

Acute ischaemic stroke during short-term travel to high altitude

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This is a case report of a young healthy adult who had acute cerebral infarcts after a short-term visit to high-altitude area. He developed acute onset of right-sided limb weakness and right hemianopia a few hours after arrival at an altitude of 3600 m by train. He was initially treated for high-altitude cerebral oedema but later computed tomography and magnetic resonance imaging confirmed ischaemic infarcts in the medial left occipital lobe and left thalamus. Subsequent investigations, including laboratory tests and imaging including an echocardiogram, revealed no culpable predisposing factors.

Case report

A 19-year-old previously healthy college student suffered an acute ischaemic stroke during travel to high-altitude area in December 2009. A few hours after arrival by train at Lhasa in Tibet, China, at an altitude of 3600 metres, he experienced sudden onset of severe headache and fatigue, with right-sided weakness, limited vision in the right, and some unsteadiness in walking. He had no nausea or vomiting, nor was he confused or having difficulty in breathing at any time. Moreover, he had no fever, chest pain, cough, or sputum production. He did not take any alcohol or drugs before and during his travel. He was brought to the local hospital, and was treated for cerebral oedema with hyperbaric oxygen. His weakness slightly improved after 3 days of hospitalisation.

He was transferred back to Hong Kong on the fourth day. Physical examination revealed right homonymous hemianopia, and reduced power (grade 4/5) in his right limbs. The cranial nerves were intact. Mild truncal ataxia was noted, but other cerebellar signs were not present. Fundoscopy examination was unremarkable, there being no papilloedema or retinal haemorrhage. Visual field tests showed macular sparing, characteristic of a posterior circulation infarct sparing the occipital pole.

Non-contrast brain computed tomography (CT) showed a hypodense lesion in medial left occipital lobe and another small hypodense focus in the left thalamus, compatible with cerebral infarcts (Fig 1a). Follow-up magnetic resonance imaging (MRI) after 2 weeks showed heterogeneously altered signals in medial left occipital lobe and left thalamus with no restricted diffusion and some blooming artefacts, indicating subacute infarcts with mild haemorrhagic transformation (Fig 1b-f). Venous sinuses were patent and the magnetic resonance angiography was normal.

Laboratory tests revealed normal complete blood counts, fasting lipids, and fasting glucose levels. Tests for immune markers and anti-cardiolipin antibody were negative. Echocardiography did not reveal any structural abnormality.

The patient's weakness gradually improved on conservative management. The patient remained stable with no residual motor deficit on subsequent follow-up. His homonymous hemianopia remained unchanged. A follow-up MRI after 7 months confirmed the presence of a chronic infarct with reduction in size of lesion in occipital lobe, and near disappearance of the small lesion in thalamus.

Discussion

Acute mountain sickness (AMS) is an illness that usually affects travellers who rapidly elevate to altitudes above 2500 m. The common symptoms include headache, fatigue, dizziness, anorexia, nausea, vomiting, and insomnia. High-altitude cerebral oedema (HACE) is regarded as the end stage of AMS, in which the cardinal features of change in consciousness, ataxia with progression to obtundation and coma ensue.^{1,2} While mild AMS is usually self-limiting, HACE is potentially fatal as a result of brain herniation. Data from neuroimaging in AMS and HACE sufferers reveal reversible vasogenic cerebral oedema of the deep white matter. Haemodynamic factors such as impaired cerebral autoregulation,

Key words

Altitude sickness; Brain edema; Cerebral infarction; Magnetic resonance imaging; Tomography, X-ray computed

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因往高地短期旅遊後出現急性缺血性中風

本文報告一名一向健康良好的年青男性往高地短期旅遊後出現急性缺血性中風的病例。病人乘火車至海拔3600米到達目的地數小時後，出現右邊肢體無力及右側偏盲。起初為病人作高海拔腦水腫治理，但其後的電腦斷層及磁共振影像確定病人內側左枕葉及左丘腦處有缺血性梗塞。病人後來進行實驗室測試及包括超聲心動圖的放射影像顯示並無發病誘因。

sustained vaso-dilatation, and elevated cerebral capillary pressure are thought to be contributory factors.³ This is distinct from the cerebral infarction, in which non-reversible cytotoxic oedema involving both gray and white matter is encountered.

Acute stroke is rarely reported during short-term visits to high-altitude regions. Both clinical and imaging findings of our patient were typical of acute cerebral infarction. Unlike the current case, HACE tends not to produce focal neurological deficits.

