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 lumbosacral plexopathy and aortic dissection

There is only one case report dealing with ischaemic lumbosacral plexopathy 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. 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. The realisation of sensory 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 pseudoaneurysm which extended from the origin of the descending thoracic aorta down to the bifurcation (type 3). The angiogram of iliac and femoral arteries showed that 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 adductor 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 aortic bifurcation 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 lower limbs was strictly normal. Ischaemic neuropathies are divided into those occurring with diseases affecting large arteries and those occurring without 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 events can be explained by the relative resistance of peripheral nerves to ischaemia. It results from their slight metabolic needs (oxygen requirement of human nerve = 0.3-0.5 ml/100 g) and their capacity for autoregulation, 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, and the importance and particular duration of arterial occlusion.

The different fibres that constitute peripheral nerves seem to be affected selectively by ischaemia. In some studies, those, which seem 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. Sensory deficits are more frequent than motor deficits. In particular, the intercostal segments do not have the typical 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 shows nerve abnormalities consistent with axonal damage. Conduction blocks are unusual.

Such ischaemic lumbosacral plexopathies have been reported after aortobifemoral graft and saphenofemoral 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. 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 glutal branches of the internal iliac artery. In our patient, ischemia 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.
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