

# Diffusion restriction in thrombosed superior ophthalmic veins: Two cases of diverse etiology and literature review

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## ABSTRACT

Thrombosis of superior ophthalmic veins (SOV) is a well known entity occurring secondary to varied etiologies. We describe diffusion restriction in thrombosed SOV in two cases of different etiologies- bilateral involvement in a patient with septic cavernous sinus thrombosis (CST) and another where embolisation of an indirect carotico-cavernous fistula (CCF) resulted in complete SOV thrombosis accompanied by clinical worsening. Our cases add to the limited literature on diffusion findings in SOV thrombosis.

## CASE REPORT

### CASE REPORT

#### CASE 1:

A 64 year old female, a known poorly controlled diabetic for 2 years presented with right sided toothache since 30 days. There was no associated mandibular swelling. A dentist advised sugar control before proceeding with any dental intervention and she was started on Insulin. 8 days later, she developed headache and right facial pain which was continuous burning type. Two days later she noticed right sided ptosis and examination revealed right ophthalmoplegia. The patient worsened further with right visual deterioration and two days later she developed left sided ptosis. A day prior to admission (29 days into illness) she had recurrent left focal seizures with secondary generalization with associated nausea and vomiting. She had no nasal or ear discharge nor cough or expectoration. No facial weakness or bulbar symptoms were noted.

On admission, she was conscious and obeying commands. Local examination revealed chemosis, proptosis, right frontal and bilateral eyelid erythema. She was totally blind in the right eye and could count fingers at one meter with the left eye. There was total right ophthalmoplegia while on the left adduction and elevation was restricted. The right pupil was dilated and sluggishly reacting, left was normally reactive. Rest of the cranial nerves were normal. Power in the left upper and lower limb was grade 3/5, with mild left hypotonia and brisk deep tendon reflexes. Plantars were flexors bilaterally. There were no meningeal or cerebellar signs. The systemic examination was normal.

Her investigations on admission revealed an increased total count of 15100 cells/ mm<sup>3</sup> (range: 5000-10000/mm<sup>3</sup>), ESR of 80 mm/hr (range: 0-10 mm/hr in females) consistent with infection & random blood glucose of 287 mg% (range). The electrolytes, hepatic and renal functions were normal. Conventional MRI showed the parenchymal lesions to be abscesses while filling defects in the cavernous sinus (CS)

were due to clots. The CST was as evidenced by filling defects within the CS secondary to clots (figure 1A, 1B, 1C). However thrombosis of the bilateral SOV was visualized only on diffusion weighted images (figure 2A, 2B). Contrast enhanced MR venogram revealed thrombosis of the superior sagittal sinus (SSS) and bilateral transverse sinuses (TS) (figure 3A). Extension of the infection into the periorbital soft tissue (figure 3B) right orbit and intracranially as multiple abscesses in the right frontal lobe (figure 4A, 4B, 4C) was noted. She was managed with insulin and appropriate parenteral antibiotics. However her general condition later worsened due to septicemia. She developed thrombocytopenia and features of disseminated intravascular coagulation - these were corrected with platelet transfusion and fresh frozen plasma. After correction of thrombocytopenia she was subjected to surgical tapping of the frontal abscess. A culture confirmed the causative organism as staphylococcus aureus. She was continued on appropriate antibiotics and inotropic support, but later developed ARDS to which she succumbed.

#### CASE 2:

A 52 year old lady presented with left frontal headache which increased on eye movements for the last 2 months. This was followed by redness, pain and protrusion of the left eye. She was non diabetic but a known hypertensive for 6 years.

Local examination revealed left axial proptosis, chemosis and conjunctival congestion. Visual acuity was finger counting more than 5 feet bilaterally. Movements of the extra-ocular muscles were restricted. Bilateral pupils were reactive. The fundus showed changes of hypertensive retinopathy bilaterally. There was no ocular bruit. Systemic examination was normal. The hematological parameters were normal as was her blood glucose, renal and hepatic function.

With the clinical possibility of a carotico-cavernous fistula (CCF), she was referred to our institute for endovascular management of CCF. Clinical evaluation at admission was similar to that at the time of first evaluation. She underwent a complete six vessel angiogram which revealed a left indirect CCF fed by meningeal and accessory meningeal branches of the left internal maxillary artery (IMA) (figure 5A) and dural branches of left ICA. The venous drainage was to CS bilaterally and then bilateral inferior petrosal sinus. The enlarged left SOV showed a large intraluminal filling defect with a thin streak of contrast along the residual lumen superiorly (figure 5B) and it drained to the facial vein. The IMA feeders were embolised with 250-355 micron polyvinyl alcohol particles. A final check angiogram of the external carotid artery (ECA) showed near total obliteration of the fistula. Minimal residual filling of the fistula was seen through the dural ICA branches, which were not considered for embolisation. She was discharged after three days with advice for left carotid compression to aid thrombosis of the residual fistula. One day after discharge the patient came back with increased proptosis and chemosis and restriction of EOM. She could perceive only hand movements at this time. Conventional MR imaging showed a T1 hyperintense clot within the left SOV. However DWI revealed diffusion restriction in the left SOV (figure 6A, 6B, 6C) which was

consistent with the acute nature of the lesion. Timing the age of the thrombus was thus possible on DWI. She was given 5000 IU of Heparin every 6 hours and 1gm i.v Methylprednisolone daily for 5 days; local antibiotics were instilled. Over the next 10 days she showed significant symptomatic improvement. Her vision also improved to finger counting at 2 feet. She was put on Warfarin to adjust the INR to 2.5- 3. Steroids were gradually tapered.

