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CASE REPORT

Use of dexamethasone in septic cavernous sinus thrombosis coexisting with bacterial meningitis secondary to sinusitis: A case report

Wesam Albqa'een†, Nikesh K Giri†, Gemma Smith, and Revin Thomas*

Department of Stroke Medicine, University Hospital of North Durham, Durham, DH1 5TW, United Kingdom

Abstract

We present the case of a 69-year-old woman who developed septic cavernous sinus thrombosis (SCST) secondary to bacterial meningitis and sinusitis. She presented to the emergency department with frontal headache, neck stiffness, photophobia, exophthalmos, and fever. Initial investigations revealed extensive opacification of the paranasal sinuses on plain cranial computed tomography (CT), and blood tests indicated a bacterial infection. She initially received antibiotics and dexamethasone for suspected bacterial meningitis. Cerebrospinal fluid analysis revealed elevated glucose and protein levels as well as white and red blood cell counts, along with a negative Gram stain. An ophthalmology assessment and CT venography indicated a strong suspicion of cavernous sinus thrombosis, further confirmed by magnetic resonance venography. Anticoagulation with antibiotics and dexamethasone was initiated, leading to significant clinical improvement. This case highlights the importance of the early recognition and treatment of SCST and the role of dexamethasone in patients with coexisting bacterial meningitis.

Keywords: Septic cavernous sinus thrombosis; Dexamethasone; Corticosteroids; Bacterial meningitis; Sinusitis; Antibiotics; Anticoagulation

†*These authors contributed equally to this work.*

***Corresponding author:**
Revin Thomas
(revin.thomas@nhs.net)

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1. Introduction

Cavernous sinus thrombosis (CST) is a rare yet serious condition that may complicate infections such as sinusitis, orbital cellulitis, and pharyngitis, particularly after a thrombophilic event. It can present as septic or aseptic. The primary etiological organisms are bacteria, with *Staphylococcus aureus* accounting for approximately two-thirds of cases. Other frequently encountered pathogens include *Streptococcus* species.[1]

CST commonly arises from the hematogenous spread of infection to the cavernous sinus through the venous system, particularly via sphenoid sinusitis. The pterygoid plexus, a valveless venous system that connects with the cavernous sinus, increases the susceptibility to impaired drainage. The fine, irregular structure of the cavernous sinus functions as a sieve, trapping bacteria and increasing the risk of thrombophlebitis.[2]

2. Case presentation

A 69-year-old Caucasian woman presented to the emergency department after a referral from her primary care physician, with a 3-day history of progressively severe

frontal headache, facial pain, fever, neck rigidity, blurred vision, and photophobia. Her medical history included chronic obstructive pulmonary disease, hypertension, and hyperlipidemia. She had no recent travel history or infection contact.

On examination, she was alert but appeared drowsy. She had significant exophthalmos in the right eye. Both pupils were reactive to light, and eye movements were normal. She also had mild chemosis. Cranial nerves were grossly intact. She had neck stiffness and a positive Brudzinski sign. Her muscle power and reflexes were normal. On auscultation, she had mild wheezing and normal cardiovascular parameters and had no rashes.

Initial laboratory workup demonstrated a high level of C-reactive protein (366 mg/L [0 – 10 mg/L]) and white blood cell (white blood cells [WBC]; 32.1 × 10^9/L [4 – 11 × 10^9/L]) and neutrophil [30.8 × 10^9/L (2 – 7.5 × 10^9/L)] counts. Cranial computed tomography (CT) revealed extensive opacification of the paranasal sinuses (Figure 1). Given her clinical presentation and laboratory and imaging findings, the leading differential diagnosis was sinusitis with a high suspicion of meningeal involvement, likely bacterial in origin. She was empirically treated with antibiotics intravenously, particularly chloramphenicol and cotrimoxazole (she was allergic to penicillin). Intravenous dexamethasone, 10 mg every 6 h for 4 days, was also initiated.

On day 2, an ophthalmology consultation was requested due to persistent exophthalmos, chemosis, and reduced visual acuity. Subsequent CT venography (CTV) revealed a small filling defect in the right internal jugular vein (Figure 2) and right cavernous sinus (Figure 3), which was suggestive of a thrombus and raised a strong suspicion of CST.

Moreover, contrast-enhanced magnetic resonance venography (MRV) revealed a filling defect in the right cavernous sinus, confirming the diagnosis of right CST (Figure 4).

