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Case Report

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OPHTHALMIC PRESENTATION OF CAVERNOUS SINUS VENOUS THROMBOSIS:A CASE SERIES

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ABSTRACT

Cavernous sinus venous thrombosis is an uncommon but dreaded neurological disease, commonly encountered in younger females. It presents with myriad of neurological and systemic symptoms and signs. However, visual disturbances can be a sole presentation with bilateral disc edema suggesting increased intracranial pressure. The predisposing factors include variety of inherited/acquired prothombotic and hypercoaguable states. Due to a varied symptomatology diagnosis is delayed or misdiagnosed which can result in life threatening complications such as herniation. With prompt diagnosis and management it is reversible. Lumbar puncture and MR Venography may aid in diagnosis and differentiation from other conditions.

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is a rare form of cerebro-vascular accident presenting with multitude of symptoms mimicking other neurological disorders leading to misdiagnosis.[1,3] It has gained recognition in recent years due to advancement in field of neuroimaging and can be life threatening.[1]

The incidence is two to five million cases annually commonly in younger population.[1,2] Headache accounts for 70-90% cases associated with vomiting and tinnitus.[4,6] However, it may present with visual disturbances as the first presentation, warranting a comprehensive ocular examination. We report three patients who presented to us with similar variable visual disturbances.

CASE DESCRIPTIONS

CASE 1

A 35 year male presented with blurring of vision since one month associated with severe throbbing headache, associated with right sided mastoid pain and tinnitus. He was on treatment for migraine and vertigo since 3 months. No history of nausea/vomiting, trauma or medical history.

The patient was conscious with stable vitals. Best corrected visual acuity (BCVA) 20/20 both eyes (OU). Red-green deficiency was noted on ishihara charts. Right eye RAPD Grade 3 was present, rest anterior segment was unremarkable. Fundoscopy revealed bilateral (B/L) disc edema of 6 and 3 DD respectively (Frisen grade 5).(Figure 1)Ultrasound-B scan showed increased optic nerve sheath diameter (ONSD). Routine hematological tests showed no abnormality. MRI brain and orbitshowed B/L dilation of peri-optic nerve subarachnoid space just posterior to the globe at the level of optic disc. Neurological examination was normal. MR venography (MRV) showed right sided transverse venous sinus thrombosis. (Figure2) Detailed blood panel for hypercoaguable states showed decreased protein S activity by 55%, raised antithrombin 3 by 130% and increased homocysteine levels of 20µmol/L. Neurology consultation was sought and a diagnosis of IIH secondary to CVST was made. Subsequent MRV showed B/L transverse and sigmoid CVST with extreme collateralization. He was treated with intravenous heparin. Follow-up showed gradual resolution until 6months.

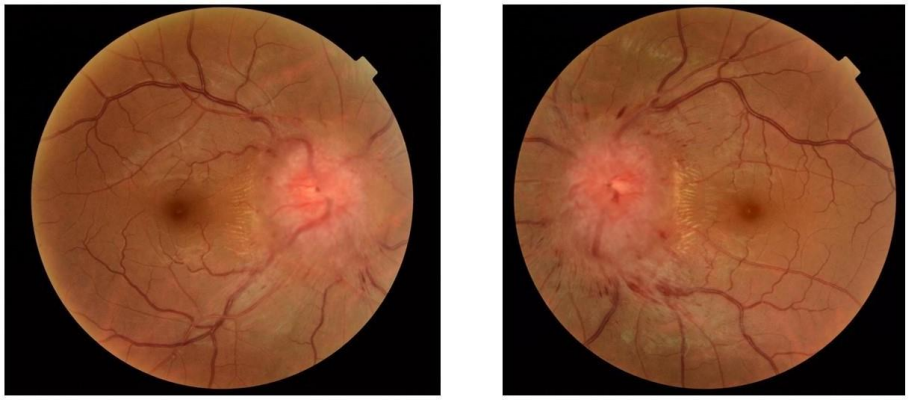
CASE 2

An 18 year old male presented with blurred vision and alternating hemi cranial headache associatedwith aura and 2 episodes of projectile vomiting within 10 days. Patient had no medical history. BCVA 20/30 OD and 20/40 OS. Fundoscopy revealed B/L disc edema of 2 and 5 DD (Frisen grade 5). (Figure3) Ultrasound B scan showing increased ONSD measuring 5.5 mm. MRI brain and orbit showed CVST in the form of partial thrombosis of right transverse sinus and complete thrombosisof right sigmoid sinus with B/L prominent CSF sleeve around optic nerve. Routine investigations were normal apart from raised homocysteine levels of >50µmol/L. The patient was started on oral anticoagulants. There was resolution of papilloedema with BCVA 20/30 at 3 months.

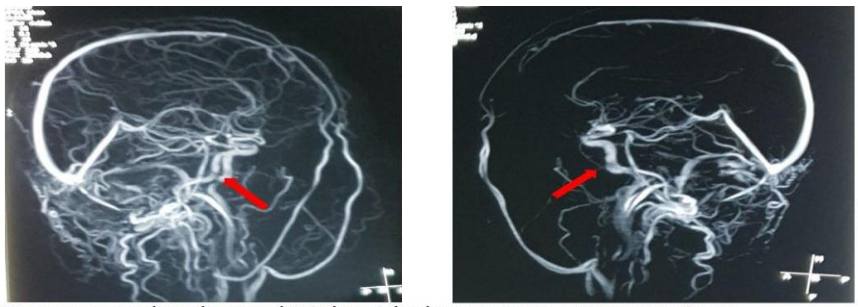
CASE 3

A 40 year male presented with complaints of blurred vision associated with throbbing headache since morning. Patient was a known diabetic and hypertensive since 15 years.