#### DISCUSSION

Le Bihan first described the clinical application of diffusion weighted imaging (DWI) [1]. Presently DWI plays an indispensable role in the imaging of acute stroke where cytotoxic edema is seen as restricted diffusion. DWI also plays an important role in the evaluation of brain tumors, infection, demyelinating diseases as also intracranial hemorrhage. DWI can assist recognition of SOV thrombosis and in conjunction with other MR sequences help arrive at an etiological diagnosis and aid in guiding the appropriate management. We describe two such cases where DWI identified SOV thrombosis

The location of the CS and its anatomical connections make it a venous receptor for a large area. The dural sinuses and the connecting cerebral and emissary veins have no valves which allow blood to flow in either direction depending on the vascular pressure gradients. This makes the CS vulnerable to septic thrombosis resulting from infection at any of the sites it drains [2]. Septic cavernous sinus thrombosis (CST) most commonly follows infections of the middle third of the face. Other antecedent sites of infection include paranasal (usually sphenoid) sinusitis, dental abscess, otitis media etc [2, 3, 4]. In about 60-70% of patients, Staphylococcus aureus is the pathogen responsible for CST [2, 3, 5]. Other organisms responsible are streptococcal species, including Streptococcus pneumoniae; gram-negative bacilli; and anaerobes [6].

The diagnosis of septic cavernous sinus thrombosis requires a high index of suspicion and confirmation by MR imaging as CT findings may be subtle and a negative CT scan cannot rule out CST reliably when the clinical suspicion is high. The direct signs of CST on imaging are the enlargement and expansion of the cavernous sinus with lateral wall flattening or convexity rather than normal concavity. In addition, multiple irregular or single large filling defects within the enhancing cavernous sinus are highly suggestive direct evidence for thrombi [2, 7, 8, 9]. Indirect signs, related to concomitant venous obstruction, consist of dilation of the superior ophthalmic vein, exophthalmos, soft tissue edema, and thrombi visualized in the veins and sinuses tributary to the cavernous sinus [7, 8, 10, 11].

Our first case demonstrated bilateral CST, SSS and bilateral TS thrombosis. There was right orbital involvement and intracranial extension of the infection. The source of infection in our patient was presumably of dental origin which likely precipitated secondary to uncontrolled diabetes. Pavlovich et al described two cases of bilateral septic CST: the first of dental origin due to poor dental hygiene and the second

a complication of sphenoid sinusitis [12]. As noted in our case, spread to the opposite eye through the inter-cavernous sinus, usually within 24 to 48 hours of the initial unilateral involvement is a common and characteristic feature of CST and an ominous complication [2, 3]. Similar to our case, systemic effects from sepsis, local effects from direct injury to cranial nerves III through VI and impaired vascular drainage from the face and eyes and possible extension into adjacent tissue may result in a catastrophic illness [2] and the course may be rapid downhill [13]. Extension of the thrombosis to other venous sinuses may occur [6]. Other described intracranial complications of CST include brain and orbital abscesses [14] and mycotic aneurysms of the ICA [15].

Early diagnosis and surgical drainage of the underlying primary source of infection in conjunction with long-term intravenous antibiotic therapy are critical for an optimal clinical outcome. Management of patients with CST must also include treatment of primary infections and of associated complications [16]. The role of anticoagulants in CST remains controversial though. The mortality from CST has now decreased to approximately 20% to 30% with extensive antibiotic coverage [2].

Evaluation of ischemia in neighboring structures such as the optic nerves as a complication of CST is best done by DWI as described in previous reports [17, 18]. On review, we noted that the literature on bilateral SOV thrombosis secondary to CST is scant. The utility of DWI in demonstrating SOV thrombosis was first described by Parmar et al [19]. Michaelides described a case of bilateral SOV thrombosis due to raised factor VIII [20]. Shankar et al have described bilateral SOV thrombosis and attributed it to hyperglycemic state of their diabetic patient; the thrombosis resolved spontaneously. However they have not described the DWI findings in their case [21].

The uniqueness of our first case is the demonstration by DWI of bilateral SOV thrombosis, the first such report to the best of our knowledge. None of the previously stated cases demonstrate bilateral diffusion restriction in thrombosed SOV on imaging.

