

## Plexopathies

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Tumors may invade the cervical, brachial, and lumbosacral plexus by direct extension from primary tumors in regional organs or by secondary invasion following metastases to regional lymph nodes. Occasionally, tumor tracks along the epineurium surrounding the trunks of the nerve plexuses. In the previously treated cancer patient, the major differential diagnostic consideration is radiation-induced plexopathy. Less frequently, the plexopathy results as a complication of regional (usually intra-arterial) chemotherapy or is due to fibrosis following surgical resection of primary tumor within the region.

Tumor plexopathy is a symptomatic complication in approximately 1 of 100 patients with cancer. In two retrospective reviews from cancer hospitals, based on 12,000 patient visits per year, the prevalence of brachial plexopathy resulting from tumor invasion was 0.43%, and the prevalence of lumbosacral plexopathy was 0.71% (Kori et al., 1981; Jaeckle et al., 1985). The actual prevalence may be higher, as not all patients with plexopathy accompanying terminal malignancy are reported. The best estimates have been obtained from breast cancer patients treated with conventional radiotherapy in whom the prevalence of brachial plexopathy at 5 years was 1.8% to 4.9%; however, this study likely included some patients with radiation-induced plexopathy (discussed below) (Pierce et al., 1992; Powell et al., 1990; Sheldon et al., 1987).

Familiarity with plexus neuroanatomy is necessary to correctly interpret the clinical findings and to distinguish between plexopathies and neurologic

dysfunction due to epidural compression by tumor, neoplastic meningitis, and other disorders of peripheral nerves. In clinical practice, physicians usually encounter patients with brachial or lumbosacral plexopathy. Cervical plexopathy is also discussed as its clinical presentation, tumor histologies, and therapeutic implications differ somewhat, although there is considerable overlap with brachial plexopathy. Finally, one must be able to distinguish between those patients with plexopathies due to radiation or other causes, which is discussed separately.

### CERVICAL PLEXOPATHY

#### Anatomy

The *cervical plexus* is formed from the ventral rami of the four upper cervical nerve roots (C1–C4). Afferent sensory fibers from the skin and soft tissues of the neck travel via the greater and lesser occipital nerves (posterior occiput and postauricular area); the great auricular and transverse cervical nerves (preauricular area, mandibular angle, anterosuperior neck, and submandibular area); and the medial and lateral supraclavicular nerves (inferior anterolateral neck) through the cervical plexus. The cervical plexus supplies motor innervation to the diaphragm and, deep cervical and hyoid muscles and via the spinal accessory nerve to the sternocleidomastoid and trapezius muscles (Truex and Carpenter, 1969).

### Pathophysiology

The cervical plexus is usually invaded by tumor from neighboring soft tissue or bony structures (Fig. 18–1). The neoplasm may invade the plexus directly or indirectly by metastatic spread to regional lymph nodes, ribs, or vertebral bodies. The most commonly associated tumors of the cervical plexus include squamous cell carcinoma of the head and neck, lymphoma, and adenocarcinomas of the lung and breast.

### Clinical Presentation

Patients with cervical plexopathy usually present with pain located in the neck, shoulder, or throat, which is often exacerbated by neck movement, swallowing, and coughing. The pain is often deep, boring, and constant, but may have intermittent sharp components or caustalgic features. On examination, the cervical musculature may be tender to palpation. One should suspect tumor plexopathy from tumor invasion if large palpable tumors or firm anterior or pos-

terior cervical chain or supraclavicular nodes are present. Local sensory loss may be demonstrable, but is often incomplete due to overlap of root zones. Often, one must distinguish postsurgical sensory loss from that due to tumor recurrence. The timing and location of sensory loss are useful points of distinction. Most commonly, surgical dissection transects superficial branches of the greater auricular nerve over the preauricular area and mandibular angle, or the transverse cervical branches, producing numbness in the skin of the upper anterior neck and submandibular area.

Tumor may involve the phrenic nerve producing a paralyzed hemidiaphragm, which can be confirmed by chest radiograph or fluoroscopy. Patients may have dyspnea and paradoxical diaphragmatic excursions. Motor dysfunction of branches to the deep cervical and hyoid muscles are usually asymptomatic. Involvement of the spinal accessory XIth cranial nerve may produce shoulder weakness and instability. Sternocleidomastoid weakness may produce head deviation or tilt. Head and neck tumors and lymphomas



**Figure 18–1.** Cervical plexopathy. Magnetic resonance imaging scan of metastatic adenocarcinoma from submandibular primary tumor to lower left cervical plexus (arrows), producing C4–C5 pain, tingling, hoarseness, and Horner's syndrome.

involving the anterior cervical lymphatic chain may grow inferiorly, producing additional involvement of the upper brachial plexus.

By definition, involvement of the cervical plexus implies a close proximity of the tumor to the cervical spine. If sharp and severe pain occurs with neck movements, or pain is elicited with percussion over the spine, there may be associated involvement of the cervical spine or impending epidural extension.

