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RESEARCH ARTICLE

UNCOMMON EXPECTED COMPLICATION OF CEREBRAL VENOUS THROMBOSIS –FAVORABLE OUTCOME A CASE REPORT COMPLICATED BY INTRACRANIAL HYPERTENSION

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ABSTRACT

Thrombosis of the cerebral dural sinuses has been well described in the literature and rarely the dural sinus obstruction can result in unremitting papilledema (raised intracranial hypertension) causing blindness. We report a case of a 21-year-old man, who was diagnosed to have extensive sinus thrombosis involving superior sagittal sinus thrombosis and developed visual deterioration. The patient did not respond to conservative treatment and a thecoperitoneal shunt was performed; following which he developed intracranial hypotension with bilateral SDH needing bilateral burr hole evacuation. Patient is doing well at follow-up with good outcome.

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INTRODUCTION

Thrombosis of the cerebral dural sinuses has been well described in the literature and can present with diverse symptomatology and neurological findings (Horton *et al.,* 2012; McDonnell *et al.,* 1997; Cunha *et al.,* 2005). It has been most commonly associated with sepsis, trauma, pregnancy, the puerperium and many other hypercoagulable states (Horton *et al.,* 2012; McDonnell *et al.,* 1997; Cunha *et al.,* 2005; Orcutt *et al.,* 1984; Couban, 1991). Rarely the obstruction of the dural sinuses produces a clinical syndrome that resembles pseudotumor cerebri, and severe vision loss can be the presenting sign of cranial venous thrombosis (Horton *et al.,* 2012; Cunha *et al.,* 2005). We report this case to emphasize that in spite of advances in diagnostic radiological techniques, it remains an under-recognized condition and because chronic papilledema may cause progressive visual loss, raised intracranial hypertension should not be considered as a benign condition and fundal changes and visual function should carefully be monitored and evaluated with a phase contrastmrv (Horton *et al.,* 2012; McDonnell *et al.,* 1997; Cunha *et al.,* 2005; Orcutt *et al.,* 1984; Couban, 1991).

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CASE REPORT

A 21-year-old man was admitted at a peripheral hospital with complaints of sudden onset of weakness of both upper and lower limbs and deviation of angle of mouth to of one day duration. There was no history of fever or trauma. He was not a known hypertensive or diabetic. His general and systemic examination was normal except high blood pressure (170/100 mmHg). At presentation his gcs was 6 which later improved to 9.On examination he was opening eyes spontaneously, obeying command but confused.. Pupils were bilaterally equal and reacting to light. There was grade 1-2/5 hemiplegia involving right upper and lower limbs. Left side power was apparently normal. Deep tendon reflexes were exaggerated on right side and normal on left side. Right plantar was extensor and left was flexor. There were no meningeal signs. With all these findings a diagnosis of cerebrovascular accident was made. He was investigated with magnetic resonance imaging (MRI) extensive superior sagittal sinus thrombosis and right transverse sinus thrombosis and involvement of straight sinus. The patient was managed conservatively with anticoagulants and gradually improved in his motor power. About two weeks later he noticed deterioration in vision in his right eye. The fundus showed bilateral papilledema. In the right eye he could perceive only finger movements at one foot; however, the

vision in the left eye was comparatively better. Repeat computerized scan (CT scan) was apparently normal. Detailed blood investigations showed hemoglobin 8.8 gm%, total leukocyte count 11.000 (polymorphs 78%, lymphocytes 21% and monocytes 1%). Biochemical investigations were normal. Tests for serum anticardiolipin antibodies and lupus anticoagulant an immunoblot spectrum were negative. The patient underwent lumbar puncture study; opening CSF pressure was 26 cm of water. CSF was drained and soon after the patient noticed improvement in his vision. CSF analysis showed normal study. Coagulation profile showed prothrombin time (control/patient 11.6/17.9) with an INR 1.58. The patient was started on oral steroids and carbonic anhydrase inhibitors. However he did not respond to conservative management and a thecoperitoneal shunt was performed following which his vision started improving and he was discharged.He was admitted two weeks later with severe headache irrelevant talk and severe disorientation and high fever. An urgent ct was done and it showed bilateral sdh had developed. A bur hole right surgery was done patient was observed. His condition improved and after 4thday of post op status it again deteriorated. A repeat ct was done and it showed parietal sdh and another burr hole surgery was done. He was observed and his condition improved and also his vision improved but he had severe leg pain on mobilization post op status. A bilateral lower limb Doppler was done which showed an acute illio femoral thrombosis in right lower limb.he was reffered to vascular surgeon for further management and heis doing well now.

DISCUSSION

The clinical signs in cases of superior sagittal sinus thrombosis include headache, focal neurological deficits, seizures and mental disturbances (Thron *et al.,* 1986). The most frequent presenting symptoms of raised intracranialhypertension include headaches, vomiting and visual disturbances with normal neurological examination (except papilledema and the occasional sixth nerve palsy) (Wraige *et al.,* 2002; Lundar and Nornes, 1990). If there is development of raised intracranial hypertension then additional sign and symptoms of raised intracranial hypertension can get superimposed, (Thron *et al.,* 1986) and severe vision loss can be the presenting sign of cranial venous thrombosis. (Cunha *et al.,* 2005) The diagnosis of cerebral venous sinus thrombosis rests on radiological imaging of the cerebral venous system. (Couban *et al.,* 1991; Higgins *et al.,* 2003) These investigations either alone or in combination will confirm the diagnosis of venous sinus thrombosis and at the same time will rule out the hydrocephalus or a space occupying lesion (Higgins *et al.,* 2003). Angiography has been shown to be the best diagnostic tool and should not be delayed if there is a clinical suspicion of thrombosis. (Thron *et al.,* 1986) Recently, MR imaging combined with MR venography has been recognized as a safe and noninvasive technique for the diagnosis of venous sinus thrombosis and also for the follow-up. (Cunha *et al.,* 2005; Couban *et al.,* 1991; Ozsvath *et al.,* 1997) The treatment of superior sinus thrombosis associated raised intracrainial hypertension is controversial and ranges from anticoagulation or thrombolysis to supportive therapy only. (Couban *et al.,* 1991) If the patient is clinically stable and responds well he/she can be managed conservatively. (Couban *et al.,* 1991) The conservative management of raised intracranial hypertensionincludes, acetazolamide, furosemide and/or corticosteroids (Lundar and Nornes, 1990; Liu *et al.,* 1994). Lack of immediate improvement is an indication for optic nerve sheath decompression, (Horton *et al.,* 1992; Cunha *et al.,* 2005; Liu *et al.,* 1994) which can subsequently be operated with implantation of a lumboperitoneal, cisternoatrial or cisternoperitoneal shunt (Lundar and Nornes, 1990). Headache in post shunted patients should warn the treating physician about SDH secondary to low pressures.

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