Our second case was that of sub acute SOV thrombosis after particle embolisation of an indirect CCF. An indirect CCF includes Barrow's type B, C, D and is supplied by dural branches of the ECA and/or ICA [22]. They are usually spontaneous and low flow. Indirect CCFs or dural cavernous sinus fistula is clinically difficult to diagnose with insidious onset of redness in the eye being the only subtle finding [23]. Treatment includes embolisation of ECA feeders and may be aided later by manual carotid compression [24].

In our patient, we embolised the ECA feeders which led to the cessation of flow to the fistula. However 4 days post embolisation the patient presented with acute pain, increased proptosis and congestion in left eye consistent with paradoxical worsening of symptoms. Paradoxical worsening has been well described in the literature and may occur soon after embolisation [25] or after few months [26]. Literature also describes paradoxical worsening with subsequent

spontaneous resolution in patients after transarterial embolisation [27, 28] stereotactic radiotherapy [29] or conservative treatment due to complete thrombosis of SOV. Chen et al have described a case where the paradoxical worsening in their case of CS dural arterio-venous fistula prompted for an urgent cerebral angiography which revealed thrombus formation in the distal left SOV and loss of the drainage via the facial vein [26]. In our case, embolisation led to arrest of forward flow to the SOV and induced thrombosis of its residual lumen which became symptomatic despite the fact that posterior venous route was intact. Chen et al state that the combination of worsening signs and evidence of thrombosis may indicate impending resolution of a cavernous-dural shunt [25]. To evaluate the cause of worsening, our patient was subjected to MR of the brain. The DWI revealed restricted diffusion in the entire left SOV. Though such a clinical scenario is described previously [27], to the best of our knowledge, ours is the first case of an indirect CCF where imaging has demonstrated diffusion restriction in SOV confirming its thrombosis.

The signal intensity of an acute infarct on DWI and ADC evolves in a time bound manner [30]. However the evolution of DWI-ADC signal intensity for intra parenchymal hematomas is dependent on different factors such as stage of the hematoma and varies [31]. The evolution of diffusion changes in intravascular clots perhaps differs when compared to that of intra parenchymal hematoma [19]. Imaging carried out in our first case was about 30 days into the illness whereas the second case was one day into the illness. Though in different time period of evolution (chronic and acute) respectively, the DWI-ADC signal corresponded to the hyper acute period of a parenchymal hematoma. Chu et al have illustrated a case where 20 days after symptom onset the thrombosed internal jugular vein demonstrated diffusion restriction [32]. Favrole et al have demonstrated similar findings in thrombosed venous sinuses on MRI performed over one month after symptom onset [33]. We thus re-emphasize what Parmar et al state in that the evolution of intravascular thrombi does not follow the evolution of intra parenchymal hematoma [19].

To conclude, DWI restriction in SOV thrombosis definitely draws our attention to a pathology which might otherwise be overlooked, though timing the intravascular clot may not be as correct. SOV thrombosis was identified correctly in both our patients. Despite appropriate management, our first patient succumbed to a different cause; our second patient however was benefited and showed significant improvement. DWI might thus be of benefit in all cases where a CS infective etiology is suspected especially when clinical possibility of orbital involvement is very high.

#### TEACHING POINT

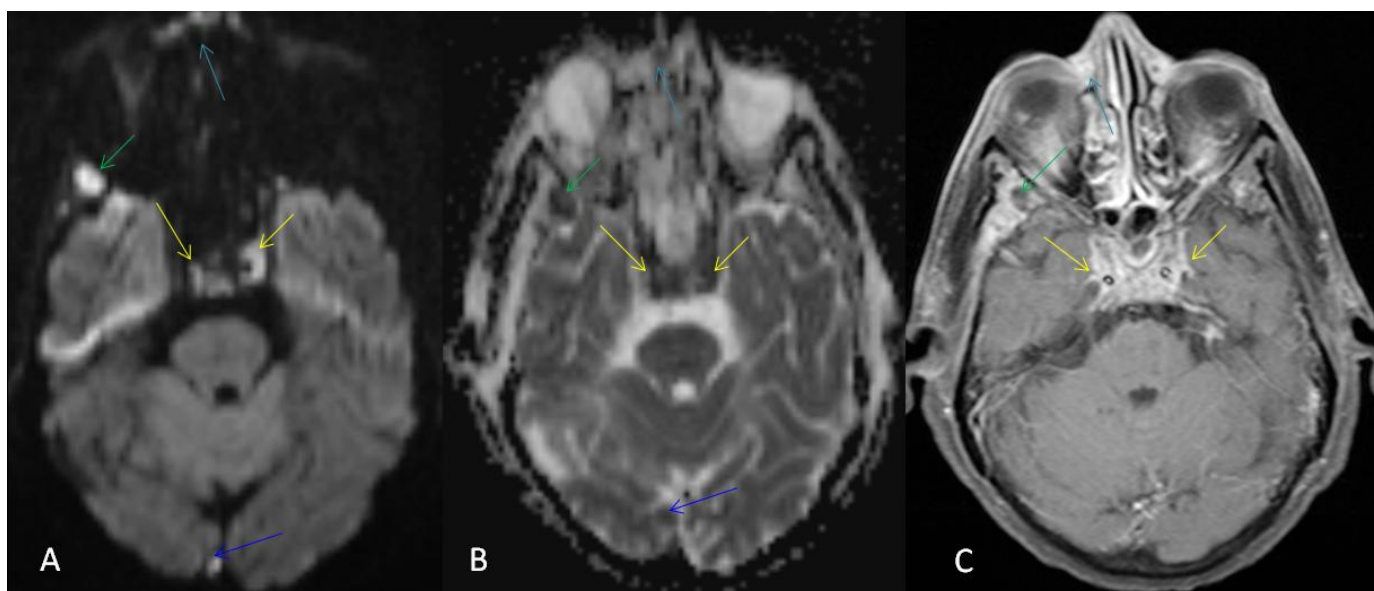
Diffusion weighted imaging might be of benefit in imaging infection of the orbital region. Attention to subtle changes in diffusion characteristics might help in identifying the lesions in time to provide the best management.

