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CEREBRAL VENOUS SINUS THROMBOSIS (CVST) DURING INDUCTION THERAPY FOR ACUTE PROMYELOCYTIC LEUKEMIA: A CLINICAL CHALLENGE

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ABSTRACT Acute Promyelocytic Leukemia (APL) is frequently associated with severe coagulopathy, manifesting as life-threatening hemorrhage or thrombosis. While treatment with All-Trans Retinoic Acid (ATRA) and Arsenic Trioxide (ATO) has revolutionized outcomes, these agents are associated with rare but serious vascular events.Here we report a case of a 35-year-old woman who developed Cerebral Venous Sinus Thrombosis (CVST) and secondary intracranial hypertension while undergoing APL induction therapy. Early diagnosis via MR Venography and prompt initiation of anticoagulation led to a favorable clinical recovery.

KEYWORDS : Cerebral Venous Sinus Thrombosis, Acute Promyelocytic Leukemia, ATRA, Arsenic Trioxide, Papilledema, Hypercoagulability

INTRODUCTION

Cerebral Venous Sinus Thrombosis (CVST) accounts for approximately 0.5–1% of all strokes and typically affects young adults, especially women. It is associated with prothrombotic states including malignancy, infections, pregnancy, oral contraceptive use, and hematologic disorders[1]. Acute Promyelocytic Leukemia (APL) is a distinct subtype of acute myeloid leukemia characterized by t(15;17) translocation and PML-RARA fusion gene. Treatment with All-Trans Retinoic Acid (ATRA) and Arsenic Trioxide (ATO) has dramatically improved survival outcomes. However, APL is associated with complex hemostatic disturbances, leading to both hemorrhagic and thrombotic complications. Cerebral Venous Sinus Thrombosis (CVST) is an uncommon but serious thrombotic event that may occur during therapy[2]. We report a case of CVST presenting with features of raised intracranial pressure in a young woman undergoing treatment for APL.

Case Presentation

A 35-year-old woman was diagnosed with APL one month prior and was receiving an induction regimen of ATRA and ATO. She had no other significant comorbidities or traditional stroke risk factors. Now presented with a five-day history of persistent headache, visual blurring, and recurrent vomiting.

On Examination: The patient was neurologically intact regarding cognition (MMSE 30/30) and motor function but exhibited bilateral abduction deficits (Cranial Nerve VI palsy).

Fundoscopy: Revealed Grade IV papilledema.MRI/MRV of the brain identified a loss of flow void in the anterior superior sagittal sinus and signs of raised pressure, including a dilated optic nerve sheath and partial empty sella. Treatment was initiated with Acetazolamide to reduce intracranial pressure and the direct oral anticoagulant Apixaban. Chemotherapy was continued under close oncological supervision of the patient.

Follow-up: At one month, the patient reported total resolution of neurological symptoms, including vision restoration and normal extra ocular movements.

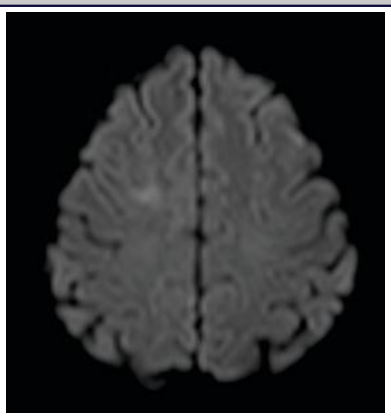

Figure 1: Diffusion Weighted Image [DWI] Brain: Subtle Hyperintense Area Seen in Right High Frontal Region With Adjacent Mild Corical Thickening and Swelling.

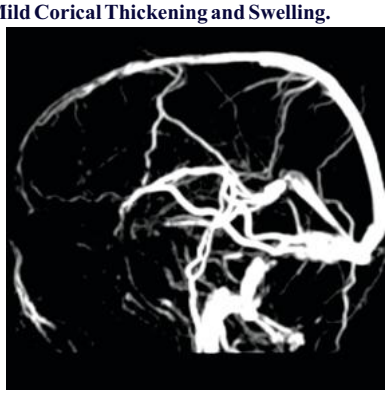

Figure 2: Shows Anterior Aspect of Superior Sagital Sinus is Narrowed in Caliber With Loss of Flow Void – Likely Indicates Thrombus

DISCUSSION

Cerebral Venous Sinus Thrombosis (CVST) is an uncommon but potentially serious neurological complication in patients with Acute Promyelocytic Leukemia (APL). Although APL is classically associated with hemorrhagic manifestations due to disseminated intravascular coagulation and increased fibrinolysis, thrombotic events are increasingly recognized during the course of the disease and its treatment. Both venous and arterial thromboses have been reported, venous thromboembolism being the most frequently described manifestation[3].

The pathogenesis of thrombosis in APL is complex and multifactorial. Leukemic promyelocytes release procoagulant substances such as tissue factor and cancer procoagulant, which activate the coagulation cascade and contribute to a hypercoagulable state. Additionally, inflammatory cytokines and endothelial injury further amplify the prothrombotic milieu. Treatment with all-trans retinoic acid (ATRA), a cornerstone in the management of APL, may also increase the risk of thrombosis by promoting platelet activation and enhancing the expression of adhesion molecules on endothelial cells, thereby facilitating thrombus formation[4].

Clinically, CVST may present with nonspecific neurological symptoms such as persistent headache, nausea, vomiting, visual disturbances, seizures, or altered sensorium. Signs of elevated intracranial pressure, including papilledema, may mimic pseudotumor cerebri and should prompt evaluation for venous sinus thrombosis, particularly in patients receiving induction therapy for APL. Early recognition is crucial, as delayed diagnosis may lead to complications such as venous infarction, cerebral edema, or permanent neurological deficits. Magnetic Resonance Imaging (MRI) with Magnetic Resonance Venography (MRV) is considered the most sensitive non-invasive modality for confirming the diagnosis[5].

Management of CVST in patients with APL poses a therapeutic challenge because of the simultaneous risk of bleeding and thrombosis. Anticoagulation therapyis generally recommended once significant intracranial hemorrhage is excluded or stabilized. Careful monitoring and multidisciplinary management are essential while continuing appropriate leukemia-directed therapies. Early diagnosis and prompt initiation of treatment are crucial for preventing long-term neurological complications and improving patient outcomes[6].

CONCLUSION

CVST is a rare but critical complication in APL patients treatedwith ATRA and ATO. Clinicians must maintain a high index of suspicion in any patient presenting with new-onset neurological or visual symptoms during induction therapy. Timely imaging and anticoagulation are essential for achieving positive clinical outcomes

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