Scientific Research Publishing

World Journal of Neuroscience, 2026, 16(1), 49-56
https://www.scirp.org/journal/wjns
ISSN Online: 2162-2019
ISSN Print: 2162-2000

Acute Invasive Rhinosinusitis Complicated by Cerebral Venous Sinus Thrombosis: A Case Report and Review of Multidisciplinary Management

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How to cite this paper: Osula, E., Abideen, Z., Shajahan, K., Arkras, Z., Abubaker, T. and Graham, S. (2026) Acute Invasive Rhinosinusitis Complicated by Cerebral Venous Sinus Thrombosis: A Case Report and Review of Multidisciplinary Management. *World Journal of Neuroscience*, **16**, 49-56.
https://doi.org/10.4236/wjns.2026.161005

Received: October 30, 2025
Accepted: February 7, 2026
Published: February 10, 2026

Abstract

Acute sinusitis is common, but intracranial complications such as cerebral venous thrombosis (CVT), meningitis, and brain abscesses are rare and potentially life-threatening. Early recognition and multidisciplinary management are critical, particularly in immunocompromised or medically complex patients. We report a 68-year-old male with a history of mantle cell lymphoma, hypertension, and chronic tobacco use, who presented with a three-day history of right-sided headache and intermittent tinnitus. He had recently been treated for herpes simplex virus type 1 with acyclovir, which he discontinued due to persistent symptoms, and was concurrently being treated for right-sided mastoiditis. Despite multiple prior emergency visits, his symptoms persisted. Initial imaging revealed right tympanic membrane opacity and opacification of the right mastoid air cells and middle/external ear, consistent with mastoiditis. MRI of the brain and MR venography confirmed thrombosis of the right transverse and sigmoid sinuses. Lumbar puncture demonstrated lymphocytic predominance consistent with viral meningitis. The patient was treated with empiric broad-spectrum antibiotics, corticosteroids, and anticoagulation, and later transitioned to apixaban. Multidisciplinary management involved neurology, otolaryngology, infectious disease, and hematology teams. This case highlights that subtle neurological symptoms, such as isolated headache or tinnitus, may indicate serious intracranial complications of sinusitis. Advanced imaging, particularly MRI and MR venography, is essential for early detection. Coordinated multidisciplinary care, timely therapeutic intervention, and awareness of atypical presentations in high-risk patients are vital to improve outcomes and prevent potentially fatal sequelae.

Keywords

Sinusitis, Mastoiditis, Cerebral Venous Thrombosis, Intracranial Complications, MR Venography, Multidisciplinary Management

1. Introduction

Acute rhinosinusitis is a prevalent clinical entity encountered in both outpatient and hospital settings, characterized by inflammation of the mucosa of the nasal cavity and paranasal sinuses. It affects millions of individuals annually, with viral etiologies accounting for the majority of cases. However, approximately 0.5% - 2% of cases progress to bacterial infection, necessitating medical intervention [1]. While most infections resolve without complication, a small but clinically significant proportion may result in serious extracranial and intracranial sequelae. These complications typically arise when infection extends beyond the sinus walls into adjacent structures, including the orbit, meninges, or cerebral venous system. Risk factors for such progression include virulent pathogens, anatomical obstruction, immunosuppression, prior sinus surgery, or delayed recognition and treatment [2] [3].

Intracranial complications of sinusitis, though uncommon, are associated with substantial morbidity and mortality. They encompass epidural and subdural empyema, meningitis, brain abscess, cavernous sinus thrombosis, and cerebral venous sinus thrombosis (CVST) [4] [5]. CVST, in particular, is a rare but life-threatening condition characterized by thrombus formation within the dural venous sinuses. This impairs venous drainage, promotes cerebral edema, increases intracranial pressure, and predisposes to venous infarction and hemorrhage [6] [7]. Early recognition is challenging because presenting symptoms, including headache, fever, rhinorrhea, altered mental status, visual disturbances, seizures, and focal neurological deficits, often overlap with uncomplicated sinusitis [8] [9]. Consequently, delayed diagnosis is common, increasing the risk of permanent neurological deficits or death.

The pathophysiology linking sinusitis to CVST is multifactorial. Infection may spread directly through emissary veins, bony dehiscence, or via thrombophlebitis processes within the valveless venous system of the skull [10] [11]. Bacterial toxins and inflammatory mediators promote endothelial injury and local hypercoagulability, facilitating thrombus formation. The superior sagittal, transverse, and sigmoid sinuses are most frequently affected, though cavernous and straight sinuses may also be involved. Certain pathogens, particularly *Streptococcus* species, *Staphylococcus aureus*, and anaerobic organisms, are more frequently implicated in intracranial extension [12].

