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A Rare Presentation of Cavernous Sinus Thrombosis in a Young Female

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Abstract

A 31-year-old female with a history of iron deficiency anemia and migraines presented with a week-long left-sided headache, dizziness, and intermittent weakness in her legs. Her symptoms persisted, leading to her admission to the emergency department (ED). On further evaluation, the patient exhibited signs of ear pain and decreased hearing, as well as erythematous, bulging tympanic membranes. A thorough workup and imaging revealed a subdural hematoma, cavernous sinus thrombosis (CST), and restricted diffusion in the left frontal lobe. Neurosurgery consultation confirmed CST as the primary cause of her symptoms. The patient was started on anticoagulation therapy and transferred emergently to a higher-level trauma center. This case underscores the importance of considering CST in patients with unexplained headache, dizziness, neurological symptoms, and possible underlying hypercoagulable states [1]-[3]. Early recognition through imaging and appropriate treatment can significantly impact patient outcomes.

Keywords

Cavernous Sinus Thrombosis, Headache, Horizontal Nystagmus, Iron Deficiency Anemia, Magnetic Resonance Venography (MRV)

1. Introduction

Cavernous sinus thrombosis (CST) is a rare but serious condition that can lead to significant morbidity and mortality if not diagnosed and treated early. The overlapping features with benign headache conditions, such as migraine headaches and the necessity of specialized imaging techniques often make this potentially fatal condition difficult to diagnose. CST most commonly arises from infections, particularly those involving the paranasal sinuses, or from hypercoagulable states,

though it can also be seen in patients with a history of trauma, surgery, or malignancy [2] [3]. CST is characterized by the formation of thrombi within the cavernous sinus, which can lead to increased intracranial pressure, cranial nerve dysfunction, and even ischemic stroke if left untreated [2] [4].

In patients with CST, the early symptoms often are nonspecific, however, the presence of atypical signs such as hearing loss, dizziness, or visual disturbances should raise suspicion for a more serious etiology [5]. In this case, a 31-year-old woman with a known history of migraines and iron deficiency anemia presented with headache, dizziness, and weakness. A comprehensive evaluation, including neuroimaging, revealed the presence of CST, which was later confirmed through MRI and MRV. This case highlights the importance of maintaining a high index of suspicion for CST in patients with persistent or atypical neurological symptoms, especially when they present with signs not traditionally associated with migraine or other benign causes [1].

2. Case Presentation

A 31-year-old female with a history of iron deficiency anemia and migraines presented to urgent care with a week-long left-sided headache, rated 8/10, and intermittent dizziness. Of note, the patient mentioned her legs became weak and gave out the day prior while walking a short distance, and reported having a similar occurrence the week before. The patient stated she was given a prescription for Naproxen 500 mg two days prior which did not resolve her symptoms. She denied symptoms of vision changes, chest pain, shortness of breath, mental status change, seizures, and change in bowel or bladder habits. The patient denied any past surgical history, tobacco, ethanol, recreational drug use, and home medication use. On exam, the vital signs were all within normal limits and the patient was able to walk, but only with the aid of her husband. The urgent care labs revealed a hemoglobin of 7.9 and blood glucose of 150, and the patient was sent to the nearby emergency department.

Upon obtaining history at the emergency department, the patient additionally admitted to symptoms of nausea and bilateral ear pain with decreased hearing. The patient described that the headache began on the left side of her head and radiated throughout the rest of her head. She denied known sick contacts, recent travel, cough, congestion, fever, sore throat, chills, dizziness, lightheadedness, numbness or tingling, focal weakness, chest pain, shortness of breath, dyspnea, abdominal pain, vomiting, diarrhea, neck pain, back pain, and rash. The patient's vitals again were all within normal limits and on exam her external auricles were normal appearing bilaterally without postauricular erythema or edema, mastoid tenderness was not present, however tympanic membranes were bilaterally erythematous and slightly bulging. The patient was neurologically intact and her gait was normal. The differential diagnoses for the patient included subarachnoid hemorrhage, intracranial hemorrhage, ischemic stroke, temporal arteritis, venous sinus thrombosis, meningitis, encephalitis, intracranial mass and migraine head-

