is expedited by breathing oxygen, preferably under pressure.

The classical structural damage caused by carbon monoxide poisoning is a diffuse hypoxic cerebral injury leading to infarction. The most common lesions are of the pallidum bilaterally and are predominantly in the anterior two thirds, and probably result from the sensitivity of this functional end arterial area to hypoxaemia and hypotension. A unilateral pallidal lesion and marked asymmetrical white matter involvement has been described.1 The unique tension.

must be due to the sensitivity of the cerebral cortex to carbon monoxide.

under pressure.

The involvement of the globus pallidus and medial temporal lobes was seen. The precise cause of this cerebellar oedema is unknown, but could have been due to acidosis and hypoxaemia which are a common cause of widespread white matter oedema in CO poisoning. The hydrocephalus was of a transient nature since it subsided on medical therapy. Bilateral cerebellar oedema in acute carbon monoxide poisoning, presenting with acute hydrocephalus, has not been previously reported. It is important to be aware of this complication in the initial stages, as a CSF diversion procedure might be required if it does not respond to steroids.

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IgG, oligoclonal bands and cytology. Her HLA type is A1, A2, B8, B18, DR3 and DR4.

The main abnormality of the CSF was a variable lymphocytic pleocytosis in 11 lumbar punctures (range 6–268 cells). On three occasions the CSF protein was raised (0·60–0·70 g/l). Glucose levels have been normal.

During a 20 month period, five MRI brain scans have been performed. The first, a month after the initial presentation was normal. The second, three months later, showed an area of high signal in the right cerebellar hemisphere on T1 images and correspondingly an area of low signal on the IR scan. At 12 months a third MRI showed several areas of increased signal on T2 images; in the left middle cerebellar peduncle extending into the anterior aspect of the left cerebellar hemisphere, with a corresponding area on the right side, and also on the medial aspect of the right temporal lobe. These areas enhanced with intravenous gadolinium. Sixteen months after presentation a fourth MRI showed widespread T1 increased signal changes localised in the left fronto-parietal area involving both white matter and cortex, the left optic tract, the right temporal lobe, the corpus callosum and the right brainstem, extending into the right cerebellar peduncle. The areas involved were more extensive than those seen previously. Her most recent MRI scan, three months later, showed further new gadolinium enhancing lesions in the right temporal and parietal lobes, with resolution of some of the other lesions seen on earlier scans. The new enhancing right parietal lesion (fig) was biopsied stereotactically. Histology was non-specific with an excess of astrocytes in the cortex and more prominent in the white matter with oedema, narrow perivascular cuffs of myelin pallor and small numbers of foamy macrophages.

Neurophysiological investigation showed that the previously abnormal visual evoked response (VERs) became normal following the first episode of bilateral optic neuritis. After further attacks of right optic neuritis,
VERs from the right eye remained persistently abnormal (P100 latency 121 ms); also over the past eight months VERs from the left eye have become abnormal (P100 latency 118 ms). Brainstem auditory evoked responses became abnormal, on the left side six months after the onset of the illness, and on the right side 12 months after onset. These deteriorations occurred after two clinical exacerbations affecting the brainstem and preceded the third MRI scan. Electroencephalograms showed widespread delta activity most prominent frontally and also interseizure epileptiform activity.

We describe a patient who has a chronically active, diffuse relapsing and remitting meningo-encephalo-myelitis of unknown aetiology. It is similar to the case described by Monteiro and Correia differing in the frequency of relapses, and partial remission, the presence of a chronic active diffuse process and the association with abnormal MRI scans.

There has been no evidence of sarcoidosis, systemic lupus erythematosus, cerebral vasculitis, cerebral lymphoma, Behcet's disease or cerebral Whipple's disease. An empirical course of sulphamethoxazole-trimethoprim was given without benefit.

The natural history of the illness presented in this patient is not characteristic of accepted clinical patterns of multiple sclerosis. It has exhibited frequent, recurrent episodes of relapsing and active meningo-encephalo-myelitis with pyrexia and epilepsy. It is, however, conceded that the sites of involvement are elective sites in multiple sclerosis. Lumsden stated that he could find "no meaningful pathological distinctions between acute disseminated encephalomylitis and multiple sclerosis."

We believe that the description of rare but carefully documented cases of this type may provide useful information on the pathology and pathogenesis of relapsing meningo-encephalo-myelitis, and possibly also demyelinating disease. It may also be desirable to exclude the adjective "benign" from the title "benign relapsing meningo-encephalo-myelitis".

Pain arising from the oesophagus may mimic glossopharyngeal neuralgia

A 51 year old female presented with a two year history of ear pain. Lancinating attacks lasting 3–4 seconds began on the left side of the throat and radiated towards the left ear. No specific precipitants could be identified and the symptom occurred at any time of day or night. She was otherwise symptom free and had no significant past medical history. Physical examination was entirely normal. A provisional diagnosis of glossopharyngeal neuralgia was made and the patient was started on carbamazepine. The initial response was promising but the pain recurred and persisted despite increased doses of carbamazepine and a trial of sodium valproate.

Eighteen months into therapy the patient began to complain of pain behind the sternum coinciding with the pain felt behind the ear. Progressively the retrosternal component developed dysesthetic features though still episodic and related to the lancinating attacks of ear pain which remained strictly unilateral. The symptoms were now noticeably worse at night. Gastroscopy and pH monitoring were performed. A severe grade 3 oesophagitis was noted at endoscopy. Intra-oesophageal pH remained below 4·0 for 26% of the 24 hour study period (normal <3·4%). Reflux peaks correlated well with the patients symptoms. Omeprazole was started with resolution of ear pain and retrosternal discomfort over a period of 12 months review.

It has long been recognised that diseases of the oesophagus, notably gastro-oesophageal reflux and oesophageal spasm may mimic the pain of myocardial ischaemia. Oto-laryngological manifestations, though less widely known include otalgia, chronic pharyngitis and cervical pain two. However, lancinating, unilateral pain confined to the throat and ear is a pattern more consistent with glossopharyngeal neuralgia.

As medical and surgical therapy fails to relieve the pain in 30–40% of cases of glossopharyngeal neuralgia the possibility that the pain may be referred from the oesophagus should be considered.

Benign relapsing meningo-encephalomyelitis.

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