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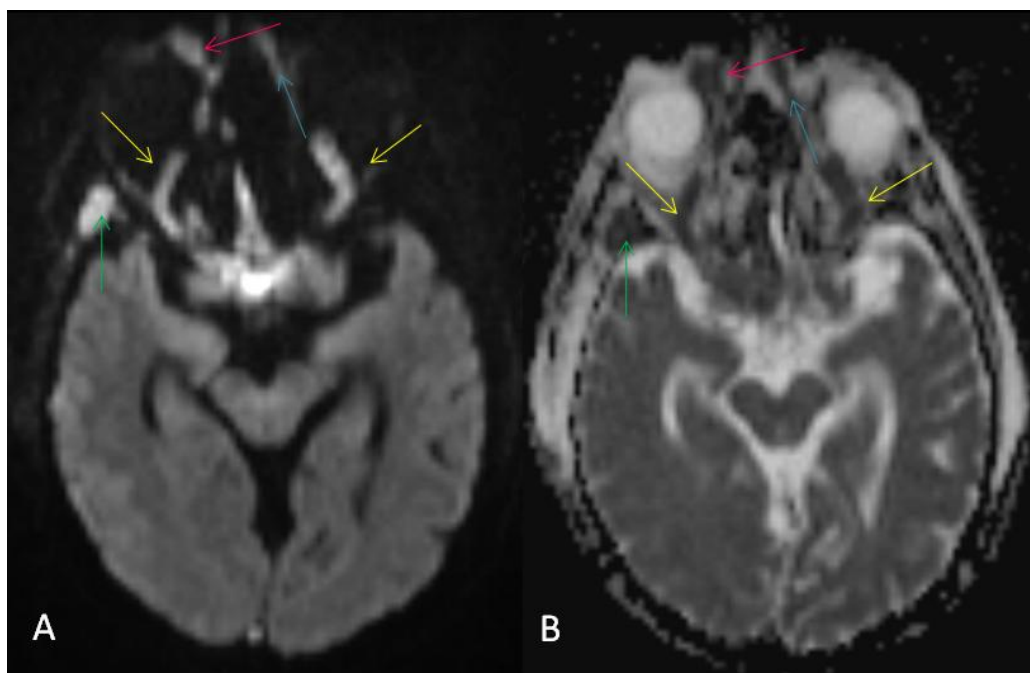
FIGURES



**Figure 1:** Case 1: 64 year old female patient, extensive venous sinus thrombosis with bilateral superior ophthalmic vein thrombosis.