Moreover, it produces diffuse changes similar to any other kind of cerebral oedema on CT. On the other hand, certain characteristic MRI findings of HACE have been reported, namely hyperintensities around the splenium, and callosal microhaemorrhages,^{4,5} as illustrated in another case (Fig 2).

Cerebral venous thrombosis is one of the differential diagnoses of altitude-related disorders. The symptoms of headache and haemorrhagic transformation are commoner in venous infarcts. Venous flow can be jeopardised during dehydration and there have been a few reports of cerebral venous infarction developing in high-altitude region^{6,7} and during long-distance air travel.⁸ In our patient, the medial cortical infarct could be attributed to blocked superficial cortical vein(s). Isolated cortical vein thrombus can appear as cord-like lesions that show high signals on T1-weighted images, low signals on T2*-weighted images or high signals on diffusion-weighted images.⁹ However, this condition is difficult to exclude without an MR or digital subtraction

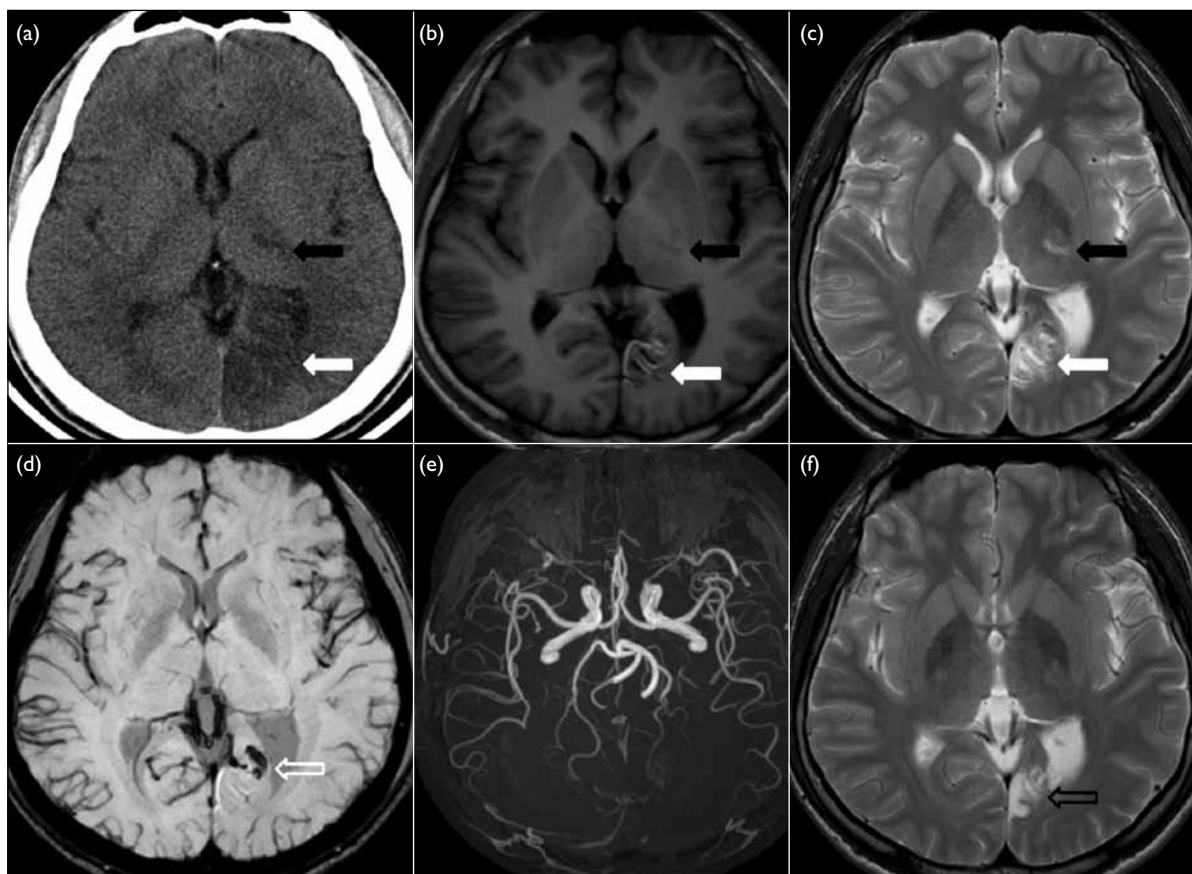


FIG 1. Urgent (a) computed tomographic brain at 4 days and (b-d) magnetic resonance imaging (MRI) at 2 weeks show cerebral infarcts involving the left medial occipital lobe (white arrows) and left thalamus (black arrows). There were also T1 hyperintensities along part of the occipital lobe cortex, and blooming on susceptibility weighted images (d, white open arrow), suggesting haemorrhagic transformation or cortical laminar necrosis. A diffusion-weighted image (not shown) revealed no restricted diffusion that was consistent with subacute infarcts. Contrast-enhanced MRI demonstrated luxury perfusion in the occipital lobe lesion (not shown). (e) The magnetic resonance angiography shows neither steno-occlusive disease nor other vascular anomalies. (f) Follow-up MRI after 7 months shows shrinkage of the occipital lobe lesion associated with negative mass effect and disappearance of the left thalamic lesion (black open arrow)

