LETTERS TO THE EDITOR

Focal neurological deficits and migraine at high altitude

The development of transient focal neurological deficits at high altitude is uncommon but well recognized.1 In the absence of concomitant altitude illness a thromboembolic aetiology has usually been presumed. In this case transient focal neurological deficits occurred at high altitude in clear association with migraine.

A 45-year-old right-handed white man had lived and worked at an altitude of 3840 m in the Nepal Himalaya for two years. He had a long history of migraine, with between 20 and 30 attacks (with and without aura) each year. His attacks with aura typically included homonymous visual disturbances, with only one episode before his high altitude sojourn involving additional focal neurological features. This occurred seven years previously and incorporated subtle dysgraphia lasting 30 minutes and heaviness in the right upper limb that persisted for a few hours. He had a strong family history of migraine, including one son who experienced a short-lived hemiplegia during one attack.

While living at high altitude his migraines did not occur more often than usual, but several attacks were associated with focal neurological deficits. On three occasions these attacks were preceded by numbness of one arm (twice on the right, once on the left) that resolved within 30 minutes of treatment with ergotamine. All three attacks occurred at altitudes of around 3800 m and were not associated with any particular activity. The most dramatic focal event occurred while climbing on a 6100 m mountain. He was feeling unwell with an acute acetazolamide-perfused prophylaxis for acute mountain sickness and had acclimatised well to a high camp at 5600 m. Having spent the night at this camp, he climbed for one hour to an altitude of around 5900 m when his vision started to blur. This progressing to flashing lights near the centre of vision, with half-field predominance and scotomata patchy visual loss, all typical of his migraine aura. Ten minutes later, while increasing the pitch of his companions that he would be unable to ascend further, he found that he had difficulty finding the correct words. He was accompanied down to base camp (5000 m) over one hour during which time a mild left-sided frontal headache developed, he became unable to speak, and moderate weakness developed in his right arm such that he had difficulty holding objects. At no stage did he have problems understanding the speech of others or difficulty walking. On further descent to 4300 m over three hours his symptoms had disappeared and his weakness and speech had improved considerably. At this stage he was examined by a physician who found very mild limb weakness of the right arm and a mild expressive dysphasia. Later that day he descended to 3840 m without additional problems.

Facilities for investigation at this stage were limited. On arrival at 3840 m his arterial oxygen saturation by pulse oximeter was 87% (his normal value for this elevation) and blood pressure was 118/80; packed cell volume measured one week later was 0.60. Clumsiness in the right arm lasted another two days, while subtle word-finding difficulty persisted for one week. He moved to sea level one month later and has remained well for over a year after this event, although he continues to experience regular "uncomplicated" aura attacks as previously. Subsequent neurological review showed no residual deficits.

This case is of interest for several reasons. Firstly, the development of focal neurological features preceeding headache and nausea in a typical time course, together with the complete resolution of symptoms provided support the diagnosis of migraine with aura. The neurological signs developed over a longer period than usual with stroke and the lack of other features of altitude illness makes high altitude cerebral hypoxia unlikely.

A second area of interest relates to the fact that, whereas this patient did not experience more frequent migraines while living at high altitude, his attacks incorporated more focal neurological features than he had previously experienced. Furthermore, the most dramatic event occurred at extreme altitude where his arterial oxygen saturation would have been only about 75%. The high altitude environment significantly modifies the cerebral hemodynamics and may be associated with low cerebral metabolic rate.1 Evidence suggests that migraine may result from episodes of ischaemia, possibly through triggering a spreading cortical depression.2 In the case presented here, one could also postulate that attacks may have had an additive effect with migraineous cerebral hyperperfusion in causing prolonged neurological deficits.

Knowledge about whether migraineurs experience everyday focal deficits, or frequent episodes at high altitude is incomplete. One South American study found a higher prevalence of migraine in residents living at 4328 m than in a sea level population.4 Susceptible headache sojourners have had recurrent migraine attacks triggered by ascent to certain altitudes, but these accounts are largely anecdotal. A migraine-like syndrome developed repeatedly in susceptible persons exposed to simulated altitudes of between 9000 and 11400 m in a decompression chamber.5 One well documented case involved a mountaineer who experienced transient right sided sensorimotor disturbances, dysphasia, blurred vision, and nausea associated with headache while climbing above 5000 m on two separate occasions.6 Detailed investigations of symptomatic episodes showed no abnormalities. Although lacking a clear history of migraine, this presentation suggests the diagnosis of migraine with aura. Apart from hypoxia, there are other potential trigger factors for migraine present at high altitude such as exercise, poor food and fluid intake, photic stimuli, cold temperatures, and sleep deprivation.

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Chickenpox and multiple sclerosis: a case report

Multiple sclerosis is a common, initially mostly relapsing-remitting, demyelinating disease of the CNS. Despite vigorous effort, the aetiology has not yet been elucidated. It is believed that, on the basis of a specific immunogenetic background, exogenous factors may trigger an immunological process that leads to focal demyelination in the CNS.2 Moreover, the precipitation of individual exacerbations in affected patients may probably also be triggered by exogenous factors. Viral infections have been discussed as an aetiological factor of the disease and have been reported in recurrent chickenpox and mumps attacks.2,12 We report here the precipitation of an acute exacerbation of multiple sclerosis by varicella in an adult.

One week before admission, the 27 year old white male patient experienced the rapid appearance of successive crops of vesicles on skin and mucous membranes typical of varicella, accompanied by pruritus, slight fever, malaise, and anorexia. He acquired the disease from his 31-year-old sister, who had had chickenpox two weeks earlier and was in the convalescent stage at that time. The patient had not had chickenpox during childhood. Five days after the onset of the rash he felt transient sensory impairment in both legs, unsteadiness of gait, and weakness of both legs until he was unable to walk. His medical history was unremarkable except for hypothyoxia of the right hand that resolved spontaneously one year earlier. The present medical examination disclosed only one chickenpox rash in varying stages of development and exudation in the trunk, face, and scalp. MRI, and cervical spinal cord at one scan. The neurological examination, the cranial nerves were unremarkable except for cogwheeling of pursuit movements of the eyes. There was
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