion with DWI hyperintensity and corresponding low ADC values [4]. On

the contrary, vasogenic edema does not demonstrate restricted diffusion and instead, demonstrates DWI hyperintensity with high ADC values due to the T2 shine-through effect. Vasogenic edema may be reversible when thrombosis is treated. Cytotoxic edema representing infarction from ischemia is not typically reversible.

The thalamostriate vein (TSV) is the largest tributary of the ICV and in 77% of cases, only the TSV drains the basal nuclei (Figure 3) [2]. Thus, not only the thalami but also the basal ganglia are at risk of edema and infarction in ICV thrombosis.

In our patient's case, there was extensive T2W hyperintense vasogenic edema in bilateral basal ganglia and thalami, disproportionate to the small focus of restricted diffusion in the left thalamus, raising suspicion of internal cerebral vein thrombosis. On T2W image, the left ICV was hypointense and on TOF images, it did not display any increased signal. However, increased susceptibility of the left ICV, straight sinus and vein of Galen on GRE images suggested possible thrombosis.

Contrast-enhanced CT venogram subsequently confirmed thrombosis in the left ICV and partial thrombosis of the straight sinus and vein of Galen. Edema in the contralateral right thalamus and basal ganglia can be explained by thrombosis of the vein of Galen and straight sinus draining the patent right ICV. This case also supports the findings of Idbaih et al. where GRE sequence is more sensitive than T1W and T2W sequences in detecting the acute thrombus when it is not yet T1W hyperintense and appears T2W hypointense and indistinguishable from the hypointense flow voids of patent veins.

Treatment & prognosis

Patients diagnosed with cerebral venous thrombosis are often medically treated with anticoagulation. Initial treatment usually involves the administration of subcutaneous low-molecular-weight heparin followed by conversion to oral anticoagulation therapy, the duration of which would depend on the patient's risk factors for thrombosis [1]. American Heart Association/American Stroke Association (AHA/ASA) guidelines recommend anticoagulation with subcutaneous low-molecular-weight heparin for initial treatment with conversion to oral anticoagulants for a period of 3 to 12 months [1]. However, patients who are at high risk for recurrent thrombosis may require lifelong anticoagulation therapy [1] (Table 2).

In cases which do not respond to anticoagulation therapy, endovascular treatment is sometimes initiated for thrombectomy and thrombolysis for clinically deteriorating patients [1]. In severe cases of raised intracranial pressure with the risk of herniation, surgical decompressive craniectomy can be a life-saving procedure [1].

As per the AHA/ASA guidelines, follow-up imaging with CT or MR venography to assess recanalization of thrombosed cerebral veins may be considered in 3-6 months from the time of diagnosis for clinically stable patients or performed when symptoms worsen [1].

Deep cerebral venous thrombosis carries a poorer prognosis compared to superficial cerebral venous thrombosis, with 29.4% of patients with deep CVT experiencing lifelong dependence or death [3]. This is likely due to the deep brain structures that are involved in deep cerebral venous thrombosis, such as the basal ganglia and thalami, which not only serve motor and sensory functions, but also play a role in the patient's consciousness, cognition, emotions, learning, language and memory abilities.

Literature review

For our literature review, articles with "internal cerebral vein thrombosis" on PubMed were reviewed, with 7 cases of unilateral ICV thrombosis identified. An additional 2 cases were from an article by Hu et al. which identified another 7 cases of unilateral ICV thrombosis published in literature [7]. In total, there are 16 cases of unilateral ICV thrombosis, 12 (75%) of which were reported to have left ICV thrombosis. Table 5 summarizes the details of the 9 cases we have collated.

The pathophysiology behind the apparent tendency for the left internal cerebral vein to be thrombosed compared to the right is not well understood. We hypothesize that this may be related to asymmetric venous drainage in the brain as suggested by Philpott et al. where smaller and poorer left-sided venous drainage predisposes to left-sided thrombosis [8]. Our patient had right transverse sinus dominance which may have resulted in poorer left-sided venous drainage. More research is required to determine the likelihood of this hypothesis.

Differential diagnoses

The most common clinical suspicion for a patient presenting with neurological deficits is that of arterial stroke. In the setting of bithalamic edema and restricted diffusion, arterial infarcts caused by artery of Percheron or basilar artery tip thrombosis are considerations. However, CVT should be suspected when imaging findings are atypical of an arterial infarct. Other differentials worth considering in bithalamic lesions include infective causes such as viral encephalitis, metabolic/toxic disorders, and less commonly primary bilateral thalamic glioma.

