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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¹ Alfrey, A C, *et al*, *New England Journal of Medicine*, 1976, **294**, 184.

² Cartier, F, *Nouvelle Presse Médicale*, 1978, **7**, 97.

³ Buge, A, *et al*, *Nouvelle Presse Médicale*, 1978, **7**, 2053.

⁴ Boukari, M, *et al*, *Nouvelle Presse Médicale*, 1978, **7**, 85.

⁵ Platts, M M, *et al*, *British Medical Journal*, 1977, **2**, 657.

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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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¹ Russell, R, *Quarterly Journal of Medicine*, 1959, **28**, 471.

² Bulgen, D Y, and Hagleman, B L, *Hospital Medicine*, 1976, **2**, 39.

³ Lewis, R R, *et al*, *Stroke*, 1978, **9**, 465.

⁴ Beasley, M G, *et al*, in *Doppler Ultrasound in the Study of Central and Peripheral Circulation*, ed J P Woodcock and R F Sequeira, chapter 7. Bristol, University of Bristol Press, 1978.

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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.