Visual Impairment in Cerebral Venous Thrombosis: The Various Shades of Gray

Visual impairment can complicate Cerebral Venous Thrombosis (CVT) through multiple mechanisms, contributing to its morbidity. It can be the only presenting symptom in patients with benign intracranial hypertension (BIH)-like presentation of CVT due to raised intracranial pressure (ICP) caused by venous thrombosis without venous infarcts.[1] Venous infarcts involving the occipital cortex causing cortical blindness can be a rare presentation of CVT.[2]

Visual impairment can occur while on treatment during the acute phase due to posterior cerebral artery compression secondary to herniation in large venous infarcts. The primary pathology causing the CVT, such as infectious causes or systemic vasculitis, can also affect the retinal artery or veins and cause visual impairment. Visual impairment can also occur as a late complication in CVT following the development of a secondary dural arteriovenous fistula (DAVF).

However, it is the BIH-like presentation of CVT that can often be misdiagnosed with disastrous consequences. In a series evaluating BIH patients,[3] 10% had CVT.

It is very important to identify these patients early to avoid the progression of visual deterioration and blindness. Before starting treatment, a primary or secondary DAVF should be ruled out.[4] Anticoagulants and acetazolamide should be started early.

Serial ultrasounds to measure the optic nerve head diameter can be used for monitoring the effect of treatment.[5] In chronic cases, monitoring can be done using optical coherence tomography (OCT) or visual fields.

Serial therapeutic lumbar punctures can be conducted if there are no contraindications or in cases of deterioration. Theco-peritoneal shunt (TPS) can be attempted; however, the complication rate with TPS is high. The literature on the safety, efficacy, and outcome of ONF and TPS in acute CVT is lacking. Reddy TA, *et al*.,[6] the authors have described the safety and outcome of the optic nerve sheath fenestration (ONSF) or the TPS in CVT cases with the progressive blurring of vision with papilledema. The complication rate was 47% (29.4% had major complications). One of the reasons postulated for this is the rupture of the bridging vessels that run between the dura and the surface of the brain with the sudden downward displacement of the brain post-shunting. It will be wise to use only programmable shunting for CVT patients with BIH-like presentations.

ONSF[7] is a procedure that incises the optic nerve sheath in the intra-orbital segment, reducing the pressure around the optic nerve. Unilateral ONSF can decrease pressure and papilledema in both ipsilateral (operated) and contralateral (unoperated) eyes. Unfortunately, this procedure is not available in many centers, and neurologists need to encourage their ophthalmology colleagues to take it up.

Endovascular procedures[8] to open up the blocked venous channels can also be attempted to prevent blindness in refractive cases. However, there is no clarity on the timing or the ideal patients who can benefit.

Thus, we see that in cases of CVT with visual impairment, there are many gray areas. One needs to be vigilant to pick up these cases early and monitor the vision and ICP closely. There is no clarity on what should be the best treatment strategy for saving the vision or the ideal timing for intervention. Often, the treatment may need to be tailored for a given patient[9] or a combination of interventions may be beneficial. Further research and studies are needed on this rather uncommon but dangerous complication of CVT. This article adds to the sparse literature available on this topic.

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