# Sudden onset hemiplegia at high altitude

Henry C Chandler,<sup>1</sup> A Mellor<sup>2,3</sup>

<sup>1</sup>York Garrison Medical Centre, Imphal Barracks, York, UK <sup>2</sup>Department of Cardiothoracic Anaesthesia, James Cook University Hospital, Middlesbrough, UK <sup>3</sup>Academic Department of Anaesthesia and Critical Care Medicine, RCDM, Birmingham, UK

#### Correspondence to

Capt Henry C Chandler, RMO 2 Signal Regiment, Medical Centre, Imphal Barracks, York YO10 4HD, UK; henrychandler@doctors.org.uk

Received 19 October 2014 Revised 16 January 2015 Accepted 19 January 2015 ABSTRACT

Travel to high altitude and the incumbent exposure to hypobaric hypoxia leads to a prothrombotic state. This may increase the likelihood of thromboembolic events, including stroke, in otherwise healthy individuals. While there have been sporadic anecdotal reports of 'stroke-like' syndromes at high altitude for over 100 years, there are surprisingly few detailed reports supported by imaging.

This report describes a case of posterior circulation infarct thought to be due to a paradoxical embolus through a patent foramen ovale. The relationship between high-altitude physiology, increased incidence of thromboembolism and the significance of patent foramen are discussed in the report.

#### **INTRODUCTION**

Millions of lowland dwellers visit or commute to elevations above 3000 m annually. Sojourns at high altitude lead to hypoxia, an increased haematocrit and reduced plasma volume, all of which may result in a prothrombotic state. Despite this theoretical risk, reports of pathology related to thrombosis or emboli at high altitude are rare.

#### CASE

While descending from a trek to Everest Base Camp (trekking in the Himalayas) at an altitude of 3400 m, a previously fit and healthy 22-year-old men presented with sudden onset right-sided weakness, slurred speech, double vision, facial droop and depressed conscious level. The majority of his symptoms resolved after about 30 min, apart from the double vision as a result of paralysis of the superior oblique muscle of the left eye. His rapid recovery and lack of preceding headache made a diagnosis of high-altitude cerebral oedema (HACE) unlikely and a provisional diagnosis of a transient ischaemic attack was made. Initial treatment with aspirin 300 mg was given.

After 45 min, the patient began to deteriorate with worsening visual symptoms, slurred speech and decreasing conscious level. As HACE could not be excluded, dexamethasone 8 mg was given intramuscularly and the expedition leader was alerted to arrange evacuation. Nightfall prevented helicopter evacuation and a local medical clinic supplied basic observation kit, equipment for intravenous access and an oxygen concentrator. Oxygen saturations were between 88% and 92% (as expected at 3400 m), pulse 90 and regular, BP 98 mm Hg systolic. The patient was transferred from his hostel to the local clinic in Namche Bazaar by stretcher and 2 L supplemental oxygen and 500 mL Hartmann's solution were given.

Telephone advice from the UK (AM) was provided and supported a diagnosis of a cerebrovascular accident (CVA), although it was considered reasonable to

### Key messages

- ► The physiological response to high altitude can lead to a prothombotic state.
- Neurological symptoms at high altitude do not immediately indicate high-altitude cerebral oedema.
- When providing medical support in remote locations, it is vital to have a robust plan to treat medical emergencies with limited resources.

continue administration of dexamethasone, especially as HACE may present with focal deficits and mimic stroke.<sup>1</sup> Overnight the casualty's level of response fluctuated with episodes of profoundly depressed conscious level, on one occasion necessitating a jaw thrust to maintain his airway. The following morning, helicopter evacuation to Kathmandu took place. Investigations carried out there included CT head, CXR and baseline bloods that showed no evidence of cerebral oedema or pulmonary oedema. The haemoglobin was slightly elevated at 17.6 g/dL in keeping with his recent spell at high altitude. On review on day 4, the patient reported altered vision and had evidence of an abnormal gait.

Following repatriation to the UK, a magnetic resonance angiogram performed at Ninewells Hospital, Dundee, revealed a recent left-sided posterior circulation infarct as demonstrated by foci of increased signal on T2-weighted MRI images shown (Figures 1 and 2). An echocardiogram revealed a patent foramen ovale (PFO) (demonstrated on Valsalva manoeuvre). A diagnosis of a paradoxical embolus through the PFO leading to the cerebral infarct was made. At 1-month follow-up, the patient had made an almost complete recovery.

