

# Cerebral venous infarction during a high altitude expedition

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## ABSTRACT

**Bilateral venous infarction of the brain due to thrombosis of the deep cerebral venous system is relatively rare, accounting for approximately 3–8 percent of all cases of cerebral venous thrombosis (CVT). Known risk factors include the use of oral contraceptives, pregnancy, puerperium, malignancy and thrombophilic states. CVT, in the setting of acute mountain sickness (AMS), has rarely been reported. We present an unusual occurrence of bilateral deep subcortical venous infarction in a previously-well, 39-year-old woman, who developed AMS during a high altitude expedition in Nepal. The possible mechanisms responsible for this unfortunate event include dehydration with resultant relative polycythaemia and raised intracranial pressure at high altitudes. CVT should be considered in mountain climbers presenting with progressive neurological deterioration that is not solely attributable to AMS.**

**Keywords:** acute mountain sickness, cerebral venous thrombosis, high altitude

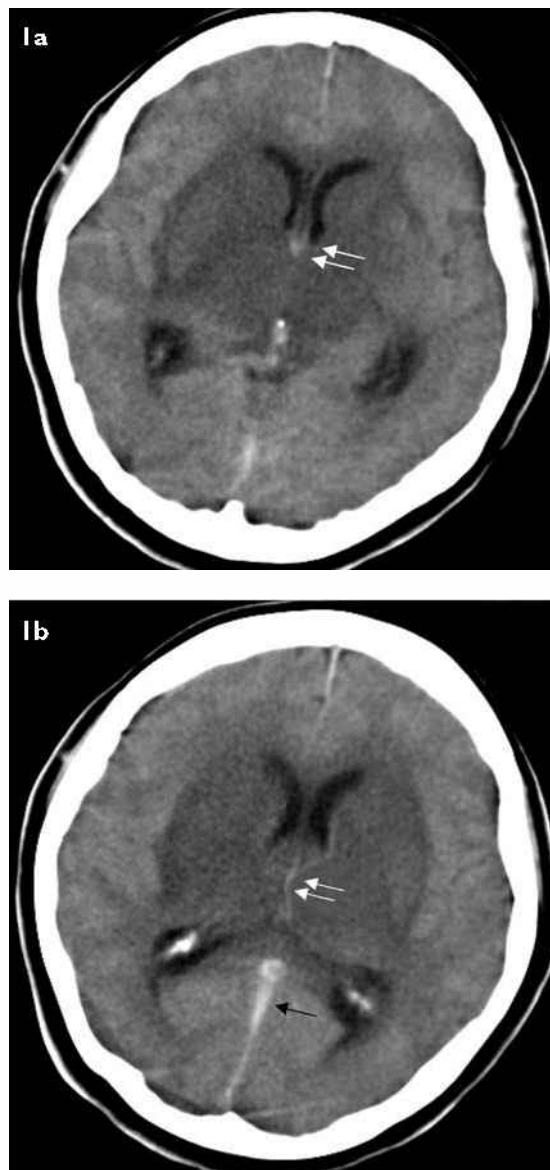
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## INTRODUCTION

Early diagnosis of cerebral venous thrombosis (CVT) of the deep venous system can be elusive and challenging as most patients initially present with non-specific symptoms of headache. We describe an unusual case of bilateral deep subcortical infarctions due to CVT in the setting of a high altitude climb.

## CASE REPORT

A 39-year-old right-handed Chinese woman, without a past medical history, was in Nepal for a 14-day mountain climbing expedition. On the fourth day of the climb, at approximately 4,000 m above sea level, she developed dizziness, headache and vomiting; symptoms thought to be due to acute mountain sickness (AMS). Although she descended to 2,000 m, her symptoms got progressively more severe, and she became increasingly drowsy and confused. She had to be carried down and



**Fig. 1** (a, b) Unenhanced axial CT images of the brain at the time of presentation show symmetrical hypo-attenuation of the deep nuclei bilaterally, indicating oedema/ischaemia, with a hyperdense appearance of the internal cerebral veins (double white arrows) and straight sinus (arrow), suggestive of thrombosis of the deep venous system.

was sent to a local hospital.