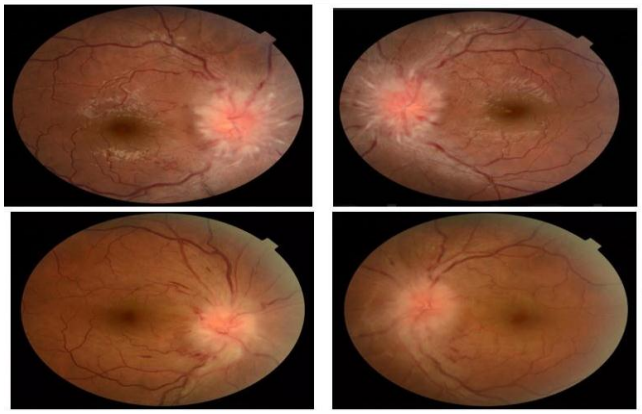
BCVA was 20/30 OU. Normal pupillary reflexes. Fundoscopy showed B/L disc edema of 4 and 6DD with no evidence of diabetic or hypertensive retinopathy. (Figure 3) Blood investigations revealed fasting and postprandial blood sugars of 185mg/dl and 325mg/dl respectively. Investigations for hypercoaguable states were normal. MRI showed extensive dural venous sinus and superficial cortical vein thrombosis without any venous hemorrhagic infarct. Follow-up visit with oral anticoagulants showed resolving disc edema with BCVA 20/20 OU at 4months.



Case 1: Bilateral Optic disc edema of of 6 and 3 DD respectively with flame shaped hemorrhages and exudates temporal to the disc.



- MR venography showed Right sided transverse venous sinus thrombosis as shown by absence of normal flow void on both T1- and T2-weighted images indicated by arrow heads .



- Case 2 Bilateral optic disc edema of 2 and 5DD respectively

- Case 3: Fundus photo showing bilateral gross disc edema of 4 and 6 DD.

DISCUSSION

IIH is encountered in young obese females with increased intracranial pressure without any CSF changes or any intracranial pathology. The main differential diagnosis of IIH is CVST. Diagnosisof CSVT becomes a challenge as the patient may present with isolated IIH in one-third of the patients.[10] It can occur in any age group, mean age being 37 years.[6,7] Risk factors include a variety of conditions like thrombophilia, inflammatory

bowel disease, Inherited or acquired hypercoaguable disorders, dehydration, pregnancy, oral contraceptives, substance abuse, head trauma.[5]

Thus, necessitating a complete blood investigation panel , including complete blood count with ESR, protein C/S, factor V Leiden, homocysteine, antithrombin III, APLA, lupus anticoagulant, cardiolipin, prothrombin time, and activated partial thromboplastin time to rule out underlying hypercoagulable states.

The sequence of involvement of sinuses are left followed by right transverse sinus, superior sagittal, and lastly cavernous sinus.[8] CVST can present as focal neurological deficits,isolated IIH,subacute diffuse encephalopathy, painful ophthalmoplegia involving third, fourth, or sixth cranial nerves.[6] Papilledema being the most common ocular sign observed.[8]

Diagnosis may need to be established based on localization of the thrombus and its differentiation from IIH and other neurological conditions. Neuroimaging includes CT scan and MRI as the first diagnostic modality. CT scan was found to be normal in 26% cases while MRI alone is 64% sensitive due to signal intensity variation of the thrombus governed by the duration classified as acute, subacute or chronic.[9] MRI shows absence of flow void in T1 and T2weighted images, however in chronic course it may look completely normal.[10] Thus, diagnostic sensitivityincreases to 100% when MRI is combined with MRV.[7,8,10]

Therefore, in a case of papilledema with suspected IIH, contrast-enhanced MRV becomes an important imaging modality. Lumbar puncture may aid in diagnosis.

Current therapeutic practice for CVST includes the use of anticoagulants such as intravenous heparin or subcutaneous low molecular weight heparin, use of thrombolytics, and symptomatic therapy including control of seizures and elevated intracranial pressure.[11]Endovascular treatment (chemical thrombolysis, mechanical thrombectomy) may be an option in patients who do not respond to anticoagulation treatment.[9]

CONCLUSION

CVST can present solely with ocular symptoms as the presenting complaint with or without neurological symptoms. While evaluating any patient of papilledema presenting with a normal unenhanced brain imaging, CVST should always be kept in mind. MRV and lumbar puncture may aid in diagnosis. If left untreated, complications such as transtentorial herniation may lead to death. It is crucial to differentiate CVST from IIH as the treatment for both are different and CVST can be life threatening causing stroke and death.[9]

Declaration of Patient Consent
A valid informed consent was taken by the patient and attender to use the case details and clinical pictures/ investigation reports for academic purposes.

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Conflicts of Intrest
Nil.

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