

## Case Report

# Superior sagittal sinus and transverse sagittal sinus thrombus secondary to occult meningioma

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### ABSTRACT

The superior sagittal sinus is the largest of the venous sinuses. It receives blood from the frontal, parietal, and occipital superior cerebral veins and the diploic veins, which communicate with the meningeal veins. The superior sagittal sinus drains into the transverse sinuses. Central nervous system tumors like meningioma, glomus tumor, and medulloblastoma, often directly compress the veins and sinuses of the brain. Major sites of the occlusion include superior sagittal sinus (SSS) and transverse sinus. Initial days cerebral venous sinus thrombosis (CVST) was diagnosed only on autopsy. Since the advent of modern investigative modalities like magnetic resonance imaging (MRI), Computerised Tomography Angiography (CTA) and Magnetic Resonance Venography (MRV), more and more cases are being diagnosed confidently.

**Keywords:** CNS tumor, CVST, Meningioma, Superior sagittal sinus, Temporal sagittal sinus

### INTRODUCTION

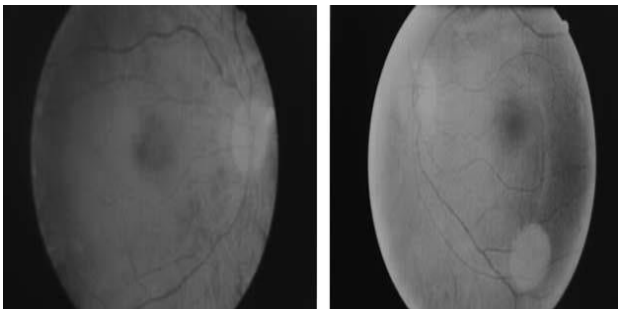
The superior sagittal sinus is the largest of the venous sinuses. It receives blood from the frontal, parietal, and occipital superior cerebral veins and the diploic veins, which communicate with the meningeal veins. The superior sagittal sinus drains into the transverse sinuses.<sup>1</sup> Occlusions of veins that drain was first published in 1820s. Ribes from France described the first case of dural sinus thrombosis.<sup>3</sup> Central nervous system tumors, as well as, systemic malignancies and solid tumors outside the central nervous system, can cause CVST. Central nervous system tumors include meningioma, glomus tumor, and medulloblastoma. These tumors often directly compress the veins and sinuses of the brain. Major sites of the occlusion include superior sagittal sinus (SSS) and transverse sinus. Inflammatory CVST is secondary to

infections in the “danger triangle of the face,” whereas noninflammatory CVST is typically associated with hypercoagulable state. In a small fraction of the patients, the underlying cause for CVST could not be readily identified.<sup>2</sup> Herein, we describe a female presented with superior sagittal sinus and transverse sagittal sinus thrombosis in association with meningioma. We present this case to highlight the concurrent occurrence of CVST and meningioma.

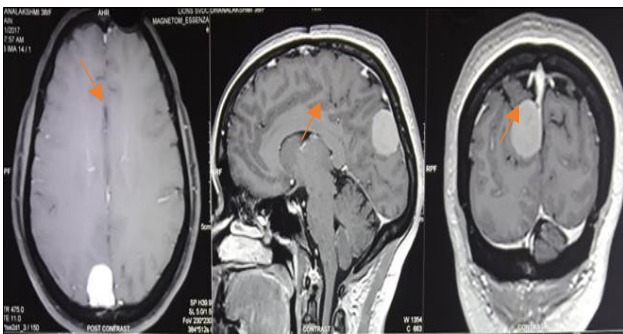
### CASE REPORT

A 38 years old female with no comorbid illness admitted with to the emergency room with 7 days history of severe headache in the occipital region, which was a non-radiating type, associated with unsteadiness of gait and multiple episodes of vomiting. She had history of fever for past 7 days. On initial examination patient was

conscious, oriented, and lethargic. Systemic examination showed no significant abnormality. With the routine blood investigations, infectious etiology has been ruled out. CT brain was performed which showed right parasagittal hyperdense lesion-? Meningioma. To substantiate CT findings, emergent brain MRI and MRV were performed. It showed rounded well-defined lesion measuring 2.6cm x 2.1 cm x 2.1 cm extra axial T2/FLAIR hyperintense and T1 hypointense space occupying lesion - features suggestive of meningioma in the parasagittal high posterior right occipito-parietal region. Metastatic evaluation revealed no additional lesion. MR venography showed compression of the superior sagittal sinus with thrombosis and left transverse sagittal sinus thrombosis.



**Figure 1: Retinal imaging upon hospitalization. Optic discs were clear.**



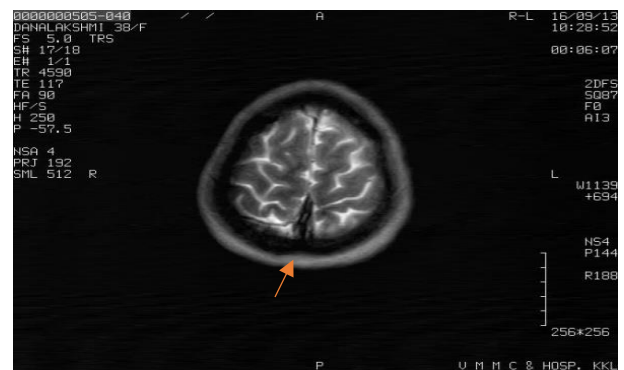
**Figure 2: Post contrast T1 weighted image showing early enhancing meningioma as pointed with arrows.**

EEG showed moderate abnormality with slow wave complexes of low to moderate amplitude at 1.5 to 3 Hz, most significant in the right parietal region but also in the central and occipital regions. The opening lumbar pressure was 23 cm H<sub>2</sub>O. Cerebrospinal fluid was normal: cell count: 0/mm<sup>3</sup>, protein: 54 mg/dL, and glucose: 81 mg/dL. Both acid fast staining and ink staining were negative.