Prompt neuroimaging is critical for early detection and optimal management. Magnetic resonance imaging (MRI) with MR venography (MRV) is considered the gold standard, providing detailed evaluation of sinus patency, thrombus char-

acteristics, and parenchymal changes such as edema or hemorrhage [13] [14]. Early intervention with targeted intravenous antibiotics and anticoagulation significantly improves prognosis, reduces thrombus propagation, and mitigates secondary neurological injury [15] [16]. Surgical intervention, typically via endoscopic sinus drainage, is indicated for abscess formation, persistent obstruction, or inadequate response to medical therapy. Given the complexity and high-risk nature of sinusitis-associated CVST, multidisciplinary management involving otolaryngology, neurology, infectious disease, and neurosurgery is essential to optimize outcomes [17] [18]. Given its rarity, diagnostic challenges, and high morbidity, each reported case contributes valuable insight into presentation, management, and outcomes. We present a case of acute invasive sinusitis complicated by cerebral venous sinus thrombosis, emphasizing the importance of early recognition, prompt imaging, and coordinated medical and surgical intervention. This report highlights both the clinical subtleties of CVST and the critical role of multidisciplinary care in preventing catastrophic neurological sequelae.

2. Case Presentation

A 68-year-old male with a history of mantle cell lymphoma, hypertension, and chronic tobacco use presented to the emergency department with a three-day history of diffuse, pounding headache predominantly affecting the right side, accompanied by intermittent tinnitus. He denied fever, neck stiffness, visual changes, or focal neurological deficits. Three to four days prior, he had been evaluated by his primary care physician for a facial lesion, diagnosed as herpes simplex virus type 1 (HSV-1) and was prescribed a seven-day course of acyclovir. During therapy, he developed progressive headache and worsening tinnitus, so he discontinued the acyclovir. Discontinuation of acyclovir did not relieve his symptoms, prompting hospital evaluation. Notably, the patient presented to another hospital three times prior to this admission for similar headache symptoms. On each visit, he underwent limited evaluation; only one visit included an ear cleaning procedure after imaging revealed a right ear infection. He was discharged on all three occasions with a "migraine cocktail," which failed to relieve his symptoms.

Upon arrival at our facility, an initial computed tomography (CT) of the brain revealed no acute intracranial abnormalities but demonstrated right tympanic membrane opacity. Temporal bone CT showed opacification of the right mastoid air cells and right middle/external ear, consistent with acute mastoiditis. Magnetic resonance imaging (MRI) of the brain revealed a filling defect in the right transverse and sigmoid venous sinuses, suggesting partial thrombosis. MR venography confirmed cerebral venous thrombosis. Lumbar puncture demonstrated lymphocytic predominance with normal protein and glucose, consistent with viral meningitis. The patient was diagnosed with cerebral venous thrombosis secondary to mastoiditis. Treatment included empiric broad-spectrum antibiotics to address potential bacterial superinfection, corticosteroids to mitigate cerebral edema, and anticoagulation with a heparin infusion later transitioned to apixaban. Multidis-

ciplinary management involved neurology, otolaryngology, infectious disease, and hematology teams.

2.1. Investigations

CT brain revealed no acute intracranial abnormalities but demonstrated right tympanic membrane opacity. Temporal bone CT confirmed opacification of right mastoid air cells and the middle/external ear, consistent with mastoiditis. MRI of the brain (sagittal view) showed a filling defect in the right transverse and sigmoid venous sinuses, suggestive of partial thrombosis (Figure 1). MR venography confirmed thrombosis in the same venous sinuses (Figure 2). Cerebrospinal fluid analysis from lumbar puncture demonstrated lymphocytic predominance with normal protein and glucose, consistent with viral meningitis. These findings collectively supported the diagnosis of cerebral venous thrombosis secondary to mastoiditis.

