

Radiological Improvement in a Patient with Cerebral Venous Sinus Thrombosis

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ABSTRACT

Introduction: Cerebral venous sinus thrombosis (CVST) is a rare stroke subtype. Treatment with warfarin or a direct oral anticoagulant (DOAC) without heparinization can be initiated in the chronic phase and has been reported to be comparably effective between the two agents. This case report aims to discuss the radiological improvement in brain magnetic resonance imaging (MRI) in a patient with CVST after three months of oral anticoagulant therapy with warfarin.

Case Description: A 51-year-old male patient with CVST and hypertension reported headache for one month and dizziness with a sensation of swaying. Laboratory examination revealed elevated total and LDL cholesterol levels. Brain MRI demonstrated an empty delta sign in the confluent sinus and absence of signal in the left transverse sinus, consistent with CVST. The patient received warfarin 2 mg orally once daily. After three months of treatment, the patient was symptom-free, and brain MRI showed that the empty delta sign had partially disappeared, accompanied by the return of the flow signal in the left transverse sinus.

Conclusion: Anticoagulant therapy for CVST should be initiated promptly, with warfarin alone considered if the onset is chronic and the patient is clinically stable.

Anticoagulant, Cerebral Venous Sinus Thrombosis, MRI, Warfarin

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INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is a rare subtype of stroke that is often unrecognized because its clinical manifestations are varied and may be asymptomatic [1,2]. CVST occurs when a thrombus forms and potentially obstructs the dural venous sinus and/or related venous structures, such as the internal jugular vein or cerebral veins, representing 0.5–1% of all stroke cases [3]. The average annual incidence of CVST is 12 cases per million people, mainly affecting females of childbearing age (female-to-male ratio 3:1) [4]. Recent studies have shown changing trends by sex, with a higher occurrence in men than in women, a phenomenon linked to increased alcohol consumption, cultural access bias, or underdiagnosis in women [5,6]. CVST can present with symptoms ranging from mild to life-threatening, depending on which sinuses and veins are affected, the extent of brain tissue damage, its chronicity, and its impact on intracranial pressure [7].

The clinical features of CVST include headache, seizures, papilledema, focal neurological deficits, and altered mental status. CVST is classified into three categories based on onset: acute (≤ 15 days), subacute (15–30 days), and chronic (>30 days) [8]. Diagnosing CVST can be difficult because of its common risk factors, varied clinical symptoms, and occasionally, inconclusive neurological imaging. However, brain magnetic resonance imaging (MRI) using a susceptibility-weighted imaging (SWI) sequence is considered the most effective diagnostic modality [1]. Treatment should begin immediately once CVST is diagnosed, with prompt initiation of anticoagulant therapy, management of the underlying cause (such as dehydration, sepsis, or discontinuation of prothrombotic medications), control of seizures, and management of intracranial hypertension, when necessary [7]. The American Heart Association and European Stroke Organization guidelines recommend initiating anticoagulation with low-molecular-weight heparin (LMWH) rather than unfractionated heparin during the acute or hyperacute phase. Patients should subsequently transition to an oral vitamin K antagonist (VKA) for three–12 months [9,10]. Warfarin may be substituted with a direct oral anticoagulant (DOAC) [1]. Warfarin or DOAC alone, without heparinization, may be suitable for use during the chronic phase. A study by Wasay et al. found similar effectiveness between warfarin and DOACs in patients with CVST when treatment was started within 3–11 days, with 29% of patients not receiving prior heparinization [11]. Therefore, this case report aimed to discuss the radiological improvement in a patient with CVST after three months of warfarin administration.

CASE DESCRIPTION

A 51-year-old male patient, aged 51 years, presented to the neurology clinic for brain MRI. He was referred from a previous hospital after non-contrast head computed tomography (NCCT) raised suspicion of cerebral venous sinus thrombosis (CVST). He reported a headache lasting one month, characterized by a pressing sensation and a pain level of 3 on the numeric rating scale (NRS). He also reported dizziness accompanied by a sensation of swaying. He denied any double vision, slurred speech, facial drooping, or unilateral body weakness. On physical examination, the Glasgow Coma Scale (GCS) score was E4M6V5, and blood pressure was 146/97 mmHg. Neurological examination was within normal limits. Laboratory testing showed a total cholesterol level of 268 mg/dL and an LDL cholesterol level of 173 mg/dL. Contrast-enhanced brain MRI demonstrated an empty delta sign in the confluent sinus and absence of signal in the left transverse sinus, indicating CVST (Figure 1).