At the local hospital, she was noted to be obtunded, with a Glasgow Coma Scale score of 8. Computed tomography (CT) of the brain (Figs. 1a & b) showed symmetrical hypodensities involving the deep grey

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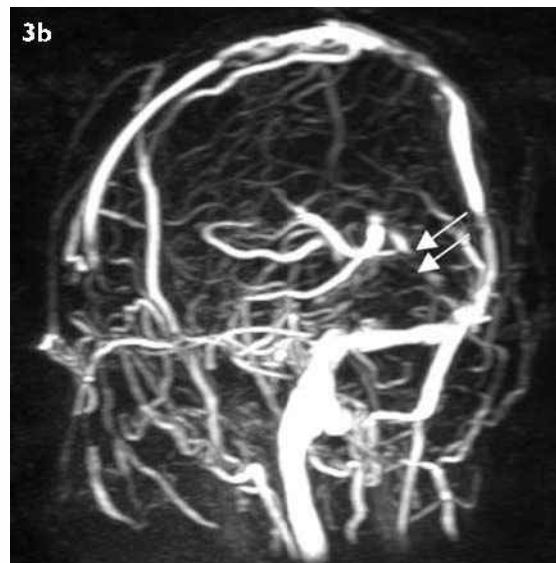
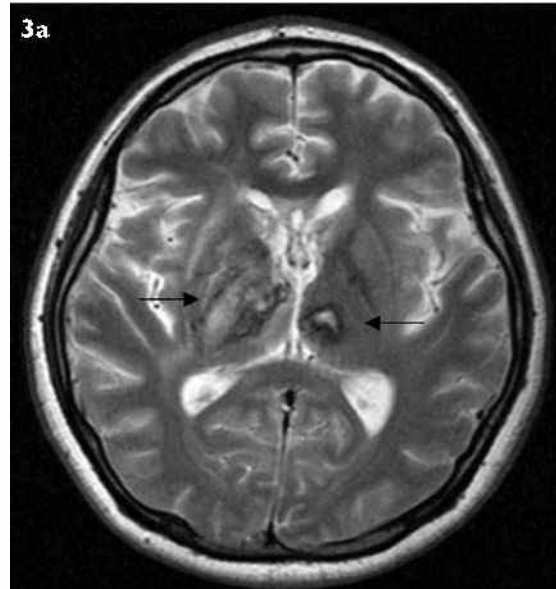
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**Fig. 2** Digital subtraction angiogram of the internal carotid artery in the venous phase shows the absence of opacification of the great cerebral vein of Galen and the straight sinus (arrows), which confirm thrombosis of these deep veins. The superior sagittal sinus and lateral sinus are patent (arrowheads).

nuclei (caudate, lentiform nuclei and thalami) bilaterally, with relative hyperdensity of the internal cerebral veins and straight sinus, suggestive of thrombosis of the deep venous system with venous infarction. In addition, there were cerebral oedema and developing hydrocephalus. Digital subtraction angiography of the cranial vessels (viewed in retrospect) showed the absence of opacification of the great vein of Galen and straight sinus (Fig. 2). Cerebrospinal fluid analysis, done at the local hospital, was unremarkable. The working diagnosis made at that time was central nervous system infection with cerebral oedema. Hence, she was treated with intravenous mannitol, dexamethasone and empirical antibiotics, and had an external ventricular drain inserted to treat the hydrocephalus.

Her neurological status improved slightly, and she was transferred to our institution in Singapore for further management, approximately one month after symptom onset. On examination, she was conscious but not responding to commands and had a right gaze preference. She was tetraparetic with increased tone in all four limbs and bilateral extensor plantar responses. Magnetic resonance (MR) imaging of the brain (Fig. 3a) showed multiple foci of T2 prolongation in the basal ganglia and thalami (right > left). Associated gradient-echo susceptibility artefacts were seen in the deep nuclei, compatible with haemosiderin deposits from prior haemorrhage. The MR venography (Fig. 3b) showed some signal attenuation in the straight sinus and at the junction of the straight sinus with the dominant right transverse sinus. This suggested that there was



**Fig. 3** MR imaging of the patient performed one month later. (a) Axial T2-W image revealed established foci of gliosis with signal susceptibilities, indicating prior haemorrhage in the bilateral deep nuclei (arrows), consistent with previous haemorrhagic venous infarcts in the deep venous territory, even though (b) MR venogram shows partial recanalisation of the straight sinus (double arrows).

recanalisation at the site of the previous thrombosis.