ache. Labs were ordered and resulted. Complete blood count revealed microcytic anemia (hemoglobin of 7.6, hematocrit of 27.4, MCV of 60.1), increased red cell distribution width of 21.7, increased immature granulocyte count of 0.04, 1+ anisocytosis, 1+ ovalocytes, 1+ hypochromia, occasional macrocytosis, 1+ microcytosis, 1+ poikilocytosis, 1+ polychromasia, 1+ target cells, occasional teardrop cells, and occasional schistocytes. Complete metabolic panel revealed potassium of 3.4, chloride of 111, increased BUN/creatinine ratio of 25.9, increased total protein of 8.3, and increased globulin of 4.8. During the patient's stay in the emergency department, she was given IV Toradol, Reglan, Benadryl, Decadron, and a fluid bolus. On re-evaluation, the patient was not in acute distress and stated that her headache had resolved. The patient was discharged with a diagnosis of headache and was told to return to the emergency department if her symptoms worsened.

Although the patient initially presented with a severe unilateral headache, intermittent dizziness, and reported leg weakness, her neurologic examination in the emergency department was unremarkable. Her symptoms improved significantly following administration of IV fluids, anti-inflammatory and anti-emetic medications, which are characteristic of a benign headache syndrome such as a migraine or tension headache. Additionally, her vital signs remained stable throughout the visit, and no acute abnormalities were identified in the lab work other than anemia. Given the absence of concerning features such as altered mental status, persistent focal deficits, fever, signs of increased intracranial pressure, or infectious source requiring urgent intervention, the clinical picture was most consistent with a primary headache disorder. Therefore, after symptom resolution and risk stratification, the diagnosis of headache was made, and the patient was deemed safe for discharge with close return precautions.

Three days later the patient returned to the emergency department with similar complaints of headache, dizziness, vomiting, and weakness. The patient stated that she was well until the night prior where she began vomiting, felt dizzy and weak, and fell to the ground. She denied loss of consciousness, head strike, fever, chills, vision changes, numbness, tingling, chest pain, shortness of breath, dyspnea, abdominal pain, and diarrhea. Vitals were taken and all were within normal limits. On physical examination, horizontal nystagmus was observed with 5/5 strength in upper and lower extremities, intact sensation, and finger-nose-finger without dysmetria. Laboratory workup was notable for a microcytic anemia with increased red cell distribution width, mild leukocytosis (WBC 11.18), and elevated absolute neutrophil count (7.45). Hemoglobin, hematocrit, and platelet levels were otherwise unremarkable. The remainder of the differential was non-contributory. Coagulation studies showed a mildly elevated prothrombin time (13.7), decreased partial thromboplastin time (21.3), and a normal international normalized ratio. A repeat complete metabolic panel did not reveal any significant differences from previous values.

Given the repeat occurrence of the patient's symptoms, CT imaging was or-

dered with low suspicion for TIA, assuming migraine versus vertigo. The radiologist consulted the provider stating that the CT showed suspicion for a subfalcine subdural hematoma, seen in Figure 1, and to consult neurosurgery for further plans. The neurosurgery team was consulted and after reviewing the head CT stated that there was concern for a sagittal sinus thrombosis, as seen in Figure 2, and recommended an emergent MRI and MRV to be ordered. The MRV head without contrast showed extensive sagittal, straight, and transverse sinus thrombosis as seen in Figure 3. The MRI brain without contrast showed restricted diffusion in the left frontal lobe, bilateral mastoid air cell effusions, and venous sinus thrombosis seen in Figure 4. The neurosurgeon and radiologist recommended emergent transfer to a level I trauma center and STAT flight was activated. While preparing the patient for transfer, she received an IV heparin bolus and drip for anticoagulation as recommended by neurosurgery. The patient remained neurologically intact when the flight arrived.

