

Closed traumatic head injury: dural sinus and internal jugular vein thrombosis

J M Caplan,¹ Z Khalpey,¹ J Gates²

Dural sinus thrombosis (DST) has an annual incidence of 3–4 per million and can result from many aetiologies. Presentation of the disease can vary considerably, as can the aetiology and delay of symptoms to clinical detection. Symptoms on presentation include headache, seizures, focal neural deficits and altered mental status. There are many aetiological risk factors associated with DST, which include hypercoagulable states, oral contraceptive use, infection and mechanical causes such as cranial trauma. DST as a result of trauma is rare and aetiologies range from mechanical falls with or without skull fracture, firework explosions, gunshots to the head, blunt trauma to the head and closed head injury. Internal jugular vein thrombosis is also a rare disease and as with DST, traumatic aetiologies are uncommon. More common aetiologies include iatrogenic causes related to catheterisation as well as infectious causes (eg, Lemierre's syndrome). A case of thrombosis of the transverse sinus, sigmoid sinus and internal jugular vein associated with a closed head injury as the result of a motorcycle accident is presented.

A 27-year-old-man was involved in a high speed (55 mph) motorcycle collision with a tree. He was wearing a helmet. At an outside hospital a maxillofacial and cervical spine computed tomography (CT) scan did not indicate evidence of injury. Given persistent headaches and upper extremity paraesthesia, he was transferred to our institution for evaluation on the day of the accident.

On arrival he complained of bilateral upper extremity paraesthesia. Neurological examination was grossly intact and unremarkable, with the exception of a hoarse voice. Head and neck CT revealed a left side basilar skull fracture lateral to the occipital condyles with fracture lines extending to the hypoglossal canal and jugular foramen (fig 1). A cervical spine magnetic resonance imaging scan and plain cervical spine films revealed no evidence of acute injury. A head and neck magnetic resonance angiography revealed a haemorrhagic contusion in the medial left cerebellar hemisphere and thrombosis of the left sigmoid sinus and left jugular vein (fig 2). Neurologists noted a partial VI nerve palsy with left beating nystagmus and a hoarse voice. Sensation was intact bilaterally.

The patient was admitted and started on subcutaneous heparin. The ENT service noted the left vocal cord was paretic in a paramedian position, with a mild posterior glottis gap; the right vocal cord was normal. Supraglottic sensation was decreased with no cough elicited, consistent with a vagal nerve paralysis, probably secondary to oedema at the site of the skull fracture. Fluoroscopic speech and swallow evaluation showed significant residue with puree and soft solids, but

without evidence of penetration or aspiration. A thin liquid diet was recommended.

On hospital day 3, repeat magnetic resonance venography revealed no interval change. In the interim, the patient was changed to a therapeutic dose of intravenous heparin (without a bolus). An interval head CT on hospital day 5 did not reveal any new haemorrhage; however, there was an area of increased attenuation in the distribution of the transverse sinus, consistent with an extension of the thrombosis. While on therapeutic heparin, he was started on warfarin. Repeat video swallow showed residue within the piriform sinuses and silent aspiration. A diet of thin liquids and thin puree followed by sips was recommended. The patient's warfarin dosing was changed to crushed pills thereafter.

At the time of discharge, the patient's headaches and upper extremity paraesthesia had resolved, an interval head CT showed no significant change and his international normalised ratio was therapeutic (goal 2–3). He was discharged home with planned follow-up in trauma clinic, neurology clinic (including laryngeal electromyography) and ENT clinic.

Three weeks after discharge, he had lost 20 pounds since the accident and had difficulty swallowing liquids and solids. His voice remained soft. The patient was subsequently lost to follow-up. A phone call for the purposes of this case report revealed that the patient stopped taking warfarin at 5.5 months; 2 weeks short of his 6-month course. He reports that his voice has not returned to normal, although it is improved, he is continuing to have difficulty with hoarseness and controlling tone. Furthermore, he continues to have difficulty swallowing, although this too is improved, requiring liquid boluses to help swallow at times.