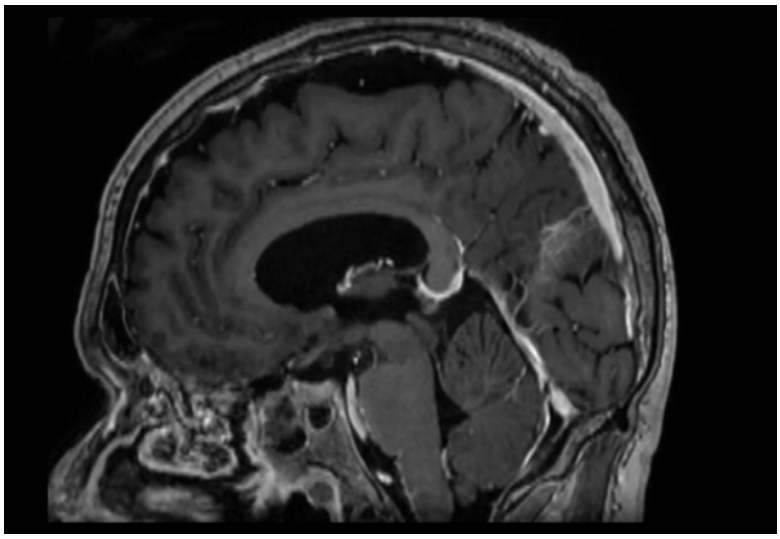

Figure 1. MRI Brain (Sagittal view): Acute right mastoiditis with filling defect in right transverse and sigmoid venous sinuses.

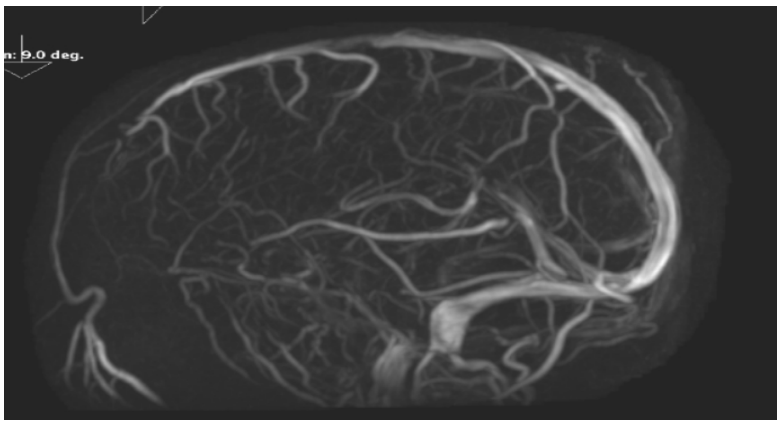

Figure 2. MR Venography (Spin view): Thrombosis of right transverse and sigmoid venous sinuses.

2.2. Differential Diagnosis

The initial differential diagnosis included migraine or tension-type headache, viral meningitis, otogenic infection (otitis media or mastoiditis), intracranial mass lesion, and cerebral venous thrombosis. Early imaging was essential in distinguishing CVT from other potential causes, especially given the atypical presentation without fever or focal neurological deficits.

2.3. Treatment

The patient received empiric broad-spectrum antibiotics to address potential bacterial superinfection and corticosteroids to reduce cerebral edema. Anticoagulation was initiated with a heparin infusion, followed by transition to apixaban (10 mg twice daily for seven days, then 5 mg twice daily). The patient underwent close monitoring in coordination with neurology, otolaryngology, infectious disease, and hematology teams. This multidisciplinary approach ensured timely intervention and prevented thrombus propagation or other complications.

2.4. Outcome and Follow-Up

Following treatment, the patient experienced complete resolution of headache and tinnitus without developing any neurological deficits. He was discharged with continued anticoagulation and informed to follow up outpatient with neurology, hematology/oncology, otolaryngology, and infectious disease. At follow-up, he remained clinically stable with no recurrence of symptoms.

3. Discussion

Cerebral venous sinus thrombosis (CVST) represents a rare but severe complication of acute or chronic rhinosinusitis and/or mastoiditis, accounting for a small proportion of intracranial infections; however, when present, it carries high morbidity and risk for neurologic deterioration [6] [16]. CVST may arise through several mechanisms, including direct bacterial invasion, thrombophlebitis of communicating veins, local osteitis with extension through the bony sinus walls, and the propagation of infection into the dural venous channels [10] [11]. The valveless nature of venous drainage between the paranasal sinuses and intracranial venous system facilitates the spread of pathogens and inflammatory mediators, predisposing to thrombus formation. Endothelial dysfunction, hypercoagulability, and impaired venous flow collectively contribute to clot propagation, cerebral edema, and venous infarction [17] [18].

