CASE REPORT

Rare Presentation of Lower Motor Neuron Facial Palsy in Cerebral Venous Thrombosis – A Case Report

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Abstract

Clinical features of cerebral venous sinus thrombosis commonly include headache, early and late seizures, and focal deficits. Cranial nerve (CN) involvement primarily includes the ocular motor nerves. Very few cases have been reported with isolated facial nerve involvement. Our case highlights a rare atypical clinical presentation of cerebral venous thrombosis (CVT). Facial nerve palsy in cerebral and cortical vein thrombosis is rare with an unclear pathophysiology and is rarely reported. We report a case of a 44-year-old male, a chronic alcoholic admitted with complaints of severe headache and raised intracranial pressure (ICP) features. He developed left-sided facial nerve palsy with impaired taste perception on the left side. Magnetic resonance imaging (MRI) of the brain and venography showed evidence of a late subacute stage of occlusion in the superior sagittal sinus, straight sinus, right transverse, and right internal jugular vein (IJV) with features of intracranial hypertension. Facial palsy in our patient is considered to be due to transient neuropraxia in the intracranial segment of the nerve. This case highlights facial palsy as a rare clinical presentation of cerebral venous thrombosis.

Keywords: Anticoagulation, Case report, Cerebral venous thrombosis, Cerebral venous sinus thrombosis, Facial palsy, Papilledema.

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Case Description

A 44-year-old male presented to the emergency department with complaints of severe throbbing headache, the predominantly occipital region which later became diffuse and holocranial, present throughout the day with aggravation on coughing and sneezing for the past 5 days. He had no visual symptoms. There was no associated nausea or vomiting. However, he gave the history of photophobia and intermittent dizziness during the headache episodes. On the day of admission, i.e., 5 days after the onset of the headache he noticed the inability to close his left eye and a deviation of the angle of the mouth to the right side. There was no history of fever, ear pain or discharge, aural fullness, tinnitus, or HOH. His general examination was normal. His external ear, mastoid, and tympanic membrane were all normal. Neurological examination revealed bilateral mild abduction restriction, left lower motor neuron (LMN) facial palsy with Bell's phenomenon, and bilateral established papilledema. His visual acquity was 6/12 improving with pinhole, color vision was normal. His visual field examination revealed central and centro-caecal scotoma in the right eye. There was no spinomotor, sensory, cerebellar, and autonomic system involvement. A clinical diagnosis of raised intra cranial pressure (ICP) headache with papilledema and left LMN facial nerve palsy was made.

Magnetic resonance imaging (MRI) of the brain with magnetic resonance venography (MRV) was done which showed evidence of cerebral venous thrombosis of the late subacute stage involving the superior sagittal sinus, straight sinus, and right transverse, and right internal jugular vein (IJV). He also had extensive collateral formation in the venous system. Routine investigations showed mild leucocytosis, normal hemoglobin, and hematocrit. His renal and liver parameters were normal. Prothrombotic and vasculitic profiles were negative.

A diagnosis of cerebral venous thrombosis with left LMN facial palsy and papilledema was made. Cerebrospinal fluid (CSF) opening pressure and CSF analysis were not done. Treatment was initiated with parenteral followed by oral anticoagulants and anti-edema measures with injection heparin 5,000 units Qid f/b Tab. Acitrom 2 mg od which was titrated according to his INR, tablet acetazolamide 250 mg 2 BD, and other supportive measures with paracetamol, amitriptyline, clonazepam, and IV fluids. Patient showed symptomatic improvement on initiation of anticoagulation. Facial muscle stimulation, physiotherapy, lid taping, and eye care were given. His facial weakness improved 4 weeks after initiation of anticoagulation.

