SHORT REPORT

Intermittent claudication due to ischaemia of the lumbosacral plexus

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Abstract

The distinct clinical syndrome of exercise induced ischaemia of the lumbosacral plexus is not a widely known cause for intermittent claudication. Eight patients with the mentioned syndrome were investigated clinically, neurophysiologically, and with imaging techniques. The clinical examination showed a typical exercise induced sequence of symptoms: pain, paraesthesia, and sensory and motor deficits. The underlying vascular conditions were high grade stenoses or occlusions of the arteries supplying the lumbosacral plexus. Spinal stenosis could be excluded in all cases. Five patients received successful interventional radiological therapy. The syndrome can be diagnosed clinically and successful therapy is possible by interventional radiology.

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Single or bilateral leg pain which forces the patient to stop walking (intermittent claudication) is usually caused either by arterial occlusive disease of the legs or by stenosis of the lumbar spinal canal. In the vascular type any muscular activity of the leg results in pain. The stenosis of the lumbar spinal canal leads to complaints only in an upright position with pain followed by paraesthesia and occasionally motor weakness. There exists a third—widely unknown—type of intermittent claudication which causes leg pain with any muscular effort similar to the vascular type. In this condition the pain is mostly localised in the pelvis and is followed by paraesthesia, weakening of the tendon reflexes, and possibly motor weakness. This special type of intermittent claudication is usually associated with stenosis of pelvic arteries including the internal iliac arteries. Thus, an exercise induced ischaemia of the lumbar plexus was assumed to be causative.¹

Methods

PATIENTS

Twenty three men (age range 44–71) were investigated between 1983 and 1998. Their case histories and clinical findings indicated exercise induced ischaemia of the lumbar

plexus. The reasons for excluding 15 patients from this study were incomplete investigations (insufficient spinal imaging in eight patients, lack of or inadequate pelvic angiography in six patients), and deficient documentation of the clinical findings (three patients). Eight patients who had an arteriography of the pelvic arteries and imaging of the lumbar spine were included in this study, one of the patients was previously described in Stoehr¹

The neurological examination was performed not only during inactivity but also after provocation of the symptoms by climbing stairs or bicycle ergometry. The goal was to assess the appearance of sensory and motor deficits during exercise. In some patients additional neurophysiological tests were performed (motor and sensory nerve conduction studies in seven cases, somatosensory evoked potentials in four cases, motor evoked potentials in three cases, EMG in two cases). All patients had several risk factors for arteriosclerosis: nicotine misuse (n=8), arterial hypertension (n=6), hyperlipidaemia (n=6), diabetes mellitus (n=4), obesity (n=4).

Results

After a walking distance of 12–2000 m five patients experienced pain in the gluteal region as an initial symptom. This was followed by paraesthesia, sensory loss, and/or weakness in one or both legs. The sensory loss was not clearly restricted to specific dermatomes. The localisation was rather diffuse and showed an ascending involvement from distal to proximal—similar to the symptoms that can be seen in stenosis of the lumbar spinal canal. When exercise was continued all patients showed increasing weakness of the muscles of leg and foot, some lost their tendon reflexes. In two patients sensory loss preceded the pain, and one patient only showed progressive sensory and motor deficits without pain. The symptoms are summarised in table 1.

The neurophysiological tests had low diagnostic value. In the nerve conduction studies three patients had mild symmetric distal polyneuropathy (1, 2, and 7), Patients 3, 5, 6, and 8 showed normal findings. In three patients (3, 6, and 7) lumbar motor evoked potentials of the abductor hallucis muscle were performed and showed no abnormalities. Immediate repeat retesting after exercise

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Table 1 Symptoms and signs during walking

Patient, age	First symptom	Second symptom	Third symptom	Other
1, 68	Hypaesthesia right leg	Pain right buttock descending to leg	Motor deficits right leg	After exercise weakening of the achilles tendon reflex and patellar tendon reflex
2, 59	Pain left buttock and upper leg	Hypaesthesia left upper leg	Motor deficits left leg	
3, 57	Pain left buttock and upper leg	Ascending hypaesthesia from foot to buttock	Paresis left leg and hip	
4, 50	Bilateral pain buttocks and groin, ascending pain from right foot	Hypaesthesia right leg and left foot	Pareses of the flexor and extensor muscles of the ankle	
5, 44	Pain buttock both sides, descending pain of the legs (right more than left)	Ascending hypaesthesia of the legs (right more than left)	Pareses of the flexor and extensor muscles of the right ankle	
6, 44	Hypaesthesia of the legs (right more than left)	Pain of the feet (right more than left)	Pareses of the flexor and extensor muscles of the ankle (right more than left)	
7, 71	Pain lower abdomen, descending to buttocks and right leg	Hypaesthesia of both feet (left more than right)	Pareses of the flexor and extensor muscles of the ankle (left more than right)	No pain
8, 47	Hypaesthesia L4-S1 left	Paresis of the flexor and extensor muscles of the ankle	Loss of the left achilles tendon reflex after exercise	

Table 2 Radiological findings and interventional therapy

Patient	Angiography	Interventional therapy
1	Occlusion right internal iliac artery, high grade stenosis left internal iliac artery	NA
2	Occlusion of both internal iliac arteries, aneurysm of the abdominal aorta	NA
3	High grade stenoses of the left common and internal iliac artery	PTA in two sessions with good clinical results
4	Occlusion of the right internal iliac artery, high grade stenosis right internal iliac artery; C/A aortobifemoral Y-bypass surgery	Multiple surgical reconstructions of the bypass without significant clinical improvement
5	High grade stenosis of the distal abdominal aorta and both proximal common iliac arteries (right more than left)	Stent implantation distal abdominal aorta and the right common iliac artery combined with PTA of the left common iliac artery with good clinical results
6	No perfusion of both internal iliac arteries; high grade stenoses of both common iliac arteries; collaterals over the median sacral artery	PTA of both common iliac arteries with good results, both internal arteries show perfusion again and no further collaterals are found
7	High grade stenoses of both internal iliac arteries; aneurysm of the abdominal aorta	PTA of both common iliac arteries with good results
8	High grade stenoses of both internal iliac arteries; Collaterals over the median sacral artery	PTA of the right common and internal iliac arteries with good clinical results

NA=not assessed; PTA=percutaneous transluminal angioplasty.

showed desynchronisation of the compound muscle action potential in two patients (6 and 7), pointing to temporal dispersion.