Clinical presentation is often heterogeneous and may evolve rapidly. Headache is the most frequent symptom reported in CVST, often severe and unresponsive to routine analgesics [8] [9]. Fever, altered mental status, diplopia, seizures, papilledema, vomiting, and focal neurological deficits may follow as venous pressure rises and parenchymal injury develops. In many cases, early manifestations mimic uncomplicated sinusitis or viral illness, delaying diagnosis and treatment [1] [2]. This reinforces the importance of heightened clinical suspicion in patients presenting

with persistent headache, orbital symptoms, or neurological changes despite appropriate antibiotic therapy for sinusitis [3] [15].

Neuroimaging plays a central role in diagnosis. Contrast-enhanced MRI with MR venography remains the gold standard due to its superior ability to visualize sinus patency, thrombus formation, and associated parenchymal changes such as edema or hemorrhagic infarction [13] [14]. CT venography may serve as an alternative when MRI is unavailable or contraindicated. Imaging the paranasal sinuses is equally essential to assess disease burden, identify bone erosion or intracranial extension, and guide surgical decision-making [4] [5]. Early imaging not only confirms diagnosis but also facilitates timely coordination with neurology, neurosurgery, and otolaryngology.

Management requires a multidisciplinary approach. Broad-spectrum intravenous antibiotics targeting typical sinus pathogens, including *Streptococcus*, *Staphylococcus aureus*, anaerobes, and gram-negative organisms, should be initiated promptly [10] [12]. Anticoagulation remains a key component of treatment in CVST, even in the presence of intracerebral hemorrhage, as it prevents thrombus propagation and fosters recanalization [6] [7]. Heparin or low-molecular-weight heparin is commonly used initially, with transition to oral anticoagulation for several months, depending on clinical response and underlying risk factors. Surgical intervention, including endoscopic sinus surgery, is indicated for cases with abscess formation, significant obstruction, or failure to respond to medical therapy [11] [15]. Drainage reduces infectious load and may prevent further intracranial spread.

Prognosis varies depending on the rapidity of diagnosis and treatment. Patients who receive early anticoagulation and surgical decompression when indicated often achieve good recovery, whereas delayed management is associated with increased risk of neurological sequelae, venous infarction, seizures, and mortality [17] [19]. Literature describes mortality rates historically reaching 20% - 30%, though improved imaging and modern management strategies have significantly reduced fatality and disability [16] [18]. Continued awareness among clinicians is vital, as delayed recognition remains a persistent cause of morbidity.

This case reinforces several clinical lessons. First, persistent or worsening headache in sinusitis warrants thorough neurologic evaluation and consideration of intracranial extension [1] [2]. Second, early MRI/MRV should be pursued when red-flag symptoms, neurological deficits, visual changes, altered mental status, or refractory pain are present [13] [14]. Third, prompt initiation of antibiotics and anticoagulation can be lifesaving, and otolaryngologic intervention may expedite clinical recovery. By presenting this case, we highlight the importance of vigilance, timely diagnostic imaging, and coordinated multidisciplinary care to prevent catastrophic outcomes in sinusitis-related CVST [4] [5].

4. Conclusions

Intracranial complications of sinusitis, though uncommon, can be life-threaten-

ing and require heightened clinical vigilance, particularly in high-risk populations such as the elderly, immunocompromised patients, or those with chronic comorbidities including uncontrolled hypertension, hyperlipidemia, diabetes mellitus, or various autoimmune conditions. This case demonstrates that even subtle neurological symptoms, such as isolated headache or tinnitus, may herald serious intracranial involvement, including cerebral venous thrombosis. Early recognition of these complications is critical, as delays in diagnosis can result in thrombus propagation, venous infarction, permanent neurological deficits, or death.

Advanced imaging modalities, including MRI and MR venography, are essential diagnostic tools in detecting early venous thrombosis that may be missed on standard CT scans. The integration of a multidisciplinary approach engaging neurology, otolaryngology, infectious disease, and hematology is vital to guide prompt intervention with anticoagulation, empiric antimicrobial therapy, and supportive care.

Finally, this case underscores the importance of maintaining a high index of suspicion for intracranial complications of sinusitis, even when classical signs of infection are absent. Timely imaging, early therapeutic intervention, and coordinated care can substantially improve patient outcomes, reduce morbidity, and prevent potentially fatal complications. Clinicians should be aware that atypical presentations in immunocompromised or medically complex patients demand thorough evaluation and proactive management strategies.

Acknowledgements

I would like to express my deepest gratitude for the invaluable guidance, support, and mentorship throughout this research. I am also grateful to HCA Florida Brandon Hospital for providing the resources and facilities necessary to complete this study. I extend my sincere thanks to my colleagues and friends for their encouragement and assistance during the research process.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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