No significant abnormality could be seen in an electroencephalograph about two weeks after the event, but a right carotid angiograph showed narrowing of a distal branch of the middle cerebral artery in the region of the trifurcation.

Comment

Acute hemiparesis with radiological findings of arterial narrowing suggest ischaemia in the right middle cerebral territory due to arterial occlusion. The fact that the hemiparesis occurred soon after the skip-jack reaction is significant. Skipjack contains a high concentration of histamine in the range of 390-900 mg/100 g tissue.[1] Another variety of fish, *Scomberomorus*, contains 0·5-7·5 mg/100 g tissue. Normally histamine absorbed from the intestine is readily metabolised in the tissues by two enzymes, n-methyl transferase and diamine oxidase (histaminase). But in the presence of isoniazid, a potent inhibitor of diamine oxidase, histamine absorbed from skipjack may reach toxic concentrations. These are thought to be the pharmacological basis of the "skipjack" reactions.

A major action of histamine in the human is capillary and arteriolar dilatation causing hypotension. Cerebral arterial occulusion may occur in predisposed individuals during acute hypotensive episodes, as in acute myocardial infarction and during anaesthesia. We think the sudden lowering of blood pressure due to histamine from the skipjack reaction precipitated the cerebral arterial occlusion in our patient. The well-known interaction between cheese and monoamine oxidase inhibitor through the mediation of tyramine has caused cerebrovascular accidents due to acute hypertension. Our case illustrates a similar food and drug interaction resulting in a cerebrovascular accident, but on this occasion probably due to acute hypotension.

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Polymyalgia rheumatica and primary biliary cirrhosis

Hepatic dysfunction is common in polymyalgia rheumatica (PMR)[1] but only one case of PMR with primary biliary cirrhosis (PBC) is recorded.[2] We report three patients with PMR and features of PBC.

Case reports

(1) A 61-year-old woman presented with an exacerbation of chronic lumbago, tiredness, and morning stiffness lasting two hours. She had had recurrent shoulder and neck pains. Examination showed thoracic scoliosis with lumbar lordosis and pronounced tenderness of girdle musculature. The erythrocyte sedimentation rate (ESR) was 66 mm in 1 h. Prednisolone 10 mg daily relieved her rheumatism completely. The ESR fell to 28 mm in 1 h. A raised serum alkaline phosphatase concentration (see table) led to hepatic investigations, which showed sharp fluctuations in serum enzymes and a titre of antimitochondrial antibody (AMA) rising to 1/1280. She remains well three years later on 10 mg of prednisolone daily.

(2) A 61-year-old woman developed mild diabetes mellitus when aged 51. Pruritis and a raised alkaline phosphatase level were noted. Two years later investigation of haematuria revealed a stag-horn calculus, hypertension, and hepatosplenomegaly. Next year her diabetes required insulin. She also had urinary symptoms, hypochondrial pains, and lost weight. The ESR was 115 mm in 1 h and the alkaline phosphatase concentration had risen. A pyonephrotic kidney was removed and her diabetes remitted. When aged 56 she developed pain in the buttocks and shoulders. Shoulder movements were limited. She had splenomegaly and xanthelasma. PMR was suspected. She responded promptly to prednisolone 10 mg daily. Needle biopsy of the liver some months later showed a multilobular cirrhosis, with heavy infiltration of the portal tracts with chronic inflammatory cells, and an absence of bile thrombi. Absence of bile pigment in the cells was consistent with corticosteroid therapy.[3] Her ESR always exceeded 45 mm in 1 h and the serum alkaline phosphatase was over 800 IU/l. The serum gamma-glutamyl transpeptidase concentration was 650 IU/l and the bilirubin was raised for the first time at 35 μmol/l (2·0 mg/100 ml). The prednisolone was tailed off without relapse of her PMR. She remained emaciated and pigmented and died from cerebellar haemorrhage.

(3) A 70-year-old woman had taken amylobarbitone for eight years. She lived in an arm chair and drank two pints (1·14 l) of milk daily. Her daughters noted depression, weight loss of four stone (25·4 kg) in two years, and increasing spinal curvature. She complained of hip and shoulder pains and stiffness lasting all day but worse in the mornings. She was thin, pigmented, had pronounced thoracic kyphosis, much pain on movement, and a waddling gait. Shoulder movements were limited and the hip muscles were tender. The ESR was 110 mm in 1 h. Her symptoms responded partially to indomethacin. On prednisolone 10 mg daily she became ambulant and could dress. Steatorrhoea and osteomalacia were found. Her blood vitamin D concentration was less than 0·8 μg/l (lower limit of normal 3·5 μg/l), and serum calcium was 1·9 mmol/l (7·6 mg/100 ml) (albumin 32 g/l). Vitamin D and calcium were added to her diet and she began to eat and maintain weight. Four weeks later she died at home.

Comment

PMR is common and underdiagnosed.[4] It is a clinical entity but not a true disease. Neoplasia and rheumatoid arthritis must be excluded. Painful, disabling, and demoralising, it responds well to corticosteroids. Temporal arteritis, which may occur in 5-50% of cases, was not found in our series. None of our patients took drugs known to induce PBC. Although PMR and PBC could coexist by chance, we believe they were related in our patients. That so few patients with both conditions have been described is surprising.* Perhaps the wider availability of mitochondrial antibody assay, although not invariably positive,[5] might lead to the detection of more cases.

[1] Knorring, J, and Wasastjerna, C, *Scandinavian Journal of Rheumatology*, 1976, **5**, 197.
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[3] Kosolcharoen, P, and Magnin, G E, *Journal of Rheumatology*, 1976, **3**, 50.
[4] Coomes, E N, Ellis, R M, and Kay, A G, *Rheumatology and Rehabilitation*, 1976, **15**, 270.
[5] Doniach, D, and Walker, G J, *Gut*, 1974, **15**, 664.

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*Since the completion of this report Dr T J Hamblin, of the Department of Immunology, Royal Victoria Hospital, Bournemouth, has informed us of his study of 146 patients with antimitochondrial antibodies. In nine of these patients polymyalgia rheumatica had been diagnosed, and a further 26 had vague "rheumaticky" symptoms, for which diagnosis was uncertain. This series will be reported in detail later.

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Serum concentrations and titres in three cases of polymyalgia rheumatica at time of diagnosis

Case No	Bilirubin	Alkaline phosphatase (IU/l)	5-Nucleotidase (IU/l)	Gamma-glutamyl transpeptidase (IU/l)	Aspartate transminase (IU/l)	Antimitochondrial antibody titre (IU/l)	Smooth muscle antibody titre	IgM (g/l)
1	Normal	296	140	435	28	1/640	Negative	3·15
2	Normal	600	Not measured	Not measured	90	Negative (twice)	Not measured	0·5
3	Raised	1260	100	244	77	1/2500	Positive (+ + +)	5·6