Pelvic radiculopathies, lumbosacral plexopathies, and neuropathies in oncologic disease: a multidisciplinary approach to a diagnostic challenge

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Abstract

The purpose of this article is to familiarize the reader with the anatomy of the major pelvic nerves and the clinical features of associated lumbosacral plexopathies. To demonstrate this we illustrate several cases of malignant lumbosacral plexopathy on computed tomography, magnetic resonance imaging, and positron emission tomography/computed tomography. A new lumbosacral plexopathy in a patient with a prior history of abdominal or pelvic malignancy is usually of malignant etiology. Biopsies may be required to definitively differentiate tumour from posttreatment fibrosis, and in cases of inconclusive sampling or where biopsies are not possible, follow-up imaging may be necessary. In view of the complexity of clinical findings often confounded by a history of prior surgery and/or radiotherapy, a multidisciplinary approach between oncologists, neurologists, and radiologists is often required for what can be a diagnostic challenge.

Keywords: Lumbosacral plexus; pelvis; pelvic malignancy; plexopathy; radiculopathy; neuropathy.

Introduction

The term lumbosacral plexopathy is generally applied to pathology involving the lumbar and sacral nerve roots, the lumbosacral plexus itself, and the proximal peripheral nerves arising off the plexus (Fig. 1).

There are a number of causes of lumbosacral plexopathy; in the context of a history of malignancy it is usually due to disease progression or recurrence. Radiation-induced lumbosacral plexopathy is rarely reported. However, when there has been prior pelvic surgery and/or radiotherapy, differentiation of fibrosis from tumour can be problematic. Often the patients will be initially imaged with computed tomography (CT) and/or magnetic resonance imaging (MRI). An understanding of the neurologic presentation, the tumour histology, and the timing and type of treatment all aid the interpretation of anatomical imaging. If these tests are normal or equivocal, positron emission tomography combined with CT (PET/CT) should be considered, as recurrent disease may well be present in sites of surgical scar tissue (Fig. 2).

There is a paucity of articles in the published literature about the presentation and diagnosis of malignant lumbosacral plexopathy. We therefore illustrate several cases from one institution demonstrating pelvic neurologic anatomy and the imaging features of malignant lumbosacral plexopathies. Several cases underwent multiple imaging modalities, the complementary roles of which are elaborated upon.

Pelvic neurologic anatomy: an overview

In understanding lumbosacral plexopathies, it is important to appreciate the differences between the various levels at which lesions can occur. These are divided into radiculopathies, neuropathies, and plexopathies.

A radiculopathy applies to pathology within the neural foramina or spinal canal involving the proximal aspect of
the nerve root. There is considerable overlap in the sensory and motor territories supplied by individual nerve roots, with autonomic fibre contribution from the paravertebral autonomic chains occurring distal to the neural foramina. Radiculopathies consequently have no autonomic fibre involvement and tend to exhibit less well-defined sensory and motor symptoms in comparison with neuropathies.

A plexopathy applies to pathology of the plexus itself, the distal nerve roots, and proximal nerve trunks. As with radiculopathies, neurologic findings are similarly poorly defined and cannot be explained by a single nerve or single root lesion. However, as the nerve roots are involved distal to the neural foramina, there is associated autonomic involvement with loss of sweating and changes in skin colour.

A neuropathy applies to pathology of the nerve trunk, and is denoted by a specific nerve distribution with sharper sensory cutoff and specific muscle weakness, which is often more defined than with a more proximal lesion. Autonomic fibre involvement is anticipated.

There is no clear anatomical point that distinguishes the distal nerve roots from the plexus and the plexus from the proximal nerve trunks. However, the more distal the involvement of the plexus, the more dense and less overlapping the sensory and motor features become as more cross-overs of nerve root contributions have occurred, giving the nerve trunks a more specific supply to the various muscles and areas of skin.

The lumbosacral plexus comprises the interconnected lumbar and sacral plexuses. The lumbar plexus is formed from the ventral rami of L1–L4 and lies between the

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**Figure 1** The lumbosacral plexus and its branches.
quadratus lumborum and psoas major muscle, within which it gives rise to the femoral, obturator, ilioinguinal, iliohypogastric, and genitofemoral nerves, and the lateral cutaneous nerve of the thigh (LCNT).

The lumbosacral trunk, which is formed by fibres from the ventral rami of L4 and L5, passes over the sacral ala connecting the lumbar and sacral plexuses. The sacral plexus is located on the anterior surface of piriformis, and comprises the lumbosacral trunk and the ventral rami of S1–S4.

