Lumbosacral plexus lesions: correlation of clinical signs and computed tomography

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SUMMARY Neurological signs and computed tomographic morphology were compared in 60 patients. The primary neurological deficit was most commonly located in the sacral ($n = 31$) or lumbar plexus ($n = 23$) and was most commonly caused by a neoplasm ($n = 40$). In 78% of the patients it correlated with the lesions detected by computed tomography (CT). CT reliably demonstrates extraspinal mass lesions, but only moderately well predicts functional signs.

Ten years ago there existed no reliable method by which to image the lumbosacral plexus. The introduction of CT has afforded anatomical demonstration of both bony structures of the spine and neighbouring soft tissues.¹⁻⁴ Aside from its vast use in radicular pathology, CT has also been helpful in detecting some of the causes of lumbosacral plexopathy.⁵⁻⁹ Since treatment depends very much on the aetiology of a lesion, CT also has therapeutic implications in lumbosacral plexopathy.

The aim of the present study was to compare systematically the clinical signs found by thorough neurological examination with the CT appearance. Based on normal neural topography, we specifically were interested in knowing whether nerve lesions can be suspected from morphological changes. We also wanted to study the role of CT in evaluating lumbopelvic plexopathy as directly, as noninvasively and as economically as possible.

Material and methods

Sixty patients with suspected lumbopelvic plexopathy were included in the study: 30 of them were analysed retrospectively. They presented with both a lumbopelvic mass lesion on CT and pain or other neurological symptoms. All had a neurological examination that preceded or followed CT by 15 days on average (extreme intervals 0 to 115 days), without any surgical intervention in between. In the prospective series of 30 patients, either lumbopelvic CT or neurological examination were requested first because of pain of

unknown origin or suspected lumbosacral plexopathy; the second test was either indicated by the disease or was performed for this protocol after informed consent had been obtained. It was performed within 0 to 22 days (average 6 days). In all 60 patients the interval between the two examinations was 11 days on average.

The clinical neurological examination was performed by a neurologist, and the patients were all scanned on a third generation CT unit (Siemens Somatom SF). Technical factors included: 5 s scan time, 125 kV, 8 mm slice thickness, and 8 to 16 mm intersection distance. The upper GI tract was marked by an oral contrast agent. For better visualisation of the rectosigmoid, contrast enema was added in selected cases. Also, in order to differentiate vessels and lymph nodes and to characterise tissue, an intravenous bolus of 60 ml of water-soluble contrast medium was often used.

Based on normal neuroanatomy (fig 1) and on the lesions detected by CT from the level of the third lumbar vertebra down to the pelvic floor, those nerves which were potentially damaged were noted. Primary and additional clinical and radiographic findings and their aetiology were then correlated.

Results

In both the retrospective and the prospective subgroup three types of clinical problems gave rise to further investigation: (a) the search for a possible neoplasm or the staging of a known neoplasm ($n = 28/60$, 47%), (b) a neurological deficit ($n = 29/60$, 48%), and (c) the consequences of trauma ($n = 3/60$, 5%).

In 55 patients at least one pathological clinical sign was found by the neurologist (table 1). The primary neurological lesion most commonly involved the sacral plexus and its nerves ($n = 31/55$, fig 2) or the lumbar plexus and its nerves ($n = 23/55$, fig 3). In 47

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Fig 1 Lumbo-sacral neural anatomy: anteroposterior view and corresponding cross-sections at five important levels.