Table 6 details the imaging features of the above conditions that can mimic that of ICV thrombosis and imaging findings that distinguish them from ICV thrombosis.

Diagnostic accuracy and imaging pitfalls in CVT

In unenhanced CT, the "dense clot" sign has approximately 64% sensitivity and 97% specificity for acute thrombosis within the first week of onset [5]. It can be falsely positive in patients with raised hematocrit such as in dehydration and polycythemia. It is also harder to detect the thrombus when its density decreases after the first week. Indirect signs of CVT are present in 60-80% of CVT cases, such as ischemia and intracranial hemorrhage

[9]. Nonetheless, up to 30% of patients with CVT have normal CT brain findings [9].

CT venography has a high sensitivity and specificity in detecting CVT comparable with digital subtraction angiography in studies with small to moderate sample size [9]. When compared with TOF MR venography, it better demonstrates the venous anatomy for smaller veins with low flow, with equivalent accuracy in detecting CVT [10].

Compared with CT venography, MRI has a higher sensitivity for demonstrating cerebral edema and smaller lesions not well seen on CT [9]. Non-contrast MR venography utilizes flow-related techniques to evaluate for thrombosis. Phase-contrast MRI has 100% sensitivity and 71% specificity in detecting CVT but its sensitivity decreases for smaller veins in cortical vein thrombosis [9]. TOF MR venography has a sensitivity of 48-100% albeit with a wide 95% confidence interval [9]. Non-contrast MR venography may falsely reflect absent flow signal in vessels with slow or in-plane blood flow [11]. Hypoplastic/ atretic dural venous sinuses may not be visualized on non-contrast MR venography [11].

Contrast-enhanced MRI is superior to non-contrast MRI in detecting CVT, with a sensitivity of 86-97% and specificity of 52-100% compared to 55-97% and 28-95% respectively for non-contrast MRI [9].

With regards to the diagnostic accuracy of CT versus MR imaging, a 2018 meta-analysis concluded that both have high sensitivity and specificity for detecting CVT [9].

CONCLUSION

Unilateral internal cerebral vein thrombosis is a rare but potentially life-threatening condition that requires prompt diagnosis and early intervention. It may present a diagnostic challenge due to findings on both CT and MR imaging that can commonly be mistaken for other etiologies. It is thus imperative for the radiologist to recognize the imaging features of ICV thrombosis, have a high index of suspicion and request for further appropriate imaging to confirm the diagnosis. Familiarity with this entity will ultimately improve patient outcomes with timely intervention once the accurate diagnosis is made.

TEACHING POINT

The imaging findings of internal cerebral vein (ICV) thrombosis can be easily mistaken for other diagnoses, such as an arterial infarct when restricted diffusion is seen in the thalamus. It is crucial for the radiologist to recognize indirect and atypical findings that would raise suspicion of ICV thrombosis, such as disproportionate thalamic edema and increased susceptibility in a thrombosed vessel, following which, to recommend appropriate dedicated imaging to confirm the diagnosis.

QUESTIONS & ANSWERS

Question 1: Regarding the clinical presentation of cerebral venous thrombosis:

1. Vomiting is the most common symptom.
2. Confusion is the most common symptom.
3. Headache is the most common symptom. (Applies)
4. Paralysis is the most common symptom.
5. Nausea is the most common symptom.

Explanation: Headache is the most common symptom. [A study of 624 cases of CVT in adults reported headache and seizures as the most common symptoms (88% and 88.9% respectively), followed by paresis (78%) [3].]

Question 2: Regarding the blood supply of the internal cerebral vein:

1. The thalamostriate vein is the largest tributary. (Applies)
2. The anterior septal vein is the largest tributary.
3. The direct lateral vein is the largest tributary.
4. The deep medullary vein is the largest tributary.
5. The vein of Galen is the largest tributary.

Explanation: The thalamostriate vein is the largest tributary of the internal cerebral vein. The internal cerebral vein (ICV) drains into the vein of Galen. [The thalamostriate vein (TSV) is the largest tributary of the ICV and in 77% of cases, only the TSV drains the basal nuclei [2].]

Question 3: Regarding the brain structures affected in internal cerebral vein thrombosis:

1. The basal ganglia are commonly affected. (Applies)
2. The cerebral cortex is commonly affected.
3. The ventricles are commonly affected.
4. The thalami are commonly affected. (Applies)
5. The cerebellum is commonly affected.

Explanation: The basal ganglia and thalami are commonly affected as the internal cerebral vein (ICV) drains the deep structures of the brain. The rest of the structures given in the multiple choice are not typical locations commonly affected by ICV thrombosis. [Deep cerebral venous thrombosis often affects bilateral thalami and may involve the basal ganglia and deep white matter [4].]

