

Aggressive Manifestation of Cerebral Venous Sinus Thrombosis: Rapid Progression from Hemiparesthesia to Spastic Tetraparesis Within 24 Hours in a Young Male – A Case Report

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ABSTRACT

Cerebral venous thrombosis (CVST) is an uncommon cause of stroke, accounting for less than 1% of cerebrovascular events worldwide, and predominantly affects young adults with highly variable and nonspecific neurological manifestations. Delayed recognition may lead to significant neurological deterioration due to venous congestion, cerebral edema, and venous infarction. We report a 33-year-old male who presented with acute right-sided hemiparesthesia upon awakening, preceded by five days of persistent occipital headache. Within hours of admission, he developed right-sided hemiparesis, dysarthria, supranuclear cranial nerve VII and XII palsy, and recurrent generalized seizures. Non-contrast computed tomography revealed a hypodense lesion in the left parietal lobe with hemorrhagic components consistent with venous infarction, along with hyperdensity in the superior sagittal sinus suggestive of cerebral venous sinus thrombosis. The patient was managed with antiepileptic therapy and close supportive care. Within 24 hours, he experienced rapid neurological deterioration progressing to spastic tetraparesis. This case highlights an aggressive clinical course of cerebral venous sinus thrombosis and underscores the importance of early recognition in young patients presenting with acute neurological deficits, particularly when preceded by persistent headache or accompanied by seizures, to prevent delayed diagnosis and severe neurological complications.

Keywords: Cerebral Venous Thrombosis, Cerebral Venous Sinus Thrombosis, Venous Infarction, Tetraparesis, Seizure

INTRODUCTION

Among cerebrovascular diseases, cerebral venous thrombosis (CVST) stands apart as a rare but distinct entity, accounting for less than 1% of all stroke cases worldwide (Almegren, 2025; Liu et al., 2024)). Unlike arterial ischemic stroke, CVST disproportionately affects younger adults, particularly those below 50 years of age, making it a clinically important cause of stroke in this population (Almegren, 2025; Zuo et al., 2025). Growing clinical awareness alongside advances in neuroimaging have contributed to the increasing recognition of this condition in recent years (Zuo et al., 2025).

Mechanistically, CVST arises from thrombosis of the cerebral veins or dural venous sinuses, which obstructs venous outflow from the brain. This obstruction produces venous congestion, raised intracranial pressure, and reduced cerebral

perfusion, ultimately culminating in cerebral edema, venous infarction, and hemorrhagic transformation of the affected brain parenchyma(Wang et al., 2024). The extent of resulting neurological injury depends on several interacting factors, including the site and extent of thrombosis, the adequacy of collateral venous drainage, and the presence of concurrent parenchymal lesions(Khan et al., 2020).

The clinical picture of CVST is notoriously heterogeneous and often nonspecific, a feature that frequently delays diagnosis. Headache is the most commonly reported presenting symptom, occurring in the majority of patients, while other manifestations span seizures, focal neurological deficits, papilledema, and altered consciousness (Almegren, 2025; Jamadar et al., 2025). Because these features overlap considerably with numerous other neurological conditions, CVST is prone to early misdiagnosis, particularly in young patients who lack conventional vascular risk factors (Zuo et al., 2025).

Several venous sinuses may be affected in CVST, with the superior sagittal and transverse sinuses reported as the most frequently involved structures; multi-sinus involvement is also commonly observed in clinical practice (Mirdamadi et al., 2024; Zuo et al., 2025). Thrombosis at these sites frequently results in venous infarction, which often manifests as hemorrhagic infarction owing to impaired venous drainage and disruption of the blood–brain barrier (Wang et al., 2024). The presence of parenchymal brain lesions and associated neurological deficits has, in turn, been linked to poorer functional outcomes among CVST patients (Lin et al., 2025).

Neuroimaging is indispensable to establishing the diagnosis of CVT. Non-contrast computed tomography (CT) may reveal indirect clues such as venous infarction, hemorrhagic lesions, or hyperdensity within the affected sinus, but magnetic resonance imaging (MRI) combined with magnetic resonance venography (MRV) remains the most sensitive modality for confirming venous sinus thrombosis and delineating the venous structures involved (Khan et al., 2020; Uminski et al., 2026).

