International Journal of Research in Medical Sciences
Pramanik S et al. Int J Res Med Sci. 2017 Nov;5(11):5079-5081
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20174976

Case Report

Cranial nerve palsy in cerebral venous sinus thrombosis and response to therapy

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Received: 23 August 2017
Revised: 24 September 2017
Accepted: 29 September 2017

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ABSTRACT

Clinical presentation of cerebral venous sinus thrombosis (CVST) is variable. The most common symptom of CVST is headache. Cranial nerve palsy in CVST is rare. We present a case of CVST due to oral contraceptive pill usage presented with cranial nerve palsy. A 28 year old female presented with complains of headache and blurring of vision. On further evaluation, we found left 6th cranial nerve palsy with diminished vision on both eyes (left more than right). Initial CT brain imaging was normal. Magnetic resonance venography revealed thrombosis of superior sagittal sinus, right transverse sinus, and right sigmoid sinus extending into the right internal jugular vein. At the end of 6 months of anticoagulation therapy there is marked improvement of vision and complete recovery of left 6th nerve palsy. Though cranial nerve palsy in CVST have been reported previously, new findings of this case is that more sign contralateral to site of lesion and variable response to therapy.

Keywords: Anticoagulation, Cerebral venous sinus thrombosis, Cranial nerve palsy, Oral contraceptive pill

INTRODUCTION

The clinical diagnosis of cerebral venous sinus thrombosis is very difficult because of the variable mode of onset and wide spectrum of symptoms.[1,2] Cranial nerve palsy in cerebral venous sinus thrombosis is rare. Kushner et al. reported 5 cases of single or multiple cranial nerve palsies due to thrombosis of the ipsilateral transverse/sigmoid sinus.[3] We report a case of cerebral venous sinus thrombosis presenting with cranial nerve palsy contralateral to site of lesion and its variable response to anticoagulant therapy.

The important points of reference on the posterolateral surface of the skull are asterion, inion, apex of the mastoid process and suprameatal crest. The objectives of the present study were to determine the type of asterion depending on the presence or absence of sutural bone, to measure the linear distances of asterion from various bony landmarks, the nearest distance of the same from sigmoid and transverse sinus and also the thickness at the centre of the asterion that may be of importance to anthropologists, anatomists, forensic pathologists and neurosurgeons.

CASE REPORT

A 28 year-old female presented with complains of severe headache and blurring of vision for 2 days. It was not associated with fever, rash, convulsion and any limb weakness. There was no history of recent head trauma, facial or sinus infection. She was taking combined oral contraceptive pills for last 2 years. There was no family history of similar condition noted. She was alert, conscious. Her vitals were stable. On neurological examination, she had no nuchal rigidity but had loss of vision in both eyes. There was left lateral rectus palsy

(Figure 1A). Fundus examination showed bilateral papilledema.

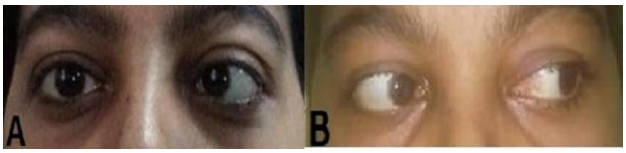

Figure 1: Case showed A- left lateral rectus palsy before therapy B- correction of palsy 6 months after therapy.

Other cranial nerves were normal. There were no other neurological abnormalities. CT scan brain (non-contrast) was normal. Magnetic resonance venography (MRV) revealed thrombosis of superior sagittal sinus, right transverse sinus, right sigmoid sinus extending into the right internal jugular vein (Figure 2-A). Automated perimeter was performed to measure visual field loss (Figure 3A, B). Visual evoked potential study showed bilateral axonal type of optico-retinal pathway dysfunction. Her blood count, P-time, INR, aPTT, protein c, protein s, anti-thrombin III, homocystine, ANA, p-ANCA, c-ANCA, antiphospholipid antibody, Lupus anticoagulant, ECG, Echocardiogram were normal.

