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Cerebral Venous Sinus Thrombosis Diagnosis: A Review of the Imaging Techniques

Mohammad Amin Shajareh Pour Salavati[1], Asma Payandeh[2], Ali Madadi Mahani[1], Seyed Mojtaba Mousavi[3]*

[1] *Student Research Committee, School of Medicine, Kerman University of Medical Sciences, Kerman, Iran.*

[2] *Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad, Iran.*

[3] *Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.*

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ABSTRACT

Thrombosis of the cerebral venous sinuses is relatively uncommon but associated with high mortality. Cerebral venous sinus thrombosis (CVST) is considered one of the most complex disorders of the nervous system. Therefore, timely diagnosis and appropriate treatment are essential to ensure effective management and improve patient outcomes. The diagnosis of cerebral venous thrombosis (CVT) requires a high index of clinical suspicion, which should be confirmed through neuroimaging—most commonly computed tomography (CT) or magnetic resonance imaging (MRI) with contrast-enhanced venography to detect thrombus formation within the venous sinuses. Laboratory investigations are also performed to identify potential underlying causes, such as coagulation disorders, systemic infections, or inflammatory conditions. Furthermore, screening for prothrombotic states is critical, as these may increase the risk of CVT. This review aims to highlight the diagnostic aspects of CVST and provide a clear overview of the imaging modalities that facilitate its early detection and diagnosis.

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Introduction

Cerebral venous sinus thrombosis (CVST) is recognized as one of the most complex disorders of the nervous system. Epidemiological studies have indicated that the annual incidence of CVST ranges from approximately 2 to 7 cases per million individuals in the general population. Moreover, CVST accounts for 0.5% to 2% of all strokes, with reported mortality rates ranging between 30% and 50% (1). Consequently, timely diagnosis and appropriate treatment are essential for effective management and improved patient outcomes, as treatment delays may result in severe neurological complications, including parenchymal hemorrhage, venous infarction, and subarachnoid hemorrhage (2). Despite the importance of prompt recognition, an estimated 25% of CVST cases remain undiagnosed until approximately 13 days after the onset of symptoms. The broad and often non-specific clinical manifestations—such as headache, confusion, focal neurological deficits, and seizures—pose significant challenges to accurate diagnosis, thereby complicating the overall diagnostic process (3).

Although magnetic resonance venography (MRV) is regarded as the gold standard for diagnosing cerebral venous sinus thrombosis (CVST), it is often unavailable in emergency settings. The diagnosis of CVST using magnetic resonance imaging (MRI) relies on the identification of abnormal signal intensities within the affected venous segments, which result from alterations in blood flow and the degradation products of

***Corresponding author**: Ali Madadi Mahani, Student Research Committee, School of Medicine, Kerman University of Medical Sciences, Kerman, Iran.*
Email: madadimahaniali@gmail.com

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hemoglobin within the thrombus (4). However, MRI poses diagnostic challenges in many cases of CVST, as the subtle and time-dependent variations in signal intensity on non-enhanced MRI can frequently be misinterpreted as normal flow-related artifacts. While MRV provides valuable diagnostic information, its use is limited by both economic and time-related constraints. Conversely, non-contrast brain computed tomography (CT) remains the only readily available imaging modality in most emergency scenarios (5). A typical finding in patients with CVST on CT is the direct visualization of a hyperattenuating thrombus within the occluded sinus. In other cases, non-contrast CT may reveal only indirect signs of thrombosis, such as diffuse cerebral edema or parenchymal hemorrhage (6).

This review aims to highlight the key aspects of CVST diagnosis and provide a clear overview of the imaging techniques that facilitate its early detection and diagnosis.

In detail, the review was conducted through a comprehensive literature search published between 2013 and 2025. The databases searched included PubMed, Scopus, and Google Scholar. The following keywords were used: *"cerebral venous sinus thrombosis," "CVST," "MRI," "MRV," "CT scan," "CT venography,"* and *"digital subtraction angiography."* The inclusion criteria consisted of peer-reviewed articles in English that focused on the imaging diagnosis of CVST. Both pediatric and adult populations were considered. Articles that primarily addressed treatment without a diagnostic context were excluded.

CVST Pathophysiology and Clinical Manifestations

The internal cerebral veins, which drain blood from the deep cortical regions, converge to form the great cerebral vein (also known as the vein of Galen). Both deep and superficial cortical veins have multiple drainage pathways that connect directly to the major dural sinuses. From these sinuses, blood flows into the straight sinus and ultimately exits the brain through the internal jugular vein. This extensive venous network provides sufficient collateral circulation in the event of an obstruction. Thrombosis most commonly involves the superior sagittal and lateral sinuses, occurring separately in approximately 70% of cases. In the remaining 30%, both sinuses may be affected simultaneously, often accompanied by involvement of the cortical and cerebellar veins (7).