Despite these diagnostic and therapeutic advances, the clinical trajectory of CVST remains difficult to predict. While many patients experience mild symptoms with gradual neurological progression, others deteriorate rapidly as a result of extensive venous infarction or intracranial hypertension(Ruderman et al., 2025). We report the case of a young male who presented with an aggressive manifestation of cerebral venous thrombosis, marked by rapid neurological deterioration from hemiparesthesia to spastic tetraparesis within 24 hours, accompanied by recurrent seizures and venous infarction.

The purpose of this report is to describe the diagnostic and management challenges posed by an aggressive presentation of CVST in a young, previously healthy male, and to draw attention to the pace at which neurological deterioration can occur once venous infarction and cortical vein involvement are established. Reporting this case from a regional emergency and neurology service in Bali, Indonesia, is particularly relevant given that CVST in this setting is uncommonly encountered, and definitive diagnostic and interventional resources — including MRI/MRV and neurointervention — are concentrated in a small number of tertiary referral centres. Delayed recognition in this context translates directly into delayed access to guideline-directed therapy; documenting the clinical trajectory and decision-making process in this case is therefore intended to help clinicians in

similarly resourced settings recognise the condition earlier and initiate a timely referral pathway.

METHODS

This report was prepared as a single case report describing the clinical course, diagnostic findings, and management of a patient with cerebral venous sinus thrombosis, drawing on an acute admission jointly managed by the Emergency Department and Department of Neurology of Bangli Regional Hospital, with referral coordination involving the Emergency Department of Klungkung Regional Hospital. The exact calendar date of admission is withheld to preserve patient anonymity, consistent with case-report de-identification standards, although the sequence and timing of events during the admission are reported in full using relative clock times. Reporting followed the CARE (Case Report) guidelines to ensure completeness and transparency.

Data were obtained through two complementary approaches: a retrospective review of the patient's presenting history and baseline clinical status as documented at first contact, and prospective, serial documentation of neurological findings, vital signs, and treatment response by the attending emergency and neurology team throughout the admission. Data sources comprised the structured admission history and physical examination, serial neurological examinations performed at each significant change in clinical status, venous blood sampling sent for complete blood count, electrolytes, renal function, glucose, albumin, and HIV serology, and non-contrast computed tomography of the brain together with a screening chest radiograph.

The case study proceeded through five stages: (1) initial triage and baseline neurological assessment in the emergency department; (2) serial neurological reassessment triggered by each new clinical finding, with vital signs and Glasgow Coma Scale recorded at every reassessment; (3) urgent non-contrast CT imaging once focal deficits and seizures emerged, to identify a structural correlate for the deterioration; (4) laboratory evaluation to exclude a metabolic, infectious, or haematological contributor to the presentation; and (5) initiation of supportive, antiepileptic, and antiplatelet therapy alongside referral planning to a tertiary centre with neurointervention capability.

Written informed consent was obtained from the patient for publication of this case report together with the accompanying images. The patient was informed that all personal identifying information would be anonymized to protect confidentiality. In accordance with institutional policy, formal ethical clearance is not required for a single case report of this nature.

RESULTS AND DISCUSSION

Case Presentation

A 33-year-old man presented to the emergency department at 09:00 with numbness affecting the right side of his body that had begun upon awakening that same morning. Five days before admission, he had developed a persistent occipital headache radiating to the back of the neck; having sought care previously, he had been told the symptom was likely related to elevated blood pressure. He also described intermittent tinnitus in the right ear.

He denied fever, diplopia, dysarthria prior to admission, vertigo, nausea, vomiting, or head trauma, and had no history of hypertension, diabetes mellitus,

cardiovascular disease, renal disease, or autoimmune disorders. He had taken analgesics and a vitamin supplement before presenting, and reported no known drug allergies.