Question 4: Regarding the imaging findings of internal cerebral vein thrombosis:

1. Vasogenic edema demonstrates restricted diffusion.
2. Cytotoxic edema demonstrates restricted diffusion. (Applies)
3. The acute thrombus may appear hypointense on T2-weighted imaging, mimicking normal flow void. (Applies)
4. The acute thrombus demonstrates decreased susceptibility on gradient echo sequence.
5. Edema in bilateral thalami appears hypodense on unenhanced CT. (Applies)

Explanation:

1. Vasogenic edema does not demonstrate restricted diffusion. There is diffusion-weighted imaging (DWI) hyperintensity but no corresponding decrease in apparent diffusion coefficient (ADC) values. Instead, ADC values can be high due to T2 shine-through effect. [On DWI/ADC, cytotoxic edema demonstrates restricted diffusion with DWI hyperintensity and corresponding low ADC values [4]. On the contrary, vasogenic edema does not demonstrate restricted diffusion and instead, demonstrates DWI hyperintensity with high ADC values due to the T2 shine-through effect.]

2. Cytotoxic edema demonstrates restricted diffusion with DWI hyperintensity and corresponding low ADC values. [On DWI/ADC, cytotoxic edema demonstrates restricted diffusion with DWI hyperintensity and corresponding low ADC values [4]. On the contrary, vasogenic edema does not demonstrate restricted diffusion and instead, demonstrates DWI hyperintensity with high ADC values due to the T2 shine-through effect.]

3. Normal flow voids in patent vessels are T2-weighted hypointense and an acute thrombus may also appear T2-weighted hypointense, mimicking a normal flow void. [GRE sequence is helpful in detecting acute thrombus where it may not be evident on other sequences, such as the T2W sequence where it is hypointense, mimicking the normal flow void of a patent vessel, and on T1-weighted (T1W) sequence where it is not yet hyperintense [6].]

4. The acute thrombus demonstrates increased susceptibility on gradient echo sequence. [A study in 2006 by Idbaih et al. found that GRE sequence had an approximate sensitivity of 90% in detecting CVT in its early stage of within a week as the thrombus would demonstrate increased susceptibility [6].]

5. Edema appears hypodense on unenhanced CT and the thalami are commonly affected in internal cerebral vein thrombosis. [Cerebral edema appears hypodense on CT and has hyperintense T2W signal on MRI.]

Question 5: Regarding the imaging modalities used in detecting cerebral venous thrombosis:

1. CT venography has high sensitivity and specificity comparable to that of digital subtraction angiography. (Applies)

2. Slow blood flow may result in loss of normal flow void in MR venography, giving a false impression of thrombosis. (Applies)

3. Cerebral edema is better detected on CT compared to MRI.

4. CT venography, when compared with time-of-flight MR venography, better demonstrates venous anatomy for smaller veins with low flow. (Applies)

5. Unenhanced CT may be normal in patients with cerebral venous thrombosis. (Applies)

Explanation:

1. This statement is true. [CT venography has a high sensitivity and specificity in detecting CVT comparable with digital subtraction angiography in studies with small to moderate sample size [9].]

2. This statement is true. [Non-contrast MR venography utilizes flow-related techniques to evaluate for thrombosis... Non-contrast MR venography may falsely reflect absent flow signal in vessels with slow or in-plane blood flow [11].]

3. MRI has a higher sensitivity in detecting subtle cerebral edema that may not be apparent on unenhanced CT. [Compared with CT venography, MRI has a higher sensitivity for demonstrating cerebral edema and smaller lesions not well seen on CT [9].]

4. Time-of-flight MR venography relies on flow-related techniques and may falsely reflect absent flow signal within small veins with low flow. CT venography, on the other hand, is able to demonstrate small veins with slow flow that are perfused by contrast-enhanced blood. [When (CT venography is) compared with TOF MR venography, it better demonstrates the venous anatomy for smaller veins with low flow, with equivalent accuracy in detecting CVT [10].]

