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Pavor nocturnus from a brainstem glioma

Pavor nocturnus or night terrors usually occur in the absence of identifiable neuropathology. This report documents pavor nocturnus associated with a brainstem lesion.

A 15 year old boy with headaches and ataxia had a brainstem tumour on CT (see fig). The patient underwent resection of a grade I cerebellar astrocytoma arising from the fourth ventricle and adherent to the brainstem. His postoperative examination disclosed dysarthria, spontaneous vertical nystagmus, bilateral sixth nerve paresis, decreased sensation on the face bilaterally, facial diplegia, sensorineural hearing loss, mild dysmetria and ataxia, and decreased reflexes with bilateral Babinski responses.

Postoperatively he developed a sleep disorder where he suddenly sat up in bed, screamed, and appeared to be staring in fright. During these episodes he was agitated and would try going over the rails of the bed or would thrash about in bed screaming. After one or two minutes, he promptly fell back to sleep. The patient had incomplete recollections of these episodes. Sometimes the only evidence of an episode was injury or blood on the floor. At other times, he recalled being frightened by images of parts of people sticking out of walls or by the belief that the bedposts were his room mates restraining him. The patient subsequently became depressed and had aggressive outbursts and paranoid beliefs. Before his tumour, he did not have a history of neurological or psychiatric difficulties, and there was no family history of a sleep disorder.

At the age of 24, polysomnography documented his night terrors. Spontaneous arousals punctuated all stages of sleep (12 a night), particularly stage three and four sleep. The patient's arousals from slow wave sleep were typically sudden and included restlessness, vocalisation, and looking accompanied by interspersed alpha and delta activity on electroencephalography (EEG). After starting clonazepam (0·5 mg at bedtime), the episodes of night terrors decreased, but he developed enuresis.

There are few documented cases of pavor nocturnus from neurological disturbances.[1] Reports suggest that night terrors may occur as a consequence of a right temporal lobe seizure focus.[2] This report documents pavor nocturnus associated with a fourth ventricular brainstem lesion.

Pavor nocturnus is usually not associated with any psychiatric or neurological disturbance when it occurs in children.[1 3] Its natural course is to disappear by adulthood,[3] suggesting a disorder of maturation of the nervous system. When night terrors begin in adolescence or adulthood, there is a likelihood of psychological difficulties.[4] Patients with night terrors may repress their anger or aggression and show elements of obsessive-compulsive and phobic behaviour as well as depression.[4] Our patient has no difficulty expressing anger and does not have obsessive-compulsive behaviours. Rather, he has a brainstem lesion disrupting non-rapid eye movement (REM) sleep and resulting in night terrors. Furthermore, clonazepam which suppresses slow wave sleep has decreased his night terrors while unmasking enuresis.

Pavor nocturnus is a consequence of partial arousal during non-REM slow wave sleep. During deep sleep, the brain continues to process subcortical sensory information such as respiratory awareness.[5] With arousal from non-REM sleep, these sensory experiences may intrude into consciousness as single, brief, frightening experiences. Patients are terrified and may even injure themselves trying to escape the frightening image or feeling. In this patient with a brainstem lesion, the mechanism of slow wave arousal may be disruption of the nucleus of the solitary tract in the dorsum of the brainstem near the wall of the fourth ventricle. Activation of this nucleus promotes sleep and may modulate the arousal properties of the ascending reticular activating system.[6] Polysomnographic studies of patients with comparable brainstem lesions would help elucidate the pathophysiology of pavor nocturnus and the proposed role of the nucleus of the solitary tract.

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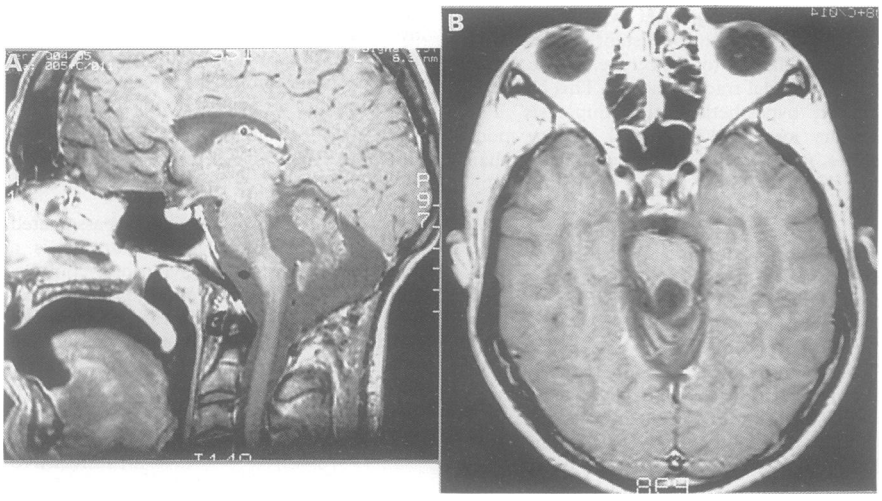

Figure Cerebellar astrocytoma adherent to brainstem.

Octreotide—a new treatment for diarrhoea in familial amyloidotic polyneuropathy

Familial amyloidotic polyneuropathy (FAP) is a well known hereditary polyneuropathy which was reported for the first time by Corino de Andrade in Portugal.[1]

This disease generally begins with sensory symptoms and signs (50%) and sexual impotence in man (30%). A less significant number of subjects have constipation (20%), loss of weight (10%) or diarrhoea (10%) as an initial symptom. After two to five years of other dysautonomic symptoms are very common, namely orthostatic hypotension and severe diarrhoea.

In a group of 60 patients followed at the neurology outpatients, we found that half had regular diarrhoea which was particularly refractory in six. To control the diarrhoea we used a low fibre diet, antibiotics (tetraciclines and metronidazole), metoclopramide and loperamide.