Cerebral venous thrombosis (CVT) arises from a disruption of local or systemic coagulation balance, leading to clot formation within the dural sinuses or cerebral veins. When the dural sinuses are involved, venous outflow is impaired and cerebrospinal fluid (CSF) reabsorption is reduced, resulting in increased venous and capillary pressure (8, 9). When collateral circulation is well developed, clinical manifestations may be limited to symptoms of elevated intracranial pressure. However, when collateral pathways are insufficient or the thrombosis extends to cortical veins, intracranial pressure may rise to levels that compromise cerebral perfusion, thereby increasing the risk of ischemia (10). CVT can also lead to focal brain edema due to elevated retrograde venous pressure. This may be accompanied by venous engorgement, scattered petechial hemorrhages that can coalesce into larger hematomas, and localized ischemic injury. Parenchymal brain lesions occur in approximately 60% of CVT cases. Unlike arterial strokes, these lesions frequently extend across arterial territories, contain hemorrhagic components in nearly two-thirds of patients, and commonly demonstrate both vasogenic and cytotoxic edema.

The clinical manifestations of cerebral venous thrombosis (CVT) are often non-specific and vary depending on factors such as the location and extent of thrombosis, the patient's age, and underlying comorbidities. Symptoms may present either in isolation—such as intracranial hypertension (ICH) or focal neurological deficits—or as a combination of both, depending on the degree of venous obstruction and its impact on brain tissue (13, 14). Approximately 40% of patients experience an acute stroke-like presentation within 48 hours of symptom onset. Headache, which may be acute or subacute, is the most common presenting symptom and is frequently observed in the absence of other neurological abnormalities. The predominant clinical features are signs of increased intracranial pressure and impaired venous drainage. These include headache (70–90% of cases), seizures (30–40%), papilledema (30–60%), focal neurological deficits (30–50%), aphasia (15–20%), altered consciousness (15–25%), coma (5–15%), and, rarely, movement disorders (15–17).

CVST Diagnostic Modalities

The diagnosis of cerebral venous thrombosis (CVT) requires a high index of clinical suspicion, which should be confirmed through imaging—most

commonly a computed tomography (CT) scan or magnetic resonance imaging (MRI) with contrast-enhanced venography to detect thrombus formation within the venous sinuses. Radiological findings suggestive of CVT may include an absence of normal blood flow in the affected sinus or evidence of ischemic changes secondary to impaired venous drainage (13, 18). Currently, there is no definitive laboratory test capable of excluding CVT in the acute phase. Nevertheless, blood tests are routinely performed to investigate potential underlying causes, such as coagulation disorders, systemic infections, or inflammatory conditions. Furthermore, screening for prothrombotic states is essential, as these conditions significantly increase the risk of developing CVT (19–21) (Figure 1).

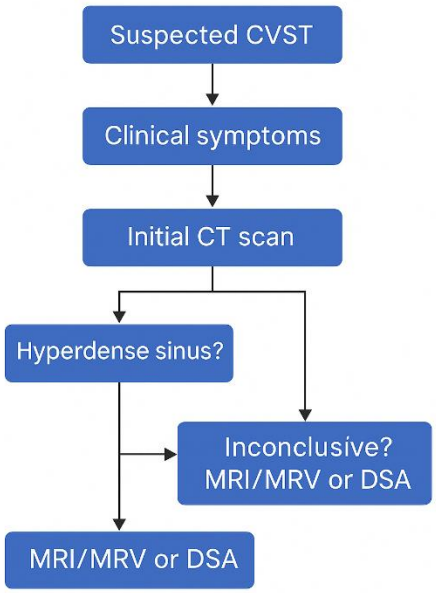

Figure 1: Graphical abstract of the Imaging workflow in CVST diagnosis

MRI and MRV

Conventional T1- and T2-weighted magnetic resonance imaging (MRI) sequences are more sensitive than non-contrast computed tomography (CT) in detecting cerebral venous thrombosis (CVT). Early MRI indicators include the absence of normal venous flow patterns and abnormal signal intensities within the dural venous sinuses (22, 23). Magnetic resonance venography (MRV) is particularly valuable in both acute and subacute settings, whether in emergency care or outpatient evaluations. It is beneficial for confirming suspected deep venous thrombosis when CT venography results are inconclusive or normal. Contrast-enhanced MRV provides superior visualization of cerebral venous structures and is less affected by irregular blood flow patterns (24, 25). Nevertheless, MRV has certain limitations. Anatomical variations, such as aplasia or hypoplasia of the transverse sinus, may be misinterpreted as thrombosis of the transverse sinus. In addition, signal loss due to in-plane flow or hyperintense thrombi can mimic normal venous flow in time-of-flight (TOF) angiography (26). Despite these challenges, both MRV and CT venography remain reliable diagnostic modalities for CVT, with MRV demonstrating greater accuracy in detecting associated parenchymal abnormalities (27).