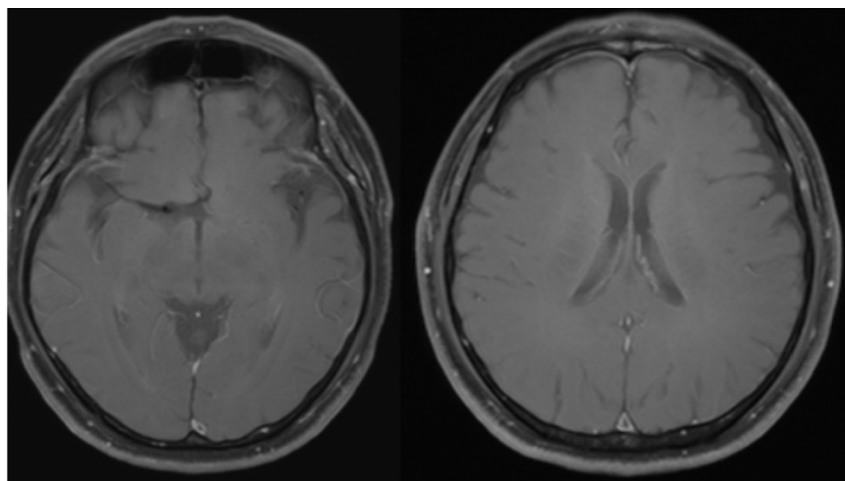


Figure 1. Brain MRI was performed with and without contrast. An empty delta sign in the confluent sinus and absence of signal in the left transverse sinus, consistent with cerebral venous sinus thrombosis (CVST).

The patient received warfarin 2 mg orally once daily, together with amlodipine 5 mg and atorvastatin 20 mg, both taken orally once daily. After three months of treatment, the symptoms resolved, with no adverse effects, such as bleeding, rash, or new neurological deficits. The International Normalized Ratio (INR) remained within normal limits (0.86). Repeat contrast-enhanced brain MRI showed that the empty delta sign

had partially disappeared, with the return of the flow signal in the left transverse sinus, and no new infarction or hemorrhage was identified (Figure 2).

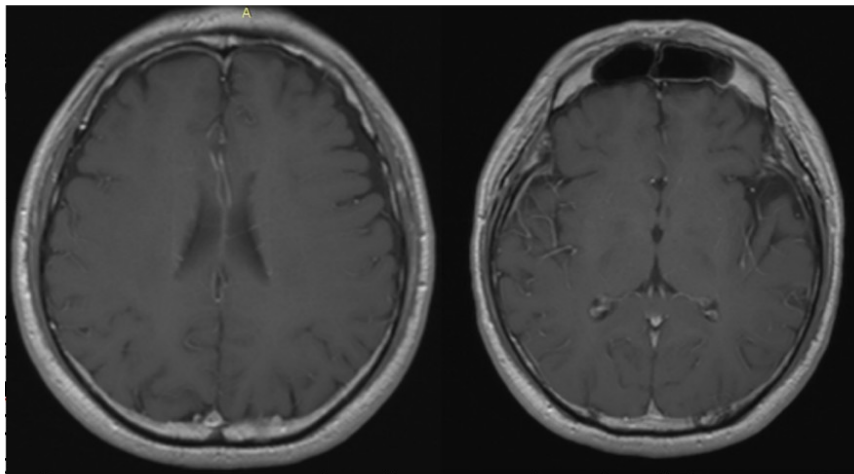


Figure 2. Brain MRI was performed with and without contrast after three months of therapy. The empty delta sign partially disappeared with the return of the flow signal in the left transverse sinus.