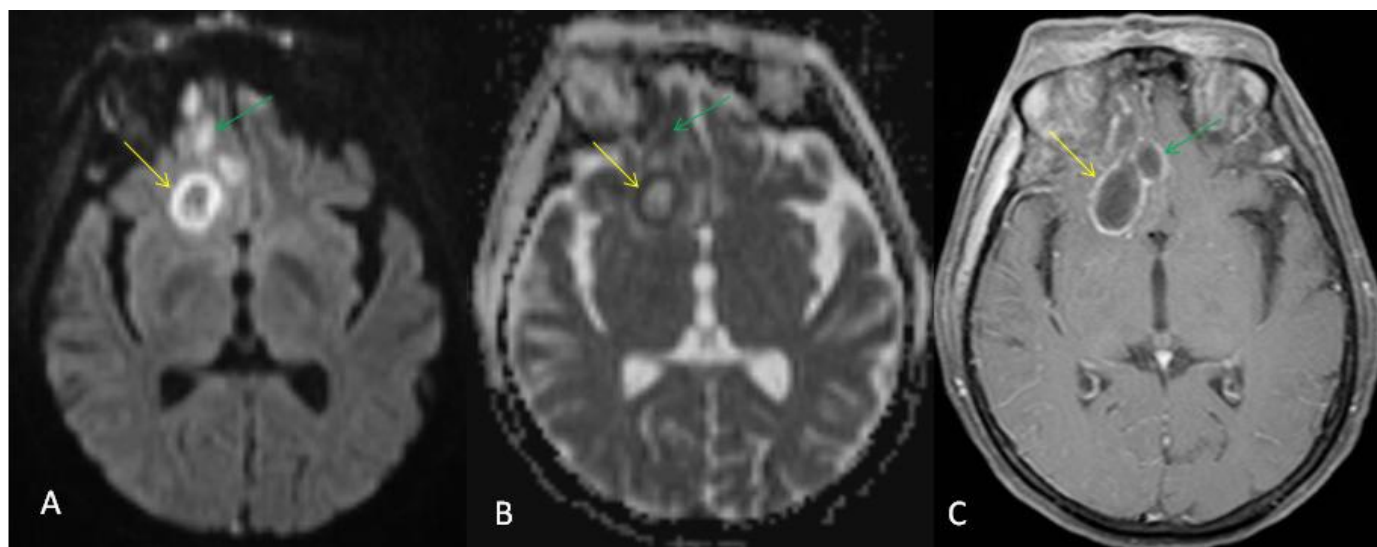
Figure 1: Diffusion weighted axial image (1.5T MRI, Siemens Avanto. TR: 3100ms, TE: 96ms, ep\_b0\_1000, 5 mm slice thickness) shows restricted diffusion in cavernous sinus bilaterally (left > right) (yellow arrows). Restricted diffusion also noted in the right temporal region (green arrow), anteriorly in the soft tissues of the nose (light blue arrow) and focally in the straight sinus (ink blue arrow). FIGURE 1B: ADC image (1.5T MRI, Siemens Avanto. TR: 3100ms, TE: 96ms, ep\_b0\_1000, 5mm slice thickness) corresponding to figure 1A shows hypointensity in the cavernous sinus bilaterally (left > right) (yellow arrows), in the right temporal region (green arrow), anteriorly in the soft tissues of the nose (light blue arrow) and focally in the straight sinus (ink blue arrow) consistent with restriction of diffusion. FIGURE 1C: Fat saturated contrast (Omniscan 0.1 mmol/kg body weight) enhanced spin echo T1 weighted axial image (TR: 847 ms, TE: 11ms, 5mm slice thickness) shows filling defects in bilateral cavernous sinuses (yellow arrows) suggestive of cavernous sinus thrombosis. Enhancing abscess noted in the right temporal region (green arrow) and nasal soft tissues (light blue arrow).





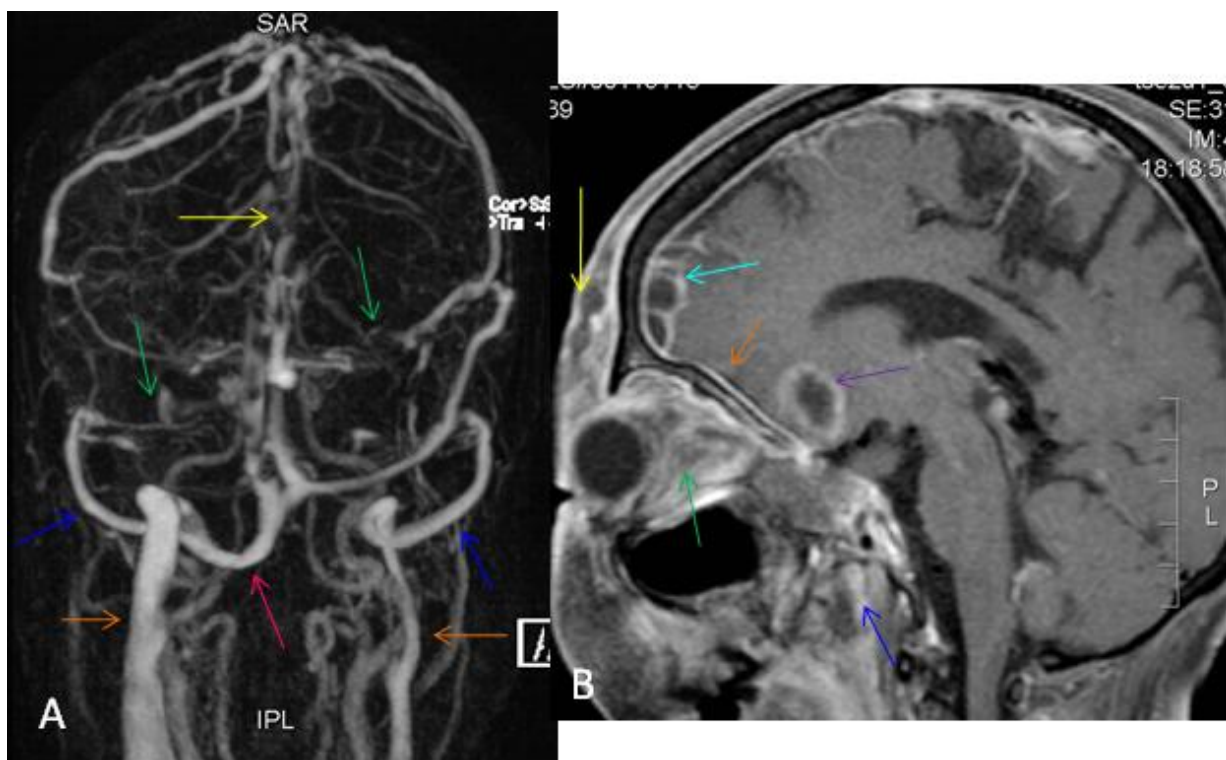
**Figure 2:** Case 1: 64 year old female patient, extensive venous sinus thrombosis with bilateral superior ophthalmic vein thrombosis.