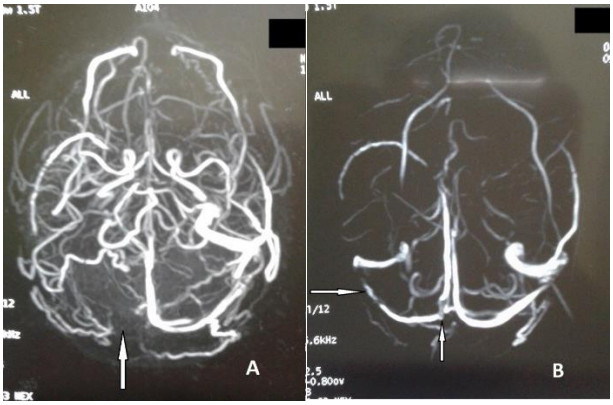

Figure 2: Magnetic resonance venography of case showing (A)- complete absence of right transverse sinus (arrow) before therapy and (B)- evidence of recanalisation in right transverse (arrows) after therapy.

She was initially treated with mannitol, subcutaneous low molecular weight heparin, acetazolamide, followed by warfarin for 6 months with strict bi-weekly monitoring of INR (international normalized ratio) to keep within 2-3.

After 6 months of therapy, her left sided lateral rectus palsy was corrected (Figure 1B) and visual field had improved significantly on right eye with minimal improvement on left eye as documented by automated perimetry (Figure 4 A, B). Repeat Magnetic resonance venography (Figure 2 B) showed narrow luminal caliber of superior sagittal, right transverse and right sigmoid bulb with evidence of recanalization.

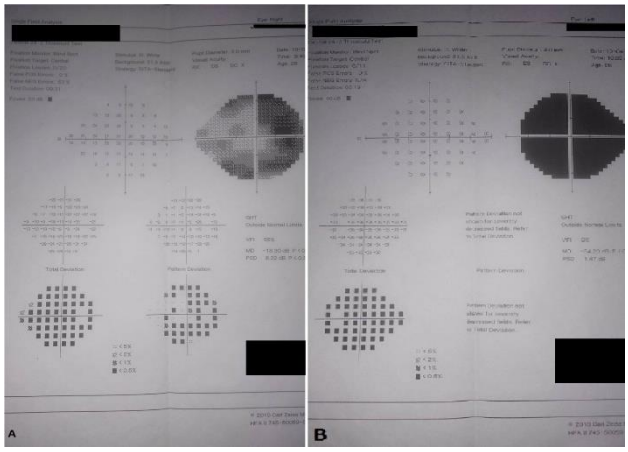

Figure 3: Automated Perimetry showed visual field defect (black area) (A-right eye, B-left eye) of case 1 before starting therapy.

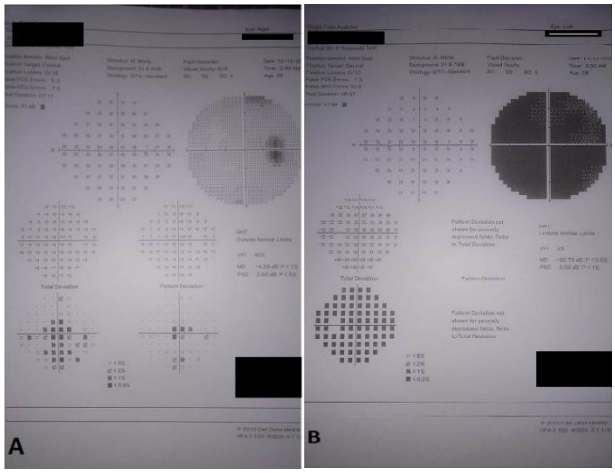

Figure 4: Automated Perimetry showed (A) significant visual field improvement of right eye, (B) minimum improvement of left eye of case 1 after therapy.