5. This statement is true. [Nonetheless, up to 30% of patients with CVT have normal CT brain findings [9].]

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FIGURES

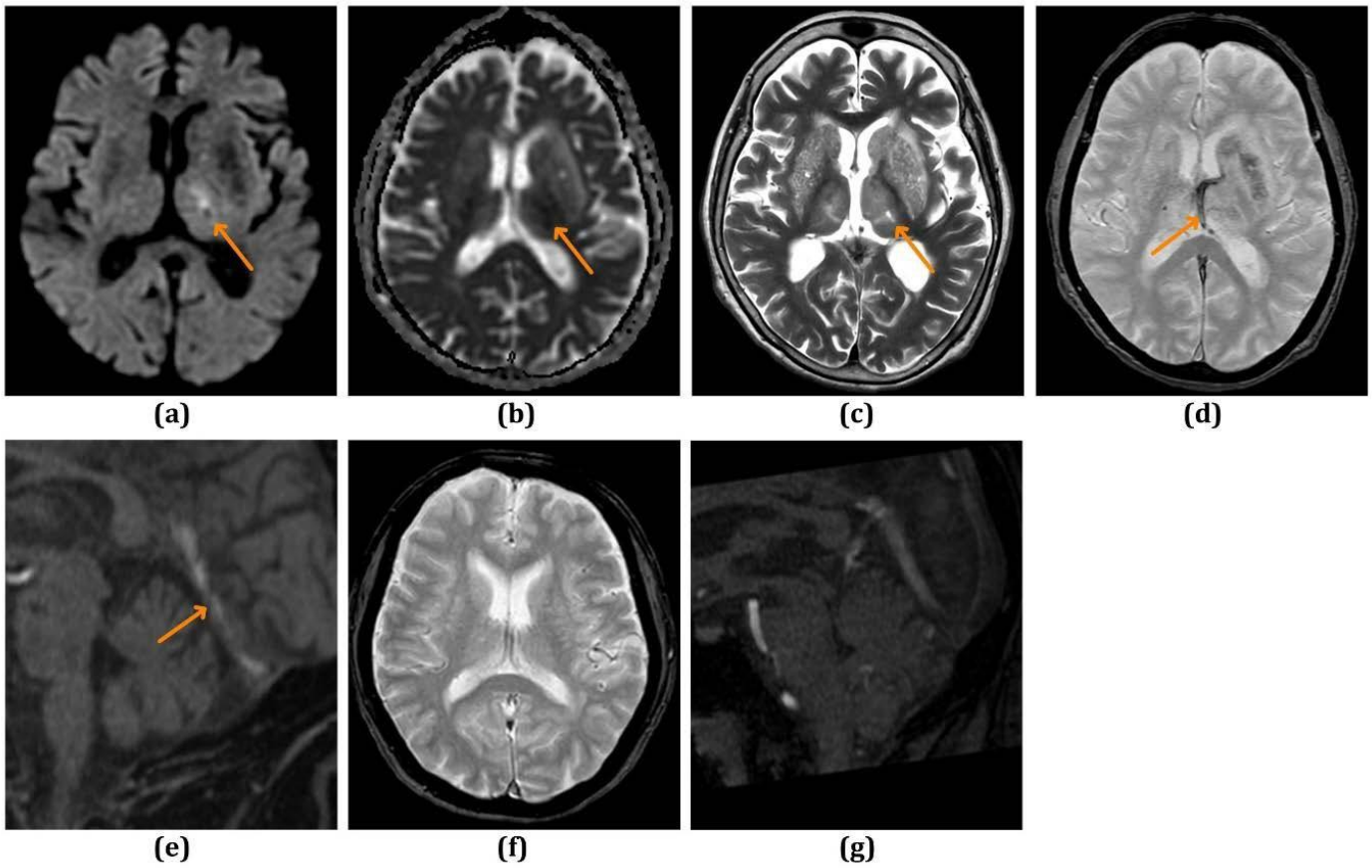

Figure 1: MRI images of a 91-year-old female with left internal cerebral vein thrombosis.

TECHNIQUE: Non-contrast MRI brain with T2-weighted fat suppressed (T2W FS) sequence, diffusion-weighted imaging (DWI) with apparent diffusion coefficient (ADC) map, gradient echo sequence (GRE), and time-of-flight magnetic resonance angiography (TOF MRA).

FINDINGS: On MRI brain, **(a)** Axial DWI image shows focal hyperintense DWI signal in the left thalamus (arrow) with corresponding **(b)** hypointense ADC signal (arrow), suggestive of restricted diffusion. **(c)** Axial T2W FS image shows slight asymmetry of the thalami with swelling of the left thalamus. Hyperintense T2W signal was seen in bilateral caudate and lentiform nuclei as well as both thalami, worse on the left (arrow). **(d)** Axial GRE image shows curvilinear susceptibility along the course of the left internal cerebral vein (arrow). Faint ill-defined susceptibility was also seen in the left caudate head and left putamen, suggestive of petechial hemorrhage. **(e)** Reformatted TOF MRA image shows linear hyperintensity along the straight sinus, raising suspicion of T1-weighted hyperintense blood products (arrow). Comparison with **(f)** axial GRE and **(g)** reformatted TOF MRA images from a normal study shows that there should not be any abnormal increased susceptibility in a patent internal cerebral vein on GRE sequence. In addition, a normal patent straight sinus will appear smooth without an abnormally elevated signal on MRA.