The sacral plexus converges toward the sciatic notch, giving rise to the sciatic nerve, posterior cutaneous nerve of the thigh (PCNT), pudendal nerve, superior and inferior gluteal nerves, and muscular branches to the pelvis and hip (Fig. 1).

**Malignant lumbosacral plexopathy**

In the context of neoplastic disease, a lumbosacral plexopathy is usually due to malignant infiltration. However, it
is important to be aware of the wide variety of disorders that can affect the lumbosacral plexus. This has been reviewed elsewhere[1].

Pain is the predominant feature at presentation of malignant lumbosacral plexopathy, and may be the only initial finding. It is typically unilateral, affecting the lower back, hip, and thigh[4,7,8]. Pain will eventually develop in almost all patients, characteristically worse at night. A majority will also have other symptoms and signs: weakness (74%), paresthesia (68%), and impaired reflexes (81%)[2]. The lag between initial pain and weakness or sensory loss may take weeks or months. Half of patients have tenderness over the sacrum or sciatic notch[6].

Predictably, tumours arising in the pelvis are most frequently implicated in lumbosacral plexopathy[2]. Malignancies within the pelvis are led in incidence by prostate carcinoma. Other pelvic tumours, though of lower incidence, have a greater propensity for lymphovascular invasion and, hence, lumbosacral plexopathy. Colorectal carcinoma is reported to be the most frequent cause of lumbosacral plexopathy[8].

In the case of malignancies arising in the pelvis, radioculopathy and plexopathy can be due to neurologic invasion/compression by the primary tumour or by its metastases in locoregional lymph nodes and nearby soft tissues or bones[9]. Extrapelvic primaries similarly may cause lumbosacral plexopathy through bone or soft-tissue metastases or, alternatively, through meningeal carcinomatosis (Fig. 3), particularly seen in leukemias, lymphoma, melanoma, and lung and breast cancer[10]. Rarely, malignancies can metastasize to the nerves themselves[11,12].

**Radiation therapy and effects in the pelvis**

In the context of a lumbosacral plexopathy following pelvic radiotherapy, disease progression or recurrence is the usual explanation; however an appreciation of the dose and area treated is helpful in ruling out radiation toxicity. As nerves tend to follow blood vessels and lymph node drainage, nerves are likely to be exposed to the lymph node treatment radiation dose.

Much of our knowledge of the tolerance doses of normal tissues is based on work completed in the 1960s and 1970s, with radiation tolerance doses of normal tissues remaining incompletely understood[13]. Consequently, disagreements arise between radiation oncologists as to the explanation for new symptoms and signs arising in a previously irradiated area. Imaging is often called upon to adjudicate in such cases.

Tolerance doses of normal tissues are typically quoted as TD 5/5 and TD 50/5, which are the 5% and 50% probability, respectively, of causing a complication within 5 years of treatment[13]. There are no quoted tolerance values for the lumbosacral plexus or pelvic nerves, but these have been calculated for the brachial plexus and pelvic organs (Table 1).

It is reasonable to extrapolate radiation sensitivity from the brachial plexus to the lumbosacral plexus. The typical pelvic nodal dose of 45 Gy in 1.8–2-Gy fractions is well under the suggested limits of 60 Gy for the whole plexus and 62 Gy for one-third of the plexus. A more contemporary source looking at dose limits for IMRT lists the maximum limit for the brachial plexus higher, at 66 Gy[14]. With the chances of developing a radiation-induced plexopathy under 5% at 5 years, these figures demonstrate why a lumbosacral plexopathy is more likely to be of malignant etiology, as the risk of local tumour recurrence is far greater. Clinically the timing of onset of radiation plexopathy is highly variable, ranging from 3 months to 30 years, with a typical latent interval of at least 12 months and with a peak at 5 years[15–17].

In contradistinction to malignant plexopathy, numbness and paresthesia are the presenting features. Eventual weakness ensues. Of note, the lumbosacral plexopathy is painless in 50% of patients, with pain rarely a presenting symptom or severe in nature[17–19]. Hence pain is the most useful distinguishing symptom between malignant and radiation plexopathy. Furthermore, the progression of radiation lumbosacral plexopathy is usually slower, lasting months or years, with stabilization and even regression known to occur[14,16,20,21].

**Investigations in suspected malignant lumbosacral plexopathy**

CT remains the cornerstone of imaging in malignant disease. However, if malignant lumbosacral plexopathy is clinically suspected, MRI is the imaging modality of choice as it has been shown to be superior to CT[22] with a sensitivity for detecting malignant brachial plexopathy of greater than 80%[23,24].

