

**Case report**

# Multiple cranial neuropathies in cerebral venous sinus thrombosis

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Received 27 February 2014; revised 13 March 2014; accepted 18 March 2014

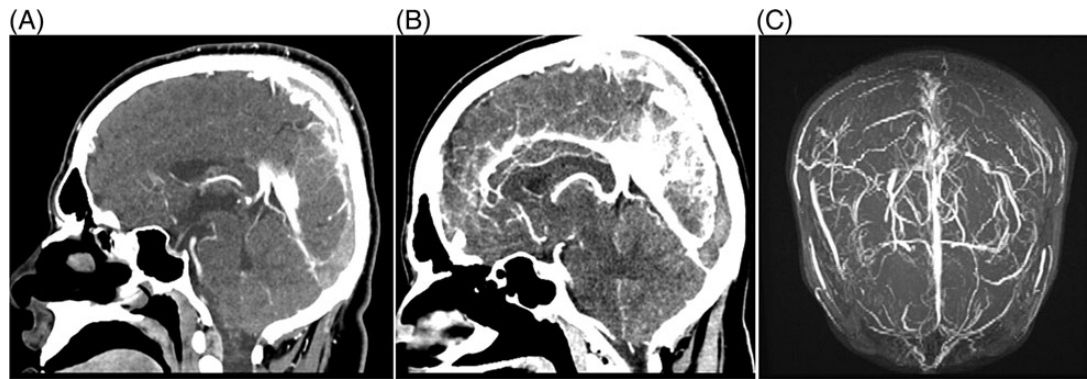
Clinical presentation of cerebral venous sinus thrombosis (CVST) is varied and often mimics many neurological disorders, making it a diagnostic challenge, and cranial nerve palsy in CVST is rare and its pathophysiology remains unclear. We report a case of a 19-year-old male with a history of whiplash injury, admitted with extensive CVST, developed right facial nerve palsy with extension of thrombus into the ipsilateral transverse sinus, sigmoid sinus and internal jugular vein. Later, he developed left facial nerve palsy with partial left oculomotor weakness. We suggest that either reversible compromised oxygen or glucose consumption within the intrinsic vascular system of the nerve, resulting in cranial nerve abnormalities. CVST should be considered in cases of trivial trauma, even in the absence of hyper-coagulable states, and it can have atypical presentation like multiple cranial neuropathies.

## INTRODUCTION

Cerebral venous thrombosis (CVT) is a relatively uncommon condition afflicting mostly young adults [1]. The diagnosis may subsequently be very difficult, especially in the very infrequent cases following closed head injury. Cerebral venous sinus thrombosis (CVST) treatment may be conservative (anti-coagulation and anti-epileptic drugs) or requires aggressive steps such as endovascular thrombolysis, putting ventriculoperitoneal shunts and decompressive craniectomy. The estimated annual incidence is 1.5–3% cases per million in adults and 6.7% per million in children [2]. Dural sinus thrombosis usually involves the sagittal sinus (70–80%), transverse and sigmoid sinuses (70%), and may extend to the cerebral veins [2]. CVST is often multifactorial, meaning that the identification of a risk factor or even of a cause should not deter a search for other causes [3]. It has been reported that prothrombotic risk factors or other direct causes, such as pregnancy, head trauma, infection and deep venous thrombosis, were identified in ~85% of the patients [2]. Cranial nerve palsy in CVT is rare and its pathophysiology remains unclear. We describe a case of CVST secondary to trivial head injury with multiple cranial nerve palsies, which was aggressively treated with ventriculoperitoneal shunt placement.

## CASE REPORT

We describe a 19-year-old male, student, right handed, who had whiplash injury while driving a car, 1 week prior to his presentation. According to him, his car was rear ended by another vehicle from behind. It was not a very hard impact and there was a little damage to both vehicles. He remained well for 4 days then his symptoms started with acute, severe headache, associated blurred vision and neck pain. His examination showed neck stiffness and bilateral severe papilledema with decreased visual acuity in the left eye to finger counting. He had extensive CVT involving the left transverse sinus (TS) and superior sagittal sinus (SSS) (Fig. 1A). Lumbar puncture showed an opening pressure of 63 cm. He was started on i.v. heparin. After transient improvement, headache and vomiting worsened. Three days later (9 days after injury), he developed right facial lower motor neuron (LMN) nerve palsy. Repeat MRI showed extension of thrombus in the straight sinus and left TS (Fig. 1B) Repeat lumbar puncture was done, with an opening pressure of 55 cm of CSF. Two days later (11 days after injury), he developed left facial nerve LMN type weakness as well as partial left oculomotor palsy. Repeat venogram showed thrombus extension into the right internal jugular vein (Fig. 1C). The patient was offered lumboperitoneal shunt after that he improved gradually. Thrombophilia



