aluminium concentration was ten times higher than normal, while no other blood values were abnormal. Finally, the interruption of oral aluminium intake and the reduction of the aluminium concentration in the dialysis fluid were followed by progressive clinical improvement and cure within one month.

It is difficult to determine whether aluminium intoxication is due mainly to digestive absorption after oral intake or to the high concentration of aluminium in dialysis fluid. Generally the concentration of aluminium in dialysis fluid plays an important part in intoxication,²⁻⁵ though prolonged ingestion of aluminium salts during chronic renal failure may be responsible for causing encephalopathy. The fact that encephalopathy due to aluminium toxicity may be reversed by stopping aluminium administration indicates the importance of performing repeated measurements of blood aluminium concentrations in patients undergoing haemodialysis and of controlling the aluminium content in dialysis fluid. When considered with EEG and clinical data these measurements may lead to early diagnosis of aluminium toxicity and reversal of the usually fatal course of dialysis encephalopathy.

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Occlusion of external carotid artery causing intermittent claudication of the masseter

Intermittent claudication of the masseter is an unusual but well-recognised symptom normally associated with temporal arteritis.^{1,2} We report a case in which it was caused by occlusion of the external carotid artery.

Case report

A 59-year-old man was admitted to hospital in 1976 after suddenly developing aphasia and inability to control his right hand. These symptoms improved rapidly. Three months previously he had developed cramp-like pain around the angle of the right mandible, which first occurred within two to three minutes of chewing food, especially meat. He had the pain only when chewing. It went almost immediately he stopped chewing, although a dull ache remained for about five minutes. The rate of onset and severity of the pain gradually increased. When he was admitted he could eat meat only if it had been minced. He had had myocardial infarction in 1966 and in 1975. In 1967 he developed intermittent claudication in the calves of both legs and in 1971 he was treated for hypertension. He smoked 40 cigarettes a day.

On examination in hospital the central nervous system was normal and there were no carotid bruits. Neither the temporomandibular joints, the salivary glands, nor the superficial temporal arteries were tender, but pulsations of the right superficial temporal artery were reduced. No significant abnormality was noted in the mouth. The skin of the face and scalp showed no evidence of impaired blood supply. The ESR was 8 mm in 1 h and x-ray examination of the right temporomandibular joint was normal. An ECG showed no evidence of a recent myocardial infarct. Both carotid bifurcations were visualised by a non-invasive technique using Doppler-shift ultrasound.^{3,4} This showed a totally occluded external carotid artery with a normal internal carotid artery on the right side and a normal carotid bifurcation on the left side (figure). These findings were confirmed by an arch aortogram. The episode of aphasia and incoordination of the right hand was

Ultrasound images of both carotid bifurcations taken in supine position (lateral views). Above: normal left junction showing internal carotid inferior to external carotid artery. Below: image of right carotid bifurcation showing patent internal carotid and completely occluded external carotid artery.

thought to be due to a transient ischaemic attack. Although the precise cause of this attack was uncertain he was treated with aspirin and dipyridamole and had no further symptoms. The pain in the jaw remained unchanged for a further three months. It then gradually improved and after another three months he was able to chew without discomfort.

Comment

We think the pain on eating in this patient was due to intermittent claudication of the masseter. We believe this is the first report of it being caused by occlusive disease of the external carotid artery. It persisted for a total of nine months and we assume that its disappearance was due to a collateral blood supply developing. The patient had generalised occlusive arterial disease. An unusual feature in the extracranial cerebral arteries was the severe disease of the external carotid with no demonstrable disease in the internal carotid artery. Probably atheroma at the origin of the external carotid artery was the principal cause for the total occlusion of this vessel, but possibly it was caused by an embolus from the heart, a result of ischaemic heart disease, rather than by atheroma. An embolus might have also caused the transient ischaemic attack.

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Thrombocytopenia associated with legionnaires' disease

Legionnaires' disease (LD) commonly presents as an acute and fulminating pneumonia. The causative organism is a Gram-negative pleomorphic bacillus. We report a case which was associated with a transient thrombocytopenia and cerebellar ataxia for which no other cause was found.

Case report

A 22-year-old driver complained of six days' malaise, left-sided pleuritic chest pain, and sweating. Three days later he developed a headache, slurred speech, an unsteady gait, and his tongue felt too large for his mouth. Lobar pneumonia was diagnosed and treatment with oral penicillin begun. After an epistaxis three days later his condition deteriorated and he was admitted to hospital. On admission he was dyspnoeic and ill, pulse 112/min, and temperature 39°C. He had the classical signs of a left upper lobe pneumonia. He also had widespread bronchospasm. He was dysarthric and hypotonic with mild neck stiffness. His white cell count was $8.7 \times 10^9/l$ ($8700/mm^3$) but the neutrophils showed toxic granulations and a left shift. The haemoglobin was 15.8 g/dl and the platelet count $40 \times 10^9/l$ ($40\,000/mm^3$). Bone marrow aspirate was histologically normal and, apart from a slight rise in the fibrin degradation product concentration to 40-80 mg/l, there was no other evidence of disseminated intravascular coagulation. Normal commensals were grown from the sputum and cultures from the blood and cerebrospinal fluid were sterile.