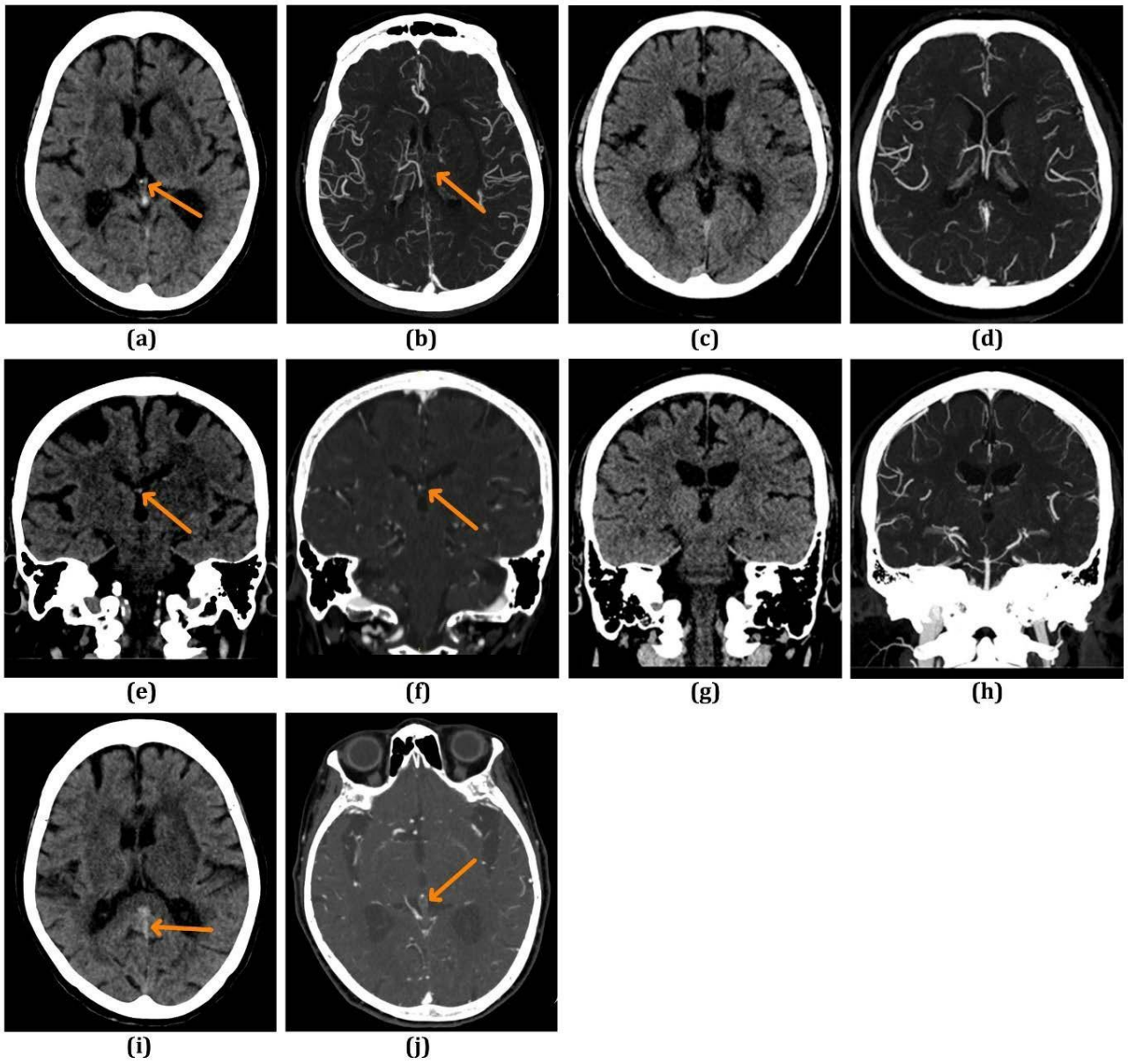

Figure 2: CT images of a 91-year-old female with left internal cerebral vein thrombosis.

