the aneurysm no intimal tear could be seen as it was very friable. The aneurysmal sac was excised. Arterial continuity was restored by using a graft. Pulsations were good and no significant bleeding occurred from the graft or the anastomosis. The post operative course was uneventful and he returned to work. His hoarseness of voice improved but not the Horner's syndrome.

The usual presentation of these lesions is a symptomless pulsatile cervical mass; neurological symptoms may be the presenting features. Rapid expansion can result in dysphagia and respiratory obstruction. The hoarseness of voice in this case was probably due to compression of the larynx by the aneurysm as he had no vocal cord palsy. Horner's syndrome was due to cervical sympathetic chain involvement.

In India it is a common practice among the rural population to have manipulation of neck with oil massage for relief of neck pain, by a barber. The manipulation consists of sudden jerky and forcible flexion, extensions and rotatory movements. Hyperextension and rotation movements of neck can pull the internal carotid artery tightly against the lateral mass of either the C1 or C2 vertebra as the carotid is firmly rooted in the carotid canal at the base of the skull. Subsequent stretching or compression of the artery against the bony prominence can result in intimal tears and subsequent pseudoaneurysm formation. This mechanism was probably responsible for aneurysm formation in this case. Cases of cervical manipulation presenting with signs and symptoms of an acute internuclear opthalmooplegia and brainstem dysfunction have been reported.

Unusual idiosyncratic reactions to carbamazepine

Sir: Two patients developed unusual idiosyncratic reactions to carbamazepine. H.T. aged 59 years had a past history of alcohol abuse and chronic pancreatitis. His medication included atenolol 100 mg/daily, isosorbide dinitrate 10 mg/t.d.s. and Gaviscon (a combination of alginic acid, magnesium trisilicate, aluminium hydroxide and sodium bicarbonate). He developed blackouts associated with vomiting and was prescribed carbamazepine 200 mg/b.d. After receiving carbamazepine for 2 days he slept for 16 hours and on recovery exhibited bizarre, uncontrollable movements of both hands and arms, throwing any objects he grasped. The movements lasted 12 hours and no further carbamazepine was given.

J.T. aged 15 years had episodes of loss of consciousness which were later shown to be psychologically determined. On valproate he developed hypersomnia and was changed to carbamazepine 200 t.d.s.. He developed a rash and the dose was reduced to 100 mg/t.d.s. The rash cleared and he remained well for 5 months before complaining of tiredness and dizziness. A month later he started to walk slowly with small, shuffling, stiff limbed steps. His whole body was rigid and on examination he had repeated attacks of shivering involving most of his body. He recovered completely within a week of stopping his medication.

The shivering dystonia exhibited by J.T. is similar to the dose dependent dystonic movements induced by carbamazepine. Ephemeral hemiballistic movements have not been reported with carbamazepine. Theoretically, interaction with atenolol or isosorbide could have raised the serum concentration of the carbamazepine; alternatively interaction with metal-containing drugs as described by Okada et al may have altered the pharmacodynamics of carbamazepine and explained the unusual nature of the dyskinetic movements.

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References


Autonomic neuropathy in systemic lupus erythematosus

Sir: In describing a case of acute autonomic neuropathy in association with systemic lupus erythematosus (SLE), Hoyle et al remarked that autonomic function had yet to be assessed in any series of patients with this disease. In as much as this remains the position regarding the English language literature, we now report our findings in 14 such cases.

The patients were participants in a wider study of autonomic function in Raynaud's phenomenon (RP), a detailed account of which is in preparation. Five had RP, while the remaining nine were randomly selected from our SLE clinic population to serve as RP case controls. When enrolled, none disclosed symptoms typical of autonomic insufficiency.

Each patient fulfilled established criteria for the diagnosis of SLE. All were black females, their ages ranging from 21 to 50 (median 34) years. None had diabetes, renal failure or clinical signs of cardiovascular disease. All were non-smokers and denied alcohol consumption. In no case was medication being taken that reputedly affects adversely peripheral autonomic or somatic nerve function.

Specifically, 12 patients were
Unusual idiosyncratic reactions to carbamazepine.

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