Clinical Findings

On arrival, the patient appeared moderately unwell but fully conscious. Initial vital signs showed a blood pressure of 140/70 mmHg, heart rate 92 beats/min, respiratory rate 16 breaths/min, temperature 36.9°C, and oxygen saturation 98% on room air. General examination was unremarkable. Neurologically, he scored E4V5M6 on the Glasgow Coma Scale, indicating full consciousness, and exhibited right-sided hemiparesthesia without motor weakness. Cranial nerve examination was normal, there were no meningeal signs, and pathological reflexes were absent.

Clinical Course

Approximately five hours after admission, at 14:00, the patient developed new deficits: weakness of the right side of the body with slurred speech. Repeat neurological examination revealed right supranuclear facial (cranial nerve VII) and hypoglossal (cranial nerve XII) palsy, together with flaccid right hemiparesis graded 3/5 on motor testing; the Babinski reflex was equivocal.

At 17:00, he had a generalized tonic seizure with whole-body convulsions and upward eye deviation lasting approximately 30 seconds, followed shortly afterward by a second episode lasting about one minute. Seizure activity resolved with intravenous diazepam, and the patient regained full consciousness roughly an hour later before being transferred to the intensive care unit for close monitoring. During ICU observation he had seven further brief seizures of 10–30 seconds each, remaining conscious and responsive between episodes; the last was recorded at 21:30. Given the progressive neurological decline, plans were made to refer him to a tertiary care center.

By 06:00 the following morning, the patient reported generalized weakness of all four limbs together with persistent headache. Vital signs at that time were blood pressure 150/90 mmHg, heart rate 78 beats/min, respiratory rate 20 breaths/min, temperature 36.7°C, and oxygen saturation 100% on 3 L/min via nasal cannula. He remained fully conscious (GCS E4V5M6). Neurological examination showed persistent right supranuclear CN VII and CN XII palsy, with motor strength of 2/5 in both upper extremities and 3/5 to 4/5 in the lower extremities, increased tone with spasticity, and an equivocal Babinski reflex — findings consistent with evolving spastic tetraparesis.

Diagnostic Assessment

Radiological Examination. Later that day, following the onset of hemiparesis and dysarthria, non-contrast multislice computed tomography (MSCT) of the brain was performed in axial, coronal, and sagittal planes to investigate the patient's acute neurological deterioration. Imaging demonstrated a hypodense lesion in the left parietal lobe with focal hemorrhagic components, consistent with acute ischemic infarction with hemorrhagic transformation (Figure 1), together with hyperdensity and increased attenuation within the superior sagittal sinus suggestive of cerebral venous sinus thrombosis (Figure 2).

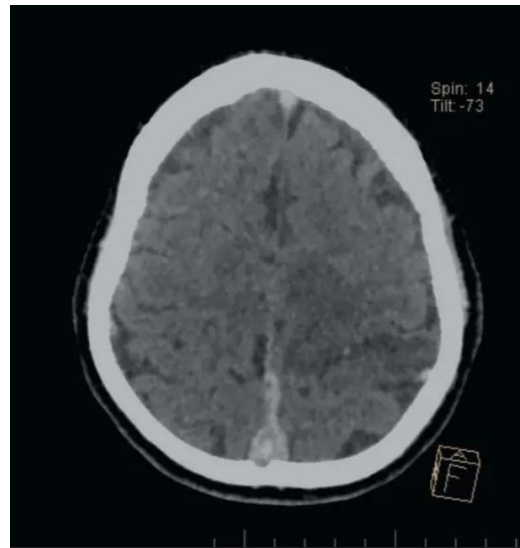


Figure 1. Non-contrast axial CT scan showing a hypodense lesion in the left parietal lobe with a hemorrhagic component consistent with venous infarction.



Figure 2. Non-contrast coronal CT scan showing hyperdensity within the superior sagittal sinus suggestive of cerebral venous sinus thrombosis.

The ventricular system was normal with no hydrocephalus, cortical sulci and gyri were preserved, and there was no midline shift; the pons and cerebellum appeared normal, and no abnormal intracranial calcification was seen. The orbits, mastoid air cells, and paranasal sinuses were unremarkable. Taken together, these findings were consistent with cerebral venous sinus thrombosis involving the superior sagittal sinus, complicated by venous infarction of the left parietal lobe with a hemorrhagic component. A chest radiograph obtained during initial evaluation to assess cardiopulmonary status showed no significant abnormality (Figure 3).