In suspected lumbosacral plexopathy the lumbar spine and pelvis should be imaged, as up to 40% of patients have epidural tumour extension. Gadolinium-enhanced imaging is considered where meningeal carcinomatosis is suspected. However, leptomeningeal enhancement
following gadolinium in the absence of tumour recurrence has been reported in polyradiculopathy presenting 15 years after radiotherapy[25]. Cerebrospinal fluid analysis is complementary to gadolinium-enhanced MRI for suspected meningeal metastatic disease[26,27].

MRI diagnosis of malignant lumbosacral plexopathy is usually based on a mass in direct contact with the plexus or the expected course of the clinically affected nerve. A minority may show thickening of the plexus or altered signal within the nerve[2,28] (Fig. 4). Oedema occurs in denervated muscle in the subacute phase, resulting in high signal on T2-weighted and short-tau inversion recovery imaging (Fig. 5), which may be reversible if the lumbosacral plexopathy recovers[28,29]. In malignant

**Table 1** Normal tissue tolerance to therapeutic irradiation in cGy by volume of tissue irradiated (modified from Ref.[13])

| Organ          | TD 5/5 volume | TD 50/5 volume | Selected end point                        |
|---------------|---------------|----------------|-------------------------------------------|
|               | 1/3           | 2/3            | Whole                                     |
| Brachial plexus | 6200          | 6100           | 6000 7700 7600 7500                     |
| Bladder       | N/A           | 8000           | 6500 8500 8000                            |
| Small intestine | 5000          | 4000           | 6000 5500 Obstruction, perforation, fistula |
| Rectum        | Volume 100 cm³ | 6000           | Volume 100 cm³ 8000 Severe proctitis, necrosis, fistula stenosis |

Tolerance doses of normal tissues are typically quoted as TD 5/5 and TD 50/5, which are the 5% and 50% probability, respectively, of causing a complication within 5 years of treatment.

N/A, not available.
Lumbosacral plexopathy recovery is not typically anticipated, and fatty atrophy of the denervated muscles ensues, resulting in loss of bulk and increased signal on T1-weighted imaging (Fig. 4) [29,30]. If the results of the MRI are noncontributory, PET/CT is a reasonable next investigation, as it has been shown to identify plexus involvement not seen with anatomical imaging alone [31,32] (Fig. 2). Furthermore, when initial imaging is negative, but the clinical history and examination are suspicious for malignant lumbosacral plexopathy, repeat imaging after an interval may demonstrate tumour not apparent on the initial scan.

In malignant lumbosacral plexopathy, imaging and electromyography (EMG) may reveal bilateral involvement in a significant minority of patients with unilateral symptoms. EMG can be of use in distinguishing radiation from malignant plexopathy [21,33].

Prognosis and treatment

As already outlined, malignant lumbosacral plexopathy is typically more rapidly progressive and debilitating than radiation lumbosacral plexopathy. Patients diagnosed earlier in the course of their malignant lumbosacral plexopathy with lesser neurologic deficits are more likely to respond to treatment. Radiotherapy is often used, but in the setting of a previously irradiated field it is used with caution.

Pain relief with radiotherapy is achieved in approximately 50% of patients [8,15,34,35]. Reversal of the neurologic deficit is realized in only one-third. Pain unresponsive to analgesia and radiotherapy may require nerve blocks, chemical rhizotomy, or chordotomy.

Pelvic functional neuroanatomy in detail

Table 2 summarizes the pelvic anatomy and sensorimotor innervation of the branches of the lumbosacral plexus.

Cauda equina

Malignant disease affecting the cauda equina may be compressive from bone-centric tumour or be due to diffuse or nodular meningeal disease (Fig. 3). Presentation is usually with low back pain. Anaesthesia may be saddle in distribution, with variable lower limb involvement. Leg weakness and sphincter dysfunction may also be present. The level of involvement depends on the extent of disease, and in the case of meningeal metastases, headache and cranial neuropathies should be sought.

Femoral nerve (L2–L4)

The femoral nerve emerges from beneath the lateral border of the psoas muscle and descends anteroinferiorly beneath the fascia of iliacus. It passes under the inguinal ligament lateral to the femoral artery. The saphenous nerve is the sensory continuation of the femoral nerve. Motor supply is to iliacus, sartorius, pectineus, and quadriceps femoris. Sensory innervation is to the skin of the anterior thigh, knee, medial calf, ankle, and foot. Femoral lumbosacral plexopathy presents with pain in the iliac fossa and inguinal region, buckling of the knee, loss of the knee jerk, and sensory and motor impairment as above (Fig. 6).