The findings of lumbar imaging were unremarkable, in all patients a spinal stenosis could be excluded. The pelvic arteriography had the highest diagnostic validity (table 2). Six of eight patients had bilateral stenoses of the internal iliac arteries. One patient had a severe stenosis of the distal abdominal aorta combined with both sided stenosis of the common iliac artery. One patient had an ipsilateral (to the affected leg) combined stenosis of the internal and common iliac artery. Five patients were treated with interventional radiological therapy of the underlying stenosis with percutaneous transluminal angioplasty, in one patient two overlapping Palmaz stents (Johnson and Johnson, Haan, Germany) were also implanted. Immediately after interventional therapy three of five patients had no more complaints. The other two had a remarkably increased walking distance until the onset of symptoms.

Discussion

The blood supply of the lumbar and sacral plexus usually derives from branches of the internal iliac artery (iliolumbar artery, superior and inferior gluteal artery, lateral sacral artery), and the deep iliac circumflex artery.² Acute ischaemic lesions of the lumbosacral plexus are caused by high grade stenoses or occlusion of the iliac arteries or of the distal abdominal aorta. The internal iliac artery plays the

predominant part. However, the most frequent cause of such acute ischaemic lesions of the lumbosacral plexus is surgery of the aortic bifurcation and the pelvic arteries^{3,4} or radiation therapy.⁵ Finally intra-arterial injections of cytostatic agents into the iliac arteries⁶ or accidental intra-arterial injections of vasotoxic agents into the gluteal arteries⁷ may result in persistent ischaemic plexopathy. Distinct from those persisting plexopathies with acute onset there is only an intermittent ischaemic plexopathy during walking with relapsing pain and sensomotoric deficits in the patients described.

PATHOGENESIS OF ACTIVITY DEPENDENT ISCHAEMIC PLEXOPATHY

Reduced perfusion within the area of the internal iliac artery can result in temporary ischaemic lesion of the lumbosacral plexus—appearing only during muscular activity of the legs. The neurophysiological finding of temporal dispersion of lumbar motor evoked potentials after exertion proves the involvement of the peripheral nerve and excludes ischaemia of the lower spinal cord or conus medullaris which additionally seems unlikely regarding the accompanying pain.

Peripheral nerves on the one hand have a high tolerance to ischaemia due to the double blood supply.⁸ On the other hand the peripheral nerve has a significantly increased energy metabolism during activity⁹ and a low capability of autoregulation of the blood supply.¹⁰ Therefore, it must be assumed that during inactivity the perfusion of the plexus is still suf-

ficient. During activity of leg muscles—supplied by branches of the external iliac arteries—however, a steal phenomenon must be supposed that privileges the leg muscles over the pelvic organs. Thus, localised pelvic pain results, followed by paraesthesia and sensomotoric deficits in the area of the lumbosacral plexus. After a rest of a few minutes the symptoms resolve completely.

CLINICAL DIAGNOSIS

The clinical diagnosis of this type of intermittent claudication due to exercise induced ischaemia of the lumbosacral plexus is based mainly on two specific features:

Firstly, as in the more frequent type of intermittent claudication due to arterial occlusive disease of the legs the symptoms appear in correlation with the strength of muscle activity. In early stages of the disease complaints only occur during walking uphill or riding a bicycle. This allows a distinction from the intermittent claudication due to spinal stenosis, where symptoms predominantly appear during walking downhill. Patients with spinal stenosis can ride a bicycle for a long distance without complaints as a result of the kyphosis of the lumbar spine with widening of the lumbar canal.

Secondly, in addition to pain, progressive sensomotoric deficits in the area of the lumbosacral plexus occur during exertion. This cannot be seen in patients with peripheral arterial occlusive disease. Moreover, the localisation of the pain in the buttock differs from this condition.

It should be noted that the neurological examination of the inactive patient usually discloses no abnormality. However, exercise can provoke the neurological symptoms in a typical sequence: pain, paraesthesia, sensory and motor deficits, and loss of tendon reflexes.

TREATMENT

First line therapy is the treatment of the underlying vascular stenoses. Because surgical reconstruction of the pelvic arteries is limited, radiological interventional therapy is preferred in

most cases. Such a therapy can only be successful if treatable stenoses within the blood supply of the lumbosacral plexus are found. Thus an exact angiographic demonstration of the pelvic arteries and their collaterals is necessary. This requires a selective catheterisation of the internal iliac artery. As patients with such a condition usually have several arteriosclerotic risk factors, besides the interventional approach the control of the risk factors is essential. This is especially important as microangiopathic changes may play an additional pathogenetic part.

Conclusion

Walking induced ischaemia of the lumbosacral plexus can lead to intermittent claudication. Quality of life is severely reduced. As in some patients the underlying vascular pathology can be successfully treated, early clinical diagnosis is essential. The knowledge of this syndrome is necessary to prevent misdiagnosis and futile attempts of treatment.

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