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

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Painless ischaemic lumbosacralplexopathy and aortic dissection

There is only one case report dealing with ischaemic lumbosacralplexopathy without simultaneous signs of limb ischaemia. 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. On the 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. The motor dysfunction was noted and deactivation was normal. Distal pulses were normal and there were no signs of muscle or skin necrosis. His CSF was normal. Aortic angiography showed a large atheromatous plaque at the bifurcation extending from the origin of the descending thoracic aorta down to the bifurcation (type 3). The angiogram of iliac and femoral arteries revealed distal arteries of lower limbs was normal.

An EMG taken two weeks later showed almost no voluntary motor unit potentials in the iliopsoas, quadriceps, glutaei 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 aorta causing haemopericardium and the patient died four weeks later. A necropsy was not performed.

Our patient probably had an acute ischaemicplexopathy. The sudden beginning of the clinical picture "like an apoplexy" was in favour of a vascular etiology. The diagnosis of more distal neuropa-thies was not suspected as an angiogram of the lower limbs was strictly normal. Ischaemic neuropa-thies are divided into those occurring with disease affecting large arteries and those occurring with disease affecting small arteries, 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 rarerness of such conditions cannot 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 the reserve of nutrient vessels, which provides a generous blood flow (43 ml/100 g/min). The nutrient arteries that supply peripheral nerves enter the epineurium and form a collateral vascular network of interfascicular 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, the importance of the muscle and particularly duration of arterial occlusion.

The different fibres that constitute peripheral nerves seem to be altered selectively by ischaemia. In some studies, axons, 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 vuln-erability of large fibres.

Clinical features of ischaemic neuropathy are generally stereotyped. Sensory deficits are more frequent than motor deficits. In particular, the most common pain burning 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 may be diagnostic, but large abnormalities consistent with axonal damage. Conduction blocks are unusual.

Such ischaemic lumbo-sacralplexopathies have been reported after aortobi-femoral 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 infe-rior gluteal artery. Only one case of painful ischaemic lumbo-sacralplexopathy due to an aortic dissection has been reported.1

Anatomically, the lumbo-sacralplexus is supplied by five lumbar arteries, which orig-inate from the abdominal aorta, the deep circumflex iliac artery, a branch of the external iliac artery, and the iliolumbar and gluteal branches of the internal iliac artery.2 In our patient, the importance of the lumbo-sacralplexus 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 bifurca-tion.

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 neuropa-thies.

Absence of pain has rarely been reported in ischaemicplexopathies. Clinical and patholog-ical studies of nerves in painful neuropa-thies 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 modulated.3

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