they may relate to the high frequency of the 11 778 mutant allele in the retinal cell layer and optic nerve,⁵ are still unclear.

P MONTAGNA
G PLAZZI
P CORTELLI
V CARELLI
E LUGARESI
Institute of Neurology
P BARBONI
Institute of Ophthalmology
M FIOCCCHI
Clinical Laboratory,
S Orsola Hospital,
University of Bologna,
Bologna, Italy

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

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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 a 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. Neurological examination showed a 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. No bladder dysfunction was noted and defecation was normal. Distal pulses were normal and there were no signs of muscle or skin necrosis. His CSF was normal. Aortic angiography showed a thoracoabdominal dissection extending from the origin of the descending thoracic aorta down to the bifurcation (type 3). The angiogram of iliac and femoral arteries and of distal arteries of lower limbs was normal.

An EMG taken two weeks later showed almost no voluntary motor unit potentials in the iliopsoas, quadriceps, glutei and thigh 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 aetiology. The diagnosis of more distal neuropathies was not suspected as an angiogram of the arteries of the lower limbs was strictly normal. Ischaemic neuropathies are divided into those occurring with diseases affecting large arteries and those occurring with diseases, such as diabetes, 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 rareness of such neuropathies 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 rich vascularisation, 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 inter-fascicular 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, and the importance and particularly duration of arterial occlusion.

The different fibres that constitute peripheral nerves seem to be altered selectively by ischaemia. In some studies, anoxia seems to affect the small fibres before the large ones and Schwann cells seem to be more resistant to anoxia 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 develop deep burning 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 inferior gluteal artery. Only one case of painful ischaemic lumbosacral plexopathy due to an aortic dissection has been reported.¹

Anatomically, the lumbosacral plexus 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 iliofemoral and gluteal branches of the internal iliac artery.⁴ In our patient, ischaemia of the lumbosacral plexus 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 neuropathies.

Absence of pain has rarely been reported in ischaemic plexopathies. Clinical and pathological studies of nerves in painful neuropathies 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 neuropathies 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 LLEDUC
P H CHOTEAU
Service de clinique médicale,
CH Saint Vincent,
Lille, France

Correspondence to: Dr V Lefebvre, Service de Clinique Médicale, CH St Vincent, Boulevard de Belfort 59044, Lille Cedex, France

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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 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 and a minority were mildly affected. Two patients were not receiving medication; the others were taking antiparkinsonian medication that was continued at the same dose throughout the study. Psychiatric assessment before the first treatment excluded major depressive disorder.

Neurological assessments were performed two weeks before the first and 24 hours and two weeks after the last therapy. These were scales or parts thereof, including unified Parkinson's disease rating scale (subscales for the evaluation of akinesia, tremor, and rigidity),² North Western University disability scale (subscales for the evaluation of walking, dressing, hygiene, eating, and speech),³ and Hoehn and Yahr staging system (the whole scale was used in assessing the stage of the disease).⁴

Repeated measures analyses of variance with the rejection criterion set at the 0.05 probability level were used to assess the differences between each assessment period for each subscale. Significant differences between the assessment before ECT and the two assessments after ECT were present for akinesia ($F(2,9) = 11.377$, $P = 0.0022$), tremor ($F(2,9) = 9.947$, $P = 0.0030$), rigidity ($F(2,9) = 13.941$, $P = 0.0055$), feeding ($F(2,9) = 6.789$, $P = 0.0206$), speech ($F(2,9) = 5.516$, $P = 0.0215$), and the

Hoehn and Yahr score ($F(2,9) = 7.744$, $P = 0.0065$).

Significant improvements in akinesia, tremor, rigidity, and feeding were found when the pretreatment was compared with the assessment 24 hours after ECT. These improvements were sustained, and there were also significant improvements in speech and the Hoehn and Yahr staging, when the pretreatment assessment was compared with the assessment two weeks after ECT. This pattern of improvement has not been previously reported. Walking, dressing, hygiene, and eating did not significantly improve.

Psychiatric monitoring throughout and formal assessment at 24 hours and two weeks after ECT found no serious side effects. Four patients developed transient confusion after ECT, which was not sufficient to cause withdrawal from the study. One patient developed dyskinesia, which resolved with reduction of medication.

We concluded that four ECT treatments unilaterally to the non-dominant hemisphere over eight days was useful in the management of Parkinson's disease, at least in the short term.

We are unable to report the long term effects of ECT on Parkinson's disease as our research team disbanded. Some patients are known, however, to have main-

tained improvement for at least 16 weeks. The medical literature contains reports of beneficial effects lasting from several weeks to years.⁵ Given the favourable results reported here, studies with longer follow up periods are indicated.

SAXBY PRIDMORE
POH TECK YEO
MOHAMED IQBAL PASHA
*Departments of Psychiatry and Neurology,
Royal Hobart Hospital,
Hobart, Tasmania, Australia*

Correspondence to: Professor S Pridmore, Department of Psychiatry, Royal Hobart Hospital, GPO Box 1061L, Hobart, Tasmania 7001, Australia.

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