Figure 3. Chest radiograph obtained during initial evaluation showing no significant cardiopulmonary abnormality.

Laboratory Findings. Concurrent with the initial neuroimaging, laboratory investigations were obtained to evaluate the patient's systemic status and to exclude a metabolic or infectious contributor to his neurological presentation. Complete blood count showed a white cell count of $9.17 \times 10^9/L$, hemoglobin 13.4 g/dL, hematocrit 39.8%, and platelets $252 \times 10^9/L$, all within normal limits; the differential count showed mild lymphocytosis (52.4%; absolute lymphocyte count $4.81 \times 10^9/L$).

Serum electrolytes were normal (sodium 141.7 mmol/L, potassium 3.57 mmol/L, chloride 104.1 mmol/L), as were total and ionized calcium (2.33 mmol/L and 1.16 mmol/L, respectively). Renal function was preserved, with creatinine 1.03 mg/dL, an estimated glomerular filtration rate of 98.36 mL/min/1.73 m², and urea 28 mg/dL. Random blood glucose was 82 mg/dL and serum albumin 4.55 g/dL, both normal, and HIV serology was non-reactive. Overall, the laboratory workup did not reveal an abnormality that could account for the patient's neurological deterioration.

Management

The patient received supportive and symptomatic care from admission onward. Intravenous 0.9% sodium chloride was infused at 20 drops/min, and oxygen was given at 3 L/min via nasal cannula. Antiepileptic therapy was started with an intravenous phenytoin loading dose of 900 mg over one hour, followed by a maintenance regimen of 100 mg intravenously three times daily; intravenous diazepam was given as needed for seizure control (maximum 20 mg/day), with oral clobazam 10 mg twice daily added as adjunctive anticonvulsant therapy. He also received intravenous dexamethasone 10 mg three times daily, intravenous paracetamol 1000 mg three times daily, intravenous citicoline 500 mg twice daily, and intravenous ceftriaxone 2 g twice daily. Antiplatelet therapy was initiated with a loading dose of aspirin 320 mg followed by a maintenance dose of 80 mg orally once daily. Anticoagulation, which international guidelines identify as the mainstay of definitive CVST therapy, was deliberately withheld at this stage: the presence of hemorrhagic transformation on initial imaging, together with recurrent seizures and an evolving examination, favoured a cautious, individualised approach and urgent

transfer to a centre where anticoagulation could be initiated under closer neurological and radiological surveillance, in line with guidance that hemorrhagic infarction warrants case-by-case risk-benefit assessment rather than automatic exclusion from anticoagulant therapy.

Case Summary

This report describes a young man with rapidly progressive neurological deterioration caused by cerebral venous sinus thrombosis (CVST) involving the superior sagittal sinus, complicated by venous infarction of the left parietal lobe with hemorrhagic transformation. He initially presented with right-sided hemiparesthesia preceded by several days of occipital headache radiating to the posterior neck. Within hours of admission his condition worsened, with right-sided hemiparesis, dysarthria, cranial nerve involvement, and recurrent generalized seizures. Neuroimaging confirmed an acute parietal venous infarct with a hemorrhagic component and hyperdensity of the superior sagittal sinus, establishing the diagnosis of CVST. Notably, the patient progressed within roughly 24 hours to spastic tetraparesis, illustrating the aggressive clinical course that CVST can occasionally follow.

Comparison with the Literature

CVST is an uncommon form of stroke that disproportionately affects young adults and individuals without conventional vascular risk factors. Unlike arterial ischemic stroke, it displays considerable heterogeneity in presentation, ranging from isolated headache to focal deficits, seizures, and impaired consciousness (Idiculla et al., 2020; Silvis et al., 2017). This broad spectrum of manifestations makes early diagnosis genuinely difficult, particularly when symptoms are nonspecific.