TECHNIQUE: Unenhanced CT brain and contrast- enhanced CT venography of the brain. Both axial and coronal planes were imaged. Intravenous contrast used in CT venography: 55ml Omnipaque 350.

FINDINGS: The radiologist raised suspicion for acute left internal cerebral vein thrombosis and recommended CT venogram for further evaluation. Unenhanced axial CT brain shows the **(a)** left internal cerebral vein (ICV) appearing hyperdense (arrow) relative to the right ICV with **(b)** corresponding filling defect of the left ICV (arrow) seen on axial CT venogram. There is preserved contrast opacification of the right ICV. A thin sliver of contrast is also seen in the straight sinus and vein of Galen. Hypodensities in bilateral basal ganglia and thalami on unenhanced CT represent edema detected on the prior MRI study. Compare these with axial images from a normal study **(c)** and **(d)** where both patent internal cerebral veins are iso-dense with the same attenuation and demonstrate normal contrast opacification.

Findings were correlated on **(e)** unenhanced coronal CT brain and **(f)** coronal CT venogram which again demonstrate a hyperdense left ICV with filling defect (arrows). Compare these with coronal images from a normal study **(g)** and **(h)** where again, both patent internal cerebral veins have normal attenuation with contrast opacification. **(i)** Unenhanced axial CT brain shows a hyperdense straight sinus with **(j)** corresponding filling defect seen on axial CT venogram.

Overall, these findings confirm thrombosis of the deep cerebral venous system with unilateral complete thrombosis of the left ICV and partial thrombosis of the straight sinus and vein of Galen.

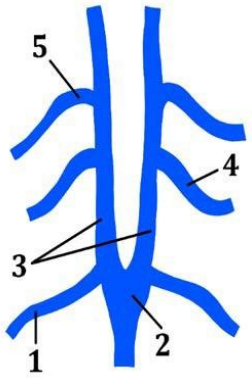

Figure 3: An illustration of the deep cerebral venous system**.** 1: Basal vein of Rosenthal (these veins join the internal cerebral veins to form the vein of Galen), 2: Vein of Galen, 3: Internal cerebral veins, 4: Direct lateral vein, 5: Thalamostriate vein

Table 1: Summary of the imaging features of left internal cerebral vein (ICV) thrombosis

Imaging modality	Imaging features of deep cerebral venous thrombosis
Unenhanced CT	• Hypodensities in bilateral basal ganglia and thalami representing edema. • Hyperdense left ICV representing the acute thrombus.
Contrast-enhanced CT venography	• Contrast filling defect in the left ICV representing thrombosis.
Unenhanced MRI	• Small focus of restricted diffusion in the left thalamus, representing cytotoxic edema from ischemia. • Disproportionate T2W hyperintensities in bilateral thalami and basal ganglia which do not demonstrate restricted diffusion, a result of concomitant vasogenic edema. • Acute thrombus within the left ICV demonstrates abnormal increased susceptibility on GRE images. • Acute thrombus in the left ICV appears T2W hypointense, indistinguishable from the normal T2W hypointense flow voids of patent vessels.

*CT: Computed Tomography, CVT: Cerebral Venous Thrombosis, GRE: Gradient Echo Sequence, ICV: Internal Cerebral Vein, MRI: Magnetic Resonance Imaging, T2W: T2-weighted

Table 2: Summary table for unilateral internal cerebral vein thrombosis

Etiology	Includes conditions that lead to a prothrombotic state.
Incidence	Unknown due to the small number of cases reported in literature.
Gender ratio	Unknown due to the small number of cases reported in literature.
Age predilection	Unknown due to the small number of cases reported in literature.
Risk factors	Thrombophilia, hematological disorders, usage of oral contraceptives, women in the puerperium period.
Treatment	Medical anticoagulation which includes subcutaneous low-molecular-weight heparin followed by conversion to oral anticoagulation therapy. Endovascular therapy for thrombectomy and thrombolysis is sometimes used in clinically deteriorating patients who do not respond to medical anticoagulation. Life-saving surgical decompressive craniectomy may be considered in severe cases of raised intracranial pressure.
Prognosis	Patients have a good prognosis if a timely diagnosis is made and early treatment initiated to reverse the outcomes. The prognosis is poor in patients who have delayed diagnosis and treatment. For patients diagnosed with deep cerebral venous thrombosis, 29.4% experience lifelong dependence or death.
Findings on imaging	Refer to Table 1

Table 3: Imaging features of cerebral venous thrombosis (CVT)

