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CASE REPORT

Cerebral Venous Thrombosis: An Unusual Cause of Complex Ophthalmoplegia in an Elderly Man

Benjamin Ng Han Sim[1*], Prakash Supahiah[2], Goh Siew Yuen[2]

[1] Medical Department, Sibu Hospital, Sibu, Sarawak, Malaysia
[2] Opthalmology Department, Segamat Hospital, Segamat, Johor, Malaysia

*Corresponding author's email: drben84@gmail.com

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ABSTRACT

Cerebral venous thrombosis (CVT) is a neurological condition occurring because of thrombosis involving the cerebral venous sinuses. This case report is an unusual clinical manifestation of cerebral venous thrombosis in a 76-year-old Chinese man who presented with restricted eye movement and double vision. Despite extensive investigation, there was no better explanation for his clinical symptom and sign apart from cerebral venous thrombosis which was confirmed by magnetic resonance venography (MRV) of the brain. Once cerebral venous thrombosis was diagnosed, he was initiated on anticoagulation and discharged with oral warfarin. This case emphasizes the need to consider cerebral venous thrombosis as one of the rare causes of complex ophthalmoplegia especially when typical cardiovascular risk factors are lacking in an individual.

INTRODUCTION

Cerebral venous thrombosis is a neurological disorder because of thrombosis involving the venous sinuses of the brain. Its diagnosis remains a challenging one because CVT manifestations are protean and non-specific. Hence, the diagnosis consideration depends on initial clinical suspicion of this entity (Piazza, 2012). The presentation of CVT varies from mild manifestation, for example, non-disabling headache to potential life-threatening manifestation, such as raised intracranial pressure and coma. We wished to present a case of cerebral venous thrombosis

in an elderly man who presented with ocular palsies. The diagnosis of CVT was incidentally discovered following brain vessel imaging as part of the evaluation for his complex ophthalmoplegia presentation (Sakaida et al., 2014). The case illustration is followed by a discussion on cerebral venous thrombosis based on the literature review.

CASE PRESENTATION

A 76-year-old Chinese man was referred to the medical outpatient clinic for restricted eye movement and double vision of two months without eye pain and redness. This symptom had caused him great limitation in term of daily activities and great anxiety as this was the first time he presented to medical attention in his lifetime.

He denied any weakness, abnormal sensation, or abnormal movement of his limbs. There was no headache, vomiting or instability. He had no constitutional symptoms and no preceding head injury or trauma. His social history was remarkable for 40 pack/year smoking since his younger days. Other aspects of history unremarkable.

Ocular examinations revealed asymmetrical bilateral ptosis more prominent over the left eye which did not obstruct his vision axis. There were no scars seen overlying upper lids and the surrounding skin was not erythematous. On gentle retraction of both eyelids, there was an inward deviation of both eyes, more prominent over the left eye. Dysconjugate gaze in all positions with diplopia appreciated.

Visual acuity of right eye 6/24 (pinhole: 6/24); left eye 6/18 (pinhole 6/12). The pupils were equal 3 mm in size with an intact direct and consensual light response without relative afferent pupillary defect. Anterior segment and fundus examination were unremarkable. Thyroid eye signs such as proptosis, lid lag and conjunctiva chemosis particularly over the horizontal rectus muscles insertion not noticeable. Features of myasthenia gravis such as fatigability and Cogan lid twitch were not present. His scalp was not tender, no features of jaw claudication to suggest giant cell arteritis. Otherwise, neurological assessment of other cranial nerves, limbs and coordination was non-contributory.

His blood pressure was 140/74 mmHg, heart rate was 73 beats/min and the temperature was normal. Auscultation of the lung and cardiovascular system revealed clear lung field accompanied by first and second heart sound without an obvious murmur and no carotid bruit. Blood investigations, lumbar puncture, electrocardiogram, transthoracic echocardiography, CT brain with contrast, magnetic resonance imaging/ venography/ arteriography of the brain were done (Table 1).

Table 1 Investigations done for this patient as inpatient and outpatient basis

Full blood count	Hb 12.6 g/dL, Total White $5.7\times10^9/mm^3$, Platelet $225\times10^9/mm^3$ (within normal limits)
Renal profile	Na 140 mmol/L, K 4.89 mmol/L, urea 4.3 mmol/L, creat 90 µmol/L (within normal limits)
Liver function test	Total Bilirubin 7.9 µmol/L, albumin 42.6 g/L, globulin 33.3 g/L, ALT 21.6 U/L; ALP 107 U/L (within normal limits)
Fasting lipid profile	Total Cholesterol 6.44 mmol/L (raised), Triglyceride 2.2 mmol/L (normal limit)
Fasting blood sugar/ HBA_1C	5.49mmol/L; 6.0% (normal limit)
Thyroid function test	TSH 2.51 µIU/mL, free T4 19.22 pmol/L (within normal limits)
Acute inflammatory markers	ESR 15 mm/hr (not raised) CRP 0.56 mg/L (not raised)

Biohazard screening	Hep B/ Hep C/ HIV/ VDRL: nonreactive
Diagnostic lumbar puncture	Appearance clear, total protein 0.56 g/L (raised), cell count = 0 Gram stain negative, direct smear for acid-fast bacilli negative. C&S no growth isolated, cytology: negative for malignancy
Electrocardiogram	Sinus rhythm
Transthoracic echocardiography	Ejection fraction 65%, good left-ventricular function, mild valves regurgitation, normal chambers size, no thrombus
CT brain with contrast	Multifocal cerebral old lacunar infarcts, no evidence of focal enhancing brain parenchymal lesion
Magnetic resonance imaging/ venography/ arteriography of brain (Figure 1)	Dural sinus thrombosis involving the left transverse and sigmoid sinuses extending to the left internal jugular vein. There is underlying cerebral atrophy with both centrum semiovale and left frontal old lacunar infarcts. There is no evidence of focal lesion or abnormal signal within the brain stem. Both intracranial and extracranial arteries were normal.