Discussion

Cerebral venous sinus thrombosis is an uncommon vascular condition accounting for about 1–2% of cerebral infarction. Its incidence is 1–2 cases per 100,000 patients/year with increased incidence noted in middle-aged females up to 2–3/100,000

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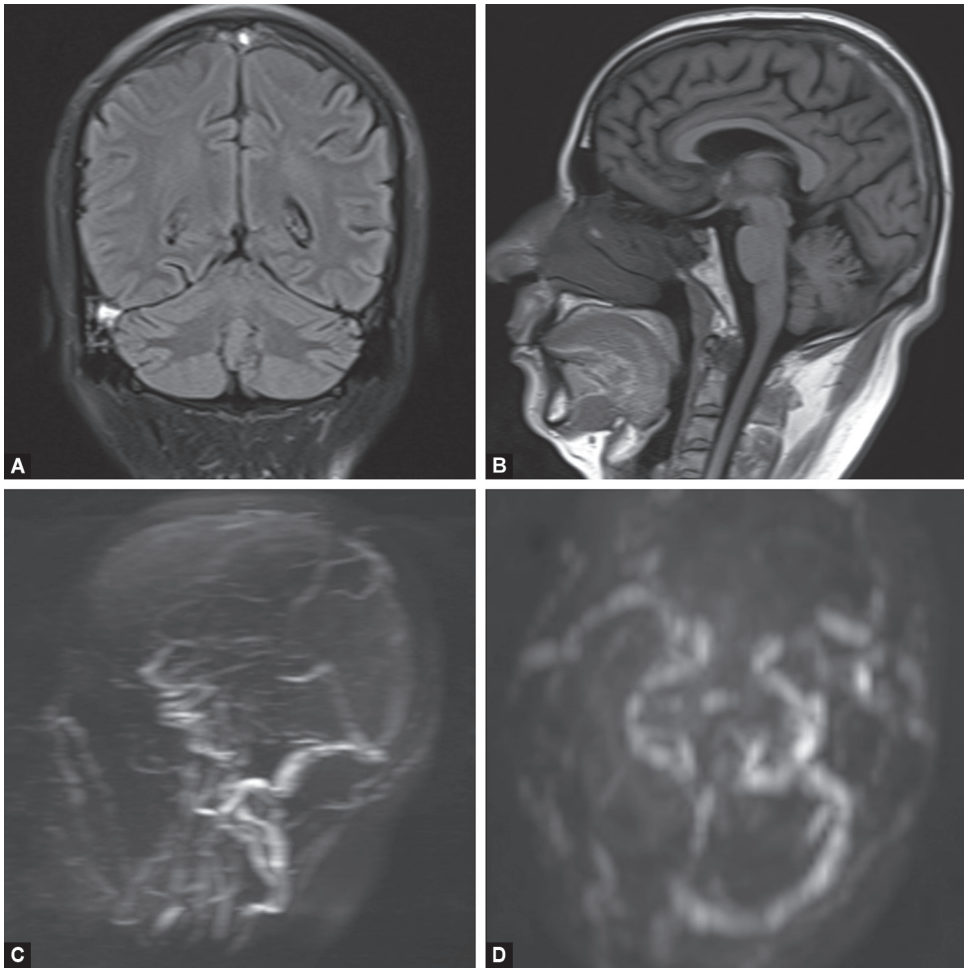
patients/year. It is the thrombosis of the superficial or deep venous system of the brain. The common risk factors that are encountered in the diagnosis of CVT are infections, congenital heart disease, pregnancy, puerperium, anemia, and polycythemia. Our patient had risk factors of chronic alcoholism and dehydration. Extensive workup towards the prothrombotic state was all normal.

Increased intracranial pressure leading to cerebral damage is usually the cause of the clinical symptoms and signs seen in CVT. Depending on the disease progression, the patient may present with an acute or subacute clinical picture. Headache, seizures, dizziness, altered level of consciousness, focal weakness, and papilledema are the usual symptoms and signs. Cranial nerve involvement is very rare.[1] Accounted for only about 10–12% of patients. The cranial nerves affected in CVT are the optic nerve, abducens, and less commonly the trochlear nerve. Very few cases have been reported showing an association between CVT and peripheral facial nerve palsy.[2] Possible mechanisms include elevated intracranial pressure, extension of thrombosis to venous channels, or pressure of the clot directly on the nerve.