DISCUSSION

Based on the patient's complaints of headache and dizziness, together with the brain MRI findings, the presentation was consistent with CVST. Two main mechanisms underlie the pathophysiology of CVST: (1) obstruction of venous circulation impairs the absorption of cerebrospinal fluid, thereby increasing intracranial pressure; and (2) thrombosis obstructs blood flow and raises venous and capillary pressure, disrupting the blood–brain barrier. The latter leads to vasogenic edema, which may subsequently cause infarction or hemorrhagic lesions. These two mechanisms account for the three primary clinical syndromes associated with CVST: focal syndrome (caused by parenchymal lesions), intracranial hypertension syndrome (caused by reduced CSF absorption), and encephalopathy (caused by a combination of both processes or by strategic lesions, i.e., involvement of both thalami). Intracranial hypertension may present as a headache, as observed in this patient. Headache occurs in almost 90% of CVST cases. Clinical symptoms usually develop gradually and may appear several weeks before diagnosis, although a small proportion of cases present with acute onset [4].

Risk factors for CVST in this patient included hypertension and dyslipidemia. Hypertension leads to structural and functional modifications in blood vessels, the endothelium, and platelets, fostering a prothrombotic state. Endothelial dysfunction, heightened platelet activation, and alterations in coagulation factors all contribute to the increased risk of thrombosis [12,13]. Dyslipidemia in this patient was characterized by elevated total and LDL cholesterol levels. Elevated total cholesterol disrupts the coagulation cascade and causes endothelial dysfunction, producing a pro-inflammatory state that ultimately increases the risk of thrombosis [14]. Elevated LDL cholesterol can heighten platelet activation, increase tissue factor expression, and correlate with levels of vitamin K–dependent coagulation factors and fibrinogen; it also promotes the activation of prothrombin, factor X, and factor VII. Furthermore, oxidized LDL stimulates the release of cellular microvesicles, which play a crucial role in mediating both arterial and venous thrombotic processes [15]. The diagnosis of CVST relies on clinical symptoms and imaging findings [4]. NCCT or MRI is often the first examination performed in patients with acute nonspecific symptoms and may reveal findings suggestive of CVST. For instance, CVST on CT or MRI may be suspected when there is (1) direct visualization of a thrombus, (2) absence of venous filling, or (3) imaging evidence of venous obstruction at the tissue level—such as edema, hemorrhagic transformation, raised intracranial pressure, hydrocephalus, and venous infarction—as well as at the vascular level, including dilated veins [9]. NCCT may show specific signs, including hyperdensity of the venous sinus or deep veins, sometimes referred to as the dense triangle sign (high attenuation in the sagittal sinus or triangular-shaped deep cerebral veins) or the cord sign (high attenuation due to thrombus in the transverse sinus). NCCT can also detect ischemia (often with some hemorrhagic

transformation), parenchymal or subarachnoid hemorrhage, or signs of edema. However, NCCT may appear normal in up to 30% of patients and, even when abnormal, may be nonspecific. Therefore, all patients with suspected CVST require additional imaging beyond NCCT [7].

Brain MRI should include T1- and T2-weighted sequences, diffusion-weighted imaging (DWI), and hemorrhage-sensitive sequences (susceptibility-weighted imaging [SWI] or T2*-gradient-recalled echo [GRE]). T1- and T2-weighted images are helpful for identifying edema and characterizing thrombus age. DWI enables the distinction between vasogenic and cytotoxic edema. For precisely identifying bleeding clots and recognizing subtle thrombosis, SWI performs better than T2*-GRE sequences [16]. In the acute phase (days 1–5), there is an intermediate T1WI signal intensity, and low T2WI and T2* or SWI signal intensity due to deoxyhemoglobin and venous enlargement. In the early subacute phase (days 5–14), there is high T1WI signal intensity and low T2WI signal, because deoxyhemoglobin is converted to methemoglobin; these changes occur centripetally in large veins [16]. Owing to extracellular methemoglobin and enhanced peripheral dural intensity, the signal is high across all sequences in the late subacute phase (days 14–21), with a central filling defect seen following gadolinium injection. In the chronic phase (beyond day 21), the degree of clot organization determines signal strength. Organized and vascularized thrombi may exhibit increased intensity, although minimal or no enhancement may also be observed, and the veins are no longer dilated [16]. The primary advantage of MRI over computed tomography (CT) is its superior soft-tissue differentiation and higher sensitivity for detecting subtle brain pathology [17]. Conventional T1- and T2-weighted MRI sequences are more sensitive than non-contrast CT for diagnosing CVST [18]. In this patient, brain MRI performed with and without contrast revealed an empty delta sign in the confluent sinus and absence of signal in the left transverse sinus, consistent with CVST.