Obturator nerve (L2–L4)

The obturator nerve descends through the substance of the psoas muscle, emerging from its medial border to extend around the pelvic side wall and exiting through the obturator foramen, where it divides into anterior and posterior branches [36]. Motor supply is to the hip adductors; adductor magnus, longus and brevis, obturator externus, gracilis ± pectineus. Sensory innervation is to a small area of skin on the upper medial thigh. The most frequent presentation is with medial thigh and groin pain [37]. Hip adduction is weak with associated hip instability and sensory loss as above, and loss of the adductor tendon reflex (variably present in normal patients) (Fig. 5).

LCNT (L2–L3)

Emerging from beneath the lateral border of the psoas muscle, the LCNT runs deep to the iliacus fascia exiting the pelvis variably, but most commonly under the lateral aspect of the inguinal ligament. Sensory supply is to the anterolateral aspect of the thigh. LCNT damage results in meralgia paresthetica, which is denoted by varying degrees of paresthesia in the sensory distribution of the LCNT, symptoms often being posture dependent.

Ilioinguinal (L1) and iliohypogastric nerves (T12, L1)

These two nerves emerge from the upper lateral psoas muscle and pass behind the lower pole of the kidney, exiting the abdominal cavity above the iliac crest between
| Nerve                  | Origin       | Pelvic course                                                                 | Motor supply                                                                 | Sensory supply                                                                 | Other clinical features                                                                 |
|-----------------------|--------------|-------------------------------------------------------------------------------|-------------------------------------------------------------------------------|--------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------|
| **Femoral nerve**     | L2–L4        | Deep to iliacus fascia, passes under the inguinal ligament lateral to the femoral artery | Quadriceps femoris; iliacus; pectineus; sartorius                            | Saphenous nerve; medial and intermediate cutaneous nerve of the thigh            | Knee jerk loss; Sensory supply—skin anterior thigh, knee, medial calf, ankle, foot     |
| **Obturator nerve**   | L2–L4        | Crosses the pelvic side wall and passes through the obturator foramen          | Gracilis; adductor magnus, longus, brevis; obturator externus ± pectineus        | Lower Medial thigh                                                              | Selective hip adduction and internal rotation weakness; loss of adductor reflex        |
| **Lateral cutaneous nerve of the thigh (LCNT)** | L2–L3        | Deep to iliacus fascia exiting pelvis under lateral aspect of the inguinal ligament | Anterolateral thigh                                                            |                                                                  |                                                                                         |
| **Ilioinguinal nerve** | L1           | Lateral wall of pelvic brim into the inguinal canal                            | Skin over inguinal ligament and small patch proximal medial thigh; base of penis; upper scrotum; mons pubis and labium majora | Purely sensory                                                                  |                                                                                         |
| **Iliohypogastric**   | T12–L1       | Behind lower pole of kidney on lateral abdominal wall superior to pelvic brim |                                                                  | Purely sensory                                                                  |                                                                                         |
| **Genitofemoral**     | L1–L2        | Descends along the psoas muscle to the inguinal ligament where it divides     | Cremaster: clinically unreliable                                               | Anterior scrotum; mons pubis; labium majora; small patch skin proximal thigh   | Foot “flop”; buttock and posterior thigh pain; loss of ankle jerk                      |
| **Sciatic nerve**     | L4–L5 S1–S3  | Deep to gluteus maximus, passing through the greater sciatic notch below piriformis | Hamstrings; dorsiflexors and plantar flexors of ankle and toes; intrinsic foot muscles | Posterior lateral leg; posterior calf; sole and dorsum of foot                  |                                                                                         |
| **Superior and inferior gluteal nerves** | L5–S2        | Exit pelvis through greater sciatic notch above and below the piriformis muscle | Superior gluteal; gluteus medius, minimus; tensor fasciae latae muscles         | Purely motor; inferior gluteal rarely solely damaged                            |                                                                                         |
| **Posterior cutaneous nerve of the thigh** | S1–S3        | Leaves the pelvic cavity through the greater sciatic notch medial to the sciatic nerve |                                                                  | Posterior thigh; lower posterior buttocks                                       | Purely sensory                                                                           |
| **Pudendal nerve**    | S2–S4        | Exits the pelvis through the greater sciatic notch; traverses the sacrospinous ligament; re-enters pelvis through the lesser sciatic notch; enters the pudendal canal | External anal sphincter; urethral sphincter and erectile vessels               | Lower anal canal and perianal skin; vagina; labia; penis; scrotum                |                                                                                         |
the muscles of the posterior abdominal wall (Fig. 7). The iliohypogastric nerve supplies the skin over the upper buttocock and above the pubis. The ilioinguinal nerve curves anteriorly into the inguinal canal supplying the skin over the inguinal ligament, upper medial thigh, base of penis, upper scrotum, or mons pubis and labium majora.