In our patient, there were features of raised ICP and unilateral (right-sided) lower motor neuron type facial palsy and its localization was made in the supra geniculate portion of the nerve (taste and tearing abnormalities). The temporal profile and the localization of LMN facial palsy without any associated features to indicate the cause of an alternative etiology suggested that the facial palsy was probably secondary to cerebral venous sinus thrombosis and raised ICP.

In one study done by Bousser and Barnet, the clinical features of CVT were classified under five categories. They include isolated intracranial hypertension, focal deficits, cavernous sinus thrombosis, encephalopathy, and rare unusual presentations like cranial nerve palsies highlighting that cranial nerve involvement apart from the ocular motor nerves is very rare in presentation. In the report given by Straub et al., they concluded that transient neuropraxia in the intracranial segment was the cause of facial palsy which was demonstrated by transcranial magnetic stimulation. In our patient, trans magnetic stimulation studies could not be done.

A literature review on patients with CVT having facial nerve palsy had been described in seven such case reports. Kuehnen et al. reported two such patients, (1) A 44-years-old male right transverse sinus thrombosis showed recovery of facial nerve palsy in 1 week and (2) A 65-year-old female with left transverse sinus thrombosis showed recovery in less than 6 weeks. Straub et al. described a 17-year-old female patient who had a risk factor of familial protein S deficiency and oral contraceptive pills (OCP) intake presenting with straight, superior, longitudinal, left transverse, sigmoid sinus thrombosis and facial nerve palsy who showed recovery in 17 days. Kulkami et al. reported a 30-year-old female



Figs 1A to D: (A) MRI Brain T2 FLAIR coronal image showing thrombus in the right transverse sinus and superior sagittal sinus; (B) T1 Sagittal image showing thrombus in the superior sagittal sinus; (C and D) MRV Image showing absent flow in the superior sagittal sinus, straight sinus and right transverse sinus

with the risk factor of anemia with superior sagittal and right lateral sinus thrombosis with recovery 1 month.[3–5] Kartal et al. reported a 17-year-old female with OCP intake presenting with straight, superior, longitudinal, bilateral transverse, and right sigmoid sinus thrombosis with recovery in 2 weeks. Kowsalya et al. reported a 39-year-old female with anemia and superior, inferior, and straight sinus, right transverse, and sigmoid sinus and frontoparietal cortical vein thrombosis who had recovery in 6 (1–7) months. A recent case report by Shah et al. demonstrated the occurrence of bilateral LMN facial nerve palsy in unilateral transverse sinus thrombosis in a 16-year-old female patient.[6]

Contrary to some of the prior case reports our patient had additional involvement of the straight sinus, superior sagittal sinus, right sigmoid sinus, and right IJV besides the transverse sinus which is postulated to lead to more pronounced ICP emphasizing the theory that raised ICP causing facial nerve palsy.[7] There are also cases reported with bilateral facial nerve palsy in patients with idiopathic intracranial pressure (IIH) and improvement following management of elevated pressure proving that increased pressure causes facial nerve palsy despite the nerve's short intracranial course and relative protection from pressure (Fig. 1).

Conclusion

This case highlights the rare atypical occurrence of LMN facial palsy in CVT which can be a false localizing sign in cases of raised intracranial pressure. Hence, CVT should be considered as one of the differential diagnoses of facial palsy in the presence of appropriate clinical presentations.

Clinical Significance

Cranial nerve involvement other than 6th and 4th CN in cerebral venous thrombosis is very rare. Facial nerve involvement in CVT is due to the transient neuropraxia that occurs in the course of the facial nerve caused by raised intracranial pressure. Resolution occurs on treatment of the underlying cause with anticoagulants.

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