Letters to the Editor

they may relate to the high frequency of the 11 778 mutant allele in the retinal cell layer and optic nerve, are still unclear.

P MONTAGNA
O PLI ZI
P CORTELLI
V CARELLI
E LUGARESI
Instituto of Neurology
P BARRONI
Institute of Ophthalmology
M PIFICCHI
Clinical Laboratory
S Orosio Hospital,
University of Bologna,
Bologna, Italy

Correspondence to: Dr Pasquale Montagna, Clinica Neurologica Via Ugo Foscolo 7, 40123 Bologna, Italy.


Painless ischaemic lumbosacral plexopathy and aortic dissection

There is only one case report dealing with ischaemic lumbosacral plexopathy without simultaneous signs of limb ischaemia.1 We present a similar case characterised by the total absence of pain.

A 69 year old man suddenly developed a weakness and numbness of the right leg. He also complained of vague abdominal discomfort but no pain was felt in the leg. His risk factors included arterial hypertension and angina pectoris. He was not diabetic. The neurological examination showed pronounced global weakness (grade 0-2/5) of the right lower limb, including the hip flexors and the gluteal muscles. The knee jerk and the ankle jerk were abolished. Sensory disturbances were found below the L2 level on the right side. Light touch and proprioception were clearly impaired whereas sensation for pain and temperature was less diminished. Numbness was well defined and the patient had no appreciation of the cold. Distal pulses were normal and there were no signs of muscle or skin necrosis. His CSF was normal. Aortic angiography showed a right subclavian dissection extending from the origin of the descending thoracic aorta down to the bifurcation (type 3). The angiogram of iliac and femoral arteries showed distal arteries of lower limbs was normal.

An EMG taken two weeks later showed almost no voluntary motor unit potentials in the iliopecto, quadriceps, glutei and adductors muscles. Recruitment of motor units was much reduced in other muscles of the right lower limb. Fibrillations and positive sharp waves were absent. Nerve conduction studies showed absent right sural and superficial peroneal sensory responses and low amplitude right posterior tibial and peroneal motor responses.

The patient had hypotensive treatment in the intensive care unit but dissection extended to the ascending aorta causing haemopericardium and the patient died four weeks later. A necropsy was not performed.

Our patient probably had an acute ischaemic plexopathy. The sudden beginning of the clinical picture "like an apoplexy" was in favour of a vascular etiology. The diagnosis of more distal neuropa thy was not suspected as an angiogram of the lower limb arteries was strictly normal. Ischaemic neuropa thy are divided into those occurring with diseases affecting large arteries and those occurring with disorders that affect small arteries. Acute ischaemia of peripheral nerves generally results from occlusion of a main proximal limb artery or from occlusion of many distal arteries. The rarity of such an event can be explained by the relative resistance of peripheral nerves to ischaemia. It results from their slight metabolic needs (oxygen requirement of human nerve = 0-3 ml/100 g) and from their blood supply, which provides a generous blood flow (43 ml/100 g/min).2 The nutrient arteries that supply peripheral nerves enter the epineurium and form a collateral vascular network of interfacicular arteries along the length of the nerve, so that it is difficult to infarct a peripheral nerve. The intensity of nerve damage varies with the number of nutrient vessels, the availability of the collateral circulation, age, sex and size, and par ticular duration of arterial occlusion.

The different fibres that constitute peripheral nerves seem to be altered selectively by ischaemia. In some studies, anaesthesia seems to affect the small fibres before the large ones and Schwann cells seem to be more resistant to axonol than axons. Nevertheless, opposite data are found in other studies, which suggest a greater vulnerability of large fibres.

Clinical features of ischaemic neuropathy are generally stereotyped.3 Sensory deficits are more frequent than motor deficits. In particular, patients may have long standing pain and neurological examination usually shows impairment of all sensory modalities or pattern of sensory loss that spares the large fibres. There are no signs of muscle necrosis. An EMG usually shows abnormalities consistent with axonal damage. Conduction blocks are unusual.

Such ischaemic lumbosacral plexopathies have been reported after aortobifemoral graft and aortofemoral bypass graft by resection of nutrient arteries, after use of an intra-aortic balloon, and after vasospasm caused by an injection of drugs into an infec tious gluteal artery. Only one case of painful ischaemic lumbosacral plexopathy due to an aortic dissection has been reported.1

Anatomically, the lumbosacralplexus is supplied by five lumbar arteries, which originate from the abdominal aorta, the deep circumflex iliac artery, a branch of the external iliac artery, and the ilioobular and gluteal branches of the internal iliac artery.4 In our patient, ischaemia of the lumbosacralplexus could be due to interruption of blood flow through the lumbar segmental arteries and branches of the iliac arteries secondary to the dissection of the aortic artery walls and of the most proximal iliac artery walls just below the aortic bifurcation.

The clinical picture in our patient was also characterised by the absence of pain whereas causalgia like burning is generally common in acute and chronic ischaemic neuropathy.

Absence of pain has rarely been reported in ischaemic plexopathies. Clinical and pathological studies of nerves in painful neuropa thyies have been limited but the occurrence of pain is usually related to a selective damage of unmyelinated and myelinated small fibres. In the same way, painless neuropa thyies seem to be related to a selective damage of large fibres, as in our patient. On the other hand, a selective loss of fibres has not been confirmed by other studies, in which the ratio between large and small fibres was not modified.5

V LEFEBVRE
J LEDUC
P H CHOTEAU
Service de clinique medicale, CH Sismondi, Lille, France

Correspondence to: Dr V Lefebvre, Service de Cllinique Medicale, CH Sismondi, Boulevard de Belfort 59044, Lille Cedex, France.


Electroconvulsive therapy for the physical signs of Parkinson's disease without depressive disorder

Faber and Trimble1 have reviewed the reports on electroconvulsive therapy (ECT) in Parkinson's disease. We conducted a prospective open trial to evaluate the short term benefit of ECT on the physical signs of Parkinson's disease without major depressive disorder. This was a repeated measures time series design where all patients received a single baseline assessment before and after two assessments after ECT, and were their own control group. Four sessions of ECT were given unilaterally to the non-dominant hemisphere over eight days. Approval for the study was given by the ethics committee, Royal Hobart Hospital and all patients gave written informed consent.

Fifteen patients (12 men) participated. Five failed to comply with all assessments and were therefore eliminated from the analyses. The remaining population (seven men and three women) had an average age of 64-8 years. The time since initial diagnosis of Parkinson's disease ranged from two months to 16 years. Most were severely affected...