CT Scan and CT venography

When cerebral venous thrombosis (CVT) is clinically suspected, an unenhanced computed tomography (CT) scan of the brain is often the preferred initial, noninvasive imaging modality (28). In the acute phase, CVT may appear as a hyperdense, elongated clot within a cortical vein—commonly referred to as the “cord sign”—which is typically visible for up to two weeks before becoming isodense with the surrounding brain tissue (29). Non-contrast CT findings are usually indirect and may include both early and late indicators of venous ischemia, such as sulcal effacement, diffuse cerebral edema, loss of gray–white matter differentiation, and ventricular compression (30). Infarcts that do not conform to arterial territories—for example, isolated subcortical lesions or multiple bilateral or unilateral areas, with or without hemorrhagic features—should raise suspicion of a venous origin. Furthermore, infarctions extending across multiple arterial territories may strongly suggest an underlying venous etiology, particularly CVT (31).
Computed tomography venography (CTV) is particularly valuable in acute and emergency settings, serving as an effective initial modality for evaluating both the deep and superficial venous systems, especially in patients who are unresponsive or uncooperative (32). The hallmark finding on CTV is a vascular filling defect, with the classic “empty delta sign” observed when the superior sagittal sinus is involved. However, diagnostic accuracy may be limited by dense bone artifacts and by arachnoid granulations, which can protrude into venous sinuses and mimic thrombotic filling defects (33). In neonates, false-positive hyperdense clot signs may occur due to naturally hyperdense venous sinuses, while an *“empty delta sign”* may also arise in the setting of

hyperdense subdural empyemas (34). Additionally, venous malformations, when dilated and congested, may appear hyperdense on unenhanced CT. On CTV, they demonstrate a characteristic pattern described as the *"caput medusae"* or *"candelabra"* sign, reflecting multiple enhancing veins converging into a single enlarged draining vein (31, 35).

Digital subtraction angiography (DSA)

Although digital subtraction angiography (DSA) is considered the gold standard for diagnosing cerebral venous thrombosis (CVT), its use is generally limited to cases in which findings from computed tomography venography (CTV) or magnetic resonance venography (MRV) are inconclusive, or when endovascular intervention is being planned, due to the invasive nature of the procedure and its associated risks (36). Typical angiographic findings include intraluminal filling defects within the dural venous sinuses or cortical veins, delayed venous outflow, and the presence of dilated collateral vessels. In some instances, abrupt termination of cortical veins accompanied by tortuous, dilated collateral channels produces the characteristic "corkscrew" appearance (37).

In addition, digital subtraction angiography (DSA) can identify vascular abnormalities such as aneurysms or dural arteriovenous fistulas, which may mimic the "corkscrew" appearance due to impaired venous drainage and vascular congestion (38). A significant advantage of DSA is its capacity to directly measure venous pressure; values exceeding 10 mm H_2O indicate an increased likelihood of parenchymal injury, thereby providing important prognostic information and guiding therapeutic decision-making (39, 40).

A comparative overview of these three imaging modalities for CVST is presented in Figure 2(Figure2).

Non-contrast CT scan vs. MRV

In this section, we compare the diagnostic utility of non-contrast computed tomography (CT) and magnetic resonance venography (MRV) in the evaluation of cerebral venous sinus thrombosis (CVST). Non-contrast brain CT remains the most widely used initial imaging modality for patients presenting with acute neurological symptoms and continues to play a critical role in the diagnostic workup of suspected acute venous sinus thrombosis. Several measurable indices—such as the mean attenuation of cerebral venous sinuses, the venous-to-arterial density ratio, the arterial–venous density difference, and the venous density–to–hematocrit ratio—have been proposed to improve the accuracy of CVST detection during CT image interpretation. Multiple studies have assessed the diagnostic performance of non-