**Figure 1:** (A) the posterior half of the sagittal sinus is found to be thrombosed with the thrombus extending into the TS reaching the sigmoid sinus on the right side. (B) progression of the intraluminal hyperdensity previously to involve currently the straight sinus and possibly the left TS with mild effacement of cortical sulci. (C) sagittal sinus, right TS, right sigmoid sinus and right internal jugular vein with minimal partial recanalization.

work-up all came out to be negative. The patient was discharged on oral anti-coagulation, and had complete recovery from bilateral facial weakness and oculomotor weakness.

## DISCUSSION

Intracranial dural venous sinus thrombosis secondary to mild closed head injury without cranial vault fractures or intracranial hematomas is an increasingly recognized entity [4]. Bousser and Barnett [3] suggested separating CVST into the following four groups: those with isolated intracranial hypertension, with focal cerebral signs, with cavernous sinus syndrome and those with unusual presentations. The cranial nerve syndrome may be allocated to the latter group. Involvement of isolated cranial nerves without focal neurological deficit is very rare [5]. Cranial nerve palsy as an isolated manifestation of CVST has been attributed to the elevated intracranial pressure, extension of thrombosis to venous channels or direct pressure from the clot itself [6]. Kuehnen *et al.* [5] have reported five patients who were initially evaluated for etiologies of single/multiple cranial nerve palsies finally turning out to be the cases of the ipsilateral transverse and sigmoid sinuses thrombosis, on evaluation. Lateral sinus (transverse and sigmoid portion) drains blood from the cerebellum, brainstem and posterior portions of cerebral hemispheres, veins from cranial nerves in the posterior fossa, the middle ear and diploic veins. According to Kuehnen *et al.* [5] thrombosis of the lateral sinus can produce venous congestion and dilatation of the cranial nerve veins, this causes reversible compromised oxygen or glucose consumption within the cranial nerve tissue due to edema and backpressure, and due to this cranial nerve palsies will develop. Straub *et al.* [7] have described a 17-year-old lady with LMN facial palsy with ipsilateral TS thrombosis; they evaluated the patient with transcranial magnetic stimulation and concluded that the facial palsy was due to the transient neuropraxia in the intracranial segment of the nerve. They explained neuropraxia due to the leakage of the fluids and ions into the endoneural space of the nerve due to

elevated venous transmural pressure in the nerve's satellite vein, which ultimately drains to ipsilateral TS [7]. This causes impairment of the saltatory current flow, with reversible slowing of the conduction or even conduction block. Among a series of 38 with CVST, Bousser *et al.* reported 3 cases with affected cranial nerves: a patient with a left III cranial nerve palsy, another one with multiple cranial nerve palsies (V–X) and the third with a right VI nerve palsy. However similar to the first descriptions, all patients presented with further symptoms of CVST: hemiplegia, deep coma, cerebellar incoordination, headache and papilledema, and angiograms showed TS thrombosis in combination with SSS thrombosis in all cases [5]. In the case series of five patients by Kuehnen *et al.*, Case 2 had ipsilateral partial third and seventh nerve involvement, with no involvement of cavernous sinus. Our patient presented with thrombosis of SSS and left TS, later on he had clinical worsening and extension of thrombus to straight sinus, contralateral TS and internal jugular vein as well, with no parenchymal lesion and development of progressive bilateral facial LMN type, and oculomotor nerve weakness.

We assumed that either reversible compromised oxygen or glucose consumption within the cranial nerve tissue due to edema and back pressure and dysfunction in the intrinsic vascular system of the nerve, resulting in cranial nerve abnormalities, or elevated venous transmural pressure in the nerve's satellite vein, which belongs to the affected drainage territory of the TS secondary to raised intracranial pressure, might have caused disruption of venous blood–brain barrier [2]. Our case presents many different findings: trivial head injury led to extensive CVST, with progressive cranial neuropathies and required aggressive management. CVST should be considered in cases of trivial trauma, even in the absence of hypercoagulable states, and it can have atypical presentation like multiple cranial neuropathies.

## FUNDING

None.

**CONFLICT OF INTEREST STATEMENT**

None declared.

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