Headache, the most frequently reported presenting symptom of CVST, may precede other neurological manifestations by several days (Idiculla et al., 2020; Ulivi et al., 2020). Many patients follow a subacute course reflecting progressive venous congestion and intracranial hypertension, though more aggressive presentations have also been described, particularly when major venous sinuses or extensive venous infarction are involved (Silvis et al., 2017).

Seizures are another common manifestation and occur more frequently in CVST than in arterial ischemic stroke (Wasay et al., 2010). Their occurrence has been linked to cortical venous involvement and the presence of parenchymal lesions, particularly hemorrhagic venous infarction (Mehvari Habibabadi et al., 2018), with cortical irritation from venous congestion and hemorrhagic injury thought to underlie seizure generation in these patients.

Thrombosis of the superior sagittal sinus is among the most frequently reported forms of CVST and is commonly associated with venous infarcts affecting the frontal and parietal lobes (Coutinho et al., 2014). Depending on which cortical regions are involved, such lesions can produce focal deficits including hemiparesis, sensory disturbance, or aphasia.

Compared with previously reported cases, our patient showed a strikingly rapid evolution of neurological deficits, progressing from hemiparesthesia to tetraparesis within a short interval. Although most CVST follows a subacute course, rapid deterioration can occur when extensive venous congestion, venous infarction, or intracranial hypertension are present (Boussier & Ferro, 2007), underscoring the

value of early recognition and prompt neuroimaging whenever stroke symptoms appear atypical.

The demographic profile of this patient is broadly consistent with previously described epidemiological patterns of CVST. A Chinese case series of 31 patients reported a mean age of 39 years with 42% male representation (Liu et al., 2024), while a Pakistani cohort reported a similar mean age of 31 years but with a marked female predominance related to peripartum risk factors (Khan et al., 2020). At 33 years old, our patient falls squarely within the young-adult age range typical of CVST, though as a male without hormonal risk factors his demographic profile diverges from the female-predominant pattern seen in much of the literature. A large emergency-department-based observational study from the United States further noted that CVST is now recognized more frequently in older age groups than previously assumed, a shift in epidemiology that clinicians across care settings should bear in mind (Ruderman et al., 2025).

Pathophysiological Considerations

The pathophysiology of CVST centers on thrombosis of the cerebral veins or dural venous sinuses, obstructing venous drainage from the brain. This obstruction produces venous congestion, raised intracranial pressure, and reduced cerebral perfusion, ultimately resulting in vasogenic edema and venous infarction (Ferro et al., 2016; Saposnik et al., 2011). Unlike arterial infarction, venous infarction frequently undergoes hemorrhagic transformation due to rupture of congested venous vessels and disruption of the blood–brain barrier (Ranjan et al., 2023).

Thrombosis of the superior sagittal sinus can impair drainage from extensive cortical venous territories, particularly the frontal and parietal lobes; as venous pressure rises, cerebral edema and venous infarction may develop, producing focal deficits (Ranjan et al., 2023). Involvement of cortical veins can also trigger cortical irritation and an inflammatory response that contributes to seizure activity (Coutinho, 2015).

In this case, the parietal venous infarct with its hemorrhagic component likely contributed to the patient's recurrent seizures and progressive deterioration. Increasing venous congestion and an expanding infarcted area may have worsened cortical dysfunction and involved bilateral motor pathways — mechanisms that plausibly explain the rapid progression from unilateral symptoms to tetraparesis observed here.

The emergence of tetraparesis from what appeared, radiologically, to be a unilateral venous infarct deserves particular comment. Several overlapping mechanisms may account for bilateral motor involvement despite an apparently one-sided structural lesion. First, extensive cerebral edema from severe venous congestion can produce a mass effect and globally elevated intracranial pressure, disturbing function in both hemispheres even when the structural imaging appears asymmetric. Second, the patient's recurrent seizures may have produced transient postictal neurological dysfunction that compounded the clinically apparent motor deficit. Third, because the superior sagittal sinus inherently drains both cerebral hemispheres, its thrombosis can plausibly cause bilateral dysfunction even though the hemorrhagic infarct visualized on CT was concentrated on one side. Recognizing these overlapping mechanisms matters clinically, since neurological

deterioration in CVST does not always track proportionally with the extent of the lesion seen on initial imaging.