DISCUSSION

The incidence of dural sinus thrombosis (DST) is three to four cases per million people per year.¹ Risk factors for DST formation include prothrombotic conditions, infections, inflammatory diseases, haematological conditions, drugs (eg, oral contraceptives), mechanical causes, tumours and iatrogenic causes such as lumbar punctures.¹ Among the mechanical causes are traumatic head injury, as occurred in our patient.

In a prospective study of 624 patients with cerebral vein thrombosis and DST, the superior sagittal sinus was most often

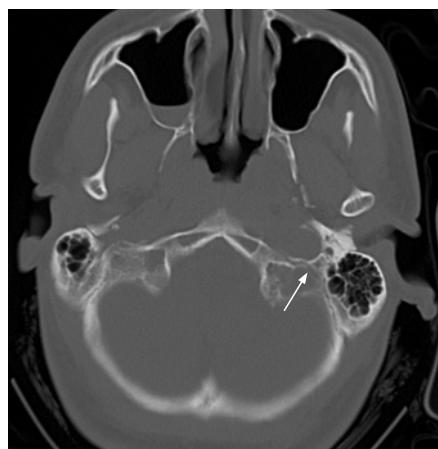


Figure 1 Axial computed tomography section showing left-sided skull base fracture (arrow) with fracture lines extending to the jugular foramen.

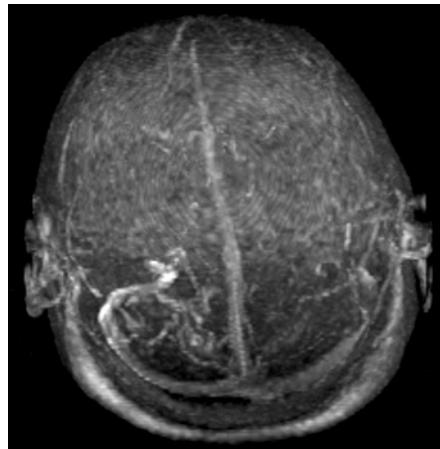


Figure 2 Three-dimensional magnetic resonance venography projection showing decreased flow within the sigmoid sinus and internal jugular vein.

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involved (62%), followed by the lateral sinuses (44.7% and 41.2% left and right, respectively).² Jugular veins were involved in 11.9% of cases. Thrombophilia, haematological conditions and infection were among the most common risk factors for all study participants. Pregnancy, puerperium and oral contraceptive use were among the most common risk factors in women. Mechanical precipitants were identified in only 28 patients (4.5%), with cranial trauma identified in only seven (1.1%).

DST can present with headaches most frequently.¹ Other manifestations include focal neurological signs, hemispheric symptoms secondary to cortical lesions, seizures, behavioural symptoms, coma and even death.¹

Treatment is aimed at preventing cerebral herniation and the prevention of extension or new formation of thrombus. The risks of haemorrhage with anticoagulation should be balanced against the possible complications of the thrombosis, including pulmonary embolism and the development of new venous infarcts.^{1,3} A Cochrane literature review revealed that anticoagulation for patients with DST appeared safe and was associated with a non-statistically significant

risk reduction of death and dependency.³ The role of thrombolysis (either systemic or local) in the management of DST has not yet been fully elucidated.

Internal jugular vein thrombosis (IJVT) is also rare, often associated with infectious aetiologies (eg, Lemierre's syndrome), or vascular trauma associated with catheterisation. IJVT secondary to trauma is uncommon.⁴ In one case the authors report traumatic bilateral IJVT associated with increased intracranial pressure, treated endovascularly with urokinase, followed by stenting and anticoagulation.⁴ Other traumatic causes reported include neck injuries while exercising and penetrating trauma to the palate.

In conclusion, we present a traumatic head injury with a skull base fracture resulting in a vagal nerve palsy with an associated DST and IJVT. The patient was bridged (via heparin) to warfarin for 5.5 months, with a gradual but progressive improvement of focal neurological symptoms. The merits of and duration of anticoagulation should be further evaluated; one could argue for a longer duration of anticoagulation; however, this would depend on the extent of the functional deficit and evidence from further large-scale

studies. This case study raises awareness of the significance of common closed head and neck injuries associated with the possible neurological sequelae of dural sinus and IJVT.

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