Treatment with erythromycin and flucloxacillin was started with a presumptive diagnosis of staphylococcal pneumonia or LD. He had a further epistaxis, but the clinical and radiological signs of infection improved. The cerebellar dysarthria worsened with the evolution of hypotonia, intention tremor, and dysidiadochokinesia in the left arm, which resolved over the next five days. Direct laryngoscopy was normal together with a normal computerised axial tomogram of the brain and normal CSF on lumbar puncture. The diagnosis of LD was based on a serum antibody titre five weeks after discharge of over 1020. No other significant antibody titres were found and a screen for infectious mononucleosis was negative. No other cause was found for the thrombocytopenia. Notably there was no evidence of disseminated intravascular coagulation and the aspirated bone marrow was normal. The platelet count gradually rose to $180 \times 10^9/l$ ($180\,000/mm^3$) five days after admission. Since then it has been normal and his subsequent progress has been uneventful apart from a deep vein thrombosis of the right calf.

Comment

Although neurological manifestations of this disease and associated gastrointestinal bleeding have been described,¹ we believe this is the first description of thrombocytopenia associated with LD.

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Functional independence in post-anoxic myoclonus: contribution of L-5-HTP sodium valproate and clonazepam

Action myoclonus is a well-recognised complication of cerebral hypoxia which may result in long-term disability and make heavy demands on nursing and related health care resources. Successful treatment with L-5-hydroxytryptophan (L-5-HTP) has suggested a selective deficiency of the neurotransmitter 5-hydroxytryptamine (5-HT); renewed interest in the pharmacological management of this incapacitating condition¹⁻³; and reduced patient dependency. Although a form of myoclonus responsive to L-5-HTP has been characterised by electrophysiological studies,³ not all cases respond satisfactorily to the 5-HT precursor, which is not surprising in view of the diffuse and variable nature of the cerebral insult in patients with post-anoxic myoclonus (PAM).

We describe a patient with severe PAM with minimal intellectual

impairment who regained functional independence only with the combination of L-5-HTP, a decarboxylase inhibitor (carbidopa), sodium valproate, and clonazepam.

Case report

A 30-year-old woman with steroid-dependent asthma sustained a respiratory arrest, the length of which was not documented. She was comatose and without focal neurological signs on admission to the intensive care unit. Assisted ventilation was continued for the 18 days she was unconscious. The day after admission spontaneous and induced myoclonus started and a single major seizure occurred. Parenteral phenobarbitone, diazepam, and oral phenytoin had no effect on the jerking.

On regaining consciousness and rational communication she was incapacitated by myoclonus, which was readily induced by any auditory or visual stimuli and voluntary or passive movements and which often became generalised. Nitrazepam, methylphenobarbitone, and carbamazepine were also unhelpful but clonazepam produced improvement. Three months later she remained completely dependent, unable to feed herself, or walk without help. In addition to spontaneous and induced focal distal, proximal, and generalised myoclonus there was a mild spastic tetraparesis and truncal ataxia. An electroencephalogram (EEG) showed central intermittent single and short-duration multiple spike and slow-wave complexes against background 6-7 Hz theta activity. One in three such discharges was associated with spontaneous myoclonus.

After cinematographic records and baseline values had been recorded, including cerebrospinal fluid 5-hydroxyindoleacetic acid (5-HIAA) and 24-hour urinary 5-HIAA concentrations, L-5-HTP and carbidopa were started with the patient's informed consent. L-5-HTP 1200 mg/day and carbidopa 300 mg/day produced a dramatic improvement, so that the patient could feed and dress herself and walk unaided. 5-HIAA concentrations were increased (in the cerebrospinal fluid from $0.26 \mu\text{mol/l}$ (49 ng/ml) before treatment with L-5-HTP to $2.2 \mu\text{mol/l}$ (422 ng/ml) after; in urine from $19 \mu\text{mol/l}$ (3.6 mg/l) to $488 \mu\text{mol/l}$ (93 mg/l)); the EEG showed more alpha-activity and less spike activity without associated jerking. The methylphenobarbitone and carbamazepine were withdrawn without effect and the clonazepam was continued. Although the generalised jerking was suppressed, the degree of overall control fluctuated considerably from day to day, and stimulus-responsive distal jerks continued.

The L-5-HTP became temporarily unavailable after nine weeks and a placebo was given with the carbidopa, resulting in increased myoclonus and reduced 5-HIAA concentrations. The addition of sodium valproate 1800 mg/day effected a significant though less dramatic improvement. With the reintroduction of L-5-HTP to the regimen of valproate, clonazepam, carbidopa, and phenytoin the patient was better than at any earlier stage. She negotiated stairs independently and there was less variability in day-to-day control and less distal jerking. Pulmonary function did not change during L-5-HTP administration.

Comment

Although most cases of PAM respond satisfactorily to L-5-HTP and clonazepam, perhaps by different mechanisms,³ some cases do not.¹⁻³ The clinical features of the myoclonus and the response to L-5-HTP in our patient suggested so-called reticular reflex myoclonus.³ The residual, stimulus-sensitive, distal jerking resembled cortical loop reflex myoclonus³ and was controlled by valproate. This may have resulted from an effect on gamma-aminobutyric acid (GABA), as experimental depletion of striatal GABA in combination with cortical damage produces focal myoclonus,⁴ and valproate has been shown to increase GABA levels.⁵ The addition of valproate in the present case also improved the day-to-day control and led to functional independence.

We suggest that valproate should be tried in patients with PAM whose response to L-5-HTP and clonazepam is suboptimal, particularly if more than one type of myoclonus is present.

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