Further diagnostic work-up for stroke in the young, including known hypercoagulable tests such as lupus anticoagulant, anticardiolipin antibodies, protein C and S, anti-thrombin 3, factor V Leiden, prothrombin gene G20210A mutation and fasting homocysteine, were unremarkable. The management was supportive. The managing team decided not to anticoagulate the patient as the MR venography had shown recanalisation of the venous thrombosis. Her neurological status, unfortunately, remained poor, and she was transferred to a rehabilitation facility for further management.

## DISCUSSION

This case has several important and unique features. First, deep CVT with bilateral venous infarctions is relatively uncommon and has rarely been reported in the setting of AMS. A high index of suspicion has to be maintained to avoid a delay in diagnosis, as in our case. Second, it offers insights into the mechanism of CVT, the importance of early diagnosis and treatment to reduce the risk of an adverse neurological outcome. Third, it highlights the potentially serious neurological complications of AMS. The classical features of thrombosis of the deep cerebral venous system are severe dysfunction of the diencephalon, reflected by coma, disturbance of eye movements and pupillary reflexes. It is often associated with a poor neurological outcome, if left untreated. The intensity of venous congestion causes haemorrhagic infarction to develop.<sup>(1)</sup> In our patient, the poor neurological status correlated with bilateral infarction involving the deep grey nuclei seen as hypodensities on CT, and foci of T2 prolongation on MR imagings (Figs. 1 & 3).

The known risk factors for CVT were ruled out in our patient – she was not on hormonal contraceptives, nor was she pregnant or in her puerperium, and the hypercoagulable work-up was unremarkable. The presumed mechanism of CVT in our patient was probably a combination of dehydration from vomiting (due to AMS), polycythaemia with relative hyperviscosity and an increase in cerebral blood flow, and hence raised intracranial pressure, compromising the low pressure cerebral venous circulation. Unfortunately, haematocrit and electrolyte levels at the time of presentation in Nepal are unavailable to verify this. The initial rise in haemoglobin concentration seen at high altitude is due to a reduction in plasma volume secondary to altitude diuresis and fluid shifts.<sup>(2)</sup> Fluid intake is restricted in most mountain climbers because of the difficulty in obtaining water. Moreover, vomiting induced by AMS, as in our patient, compounds the problem. These conditions induce relative hyperviscosity in the circulating blood which, combined with a hypobaric environment, were shown to result in increased coagulability in a rhesus monkey model.<sup>(2)</sup> Cerebral blood flow increases immediately on ascent to high altitude via vasodilatation, probably in response to hypoxia aggravated by hypoventilation, periodic breathing and exercise.<sup>(3)</sup> MR imaging studies have shown that the brain increases in volume on ascent, presumably from a combination of vasodilatation, sodium retention and increased capillary permeability.<sup>(3,4)</sup> This increase in volume elevates intracranial pressure, leading to cerebral oedema and potentially compromising the low pressure

cerebral venous circulation. Data from the International Study on Cerebral Vein and Dural Sinus Thrombosis<sup>(5)</sup> showed that coma and intracerebral haemorrhage in CVT (as seen in our patient), are independent predictors of poor outcome; and the involvement of the deep cerebral venous system is an additional unfavourable prognostic factor.<sup>(5,6)</sup> A review of the literature reveals several case reports describing CVT occurring during high altitude climbing.<sup>(7-9)</sup> Unlike our case, these cases of CVT either occurred at a much higher altitude (more than 8,000 m) or were associated with an underlying hypercoagulable state.<sup>(7-9)</sup>

The diagnosis of deep CVT can be difficult and requires a high degree of clinical suspicion, because neuroimaging studies may be normal in the early stages or may mimic other neurological disorders.<sup>(10)</sup> It is important to consider other neurological diagnoses, as the treatment for these conditions is vastly different from that of AMS.<sup>(11)</sup> In our case, the final diagnosis was made only after reviewing the history and images when the patient was transferred to our institution in Singapore. The treatment of CVT is anticoagulation, the duration of which is dependent on the presence of an underlying hypercoagulable state.

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