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 illness a thromboembolic etiology has usually been presumed. In this case transient focal neurological deficits occurred at high altitude in clear association with migraine.

A 55-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 brother who continued to experience 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. Three others 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 experienced several attacks of subacute prophyaxis for acute mountain sickness and had acclimatized 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 5500 m when his vision started to blur. This progressed to flashing lights near the centre of vision, with half field predominance and scotomatous patchy visual loss, all typical of his migrainous aura. Ten minutes later, while informing 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 altitude) 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 or speech difficulties 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" migraine 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 at high altitude is clearly associated with migraine. Focal neurological conditions occurring at high altitude in the absence of altitude illness have usually occurred without relationship to altitude.

In this case the history of migraine, the development of focal neurological features preceding headache and nausea in a typical course, together with the complete resolution of symptoms and signs, suggests a specific 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 hypoperfusion 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 florid 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 absence of further development of migraine probably indicates that cerebral hypoxia is central to the pathophysiology of migraine attacks, and that any cause of hypoxia could cause migraine, including low atmospheric pressure.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 speculate that hypoxia may have an additive effect with migrainous cerebral hypoperfusion in causing prolonged neurological deficits.

Knowledge about whether migraineur experience more attacks at altitude, or whether 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.³ Susceptible low altitude sojourners may have recurrent migraine attacks triggered by ascent to certain altitudes, but these accounts are largely anecdotal. A migraineur who developed repeatedly in susceptible sojourners to simulated altitudes of between 9000 m and 14 400 m in a decompression chamber.³ One well documented case involved a mountaineer who experienced right sided sensorimotor dysfunctions, dysphasia, blurred vision, and nausea associated with headache while climbing above 5000 m on two separate occasions.⁴ Detailed investigations after both episodes showed no abnormalities. Although lacking a clear history of migraine, this presentation suggests the diagnosis of migraine with aura. Apart from hypo-oxygenation, triggering factors for migraine may include hypoxia as exercise, poor food and fluid intake, photic stimuli, cold temperatures, and sleep deprivation.

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. Moreover, the precipitation of individual exacerbations in affected patients may probably also be triggered by exogenous factors. Viral infections have been discussed as an aetiologic factor of the disease, and recent evidence suggests that megalovirus attacks may in individual cases be linked to chickenpox.⁵ 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 wife, who had chickenpox two weeks earlier and was in the convalescent stage at that time. The patient had not had chickenpox before childhood. Five days after the onset of the rash he found himself unable to move the right side of the trunk, face, and scalp. He was admitted to hospital, and neurological examination, the cranial nerves were remarkable except for cogwheeling pursuit movements of the eyes. There was...