The American Heart Association and European Stroke Organization guidelines recommend initiating anticoagulation with LMWH rather than unfractionated heparin. Patients should subsequently transition to oral VKA for three–12 months when transient risk factors are present or indefinitely when chronic major risk factors for recurrent venous thrombosis or thromboembolism are present [10]. Treatment should be started promptly once the diagnosis of CVST is confirmed, with prompt anticoagulant therapy, management of the underlying cause (such as dehydration, sepsis, or discontinuation of prothrombotic medications), seizure control, and management of intracranial hypertension when necessary [7]. The aim of anticoagulant treatment in CVST is to halt thrombus expansion, promote recanalization, and prevent recurrent venous thromboembolism [9]. DOAC may also be used as alternatives to vitamin K antagonists, such as warfarin [1]. To date, no guidelines support the use of warfarin or DOAC alone without heparin in CVST, even in outpatient settings. Most studies comparing warfarin with DOAC in CVST have been preceded by heparin bridging therapy [19]. A study by Wasay et al. comparing the effectiveness of warfarin and DOAC in CVST showed comparable results; 71% of the participants received heparin before DOACs or warfarin, and 11% received surgical treatment [11]. The use of DOAC without bridging may be an option, as demonstrated in a study by Mutgi et al. [20]. Warfarin should be used cautiously without bridging therapy because of its slow onset of action. Therefore, warfarin or DOAC alone may be an option if the patient is clinically stable, such as when there are no signs of worsening headache or other symptoms of dizziness. INR monitoring is recommended within the first seven days of starting warfarin. The treatment duration should be at least three months and may be lifelong, depending on the cause and recurrence of CVST [7].

This patient received warfarin 2 mg orally once daily for three months. Warfarin competitively inhibits vitamin K epoxide reductase complex subunit 1 (VKORC1), which is essential for activating vitamin K, resulting in depletion of functional vitamin K stores and thereby lowering the synthesis of active clotting factors [21]. After three months of treatment, the patient was symptom-free, and the INR results were within normal limits. Repeat brain MRI revealed partial disappearance of the empty delta sign, accompanied by the return of flow signal in the left transverse sinus. A disappearing empty delta sign has been reported in multiple cases, vanishing between 12 days and 3 months, which suggests recanalization of the obstructed venous sinus. Several factors may explain this phenomenon, including the success of anticoagulation therapy and the presence of abundant venous collaterals [22]. During the chronic phase, the obstructed sinus typically develops

many new recanalization channels within the thrombus [23]. A small study by Geisbüsch et al. that assessed recanalization status using magnetic resonance angiography (MRA) and the modified Rankin Scale (mRS) in CVST patients receiving rivaroxaban showed that all patients experienced at least partial recanalization [24]. Age over 50 years and a cryptogenic etiology are recognized predictors of unsuccessful recanalization in CVST patients treated with anticoagulants [25].

CONCLUSION

In patients with suspected CVST, further investigation is necessary to confirm the diagnosis, and brain MRI is the optimal diagnostic test. Anticoagulant therapy for CVST should be initiated promptly; warfarin alone may be considered if the onset is chronic and the patient is clinically stable. The success of therapy can be evaluated both clinically and radiologically.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

INFORMED CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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COMPETING INTERESTS

The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

C.M.A. was responsible for manuscript writing and literature review. Y.I. performed data acquisition and critically revised the manuscript. M.R. provided radiological interpretation and revised the manuscript. I.R., T.K., M.A.P., and P.A. contributed to the revision of the manuscript. M.I.A.R. contributed to manuscript writing and literature review. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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