Figure legends:

1 sympathetic trunk, 2 first lumbar nerve, 3 second lumbar nerve, 4 genitofemoral nerve, 5 lateral cutaneous nerve of thigh, 6 a) iliohypogastric nerve, b) ilioinguinal nerve, 7 obturator nerve, 8 femoral nerve, 9 lumbosacral trunk, 10 sciatic nerve, 11 pudendal nerve, 12 inferior gluteal nerve (leaving the pelvis through the infrapiriform portion of the greater sciatic foramen; the superior gluteal nerve exits above it through the suprapiriform portion)

L liver K kidney P piriform muscle
shaded area: dark = bone, clear = psoas muscle

Table 1 Correlation of the main finding detected by neurological examination and by CT

Neurology		Computed tomography			
		lumbar pl.	sacral pl.	cocc. pl.	negative
lumbar plexus	23 (13 + 10)	17 (13 + 4)	2 (0 + 2)		4 (0 + 4)
sacral plexus	31 (17 + 14)		29 (17 + 12*)		2 (0 + 2)
cocc. plexus	1 (0 + 1)			1 (0 + 1)	
negative	5 (0 + 5)	2 (0 + 2)	3 (0 + 3)		
all	60 (30 + 30)	19 (13 + 6)	34 (17 + 17*)	1 (0 + 1)	6 (0 + 6)

The type of the main lesion correlated in 47 out of 60 patients (78%) (i.e. in 30/30 = 100% of the retrospective and in 17/30 = 57% of the prospective subgroup).

*1 case of non-Hodgkin's lymphoma: different side of the main lesion.

Abbreviations: pl. = plexus, cocc. = coccygeal.

In brackets: first number = retrospective, second number = prospective subgroup.

patients (78%) the principal clinical lesion correlated with the distribution of morphological changes detected by CT, although in one patient with non-Hodgkin's lymphoma the neurological loss was located on the side of the smaller mass. Twice, the main morphological lesion covered the area of the sacral plexus, but the clinical loss was lumbar plexopathy. CT was negative in five patients, in one with paraesthetic meralgia who also had bladder carcinoma, in two with quadriceps paresis (once CT was performed to rule out a haematoma with probable sequelae of poliomyelitis), and in three with questionable neurological indications for CT: a pelvic fracture, a lumboradicular compression and a symmetric peripheral neuropathy accompanying

carcinoma of the breast. In five patients of the prospective subgroup the neurological deficit expected by CT could not be confirmed by clinical examination (CT false positive); those were one each with metastases of seminoma and prostatic carcinoma, two with pelvic mass lesions due to Hodgkin's disease (fig 4), and one with a fracture of the right pubic bone that had been missed by the referring physician (fig 5). In all five cases, computed tomographic lesions, although in appropriate location, had minimal extent or just abutted the nerve that was potentially damaged.

On neurological examination, 84 individual regional lesions were identified in the 60 patients (table 2), whereas by CT 99 different lesions of plex-

Fig 2 Bilateral lesions of the sacral plexus in a 62 year old man with prostatic carcinoma. Multiple metastases in the sacral bone and the presacral soft tissues. This was the only case with urinary and fecal incontinence. He had bilateral deficits of all sacral nerves and corresponding mass lesions by computed tomography: (a) presacral soft tissue mass extending into the expanded right anterior sacral foramen that follows the course of the second (2) and third (3) sacral nerves, (b) scan performed after intravenous contrast injection 8 mm below (a). At this level, bone destruction and mass involve mainly the left side of S3 (3).

Fig 3 This 52 year old woman had a haematoma in the psoas muscle caused by anticoagulation (arrows). Neurological examination showed a left-sided lesion of the lumbar plexus from L2 to L4, including the femoral nerve and the lateral cutaneous nerve of the thigh. (a) At the level of the fourth lumbar vertebral body, the haematoma engulfs the extraspinal segments of the second (2) and third lumbar nerves (3) and the lateral cutaneous nerve of the thigh (c). It has older and more recent components, as demonstrated by areas of lower and higher density. (b) At the level of S2 sedimentation is seen within the haematoma. In contrast to the normal right side, the compartment of the left femoral nerve (f), that is located in front and between the psoas and the iliac muscle, can no longer be differentiated.

uses or nerves could be expected. Clinically, the sacral plexus and its nerves were involved most often ($n = 36/84$); lesions of the lumbar plexus, the femoral nerve or the corresponding lumbar nerves were nearly as frequent ($n = 29/84$), but lesions of the pudendal and coccygeal plexuses or the sympathetic trunk were rather rare, and were responsible for the principal

problem in only one patient.