#### DISCUSSION

Adventurous training is an important part of military life, designed to give service personnel the opportunity to push themselves doing new activities in challenging environments. This shared experience promotes teamwork, encourages initiative and develops leadership skills. Mountaineering is one such activity. While not all expeditions to very high altitudes (beyond 3500 m) require a doctor, when medical personnel do provide support, they must be aware of the specific conditions, such as acute mountain sickness, high altitude pulmonary oedema and HACE. They must also have a working knowledge of the normal physiological changes to be expected at high altitude in order to be able to recognise benign symptoms and provide the essential of all treatments, reassurance.

To cite: Chandler HC, Mellor A. J R Army Med Corps Published Online First: [please include Day Month Year] doi:10.1136/jramc-2014-000372

BMJ



1

#### **Case report**



**Figure 1** Axial T2-weighted slice through the brain at the level of the frontal sinuses and the midbrain showing a focus of increased signal in the left midbrain just medial and posterior to the red nucleus.

This episode occurred 10 days into a trekking route to Everest Base Camp, Nepal. The patient and his girlfriend were travelling with a guide and had followed a sensible acclimatisation itinerary with enforced rest days designed to minimise the likelihood of any altitude-related illness. Two days previously, they had successfully reached an altitude of 5400 m without incident. These factors, added to the fact there was no immediate improvement in symptoms following descent to lower altitudes, and in the absence of headache, made a diagnosis of HACE very unlikely.

While long-term exposure to high altitude (>3000 m) has been identified as a potential risk factor for stroke,<sup>2–5</sup> there is little in the literature describing a direct link between short sojourns to altitude, as seen with expeditions and vacations, and cerebrovascular CVAs.

The physiological response to hypobaric hypoxia found at high altitude stimulates polycythaemia, initially as a result of decreased plasma volume and subsequently as a result of increased production of erythropoietin, which in turn leads to increased red blood cell production and a subsequent raised haematocrit and haemoglobin.<sup>6</sup> Polycythaemia is known to be a risk factor for venous thromboembolism.<sup>4 7 8</sup> It is known that there is an increased incidence of deep venous thrombosis (DVT) and pulmonary emboli (PE) at high altitude, although it is difficult to know how much is directly attributable to altitude and how much due to other variables such as dehydration, exercise or immobility.<sup>6</sup> That said, this patient is not thought to have had a DVT/PE and the working diagnosis is that of a clinically insignificant venous embolism shunting through the PFO to cause his symptoms.

Ischaemic events are the most common cause of CVA in the younger age group (85.8% of cases),<sup>9</sup> with cardiogenic embolism accounting for 29.4% of these. Cardiogenic emboli have also been implicated as the most common cause for posterior cerebral artery infarction.<sup>10</sup>

PFO is thought to exist in between 15% and 35% of the population, based on autopsy studies.<sup>11</sup> A review of seven case–controlled studies by Di Tullio<sup>11</sup> reported a quadruple risk of stroke in young (<55 years) patients with a PFO. In our case, the diagnosis was made using agitated saline to demonstrate microbubbles in the left atrium, indicating a right-to-left shunt during Valsalva manoeuvre. The Valsalva manoeuvre is performed to raise right atrial pressure. The vasoconstriction of the pulmonary vasculature seen at high altitude<sup>12</sup> would also increase right atrial pressure, increasing the likelihood of right-to-left shunting.

Participation in adventurous training activities provides unique opportunities for junior doctors. Preparation in advance of an expedition can address deficiencies in factual knowledge but judgement can only be based on experience. This difficulty is compounded by the remoteness of the area and professional isolation. This casualty presented with cerebral symptoms and a presumptive diagnosis of HACE was made with the initial management plan to get the patient to lower altitudes. While the symptoms of posterior circulation infarct may resemble those of HACE, on reflection, a key feature of this case was that the casualty was descending at the time of becoming unwell. Telephone advice provided much needed reassurance to inform against urgent evacuation after dark.



**Figure 2** Paramedian slices on a sagittal fluid-attenuated inversion-recovery (FLAIR)-type sequence demonstrating a corresponding focus of increased signal in the left midbrain with extension into both thalami.