FIGURE 2A: Diffusion weighted axial image (1.5T MRI, Siemens Avanto. TR: 3100ms, TE: 96ms, ep\_b0\_1000, 5 mm slice thickness) shows hyperintense signal within bilateral SOV (yellow arrow), extending medially in the right orbital region (pink arrow). Right temporal (green arrow) and nasal soft tissues (light blue arrow) show similar signal changes. FIGURE 2B: ADC image (1.5T MRI, Siemens Avanto. TR: 3100ms, TE: 96ms, ep\_b0\_1000, 5mm slice thickness) shows decreased signal within bilateral SOV (yellow arrow), extending medially in the right orbital region (pink arrow). Right temporal (green arrow) and nasal soft tissues (light blue arrow) show similar signal changes confirming restriction of diffusion secondary to SOV thrombosis.



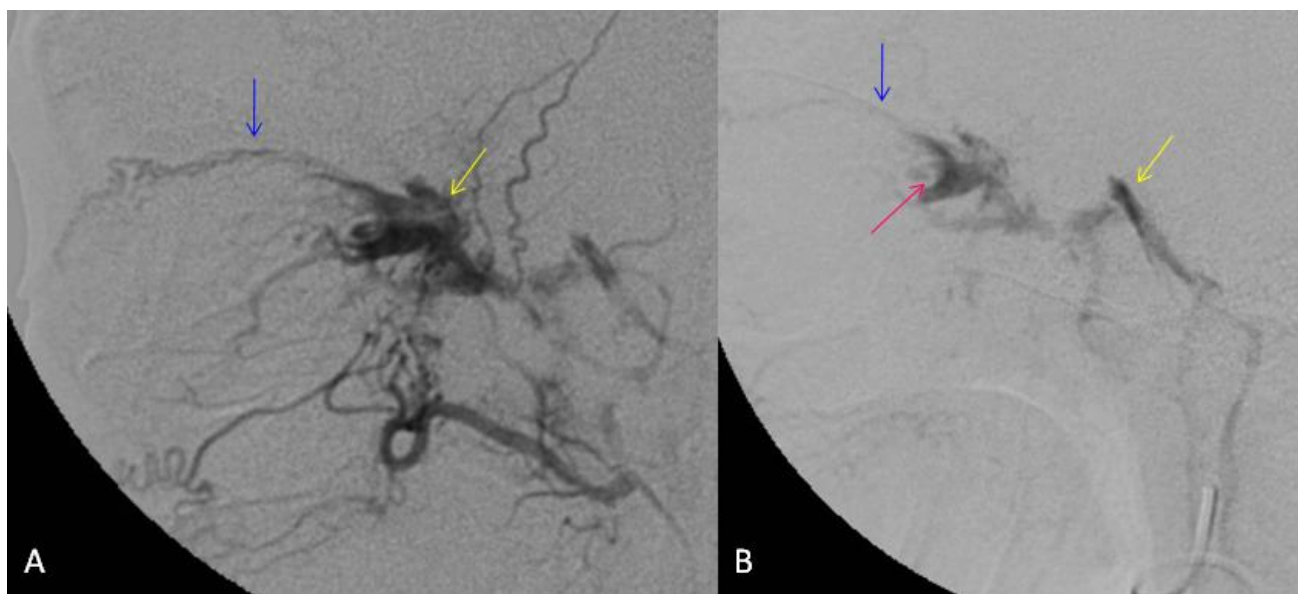
**Figure 3:** Case 1: 64 year old female patient, extensive venous sinus thrombosis with bilateral superior ophthalmic vein thrombosis.

Figure 3A : Diffusion weighted axial image (1.5T MRI, Siemens Avanto. TR: 3100ms, TE: 96ms, ep\_b0\_1000, 5 mm slice thickness) shows a peripheral rim of hyperintensity (yellow arrow) likely due to restricted diffusion. Adjacent region shows similar areas that extend anteriorly into the gyrus rectus (green arrows). Fig 3 B: ADC images (1.5T MRI, Siemens Avanto. TR: 3100ms, TE: 96ms, ep\_b0\_1000, 5mm slice thickness) show hypointensity in all corresponding areas (yellow and green arrows) that showed hyperintensity on DWI, thus confirming diffusion restriction. Fig 3C: Fat saturated contrast (Omniscan 0.1 mmol/kg body weight) enhanced spin echo T1 weighted axial image (TR: 847 ms, TE: 11ms, 5mm slice thickness) shows ring enhancement of the abscess (yellow arrow) with daughter abscess (green arrow) medially.