Cerebrospinal fluid (CSF) analysis revealed high levels of glucose (4.4 mmol/L) and protein (1 g/L), as well as WBC (18 cells/μL) and red blood cell (26 cells/μL) counts, along with xanthochromia and a negative Gram stain. CTV and MRV were further reviewed by an interventional neuroradiologist, who agreed with the diagnosis of meningitis and CST without evidence of subarachnoid hemorrhage. A remote neurosurgical consultation was sought, and therapeutic full-dose low-molecular-weight heparin (LMWH) was recommended. Blood culture identified *Streptococcus intermedius* sensitive to penicillin; however, due to the patient's penicillin allergy, targeted



Figure 1. Cranial computed tomography highlighting extensive opacification of the paranasal sinuses – marked opacification of the right sphenoid (blue arrow), left sphenoid, and ethmoid sinuses consistent with sinusitis

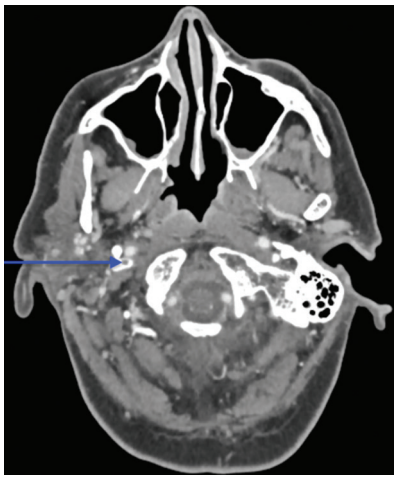


Figure 2. Computed tomography venography. The blue arrow highlights a small filling defect in the right internal jugular vein that appears darker (hypodense) than the left internal jugular vein, which appears well-opacified and bright, indicating normal blood flow with contrast filling. This darker appearance on the right indicates the absence of contrast filling in the vein, suggesting thrombus.

antibiotic therapy with chloramphenicol and cotrimoxazole was continued in accordance with institutional protocols and microbiologist advice.

The patient's presentation, laboratory data, and imaging findings were consistent with bacterial meningitis with secondary CST (SCST). Treatment decisions were made following established protocols for bacterial meningitis and SCST, which included commencing antibiotics, anticoagulation, and dexamethasone.

By hospital day 4, the patient had shown significant clinical improvement. Her neck pain and headaches

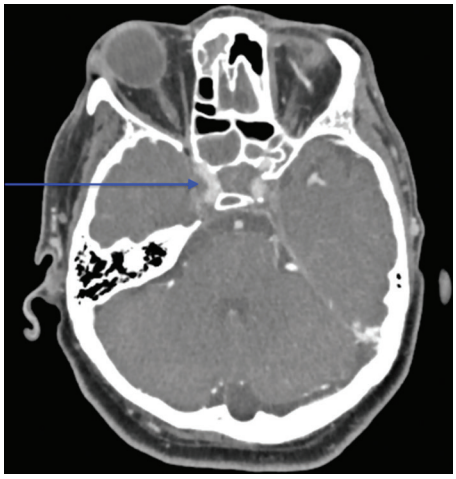


Figure 3. Computed tomography venography showing a cross-sectional view of the head, with a focus on the cavernous sinus on either side of the sella turcica. The blue arrow points to the right cavernous sinus, which appears darker and less enhanced. This hypoattenuation indicates a filling defect in the right cavernous sinus, which is suggestive of thrombosis in contrast to the left cavernous sinus that shows bright enhancement consistent with normal contrast uptake and unobstructed blood flow.



Figure 4. Magnetic resonance venography showing hypointensity on the right cavernous sinus (blue arrow) contrasting with the left side (yellow arrow) that remains bright. Normally, both cavernous sinuses appear bright due to blood flow. This filling defect on the right indicates stagnant or absent flow within the right cavernous sinus, which is consistent with cavernous sinus thrombosis

subsided. She remained on intravenous antibiotic and anticoagulation therapies for 2 weeks while in the hospital and was discharged on oral antibiotics for another 2 weeks and apixaban, with a plan to review anticoagulation on follow-up.

At her follow-up review after discharge, she reported no new or re-emergent symptoms. However, she had persistent mild headaches, which were not as severe as her initial presentation; accordingly, she was started on a trial of amitriptyline and planned for repeat MRV in 6 months to assess resolution and remained on anticoagulation therapy.

3. Discussion

The prevalence of SCST has markedly declined in the antibiotic era, and its mortality rate has significantly reduced to <30% in the postantibiotic era. However, it remains potentially lethal with long-term complications.[3] The subtle nature of early symptoms frequently results in delayed recognition and treatment initiation, causing guarded prognostication in SCST.[4] Magnetic resource imaging (MRI) and MRV are the preferred imaging modalities for diagnosing cerebral venous thrombosis (CVT).[3] Identifying CST complicating bacterial meningitis is particularly challenging due to overlapping clinical presentations.[5]