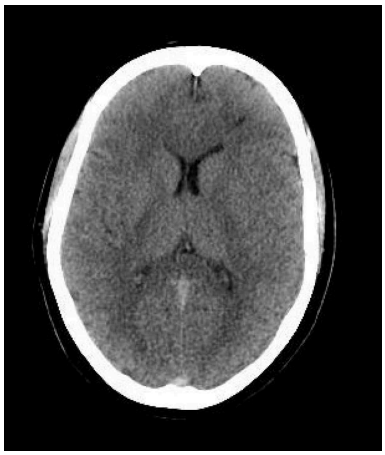

Figure 1. CT Head without contrast showing suspicion for a subfalcine subdural hematoma.

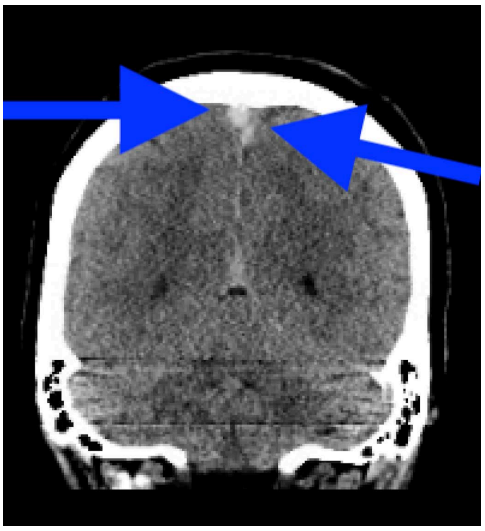

Figure 2. CT Head without contrast showing sagittal sinus thrombosis.

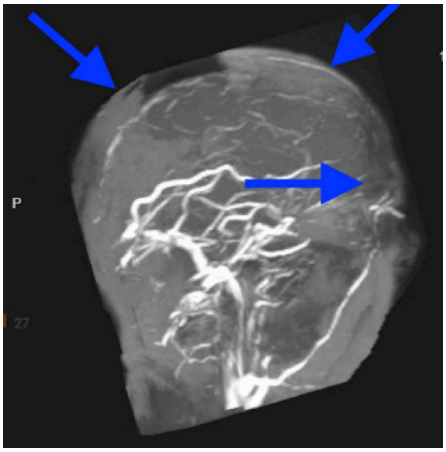

Figure 3. MRV Brain showing extensive sagittal, straight, and transverse sinus thrombosis.

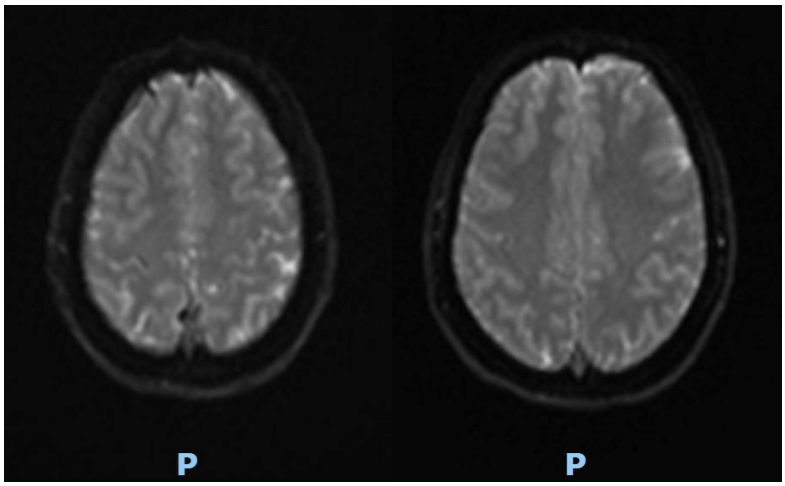

Figure 4. MRI Brain showing superior sagittal sinus thrombosis.

Post transfer it was reported that an additional MRI brain with and without contrast and MRV showed extensive dural venous sinus thrombosis with extension into bilateral frontal cortical veins and into the proximal right parietal cortical vein with several foci of venous infarction in the left frontal lobe. The patient was noted to have no neurologic deficits and was converted from heparin to lovenox during admission. ENT, ophthalmology and hematology were consulted and the patient had a negative thrombotic workup. She was discharged home on therapeutic eliquis and was advised to follow up with her primary care physician and a hematologist.