Sixty one out of these 84 regional clinical lesions were expected by CT; 23 of them were not explained by the distribution of the signs of CT, mainly coccygeal plexopathies (six out of eight missed). By contrast, based on morphological changes detected by CT, 38 other neurological lesions could be

Fig 4 Negative clinical examination with positive CT: Hodgkin's disease of stage IIIB had been known for 8 years in this 60 year old man. CT, performed because of jaundice, showed several mass lesions (arrows) but no neurological deficit was detected. (a) Iliac mass in front of the iliopsoas muscle and of the iliac crest with extension both along the lateral cutaneous nerve of the thigh (c) and the ilioinguinal nerve (i). Multiple small subcutaneous nodes on the left side are located close to the gluteus medius muscle; they are not in contact to any major nerve. (b) Internal iliac lymphadenopathy on the left side topographically relates both to the lumbosacral trunk (t) and to the first (1) and second (2) sacral nerves.

Fig 5 *Negative clinical examination with pathological CT: 82 year old woman with pain and a fracture of the right pelvic ring (arrow) not detected by the referring physician. Injury to the obturator nerve (○ marks the location where it is expected) was considered possible from the bone and soft tissue changes shown by CT although there was only a minimal peripheral contact with the nerve. Except for the paresis caused by pain, the neurological examination showed no pathological signs.*

expected which were not confirmed by clinical examination. Except for the coccygeal plexus, all locations were equally common among these false positive findings.

Bilateral neurological deficits were found in three patients. CT demonstrated bilateral mass lesions in these three as well as two other patients.

Six patients had disturbed urination; four with rectal carcinoma, one with prostatic carcinoma, and one with chondrosarcoma. Four of them had urinary incontinence, and two had urinary retention. Only one had bilateral sacral plexopathy (and radiculo-

pathy), and he also had fecal incontinence (fig 2). In the other five patients, the sacral lesion was unilateral by both clinical and radiographic examination. Malignant neoplasia was the aetiology of the neurological syndrome in 73% (40/55). Haematoma, degenerative disease and trauma were next in order of frequency (table 3). Among haematomas, iatrogenic aetiology ranked higher than spontaneous bleeding. Neoplastic lesions were most often caused by local and regional extension of visceral primary tumours, followed by non-visceral primary tumours of pelvic origin, lymphoreticular neoplasms, and by haematogenous metastases. Individual lesions of benign aetiology were most often seen in the lumbar region, those of malignant origin, in sacral distribution. Pudendal or coccygeal plexopathy and lesions of the sympathetic trunk, rather rare in our case material, were always neoplastic. Also, neoplasms, most often of pelvic origin, more frequently caused multiple neurological lesions than non-neoplastic diseases. Bilateral losses were due to a malignant schwannoma, a prostatic carcinoma, and myonecrosis.

Discussion

Lumbar and sciatic pain is one of the most frequent syndromes in western medicine and is most commonly caused by degenerative spinal disease affecting lumbosacral nerve roots, such as discopathy or bony narrowing of the spinal canal.¹⁰ A typical history and a lumbovertebral syndrome with monoradicular signs of irritation or loss on neurological examination justify the diagnosis of nerve root compression. Radiography of the lumbar spine may show corresponding lesions. Further investigations, such as myelography, spinal CT, and possibly magnetic resonance imaging,

Table 2 *Correlation of individual signs detected by neurological examination and by CT. 122 neurological and/or radiological lesions in 60 patients.*