Because anticoagulation helps prevent thrombus expansion, has anti-inflammatory properties, and promotes the penetration of antibiotics into the thrombus, it has been proposed to treat SCST. Anticoagulation in SCST is beneficial in reducing mortality, improving neurological outcomes, and decreasing residual morbidity, particularly with early initiation of treatment.[6-8] However, anticoagulation is associated with risks of intracranial and systemic hemorrhage. A meta-analysis of patients with CVT indicated a 13% reduction in the mortality rate or dependency without an increase in hemorrhagic events, even among those with pre-existing intracranial hemorrhage, supporting the cautious use of anticoagulation in CST.[9] A recent systematic review and meta-analysis of 72 studies analyzing individual data of 110 patients (of which 60 had anticoagulation, with heparin being the most commonly used, followed by LMWH and warfarin) on anticoagulation for CST revealed an adjusted odds ratio for mortality of 0.067 ($p = 0.007$) in the anticoagulant-given group. However, hemorrhagic complications occurred in two patients with hyperglycemia, indicating increased bleeding risk in this group and highlighting the need for caution in such cases. The large reduction in the adjusted odds ratio indicated that anticoagulation independently and significantly reduced the mortality risk in SCST despite the potential risk of hemorrhage.[10]

The European Federation of Neurological Societies recommends anticoagulation for 3 months for secondary cerebral venous and sinus thrombosis (CVST) with a transient risk factor, 6 – 12 months for idiopathic CVST and mild thrombophilia, and indefinitely for recurrent CVST or one CVST episode and severe thrombophilia.[11]

The use of corticosteroids in treating SCST is debated due to the inconclusive evidence of their efficacy,

prothrombotic properties, and tendency to exacerbate infection by suppressing the immune response. The benefits of decreasing orbital inflammation, cranial nerve edema, vasogenic edema, and intracerebral hemorrhage must be weighed against the adverse effects of corticosteroids. Despite limited literature supporting improved outcomes following corticosteroid administration, some studies have reported improved cranial nerve function resulting from decreased inflammation.[4,12] Notably, Ivey and Smith (1968) reported the usefulness of corticosteroids in treating adrenal insufficiency caused by pituitary dysfunction resulting from infarction or infectious spread from the cavernous sinus to sella turcica in the CST setting.[13] In 1962, Solomon *et al.* reported a case of CST that was diagnosed on hospital day 9. The condition of the patient progressed from unilateral, bilateral CST, with absolute ophthalmoplegia and bilateral ocular orbital inflammation over 6 weeks despite aggressive antibiotic and anticoagulation therapies. The patient subsequently showed improvement 2 days after the commencement of oral corticosteroids.[14] In this case, the infection might have been eliminated before starting corticosteroids, as they were initiated on day 37 when the clinical signs of sepsis had already subsided.

In 2008, Canhão *et al.* reported on the use of corticosteroids in CVT. Of the 624 adult patients included in the International Study on Cerebral Veins and Dural Sinus Thrombosis, 24% received corticosteroids with a median duration treatment of 11 days. They did not find evidence supporting the routine use of corticosteroids in the acute phase of CVT, except if indicated for treating an underlying disease other than CVT requiring corticosteroids. Corticosteroids were reported as possibly harmful and should be avoided in patients with CVT without CT or MRI evidence of parenchymal lesions.[15] Conversely, in 2014, Dinaker *et al.* reported a case of SCST with meningitis caused by community-acquired methicillin-resistant *S. aureus*, where the patient received antibiotics, anticoagulants, and dexamethasone, leading to rapid response and symptom resolution. At discharge, the patient had mild residual right hemiparesis and demonstrated full recovery at the 3-month follow-up.[16]

Given the rarity of SCST and limited literature regarding corticosteroids, we extrapolated data from other CVT studies and articles to help expand this topic. In our case, the patient showed significant improvement with corticosteroids and had no neurological deficit. Dexamethasone likely contributed to inflammation reduction, enhancing overall recovery. This supports the potential benefit of corticosteroids in managing CST with concurrent bacterial meningitis, where inflammation plays a critical role in symptomatology. However, further larger studies, particularly randomized controlled trials, may help clarify its benefits.

4. Conclusion

This case highlights the importance of early identification and aggressive management of SCST. The patient's substantial improvement may have been facilitated by the use of dexamethasone alongside antibiotics and anticoagulation. Despite the controversial role of corticosteroids in SCST, their administration in severe inflammation contributed to a positive outcome. This case supports the consideration of corticosteroids in similar clinical scenarios while emphasizing the need for further research into their role alongside conventional treatments in complex conditions such as SCST coexisting with meningitis. It also highlights the importance of a multidisciplinary approach in its diagnosis and management.

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Conflict of interest

The authors declare that they have no competing interests.

Author contributions

Conceptualization: Wesam Albqa'een, Nikesh K Giri
Investigation: Wesam Albqa'een, Nikesh K Giri
Writing – original draft: Wesam Albqa'een, Nikesh K Giri
Writing – review & editing: Revin Thomas, Gemma Smith

Ethics approval and consent to participate

Verbal consent was obtained from the patient before her participation.

Consent for publication

Verbal consent was obtained from the patient to publish her data and scan images.

Availability of data

Not applicable.

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