

form layer of the superior colliculus and periaqueductal area, somewhat less in the substantia nigra. The astrocytosis in the 1981 case was clearly visible in the same areas but much less marked than in the 1920 case.

Since our histological observations showed marked lesions only in the midbrain sections, our subsequent immunocytochemical studies were confined to these areas. Attempts to find antigens from several viruses were carried out by means of immunoperoxidase techniques using antisera against the following viruses or strains: Influenza A, strains A-2 Japan 305/57, WS/33 and A-swine 1976/31, prepared in chicken; A/New Jersey, prepared in goat; and neuroadapted NWS strain prepared in rabbit. Influenza B, strain Hong Kong, prepared in goat, herpes virus type 1, prepared in rabbit, rubella and cytomegalovirus, prepared in goat, measles and mumps viruses, prepared in guinea pig. The tests were indirect immunoperoxidase with the chicken sera, and peroxidase-antiperoxidase (PAP) with rabbit, goat and guinea pig sera.

The peroxidase tests were uniformly negative; no antigen for any of the above listed viruses or strains were detected in any of the sections examined. These results confirm the absence of herpes virus antigen in the 1920 Esiri and Swash⁴ case; and do not support previous observations by Gamboa *et al*⁵ using a different technique, direct immunofluorescence and frozen sections, who reported positive reactions with NWS strain of influenza A antiserum. Furthermore, our results have added cytomegalovirus, rubella, mumps and measles, all widespread conventional viruses, to the list of viruses whose antigens have *not* been detected in brain tissue from cases of encephalitis lethargica and post-encephalitic Parkinsonism.

All attempts have thus far failed to show a reproducible connection between a known virus and encephalitis lethargica or post-encephalitic Parkinsonism. However, the nature of the histological lesions in the present cases still strongly suggest a viral aetiology. Our negative immunocytochemical results may be due to the fact that the procedures used for antigen detection were not sensitive enough; or that the responsible virus, established or as yet undiscovered, was not among those used as probes; or that the specimens investigated were taken at a time when the antigen had already been degraded or eliminated; or, of course, that no virus is involved. It is interesting to note that in a similar disease, idiopathic Parkinson's disease, McGeer *et al*⁶

recently found evidence of an active neuropathological process at the time of death, from which they concluded that a chronic infection or an autoimmune process following an infection was a tenable hypothesis.

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Head injury induced migraine coma simulating acute extradural intracranial haemorrhage

Sir: Migraine induced by head trauma¹ and migraine coma² are uncommon but well described. We present a case in which both conditions coincided, simulating the presentation of acute extradural haemorrhage.

A 15 year old girl suffered a blow to the nose while playing hockey. She had visual scintillations which took a complex linear pattern, but did not lose consciousness and was able to play on for the 2-3 minutes remaining of the game. On coming off the field she felt light headed, nauseated and drowsy, with further scintillations. The drowsiness rapidly increased until she lost consciousness 15 minutes later. She was

admitted to hospital where she was found to be deeply unconscious, with a weak flexor response to pain, no eye opening and no verbal response. There were no focal neurological signs. A presumptive diagnosis of acute extradural haemorrhage was made and, after intubation and ventilation for air way protection, she was transferred to the regional neurosurgical unit. Computed tomography (CT) there was normal. Ventilation was discontinued, and she regained consciousness over the subsequent hour, having been unconscious for a total of 4 hours. At this stage, she was confused with a mild right monoparesis, but no other focal signs. She had a history of classic migraine over the previous year. Her aura consisted of teichopsia, which took exactly the same pattern as both the episodes of scintillation which followed her head injury, light-headedness, nausea and drowsiness, and was identical to the prodrome of her present illness. The headache was mild and often brief, accompanied by photophobia. There was no past history of her migraine following head injury. She had a family history of migraine.

Electroencephalography the day after admission was normal, as was cerebrospinal fluid examination (including immunology) on the 7th day after admission. At 5 days her monoparesis was just detectable, and she still had mild impairment of digit span. At 8 days she was completely recovered.

The patient had a typical history of classic migraine. The present episode was preceded by symptoms identical in detail to her usual aura, confirming its migrainous nature. The normal CT, EEG and CSF studies, and her complete recovery excluded serious illness. The connection with head injury is strongly supported by the time course of her illness, and the fact that even those scintillations occurring immediately after the blow were identical to her usual migrainous teichopsia.

Secondary depression of conscious level short of coma may occur without migrainous features following head injury³ and migrainous attacks precipitated by minor head injury may be accompanied by drowsiness, particularly in children.¹ The only reports of coma with clear features of migraine are of one family in which migraine coma, often precipitated by trivial head trauma, was associated with dominantly inherited cerebellar ataxia⁴ and of one sporadic case similar to our own.²

In patients with trauma-induced migraine a head injury that would otherwise have only minor or subclinical effects can precipitate a migraine attack. The reticular formation of the brainstem is thought to be important in

the pathophysiology of migraine,⁵ and dysfunction here may also be responsible for the impairment of consciousness.