Genitofemoral nerve (L1, L2)

The genitofemoral nerve exits the psoas muscle on its anterior surface, along which it runs to the inguinal ligament before dividing into the genital and femoral branches. The genital branch runs in the inguinal canal supplying the skin of the scrotum, mons, labia majora, and cremaster muscle. The femoral branch supplies a small area of skin on the anterior thigh. Sensory disturbance in genitofemoral lumbosacral plexopathy is often exacerbated by standing or hip extension. Loss of the cremasteric reflex is not a reliable sign.

Sciatic nerve (L4–S3)

The sciatic nerve exits the pelvis through the greater sciatic foramen, usually inferior to the piriformis muscle. It descends anterior to the gluteus maximus muscle between the ischial tuberosity and greater trochanter. The sciatic trunk supplies the hamstring muscles, including a portion of the adductor magnus. In the distal third of the thigh the sciatic nerve splits into the common peroneal and tibial nerves, which supply motor function to all the muscles below the knee and sensory function to the posterior and lateral calf, dorsum, and sole of the foot. Symptoms of sciatic
Lumbosacral plexopathy depend on the level of injury and the relative involvement of the tibial and peroneal divisions (Fig. 4). Most sciatic nerve lesions are partial, and so need careful distinction from more peripheral lesions. Complete proximal transection will result in paralysis of the hamstrings and muscles below the knee, with sensory loss in the distribution of the common peroneal and tibial nerves.

**Superior (L4–S1) and inferior gluteal (L5–S2) nerves**

The superior and inferior gluteal nerves similarly exit the pelvis through the greater sciatic foramen. While the inferior gluteal nerve is rarely damaged in isolation of the other nerves passing through the sciatic foramen, the superior gluteal nerve may be solely compromised as it is the only nerve to pass superior to the piriformis muscle. The superior gluteal nerve supplies the gluteus medius, gluteus minimus, and tensor fasciae latae muscles. The inferior gluteal nerve supplies the gluteus maximus (Fig. 8).

**Posterior cutaneous nerve of thigh (S1–S3)**

The PCNT leaves the pelvic cavity through the sciatic foramen medial to the sciatic nerve. It then descends anterior to gluteus maximus to the back of the thigh superficial to biceps femoris, piercing the deep fascia as it reaches the popliteal fossa. It supplies the skin of the gluteal region and the back of the thigh. Sciatic neuropathy together with disturbance of gluteal function (gluteal nerves) and/or sensory disturbance over the lower buttock and posterior thigh (PCNT) implies either a lesion at the level of the sciatic foramen or a sacral plexus lesion (Fig. 9).

**Pudendal nerve (S2–S4)**

The pudendal nerve exits the pelvis medial to the PCNT through the sciatic foramen. It traverses the sacrospinous ligament, re-entering the pelvis through the lesser sciatic foramen. The nerve then turns anteriorly, lying adjacent to the levator ani muscle within the pudendal canal (Fig. 10). There are 3 branches: the inferior rectal, the perineal, and the dorsal nerve of the penis or clitoris. Denervation of the pudendal nerve causes sensory disturbance around the anus, perineum, and genitalia. In males there may be erectile dysfunction, as the nerve carries autonomic fibres.

Figure 8 Axial CT image (a) shows metastatic renal cell carcinoma centred on the right acetabulum with extraossseous spread involving the obturator internus and sciatic notch. Axial T1-weighted image (b) shows associated atrophy of the gluteus medius and maximus, consistent with involvement of the sciatic and superior and inferior gluteal nerves.

Figure 9 Axial CT image (a) and axial T1-weighted image (b) of a patient with known melanoma presenting with signs of a sacral plexopathy. Images show diffuse metastatic infiltration of the sacrum with invasion of the left sacral foramina, with compression of the sacral nerve roots (arrows) best appreciated on the MR image.
Conclusion

Pelvic lumbosacral plexopathy in oncology patients, particularly after radiotherapy, can be diagnostically challenging. Patients will have often undergone surgery and received radiotherapy and chemotherapy, all of which can cause lumbosacral plexopathy. Collaboration between radiology, radiation oncology, and neurology is crucial to achieving a timely diagnosis. Sound knowledge of neuroanatomy, and an understanding of clinical symptomatology and the functional changes of denervation on complementary imaging modalities will assist the radiologist in making the correct diagnosis.

Conflict of interest

The authors declare that they have no conflicts of interest.

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