3. Discussion

Cavernous sinus thrombosis (CST) remains a challenging clinical entity, with its complex etiology, diverse presentation, and potential for rapid progression leading to significant morbidity and mortality. As outlined in the literature, the diagnosis and management of CST require a multi-faceted approach that incorporates

an understanding of underlying risk factors, timely diagnostic imaging, and appropriate therapeutic interventions.

3.1. Risk Factors for CST

Several risk factors predispose individuals to CST, with facial infections being the most common cause. Infections arising from the sinus, nasal furuncles, or dental abscesses, often in the danger triangle of the face, can spread to the cavernous sinus, leading to septic thrombosis. Additionally, trauma, surgery, and conditions that increase the risk of thrombosis, such as thrombophilic disorders (*i.e.*, antiphospholipid syndrome, hyperhomocysteinemia, protein C and/or S deficiencies), local or metastatic neoplasms, pregnancy, and hormonal therapies, contribute to the development of CST [1]-[3]. Notably, patients with immunocompromised states, including those with HIV, long-term corticosteroid use, or bone marrow suppression, are at higher risk for developing CST due to their increased susceptibility to infections and poor wound healing [3] [4]. The interplay between these various factors necessitates careful consideration of a patient's full medical history when evaluating for CST.

3.2. Headaches and Red Flags

Headaches are a common complaint in patients presenting with CST and often serve as a key symptom in the initial assessment. However, not all headaches are indicative of serious underlying conditions. Primary headaches, such as tension-type headaches or migraines, are usually benign and self-limiting. In contrast, secondary headaches, which are associated with more severe pathologies such as infections, tumors, or vascular events, require urgent evaluation. The presence of red flag features in the history and clinical presentation of a headache should raise concern for a secondary cause like CST. The SNNOOP10 mnemonic serves as a helpful guide to identifying these high-risk features [5] [6].

- S: Systemic symptoms, including fever, which may indicate an infectious or inflammatory process.
- N: Neurologic signs or symptoms such as focal deficits or changes in consciousness, which are concerning for vascular intracranial or cervical events, brain abscesses, meningitis, or opportunistic infections.
- N: Neoplasm history which could indicate new brain neoplasm or metastasis.
- O: Onset of the headache, particularly if it is sudden, severe, or progressively worsening.
- O: Older age, as new-onset headaches in older adults may suggest a serious condition, including cancerous etiologies or vascular events.
- P: Prior history of headache with a change in pattern, such as increasing severity or frequency.
- P: Positional headache in nature, possibly indicating intracranial hypertension or hypotension.
- P: Precipitating factors such as sneezing, coughing, or exercise, representative

of posterior fossa malformation or Chiari malformations.

- P: Pregnancy or puerperium (6 weeks after childbirth), suggesting acute intracranial or cervical vascular etiologies.
- P: Painful eye with autonomic features which can be seen in posterior fossa, cavernous sinus or pituitary gland pathology in addition to ophthalmic etiologies.
- P: Post-traumatic onset of headache related to post-dural puncture procedures, preeclampsia, hematologic etiologies including anemia, as well as hypothyroidism, hyperthyroidism, or diabetes.
- P: Pathology of the immune system including HIV or T-cell lymphoma contributing to intracranial non-vascular events such as opportunistic infections.
- P: Painkiller (analgesic) overuse or a new drug at onset of headache which can cause symptoms due to adverse drug metabolite interactions.

In the context of CST, fever, vomiting, and nuchal rigidity can be indicative of the infectious nature of the condition often seemingly pointing towards a diagnosis of meningitis, while diplopia, ptosis, and periorbital edema suggest involvement of cranial nerves III, IV, V, and VI, indicating a CST picture [5]. These symptoms, particularly when progressive, should prompt immediate imaging to assess for CST.