DISCUSSION

Cerebral venous sinus thrombosis is uncommon disorder. Several risk factors have been identified for CVST. These include medical conditions that increase the likelihood of thrombus formation such as thrombophilia, neoplasm, inflammatory condition, transient situations such as pregnancy, post-partum period, surgery, trauma, dehydration, CNS infections, and medications such as oral contraceptive pills (OCP).[4-6] Use of OCP has been shown in multiple observational studies to increase the risk CVST.[7] MR venography is considered the technique of choice for diagnosis and follow-up of CVT.[8] In early literature, Cranial nerve palsy in CVST has been attributed due to the elevated intracranial pressure, extension of thrombosis to venous channels, or direct pressure from the clot itself.[9] Lateral sinus (transverse plus sigmoid portion) drains blood from the cerebellum, brainstem and posterior portions of the cerebral

hemispheres, veins from cranial nerves in the posterior fossa, the middle ear and diploic veins. According to Kuehnen et al. thrombosis of the lateral sinus can produce venous congestion and dilatation of the cranial nerve veins. According to Kuehnen et al. thrombosis of the lateral sinus can produce venous congestion and dilatation of the cranial nerve veins which causes reversible compromised oxygen or glucose consumption within the cranial nerve tissue due to edema and backpressure, and due to this cranial nerve palsies may develop.[3] If the intracranial pressure is quite high, a sixth cranial nerve palsy may develop. Usually it presents as a false localizing sign, but it may also indicate extension of thrombus into the inferior petrosal sinus.[10] Here this case presented with bilateral loss of vision with left lateral rectus palsy.

In present case sixth nerve palsy may be due to raised intracranial pressure as a false localizing sign or involvement of superior sagittal sinus thrombosis. After 6 months of therapy, vision had improved significantly on right eye and minimal improvement in left eye as documented by automated perimetry and left lateral rectus palsy was recovered completely. We have shown a case of cerebral venous sinus thrombosis presented with cranial nerve palsy contralateral to site of lesion, more visual loss opposite to dominant site of lesion and its variable recovery after therapy. Because of its diverse etiology, manifestations and unpredictable course, cerebral venous sinus thrombosis remains a challenge for the clinicians.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Ameri A, Bousser MG. Cerebral venous thrombosis. Neurol Clin. 1992 Feb;10(1):87-111.
2. Bousser MG, Chiras J, Bories J, Castaigne P. Cerebral venous thrombosis: a review of 38 cases. Stroke. 1985;16:199-213.
3. Kuehnen J, Schwartz A, Neff W, Hennerici M. Cranial nerve syndrome in thrombosis of the transverse/sigmoid sinuses. Brain. 1998;121:381-8.
4. Bousser MG. Cerebral venous thrombosis: diagnosis and management. J Neurol. 2000;247:252-8.
5. Saposnik G, Barinagarrementeria F, Brown RD, Jr, Bushnell CD, Cucchiara B, Cushman M, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2011;42:1158. 9210.1161:STR.0b013e31820a8364.
6. McBane RD, Tafur A, Wysokinski WE. Acquired and congenital risk factors associated with cerebral venous sinus thrombosis. Thromb Res. 2010;126:81-7.
7. Dentali F, Crowther M, Ageno W. Thrombophilic abnormalities, oral contraceptives, and risk of cerebral vein thrombosis: a meta-analysis. Blood. 2006;107:2766-73.
8. Wasay M, Azeemuddin M. Neuroimaging of cerebral venous thrombosis: J Neuroimaging. 2005 Apr;15(2):118-28.
9. Kalbag RM, Woolf AL. Thrombosis and thrombophlebitis of cerebral veins and dural sinuses. In: Vinken PJ, Bruyn GW, eds. Handbook of Clinical Neurology. Amsterdam, Netherlands: North-Holland Publishing Company;1972;12:422–446
10. Martin PJ, Enevoldson TP. Cerebral venous thrombosis. Postgrad Med J. 1996;72:72-76.

Cite this article as: Pramanik S, Gazi MJ, Pal SK, Debnath NB. Cranial nerve palsy in cerebral venous sinus thrombosis and response to therapy. Int J Res Med Sci 2017;5:5079-81.