Neurological lesion	Lesion on CT in anatomical relation to					CT neg.
	lumbar pl.	sacral pl.	pudend. pl.	cocc. pl.	symp. tr.	
lumbar pl. 29 (15 + 14)	20 (13 + 7)					9 (2 + 7)
sacral pl. 36 (20 + 16)		31 (19 + 12)				5 (1 + 4)
pudend. pl. 9 (5 + 4)			7 (5 + 2)			2 (0 + 2)
cocc. pl. 8 (3 + 5)				2 (1 + 1)		6 (2 + 4)
symp. tr. 2 (1 + 1)					1 (0 + 1)	1 (1 + 0)
negative 38 (13 + 25)	10 (4 + 6)	8 (1 + 7)	9 (4 + 5)		11 (4 + 7)	
122 (57 + 65)	30 (17 + 13)	39 (20 + 19)	16 (9 + 7)	2 (1 + 1)	12 (4 + 8)	23 (6 + 17)

Abbreviations: pl. = plexus, pudend. = pudendal, symp. tr. = sympathetic trunk.
In brackets: first number = retrospective, second number = prospective subgroup.

Table 3 Aetiology of the plexus lesions

	Main per pat.	Singular lesions (more than one per patient)				
		lumbar pl.	sacral pl.	pudend. pl.	cocc. pl.	symp. tr.
Non-neoplastic						
—Total subgroups:	15 (6 + 9)	9 (3 + 6)	7 (4 + 3)			
—haematoma*	7 (5 + 2)	4 (3 + 1)	3 (2 + 1)			
—pelvic fracture	2 (0 + 2)	1 (0 + 1)	1 (0 + 1)			
—bullet injury	1 (0 + 1)	1 (0 + 1)				
—myonecrosis	1 (1 + 0)		2 (2 + 0)			
—degenerative	4 (0 + 4)	3 (0 + 3)	1 (0 + 1)			
Neoplastic						
—Total subgroups:	40 (24 + 16)	20 (12 + 8)	29 (16 + 13)	9 (5 + 4)	8 (3 + 5)	2 (1 + 1)
—local neoplasm and lymph node metastases:						
visceral	16 (10 + 6)	8 (5 + 3)	14 (7 + 7)	6 (2 + 4)	5 (0 + 5)	
nonvisceral	12 (8 + 4)	4 (2 + 2)	10 (7 + 3)	3 (3 + 0)	3 (3 + 0)	2 (1 + 1)
—haemat. metast.	4 (1 + 3)	3 (1 + 2)	1 (0 + 1)			
—lymphoreticular	8 (5 + 3)	5 (4 + 1)	4 (2 + 2)			
All	55 (30 + 25)	29 (15 + 14)	36 (20 + 16)	9 (5 + 4)	8 (3 + 5)	2 (1 + 1)

*localisation of haematomas:

—lumbar plexus: iliopsoas muscle (2 × due to anticoagulation, 1 × due to catheterisation of the femoral artery, 1 × postoperatively after hip joint prothesis).

—sciatic lesion: gluteal muscles (2 × due to anticoagulation, 1 × idiopathic haemorrhagic diathesis).

In brackets: first number = retrospective, second number = prospective subgroup.

are chosen according to the potential therapeutic implications (table 4). However, when local signs of spinal disease are absent in spite of leg weakness, sensory loss and reflex asymmetry, when sweating is disturbed, or when the cause of an expected radicular lesion cannot be found radiographically, peripheral

plexopathy becomes more likely.^{11 12} Since the lumbosacral plexus and its major nerves lie deep in the retroperitoneum, palpation is rather limited in detecting and localising their pathology, even in the presence of a mass lesion. Thorough neurological examination, however, by demonstrating involvement of several segments or of the sympathetic trunk, can generally differentiate plexopathy and peripheral nerve lesions from radicular disorders. Moreover, electromyography may be helpful in localising the lesion. At this point, in spite of a fairly precise topographical localisation of the disturbance, the underlying pathological process often remains unknown. Unless clinical signs in combination with laboratory findings point to the aetiology, whole body CT is indicated. This method differs from spinal CT in slice thickness, intersection distance, section plane, field of view, and gastrointestinal preparation.¹ Guided by the neurological signs, CT can reliably differentiate between plexopathies caused by mass lesions and entities without any space occupation. Negative CT points to peripheral, often diabetic neuropathy, or, with an appropriate history, to traumatic or radiation-induced plexopathy. In our case material, however, CT commonly showed a mass lesion, most often a visceral or non-visceral pelvic neoplasm.^{7 9 13-16} Less often, lymphoreticular systemic disease, haematogenous metastases, and non-neoplastic masses, such as iatrogenic and spontaneous haematomas, are responsible for the plexus lesion.^{3 5 9 13 17}