#### CONCLUSION

This report describes a case of posterior circulation infarct attributed to a paradoxical embolus through a PFO. While sporadic reports of 'stroke' have appeared in the literature since 1896, very few are supported by conclusive imaging as available in this case. The normal physiology of high altitude, resulting in a prothrombotic state combined with pulmonary hypertension may have been a contributory factor. The case highlights the need for expedition medical officers to have specific knowledge and, most significantly, a plan to obtain advice in case of emergency. Whilst this case affected a civilian and not a service person, it is important to ensure follow-up for all patients to whom we have a duty of care. The subsequent diagnosis will undoubtedly lead to a change in this person's recreational lifestyle, but if it had been a soldier or, perhaps moreso, RAF flight crew there would be significant impacts on that person's employability and deployability. As such, it may be worth considering screening in the form of a transthoracic echocardiogram all service personnel expecting to deploy to high altitudes.

Acknowledgements The author would like to thank Lt Colonel DR Woods RAMC, consultant advisor (Army) in Medicine, senior lecturer in Military Medicine, visiting professor at Carnegie Research Institute, Leeds Beckett University, for his assistance and input in editing the final version. Dr E Sammler, ST5 Neurology and the Radiology Department at Ninewells Hospital, Dundee, for the information provided concerning the patients' follow-up care and imaging. The authors also thank Maj R Rushambuza RAMC, consultant radiologist Op HERRICK 20, for his assistance in putting together the representative images and the patient for providing his consent to publish this case.

**Contributors** HCC was involved in actively managing the patient with telephone advice from AM. HCC and AM prepared the manuscript. HCC was responsible for submitting the paper.

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

#### REFERENCES

- Yanamandra U, Gupta A, Patyal S, et al. High-altitude cerebral oedema mimicking stroke. BMJ Case Rep 2014;2014:pii:bcr2013201897.
- 2 Jha SK, Anand AC, Sharma V, *et al*. Stroke at high altitude: Indian experience. *High Alt Med Biol* 2002;3:21–7.
- 3 Jaillard AS, Hommel M, Mazetti P. Prevalence of stroke at high altitude (3380 m) in Cuzco, a town of Peru. A population-based study. *Stroke* 1995;26:562–8.
- 4 Al Tahan A, Buchur J, El Khwsky F, *et al.* Risk factors of stroke at high and low altitude areas in Saudi Arabia. *Arch Med Res* 1998;29:173–7.
- 5 Ortiz E, Ojeda O, Silva F. Stroke in populations located at high altitudes: review and analysis of risk factors. J Neurol Ecuador 2008;17:1–3.
- 6 Wheatly K, Creed M, Mellor A. Haematological changes at altitude. J R Army Med Corps 2011;157:48–52.
- 7 Anand AC, Jha SK, Saha A, et al. Thrombosis as a complication of extended stay at high altitude. Nat Med J India 2001;14:197–201.
- 8 Prabhakar A, Aggarwal M, Khurana P, et al. Rare case of cerebral stroke & venous thrombosis developed during high altitude expedition. *Indian J Radiol Imaging* 2006;16:313–14.
- 9 Mehndiratta MM, Agarwal P, Sen K, et al. Stroke in young adults: a study from a university hospital in north India. Med Sci Monit 2004;10:535–41.
- Helseth E, Lutsep H, Buchan A, et al. Posterior Cerebral Artery Stroke Medscape review, 2012. http://emedicine.medscape.com/article/2128100-overview (accessed 15 Oct 2014).
- 11 Di Tullio M. Patent foramen ovale: echocardiographic detection and clinical relvance in stroke. J Am Soc Echocardiogr 2010;23:144–55.
- 12 Sandberg C, Naylor J. Respiratory physiology at altitude. J R Army Med Corps 2011;157:29–32.



## Sudden onset hemiplegia at high altitude

Henry C Chandler and A Mellor

J R Army Med Corps published online February 16, 2015

Updated information and services can be found at: http://jramc.bmj.com/content/early/2015/02/16/jramc-2014-000372

These includ	le:
--------------	-----

References	This article cites 11 articles, 2 of which you can access for free at: http://jramc.bmj.com/content/early/2015/02/16/jramc-2014-000372 #BIBL
Email alerting service	Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to: http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to: http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to: http://group.bmj.com/subscribe/