3.3. Imaging in CST Diagnosis

Imaging is an essential component in the diagnosis of CST, given that clinical symptoms alone are often insufficient to confirm the condition. CT and MRI are the primary imaging modalities used to visualize the cavernous sinus and surrounding structures, each with unique advantages. While CT scans, particularly with contrast, can help detect certain signs of CST, such as asymmetric sinus enlargement and dilated superior ophthalmic veins, MRI with MR venography (MRV) offers greater sensitivity in identifying venous thrombi. It is also more effective in revealing filling defects, vascular involvement, and other indirect signs of CST like orbital edema and dural enhancement [2] [5]. However, CT alone may not provide sufficient detail to definitively diagnose CST, particularly in its early stages when radiographic signs may be subtle. Therefore, a combination of both CT and MRI is often required for a comprehensive evaluation. Furthermore, specialized techniques such as CT venography and MRV offer high sensitivity for detecting venous sinus thrombosis, with CT venography demonstrating up to 95% sensitivity, making it particularly useful when venous thrombosis is suspected [5].

3.4. Iron Deficiency Anemia and Thrombocytosis

A potential, yet often overlooked, risk factor for CST is iron deficiency anemia (IDA), particularly due to its association with reactive thrombocytosis. In IDA, the body compensates for low iron levels by increasing the production of platelets, a phenomenon known as thrombocytosis which is defined as a platelet count above 400×10^9/L. The mechanism behind this process involves the upregulation

of thrombopoietin, a hormone that stimulates megakaryocytes to produce platelets. In states of iron deficiency, the lack of iron inhibits its normal regulatory role in thrombopoiesis by creating a shift in hematopoietic progenitor cell differentiation, favoring megakaryocyte over erythroid lineage development, thereby leading to an increased platelet count. This reactive thrombocytosis can create a hypercoagulable state, elevating the risk of venous thromboembolism, including cavernous sinus thrombosis. Notably, while thrombocytosis associated with IDA is often considered a benign condition, it can contribute to the thrombogenic environment necessary for CST, especially in patients who may already be predisposed due to other risk factors like diabetes, an immunocompromised state, or trauma [7] [8].

3.5. Diagnosis and Treatment

Early diagnosis of CST is paramount, as prompt treatment can significantly improve outcomes. The clinical diagnosis of CST is often supported by imaging findings, with MRI or CT revealing characteristic signs such as asymmetric cavernous sinus enlargement, filling defects, and dilated ophthalmic veins [2] [5]. Additionally, laboratory tests such as elevated D-dimer, ESR, and CRP levels can further support the diagnosis, although these are nonspecific. Blood cultures should be obtained to identify the causative organism in cases of septic CST [4].

The treatment of CST typically involves a combination of antibiotics, anticoagulation, and sometimes surgical intervention. In septic CST, empiric therapy with third-generation cephalosporins, vancomycin, and metronidazole is essential, with therapy tailored based on microbiological findings [2] [5]. Anticoagulation is a controversial aspect of CST management, with some studies suggesting that it may prevent thrombus progression, while others show no clear benefit [4]. Corticosteroids may also be considered to reduce inflammation and vasogenic edema around the affected cranial nerves and orbital structures, though their use remains debated [2] [3]. In cases where abscesses or tumors are identified as the underlying cause of CST, surgical drainage or resection may be necessary.

4. Conclusion

Cavernous sinus thrombosis is a rare but serious condition that demands a high level of clinical suspicion. The complexity of its etiology—ranging from infectious causes like sinusitis to thrombophilic disorders—requires a comprehensive diagnostic approach, including a detailed clinical history, imaging, and laboratory tests. The role of imaging is critical in confirming the diagnosis, with CT and MRI playing complementary roles in detecting the presence of thrombus and associated complications. Moreover, understanding the red flags associated with secondary headaches, such as fever and neurologic deficits, can help identify patients at risk for CST. Early diagnosis and intervention are essential to improve outcomes, highlighting the importance of a multidisciplinary approach to patient care.

Conflicts of Interest

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

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