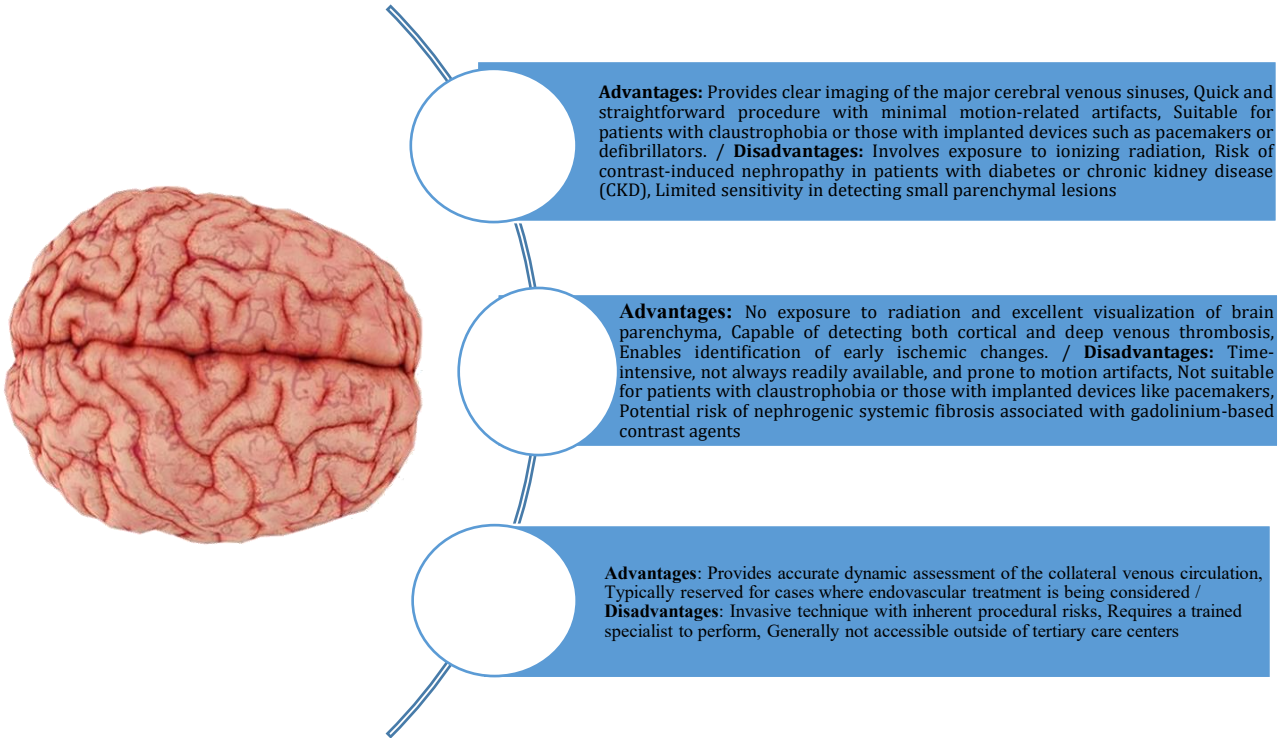

Figure 2: Advantages and disadvantages of CVST imaging modalities.

contrast CT in this setting, and their findings are summarized in Table 1(Table1).

Table 1: The comparison of the diagnostic value of the non-contrast CT scan and MRV for CVST

Study	Population	Variable	Optimal value	Sensitivity (%)	Specificity (%)
Tayyebi et al (2020) (30)	50 cases 73 control	Average venous density (HU)	61	82	100
		Venous-arterial density ratio (HU)	1.43	100	78
		Venous-arterial density differentiation (HU)	24	80	100
Singh et al (2019) (41)	38 cases 42 control	Average venous density (HU)	68	97.4	100
		Venous-arterial density ratio (HU)	-	-	-
		Venous-arterial density differentiation (HU)	-	-	-
Shayganfar et al (2019) (42)	35 cases 70 control	Average venous density (HU)	60.4	71.4	100
		Venous-arterial density ratio (HU)	-	-	-
		Venous-arterial density differentiation (HU)	-	-	-
Besachio et al (2013) (43)	25 cases 26 control	Average venous density (HU)	65	74	96
		Venous-arterial density ratio (HU)	-	-	-
		Venous-arterial density differentiation (HU)	15	80	94

HU: Hounsfield

Comparison with Diagnostic Guidelines

According to the European Stroke Organization (ESO) guidelines (44, 45), the preferred imaging modalities for CVST diagnosis are MRI combined with MRV or CT venography in acute settings. Our review is consistent with these recommendations, particularly in emphasizing MRV's superiority in tissue detail and CT's applicability in emergencies. However, despite the guideline emphasis on early imaging, our review of the studies shows that non-contrast CT often remains the primary modality due to its accessibility, especially in emergency settings. This divergence underscores the need for better implementation of guideline-based imaging pathways in clinical practice.

Conclusion

CVST is a critical condition that can lead to severe complications, including death. In addition to clinical manifestations, imaging modalities are essential for the rapid diagnosis of CVST. Different techniques, including MRI, MRV, CT, CT venography, and DSA, can be used in the diagnosis of CVST, each with its own advantages and limitations. However, the most essential factor is that both clinical manifestations and imaging modalities contribute to the most accurate and timely diagnosis of CVST, enabling the most effective treatment.

Declarations

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N.A

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