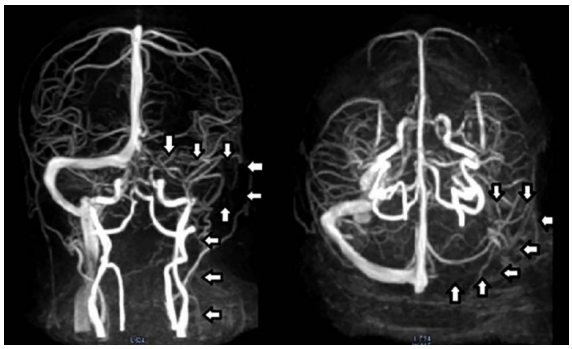

Figure 1 Magnetic resonance venography (MRV) illustrating thrombosis of the left transverse, sigmoid sinus and internal jugular vein (white arrows) in the coronal (left) and axial (right) planes

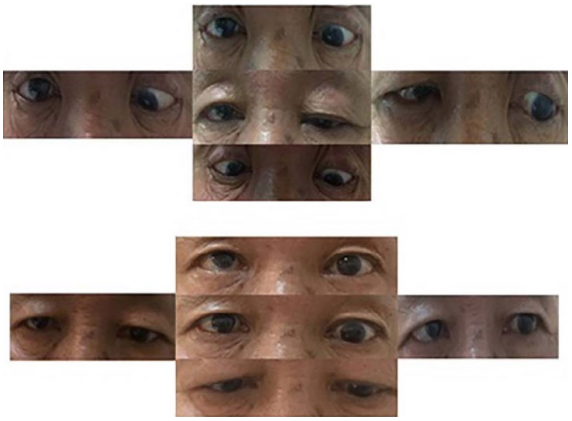

Figure 2 Ophthalmoplegia and bilateral ptosis before (upper panel) and after (lower panel) commencement of anticoagulation (at 1st month of therapy)

Treatment

Once cerebral venous thrombosis was diagnosed, he was initiated on anticoagulation and discharged with oral warfarin once INR target of 2 to 3 achieved.

Outcome and Follow-Up

The ptosis and ophthalmoplegia improved with anticoagulation therapy (Figure 2). He tolerated warfarin without any complication like bleeding. At the fourth month of treatment, his vision improved with best-corrected visual acuity of 6/12 for the right eye and 6/15 for the left eye without diplopia in all directions of gaze. He can achieve independence in activities of daily living.

DISCUSSION

This case of complex ophthalmoplegia posed a diagnostic challenge given that the initial workups were non-contributory toward a diagnosis. In fact, the diagnosis of cerebral venous thrombosis came as an incidental finding when an MRI brain was requested to look for brainstem or base of skull pathology. As the patient is an elderly man, a serious diagnosis such as malignancy with basal of skull metastasis and giant cell arthritis need to be ruled out. The most common malignancy in an elderly Chinese man that might present in this pattern would be nasopharyngeal carcinoma and we have ruled this out by

requesting our colleagues for formal ear, nose and throat assessment (Chang & Adami, 2006). Giant cell arthritis was unlikely given the lack of suggestive symptom and normal inflammatory markers. The same reason applied to other differential diagnoses like thyroid eye disease, myasthenia gravis and Miller Fischer Syndrome. Chronic progressive external ophthalmoplegia or better known as Kearns-Sayre Syndrome occurs in the younger age group with positive family history.

Cerebral venous thrombosis (CVT) is a neurological condition occurring because of thrombosis involving the cerebral venous sinuses. In more than 85% of cases, at least a predisposing risk factors like dehydration, pregnancy, postpartum period and malignancy can be identified (Piazza, 2012). However, in up to 13 per cent, there is no underlying aetiology or risk factor for CVT as exemplified by this case.

The clinical manifestation for cerebral venous thrombosis is heterogeneous with coma and death at one end and mild symptomatology in the other. The most common symptom for cerebral venous thrombosis is a headache which is reported in the range of 84.2 to 90% (Chang & Adami, 2006; Wang et al., 2015; Sakaida et al., 2014). However, in this case, the presenting symptom is double vision. From our knowledge, there is no literature which describes complex ophthalmoplegia as a manifestation of cerebral venous thrombosis apart from cavernous sinus thrombosis (Tanislav et al., 2011). Imaging had ruled out cavernous sinus thrombosis in his case. The closest resemblance to this case is the description of multiple cranial nerve palsies in the lateral sinus, jugular or posterior fossa veins thrombosis (Kuehnen et al., 1998). The ophthalmoplegia may be a false localising sign due to the high intracranial pressure resulting from thrombosis (Larner, 2003). The nerves supplying extraocular muscles may be stretched because of brain stem displacement due to the raised intracranial pressure.

CONCLUSION

This case emphasizes the need to consider cerebral venous thrombosis as one of the rare causes of complex ophthalmoplegia especially when typical cardiovascular risk factors are lacking in an individual.

CONFLICT OF INTEREST

The authors declare that they have no competing interests in publishing this article.

CONSENTS

Written informed consent was obtained from the patient to publish the case with its related pictures. A copy of the written consent is available for review by the Chief Editor.

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