Table 4 Diagnostic decision tree for lumbosacral plexus lesions

The situation is slightly different for patients with known lumbopelvic or metastatising neoplasm and

backache, whether neurological signs are present or not. CT is often indicated for staging, for tumour characterisation, or for treatment follow up. Even when there is no clinical evidence of tumour activity, the high incidence of lumbosacral plexopathy in our and other authors' experience suggests that CT should be used early. In the majority of advanced neoplasms operative verification cannot be obtained. Therefore, we were primarily interested in correlating CT morphology, topographic anatomy of peripheral nerves, and neurological signs.

Analogous to the brachial plexus^{6,18} CT is highly sensitive for the mass lesion causing the main lumbosacral neurological lesion. It was true positive in 49 of 55 patients, and all false negative cases had non-neoplastic or radicular disease. However, the centre of the tumour is not necessarily identical with the position of the nerve that is most severely affected. Additionally, all nerves potentially involved according to the topographic extent will not be affected clinically. The most critical point appears to be whether the nerve can escape into loose tissue at the periphery of a mass lesion or will be compromised soon by fixed osseous, muscular, or connective tissue structures. However, these nerves are generally not identified by CT. Another important factor is the nature of the tumour, whether it grows purely expansively, or whether it infiltrates into the neighbourhood and thus, the nerve. Therefore, predicting a neurological lesion from macromorphology, as demonstrated by CT, is not accurate. A functional nerve lesion is more likely present when the mass lesion obliterates anatomic landmarks around the pathways of the nerve (fig 3) than when it abuts it marginally or displaces it (fig 5).

On the other hand, performing CT requires precise knowledge of the three-dimensional course of the nerves clinically affected.¹⁻⁴ Discrete mass lesions must specifically be looked for. CT, even when negative, is sometimes important in differential diagnosis: in the injured patient, the negative study points to a direct nerve trauma and eliminates compression by a haematoma; in an irradiated tumour patient, it favours radiation-induced neuropathy and makes tumour relapse unlikely. In reality, all practical situations are not that typical, and may combine several aetiologies. For example, in carcinoma of the rectum a presacral soft tissue mass after abdominoperineal rectal amputation may be a postoperative or radiation-induced scar, an inflammatory complication of treatment, or may reflect residual or relapsing tumour. Although morphology helps differentiate typical cases, CT-guided fine needle aspiration biopsy or observation of the biological behaviour in a follow up study are often the only diagnostic alternatives to operative exploration. In the future,

whether magnetic resonance imaging will be able to differentiate benign and malignant tissue in this situation remains to be answered.

In conclusion, thorough clinical examination and functional localisation are paramount in the diagnosis of lumbopelvic plexopathy (table 4). They avoid unnecessary, expensive investigations and help select an individualised diagnostic pathway. CT reliably localises extraspinal mass lesions that are most often of neoplastic origin. The specific diagnosis depends on history,^{13,14} the presence or absence of a mass lesion, laboratory findings, and biopsy. Although there is a fairly good correlation between clinical neurological signs and mass lesions on CT in determining the principal nerve structure involved, the distribution of macromorphological lesions does not reliably predict the presence and severity of clinical signs. Aside from anatomical location, additional factors such as the rigidity of the surrounding tissue also influence the appearance of a nerve lesion.

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