Imaging modality	Direct signs of CVT	Indirect signs of CVT
Unenhanced CT	Hyperdense thrombosed vein, described as "filled delta", "dense clot" or "cord" sign [9].	☐ Cerebral edema which appears hypodense. ☐ Infarcts that do not conform to major arterial territorial distributions. These appear hypodense. ☐ Acute hemorrhage which appears hyperdense.
Contrast-enhanced CT venogram	Filling defect in the thrombosed vein, “empty delta” sign [9].	Similar to unenhanced CT.
Unenhanced MRI	☐ Loss of flow signal in the thrombosed vein on phase-contrast and time-of-flight imaging. ☐ Thrombus within the vein can have varying signal intensities on T1W and T2W imaging [5]. ☐ Thrombus within the vein shows susceptibility on GRE imaging [6].	☐ Vasogenic edema is T2W hyperintense, does not demonstrate restricted diffusion [4]. ☐ Cytotoxic edema is T2W hyperintense, shows restricted diffusion [4]. ☐ Hemorrhage shows increased susceptibility on GRE imaging.
Contrast-enhanced MRI	Filling defect in the thrombosed vein.	Similar to unenhanced MRI.

†CT: Computed Tomography, CVT: Cerebral venous thrombosis, GRE: Gradient echo sequence, MRI: Magnetic Resonance Imaging, T1W: T1-weighted, T2W: T2-weighted

Table 4: MRI characteristics of thrombus

	Normal patent vessel	Age of thrombus < 5 days	Age of thrombus between 5 and 30 days	Age of thrombus > 1 month
T1	Hypointense	Isointense	Hyperintense	Iso- to hyperintense
T2	Hypointense	Hypo- to isointense	Iso- to hyperintense	Iso- to hyperintense

‡Table adapted from the article by Sadik et al. [5]

Table 5: Literature review of unilateral internal cerebral vein thrombosis

Author, article year	Patient age	Internal cerebral vein involved	Clinical symptoms
Akyuz M et al. [12], 2023	45-year-old	Right	Headache, left-sided weakness
Hu et al. [7], 2021	49-year-old	Left	Right hemiparesis, aphasia, altered mental status
Hu et al. [7], 2021	63-year-old	Left	Right hemiparesis, dysarthria
Tan A.P [13], 2020	15-year-old	Right	Headache, vomiting, lethargy
Herrmann KA et al. [4], 2004	47-year-old	Left	Headache, right upper limb paresis and reduced sensation
Wittkugel O et al. [14], 2004	61-year-old	Right	Left hemiparesis, drowsiness
Rousseaux M et al. [15], 1998	22-year-old	Left	Cognitive impairment
Crawford SC et al. [16], 1995	20-year-old	Left	Headache, right upper limb weakness
Crawford SC et al. [16], 1995	42-year-old	Left	Headache, altered mental status

Table 6: Differential diagnoses of internal cerebral vein (ICV) thrombosis and their imaging features

Differential diagnoses	Computed Tomography (CT)	Magnetic Resonance Imaging (MRI)	Similarities to ICV thrombosis and distinguishing imaging features
Artery of Percheron thrombosis	Unenhanced CT: • Hypodensities affecting both thalami, specifically in the paramedian distribution, representing edema [17]. • The midbrain may or may not be affected [17]. CT angiography: • No occlusion seen in the Circle of Willis (the artery of Percheron is typically not visualized even in normal patients due to its small caliber) [18]. CT venography: • Normal	Unenhanced MRI • T2W/FLAIR hyperintensities in both thalami, representing edema [17]. • Interpeduncular T2W/FLAIR hyperintensities in a "V-shaped" configuration along the surface of the midbrain [17]. • Restricted diffusion in bilateral thalami +/- midbrain, reflecting ischemic infarction [17]. MR angiography: • No occlusion seen in the Circle of Willis (the artery of Percheron is typically not seen even in normal patients due to its small caliber) [18]. MR venography: • Normal	Similarities • Bithalamic hypodensities on CT with T2W/FLAIR hyperintensities on MRI. • Restricted diffusion in bilateral thalami. • No occlusion visualized in the Circle of Willis. Distinguishing features: • The ICV is patent if CT/MR venography is performed. • The midbrain may be involved, which is not typical in ICV thrombosis.
Basilar tip thrombosis	Unenhanced CT: • Hypodensities affecting both thalami, representing edema. • Structures supplied by the posterior circulation including the cerebellum and occipital lobes can be affected with findings of hypodensities representing edema in these regions and loss of grey-white differentiation due to ischemia. CT angiography: • Loss of flow seen at the tip of the basilar artery due to thrombosis. CT venography: • Normal	Unenhanced MRI • T2W/FLAIR hyperintensities in both thalami, representing edema. These may also be present in the occipital lobes and cerebellum. • Restricted diffusion in bilateral thalami, occipital lobes and cerebellum, reflecting ischemic infarction. MR angiography: • Loss of flow void at the tip of the basilar artery. MR venography: • Normal.	Similarities • Bithalamic hypodensities on CT with T2W/FLAIR hyperintensities on MRI. • Restricted diffusion in bilateral thalami. Distinguishing features: • The occipital lobes and cerebellum may be involved, which is atypical for ICV thrombosis. • The ICV is patent if CT/MR venography is performed. • CT/MR angiography will demonstrate loss of flow in the tip of the basilar artery, while flow in the basilar artery is normal in ICV thrombosis.
Viral encephalitis (e.g. flavivirus infection)	Unenhanced CT: • Hypodensities in bilateral thalami, basal ganglia and midbrain, representing edema [19]. CT angiography: • Normal CT venography: • Normal	Unenhanced MRI: • T2W/FLAIR hyperintensities affecting bilateral thalami, basal ganglia and midbrain, usually with symmetrical distribution [17]. • Restricted diffusion in the thalami may be observed [17]. MR angiography: • Normal MR venography: • Normal	Similarities • Bithalamic hypodensities on CT with T2W/FLAIR hyperintensities on MRI. • Restricted diffusion in bilateral thalami. Distinguishing features: • The ICV is patent if CT/MR venography is performed. • The midbrain may be involved, which is not typical in ICV thrombosis.