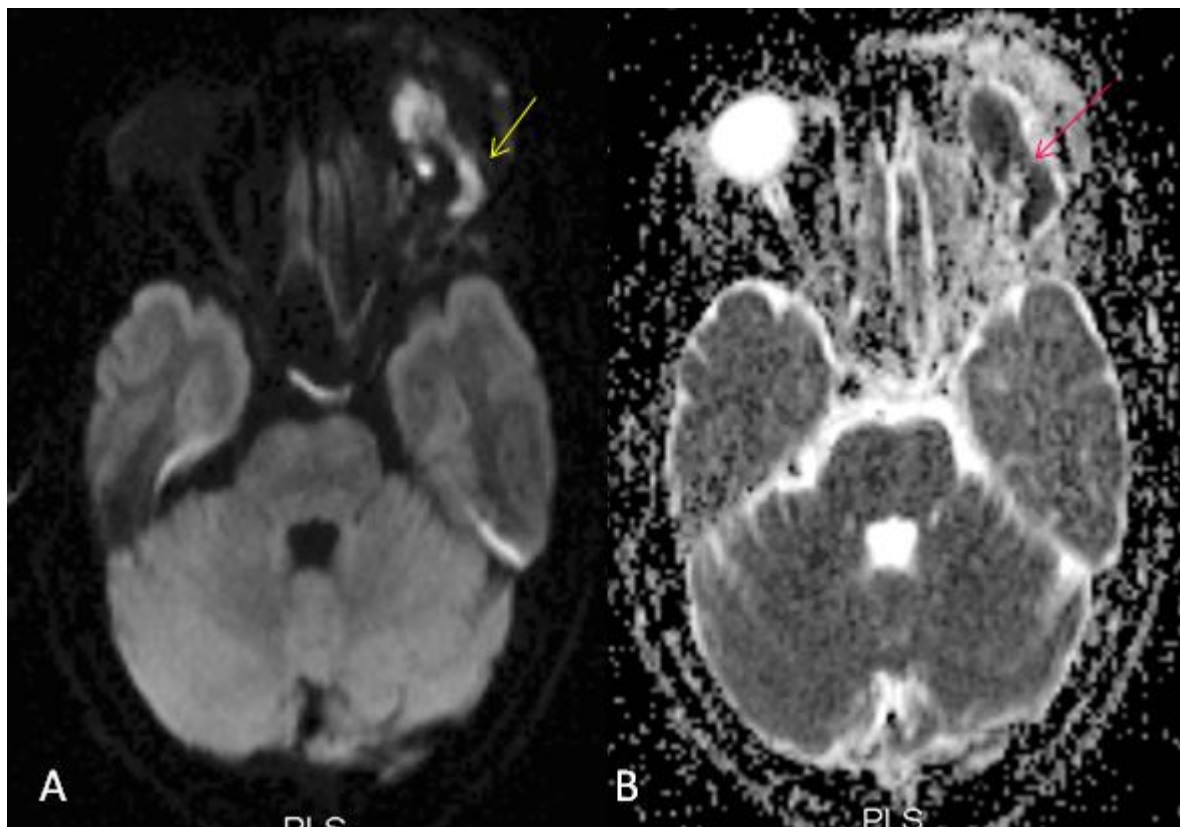


**Figure 4:** Case 1: 64 year old female patient, extensive venous sinus thrombosis with bilateral superior ophthalmic vein thrombosis.

FIGURE 4A: Contrast (Omniscan 0.1 mmol/kg body weight) enhanced MR venogram (1.5T MRI, Siemens Avanto, TR: 3.7, TE: 1.2, FLASH 3D sequence) shows thrombosis of the superior sagittal sinus (yellow arrow) and bilateral transverse sinuses (green arrows). Incidental patent occipital sinus noted (pink arrow). Bilateral sigmoid sinuses (ink blue arrows) and internal jugular veins (orange arrows) are well seen. FIGURE 4 B: Contrast (Omniscan 0.1 mmol/kg body weight) enhanced spin echo T1 weighted sagittal image shows associated complications. Multiple abscesses in the soft tissues of the forehead (yellow arrow), intraorbitally (green arrow), in the pharyngeal soft tissue (ink blue arrow); intracranially as pachymeningeal enhancement (orange arrow), subdural empyema (turquoise blue arrow) and parenchymal abscess (purple arrow).