In the present case, loss of consciousness soon after head injury very reasonably led to the diagnosis of acute extradural haemorrhage. In other cases, such features as drowsiness, confusion and hemiparesis following head injury have also led to admission to neurosurgical units.² Such patients were formerly investigated by angiography, which often caused neurological deterioration,⁵ and findings suggestive of an avascular space occupying lesion (thought to be due to oedema) led in some cases to inappropriate surgical exploration.²

Where coma or focal neurological dysfunction follow a mild head injury, and CT is negative, the diagnosis of trauma-induced migraine should be considered.

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Relapsing polyneuritis following classic Miller Fisher syndrome

Sir: The Miller Fisher syndrome is a well described form of acute inflammatory polyneuropathy, a variant of the Guillain-Barré syndrome. Initially reported by Fisher in 1956,¹ this disease is characterised by the acute onset of ataxia, areflexia and ophthalmoplegia often preceded by a viral or other illness. Despite the alarming presentation, the disease usually has a benign outcome with a spontaneous and complete recovery

within weeks. We report a case of classic Miller Fisher Syndrome followed by a bout of relapsing polyneuritis 8 weeks later.

A 21 year old white female student presented with complaints of facial paraesthesias, diplopia, generalised weakness, and a gait disturbance described as a "loss of balance sense."

Physical examination of heart, lung and abdomen was normal. Neurological examination revealed an ophthalmoplegia involving all extraocular muscles with minimal vertical gaze preserved bilaterally. Bilateral ptosis was noted, with no nystagmus. Pupils were reactive to light and accommodation. Deep tendon reflexes were slight in the upper extremities and absent in the lower extremities. Her upper and lower extremity strength was intact bilaterally, as was sensory perception. Ataxia was present, with a wide based, unsteady gait.

Lumbar puncture revealed a glucose of 58 mg/100 ml, protein of 72 mg/100 ml, and a cell count of 10/mm³ RBC and 1/mm³ WBC. Nerve conduction and EMG studies were performed. Skin temperature was within the normal range (31°C). Peroneal nerve motor velocity was 44 m/s. F wave conduction were slow at 35 mm/s. Sural responses were absent despite averaging. This was consistent with a mild demyelinating neuropathy.

The patient was diagnosed as suffering from Miller Fisher syndrome and was observed in the intensive care unit. Over the next several days, the patient experienced progressive leg and arm paraesthesias, without a deficit in limb strength. Slowly, the patient improved, and was discharged 10 days after admission with minimal ptosis, paraesthesias, facial weakness and ataxia.

The patient was seen one month later where she was found to be walking easily and feeling stronger. There was full ocular motility, with minimal ptosis of the left eye and mild right sided facial weakness. Sensory testing revealed minimally diminished vibration and position sense of hands and fingers with mildly diminished pin sensation in a stocking distribution. The gait was normal. Deep tendon reflexes were absent.

One week later she developed an exacerbation of her neurological symptoms. Ambulatory skills declined with development of foot and hand paraesthesias. On re-examination the patient was noted to have no position sense with manual testing of her hands and feet or any distal vibratory sense. Strength was good and her extraocular muscles remained intact. She had no exacerbation of her facial weakness or ptosis. Her dramatic clinical decline was consistent with a disabling relapsing polyneuropathy,

primarily sensory in nature. She was then started on a two week regimen of oral prednisone.

After one week of steroid therapy, the patient reported a dramatic improvement in her symptomatology. Unsteadiness, imbalance and diminished position were less noticeable. It was felt that she was entering remission stage of her neuropathy.

Physical examination three months after her initial diagnosis revealed persistence of the absent deep tendon reflexes. The facial weakness and ptosis, significant at the onset of her illness, were no longer apparent. Extraocular muscle movement was without limitation. Tactile sensation was normal, although position and vibration were still mildly impaired. She was able to walk without assistance and anticipated returning to her activities of normal living.

The Miller Fisher syndrome is a rare form of acquired demyelinating neuropathy,¹ a variant of Guillain-Barré syndrome, this monophasic illness develops acutely over a few days. The ataxia is often severe and debilitating. Symmetrical ocular palsies occur with greater involvement of extraocular musculature. Limb involvement is minimal or absent, although motor weakness has been known to occur. Patients usually recover without specific treatment over the course of several weeks after a relatively benign but frightening illness.

Our patient exhibited all the features of the syndrome. What distinguished our case from the others was the development of recurrent polyneuritis complicating the initial diagnosis of Miller Fisher syndrome. We did not expect her to relapse after her rather benign initial course. Interestingly, during the time of the relapse, the patient did not exhibit an exacerbation of her ophthalmoplegia or facial weakness. Her main problem was sensory with loss of position and vibratory sense of her extremities, as well as pin touch. A dramatic improvement was noted after the introduction of corticosteroid therapy.

A review of the literature has yielded similar but not identical cases. Donaghy² reported three cases of ocular palsies preceding the development of chronic polyneuritis by an interval of two to ten weeks. Two patients with ophthalmoparesis and recurrent sensory neuropathy were reported by Kaplan.³ Chalmers and Miller in 1980 reported a patient with a 2 year history of chronic progressive inflammatory polyneuropathy with a subacute exacerbation that included ataxia, areflexia, ophthalmoparesis and ptosis. Schapira³ reported a patient with a relapsing neurological deficit. He initially manifested the Miller Fisher