Wernicke encephalopathy	Unenhanced CT: • Usually normal CT angiography: • Normal CT venography: • Normal	Unenhanced MRI: • T2W/FLAIR hyperintensities affecting the dorsomedial thalami, mamillary bodies, periaqueductal grey matter and midbrain tectal plate [17]. • Restricted diffusion may be seen in the thalami for alcohol-related Wernicke encephalopathy [20]. MR angiography: • Normal MR venography: • Normal	Similarities • Bithalamic hypodensities on CT with T2W/FLAIR hyperintensities on MRI. • Restricted diffusion in bilateral thalami. Distinguishing features: • On MRI, the mamillary bodies, periaqueductal grey matter and midbrain tectal plate are commonly involved, which is not seen in ICV thrombosis. • The ICV is patent if CT/MR venography is performed.
Hyperammone-mia	Unenhanced CT: • May present as cerebral edema with effacement of the ventricles and cerebral sulci [21]. CT angiography: • Normal CT venography: • Normal	Unenhanced MRI: • Symmetrical T2W/FLAIR hyperintensities seen in the cortex of bilateral insula and cingulate gyrus, both thalami and cerebral cortex (usually sparing the occipital cortices) due to ammonia toxicity and edema [17]. • Restricted diffusion may be seen in these affected regions [17]. MR angiography: • Normal MR venography: • Normal	Similarities • T2W/FLAIR hyperintensities in bilateral thalami Distinguishing features: • The cerebral cortex, cortices of the insula and cingulate gyrus are commonly involved, which is not typical for ICV thrombosis. • The ICV is patent if CT/MR venography is performed.
Bithalamic glioma	Unenhanced CT: • Enlarged and hypodense thalami [22]. • Mass effect from enlarged thalami due to underlying tumor [17]. CT angiography: • Normal CT venography: • Normal	Unenhanced MRI: • T2W/FLAIR hyperintensities affecting bilateral thalami [17]. MR angiography: • Normal MR venography: • Normal	Similarities • T2W/FLAIR hyperintensities in bilateral thalami • Restricted diffusion in bilateral thalami. Distinguishing features: • Significant mass effect of both thalami due to underlying tumor • The ICV is patent if CT/MR venography is performed.

§ CT: Computed Tomography, FLAIR: Fluid attenuated inversion recovery, ICV: internal cerebral vein, MRI: Magnetic Resonance Imaging, T2W: T2-weighted

KEYWORDS

Unilateral internal cerebral vein thrombosis; internal cerebral vein thrombosis; cerebral venous thrombosis; computed tomography; magnetic resonance imaging

ABBREVIATIONS

ADC = Apparent Diffusion Coefficient
CT = Computed Tomography
CVT = Cerebral Venous Thrombosis
DWI = Diffusion Weighted Imaging
GRE = Gradient Echo Sequence
ICV = Internal Cerebral Vein
MRI = Magnetic Resonance Imaging
TOF MRA = Time-of-flight Magnetic Resonance Angiography
TSV = Thalamostriate Vein
T1W = T1-weighted
T2W = T2-weighted

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