Clinical Implications

This case underscores the importance of considering cerebral venous sinus thrombosis in young patients with acute neurological symptoms, particularly when headache precedes focal deficits. Because CVST can mimic arterial ischemic stroke or intracerebral hemorrhage, early neuroimaging is central to reaching the correct diagnosis (Saposnik et al., 2024).

Recognizing imaging features suggestive of CVST — dural venous sinus hyperdensity and hemorrhagic venous infarction — can accelerate diagnosis and timely management, while the rapid deterioration seen in this patient reinforces the need for close neurological monitoring and prompt treatment whenever CVST is suspected.

American Heart Association/American Stroke Association guidance recommends prompt initiation of anticoagulation once CVST is diagnosed, even in the presence of hemorrhagic infarction, since the benefit of preventing thrombus extension generally outweighs the risk of worsening hemorrhage (Saposnik et al., 2011, 2024). In this case, management centered on supportive care, seizure control, and close monitoring, reflecting both the hemorrhagic transformation present and the need for transfer to a center with more comprehensive neurointerventional capability. This also illustrates a broader challenge in managing CVST at primary and secondary care facilities, including in Indonesia, where access to MRI/MRV, subspecialist neurology services, and neurointervention remains limited outside major cities. It highlights the importance of maintaining a high index of clinical suspicion and a responsive referral pathway from the moment of first contact in the emergency department, so that patients with suspected CVST can obtain diagnostic confirmation and definitive treatment as quickly as possible.

Lessons Learned

Early recognition of cerebral venous sinus thrombosis presenting with atypical stroke symptoms is essential to prevent delayed diagnosis and neurological deterioration. Clinicians should maintain a high index of suspicion for CVST in young patients presenting with headache, seizures, or focal neurological deficits, particularly in the absence of conventional vascular risk factors. The differential diagnosis should include arterial ischemic stroke, intracerebral hemorrhage, intracranial infection, brain tumor, and demyelinating disease, all of which can present with overlapping neurological features.

Limitations

This report has several limitations worth acknowledging. First, it is based on the observation of a single case, which limits generalizability to broader patient populations. Second, the duration of follow-up was limited, precluding a comprehensive assessment of the patient's long-term neurological recovery and functional outcome. Finally, certain additional diagnostic studies — magnetic resonance venography (MRV) and comprehensive thrombophilia screening — were not performed during initial evaluation, and might have offered further insight into the underlying etiology of this patient's cerebral venous sinus thrombosis. Relatedly, although referral to a tertiary centre was arranged, this report cannot confirm whether repeat CT was performed on arrival there to verify that the venous infarct and hemorrhagic component had not progressed; interval imaging at the

receiving hospital would have been valuable both to guide the timing of anticoagulation and to corroborate the trajectory described here.

CONCLUSION

This case highlights an aggressive manifestation of cerebral venous sinus thrombosis involving the superior sagittal sinus, complicated by venous infarction with hemorrhagic transformation, resulting in rapid neurological deterioration from hemiparesthesia to spastic tetraparesis within a short period. Clinicians should maintain awareness of cerebral venous sinus thrombosis as a potential cause of acute neurological deficits in young patients, particularly when symptoms are preceded by persistent headache or accompanied by seizures. Timely neuroimaging and appropriate management remain essential to prevent further neurological deterioration and to optimize patient outcomes.

ACKNOWLEDGMENTS

The authors thank the management and colleagues at the Emergency Department and Department of Neurology of Bangli Regional Hospital, and Division of Neurosurgery, Department of Surgery, Faculty of Medicine Udayana University, Ngoerah Hospital, Bali, Indonesia, for their support throughout the preparation of this case report. Written informed consent was obtained from the patient for publication of this case report and its accompanying images, with all personal information anonymized to protect confidentiality. In accordance with institutional policy, formal ethical approval was not required for this single case report. The authors declare no conflict of interest and report that no external funding was received for the preparation of this report.

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