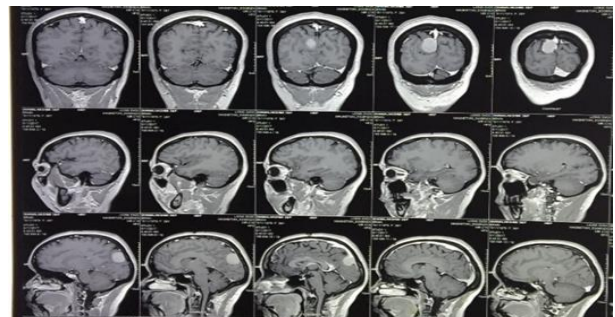
The workup for secondary causes of hyper coagulable state performed including antinuclear antibodies, ANCA, antiphospholipid antibody, anticardiolipin antibody, and lupus anticoagulant antibody, as well as protein C, S, and Factor V Leiden, which were negative. Her homocysteine level was within normal limits.

**MRI brain**

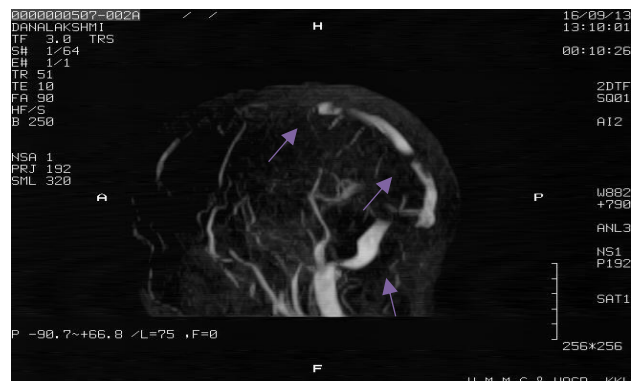
Evidence of rounded well defined extra axial T2/FLAIR hyperintense and T1 iso to minimally hypointense space occupying lesion with diffusion restriction in DW image seen in the parasagittal high posterior right occipito-parietal region adjacent the posterior flax cerebri. The lesion measuring 2.6cm (cranio-caudal) x 2.1 cm (max AP) x 2.1 cm (max. width) in size. Minimal perilesional edema anterior to the space occupying lesion. The lesion is seen closely appose to the adjacent superior sagittal sinus. Post contrast study shows dense enhancement of the lesion.



**Figure 3: Serial images of the enhancing meningioma in delayed phase showing delayed wash out.**



**Figure 4: T2WI showing flow void with interspersed hyperintensity.**



**Figure 5: MRV showing hypointensity along superior sagittal sinus and transverse sagittal sinus as indicated by the arrows.**



**Figure 6: MRV showing hypointensity in the transverse sagittal sinus.**

### MR venography

Minimal compression of the superior sagittal sinus seen in the posterior parietal region with minimal alteration of the flow signal in the compressed dural venous sinus. Compression of temporal sagittal sinus also noted in the left side. Right transverse and sigmoid sinus appears hypoplastic.

She was admitted to the intensive care unit, and heparin was started with the diagnosis of CVST. Patient underwent right occipital craniotomy and excision of right occipital parasagittal meningioma. Histopathological studies showed multiple fragments of a neoplasm composed of sheets and whorl of meningothelial cells with moderate pale eosinophilic cytoplasm, indistinct cytoplasmic borders, and spindle shaped ovoid nucleus and intervening areas of haemorrhages. F/S/O Meningothelial meningioma WHO grade I. On 4th POD haemostasis was obtained. She is managed with parenteral antibiotics, antiepileptic, IVF, head elevation and other supportive. Post-operative period was uneventful. She is discharged on POD 14. The patient recovered rapidly after the surgery. A follow-up that consisted brain CT at 3 months revealed no recurrence.

### DISCUSSION

Systemic thrombosis is well recognized in cancer patients, although CVSTs are uncommon in cancer. Cerebrovascular lesions are seen in just under one fifth of patients with cancer, resulting from 4 kinds of disorders sometimes intermingled in highly developed disseminated cancer: direct effects of the tumor, coagulopathies, infections, and therapeutic side effects 4-7; the latter 2, more than ever are seen in haematological malignancies.<sup>5</sup> Furthermore, CVST accompanied by squamous cell metastatic cervical mass, non-Hodgkin's lymphoma, and cerebral metastases of a colorectal cancer have been described.<sup>8-10</sup> The CVST infrequently presents as a paraneoplastic syndrome.<sup>6</sup> The CVST may accompany brain tumors such as brain glioma, and meningioma; it is noticeable that in brain tumors, the

direct invasion of sinuses by tumors (especially in meningioma) provokes the development of CVST. Meningioma is typically located on the convex surface of the meninges, cerebral falx, or sinus wall. The cellular origin of these tumors is unclear but may involve dural fibroblasts or arachnocytes (meningothelium), particularly in those forming the arachnoid villi. The venous sinus is one of the frequently involved structures because clusters of arachnoidal cells penetrate the dura in large number in the vicinity of venous sinuses.<sup>15</sup> In our case, the following factors might have contributed to the formation of CVST: Tumors compressing the sinuses, provoking the advent of clot in the sinus. Systemic anticoagulation is the first-line treatment for cerebral venous thrombosis, but identifying and managing the underlying and contributing factor (s) are more important.<sup>12,13</sup> In patients not responding to anticoagulation treatment, endovascular thrombolysis may be warranted.<sup>14</sup>

### CONCLUSION

Brain tumors should be suspected in corticovenous sinus thrombosis patients with no apparent underlying cause or contributing factors. Patients should be followed up carefully.

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