**Figure 5:** Case 2: A 52 year old female patient with left sided indirect carotico cavernous fistula with post- embolisation left superior ophthalmic vein thrombosis. FIGURE 5A: Digital subtraction angiogram. Pre embolisation: Left ECA arterial phase lateral view shows early filling of the cavernous sinus (yellow arrow) that drains anteriorly to the partially thrombosed SOV (ink blue arrow). FIGURE 5B: Digital subtraction angiogram. Post embolisation: Left ECA venous phase lateral view shows venous drainage of the fistula posteriorly to the inferior petrosal sinus (yellow arrow) and anteriorly to the SOV. Note the partial filling of the residual patent SOV lumen superiorly (ink blue arrow) and the meniscus sign (pink arrow) within the SOV representing the intravascular clot.



**Figure 6:** Case 2: A 52 year old female patient with left sided indirect carotico cavernous fistula with post- embolisation left superior ophthalmic vein thrombosis. FIGURE 6A: Diffusion weighted axial image (1.5T MRI, Siemens Avanto, TR: 3500, TE: 105, 5mm slice thickness, ep \_ b 1000t) shows hyperintense signal in enlarged left SOV (yellow arrow). FIGURE 6B: ADC image 1.5T MRI, Siemens Avanto , TR: 3500, TE: 105, 5mm slice thickness, ep \_ b0\_ 1000) shows corresponding hypointense signal (pink arrow) consistent with restricted diffusion.

<b>Etiology</b>	Septic cavernous sinus thrombosis, carotico- cavernous fistula, hypercoagulable states, as a sequel to infection in adjacent regions, orbital neoplasm.
<b>Incidence</b>	Very rare
<b>Gender</b>	Not known due to rarity of lesion
<b>Age</b>	Any age
<b>Risk factors</b>	Infections of head, neck, face, hypercoagulable states, vascular lesions involving superior ophthalmic vein.
<b>Treatment</b>	Antibiotics , control of infection at the primary site
<b>Prognosis</b>	High morbidity , reduced mortality < 30%, secondary to antibiotics
<b>Findings on imaging</b>	USG: Mixed echogenic thrombus in SOV , NECT: Hyperdensity in SOV region, CECT: Filling defect in SOV (may or may not extend to adjacent cavernous sinus); MRI: T1 andT2 hyperintensity, DWI/ADC: restricted diffusion. DSA: vascular aetiology if responsible can be seen, partially thrombosed SOV may be occasionally seen.

**Table 1.** Summary table for superior ophthalmic vein thrombosis



	<b>CAVERNOUS SINUS THROMBOSIS</b>	<b>CAROTICO CAVERNOUS FISTULA</b>	<b>ORBITAL CELLULITIS</b>	<b>SUPERIOR OPHTHALMIC VEIN THROMBOSIS</b>
<b>X-ray</b>	May show adjacent paranasal sinusitis	Fractures, if any, in case of traumatic CCF	May show adjacent paranasal sinusitis	May show adjacent paranasal sinusitis
<b>USG</b>	May show no flow in SOV suggestive of thrombosis (if any)	Prominent/enlarged SOV if CCF drains anteriorly	Inflamed tissues, associated complications such as SOV thrombosis (no flow)	Mixed echogenic thrombus in SOV
<b>CT</b>	Enlarged CS, adjacent associated infective lesions. Thrombosed CS is hypodense on NECT; CECT: filling defects in CS	Associated fractures if any in traumatic CCF, thrombosed SOV, prominent CS	Mixed density lesions in involved area, bony erosion; CECT: enhancing soft tissue mass	CECT: filling defect in SOV (may or may not extend to adjacent cavernous sinus)
<b>MRI T1</b>	Associated parenchymal lesions may be seen	Prominent CS	Iso-hypo intense areas	Hyperintensity
<b>MRI T2</b>	Areas of hyperintensity in parenchyma may be seen	Prominent CS, flow voids if any in CS, SOV flow void, parenchymal lesions if any	Hyperintense areas	Hyperintensity
<b>DWI/ADC</b>	Restricted diffusion in case of acute - subacute thrombosis	Restricted diffusion if any acute infarcts; thrombosed SOV.	Restriction of diffusion to varying degree	Restricted diffusion
<b>CEMRI</b>	Enlarged CS with filling defects	Filling defects in the thrombosed vascular segment	Enhancing soft tissue	Partially thrombosed SOV may be occasionally seen.

**Table 2.** Differential diagnosis findings for superior ophthalmic vein thrombosis.

**ABBREVIATIONS**

DWI : Diffusion weighted imaging  
 SOV: Superior ophthalmic veins  
 CST: Cavernous sinus thrombosis  
 CCF: Carotico-cavernous fistula  
 MRI: Magnetic resonance imaging  
 CS: Cavernous sinus  
 SSS: Superior sagittal sinus  
 TS: Transverse sinuses  
 IMA: Internal maxillary artery  
 ICA: Internal carotid artery  
 ECA: External carotid artery  
 EOM: Extra ocular movements  
 INR: International normalized ratio

**KEYWORDS**

thyroid abscess; suppurative thyroiditis; bacterial throat infection

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