venogram. Anyhow, venous infarct alone does not explain the small infarct in left thalamus. In this patient, the concomitant involvement of two sites raises the suspicion of embolic events, whilst vascular spasm involving the posterior circulation seems less likely.

Significantly increased risk of strokes has been reported in soldiers stationed at high altitude for months, which has been associated with secondary polycythaemia and much less commonly, protein C and S deficiency.¹⁰ However, acclimatisation changes appeared unlikely in our patient, owing to his visit being short term. Although pre-existing neurovascular or haematological diseases that increased the risk of acute cerebral infarcts under hypoxic condition is a possibility, there was no culpable structural or biochemical abnormality in our patient.

The management of AMS and HACE is completely different from that of cerebral infarction or cerebral venous thrombosis. For the former, descent, supplemental oxygen, hyperbaric therapy, and dexamethasone are effective options, whilst exertion, alcohol, and sedatives should be avoided. Acetazolamide may also be useful if descent is delayed.

In our patient, although the exact mechanism of stroke remains speculative, the case illustrates that acute cerebral infarct can develop during short-term visit to a high-altitude area, even in an otherwise healthy person. Heightened awareness of this entity is required for successful management.

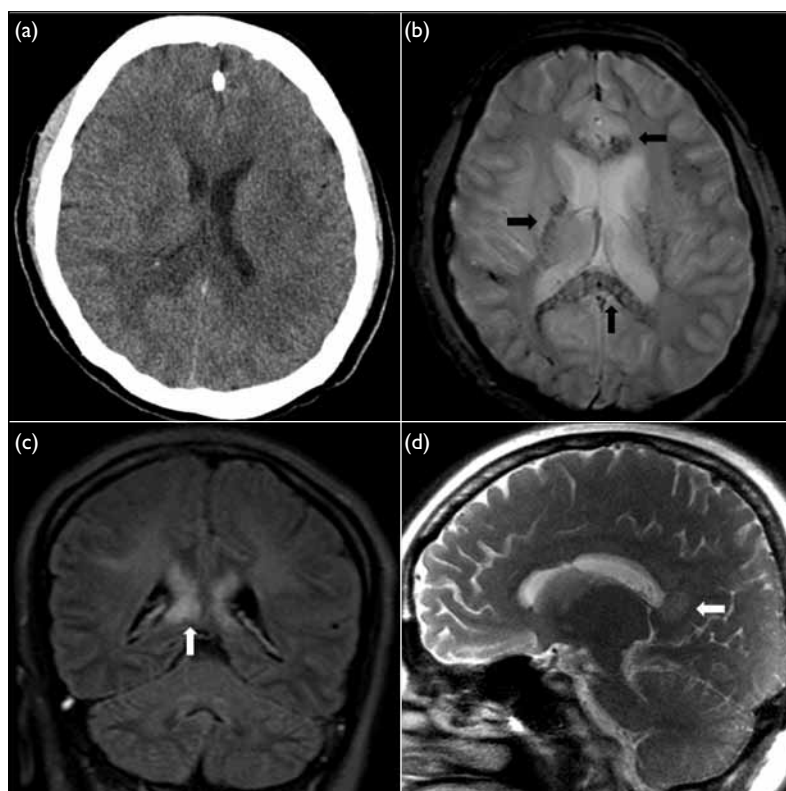


FIG 2. A 41-year-old otherwise healthy woman developed acute mountain sickness complicated with pulmonary and cerebral oedema after undertaking volunteer work for 2 weeks in Yushu, Qinghai, at an altitude of 3800 m. (a) The computed tomography performed after 2 weeks shows presumably subsiding diffuse bilateral patchy hypodensities involving cerebral white matter. (c and d) The follow-up magnetic resonance imaging (MRI) after 3 weeks show increased signals in the splenium on fluid-attenuated inversion recovery and T2 images (white arrows), and hemosiderin deposition in cerebral white matter manifested as dark spots of blooming, predominantly involving corpus callosum (b, black arrows); both characteristic MRI patterns are encountered in high-altitude cerebral oedema

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