## BRACHIAL PLEXOPATHY

### Anatomy

The brachial plexus originates from ventral rami of the lower four cervical (C5–C8) and the first thoracic roots and forms three *trunks*: a superior trunk (C5–C6); a medial trunk (C7); and an inferior trunk (C8–T1). These trunks each divide into *dorsal* and *ventral divisions*. The three dorsal divisions join together to form the *posterior cord*, which then splits into (1) the thoracodorsal nerve, to the latissimus dorsi; (2) the subscapular nerve, which innervates the subscapularis; (3) the axillary nerve, which supplies the deltoid; and (4) the radial nerve, which innervates the triceps, brachioradialis, wrist, and finger extensors and provides sensory supply to the posterior arm and forearm. The ventral division forms two cords: the *lateral cord*, which receives contributions from the superior and medial trunks, and the *medial cord*, which is a direct continuation of the inferior trunk. The lateral cord then divides into two branches: one becomes the *musculocutaneous nerve* (C5–C7, motor to biceps, brachialis, coracobrachialis and sensory innervation of the radial forearm); and the other joins a similar branch from the medial cord to form the *median nerve* (C8–T1, forearm flexors and pronators, medial hand flexor, and lumbrical muscles; and sensation to medial palm, first three fingers, and radial one-half of the fourth finger). The remaining portion of the medial cord forms (1) the *medial brachial cutaneous nerve* (T1, sensory to medial arm); (2) the *medial antebrachial cutaneous nerve* (C8–T1, sensory to medial forearm) and; (3) the *ulnar nerve* (C8–T1, ulnar forearm flexor muscles, interossei, and lumbricals; and sensation to the ulnar aspect of hand the hand and fifth finger) (Truex and Carpenter, 1969).

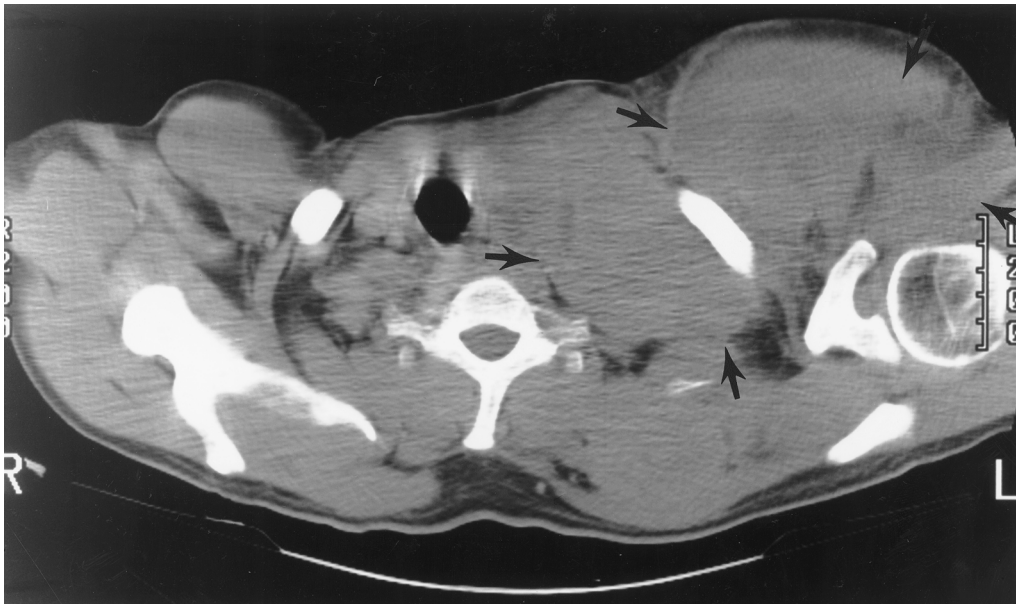
### Pathophysiology

More than two-thirds of tumors involving the brachial plexus originate from the lung or breast and generally spread to the plexus directly from the lung or from regional metastases to the axillary lymph nodes or lung apex (Kori et al., 1981) (Fig. 18–2). Early in the disease process, the tumor often invades the lower plexus, particularly the inferior trunk and medial cord, a pattern that was part of the original description of the superior sulcus syndrome (Pancoast, 1932). Conversely, tumors of the head and neck usually invade the superior trunk or posterior cord from above. Often, the clinical pattern of plexus involvement is patchy, due to irregular involvement of the plexus by growth from the relatively random arrangement of lymph node metastases. Patchy involvement is most commonly identified when formal electrophysiologic investigations are performed.

### Clinical Presentation

In a large series of patients with cancer and brachial plexopathy, pain was the most common presenting symptom (75%), followed by dysesthesias (25%) (Kori et al., 1981). The pain was typically located in the shoulder and axilla and often radiated along the medial aspect of the arm and forearm into the fourth and fifth fingers. Initially, pain was often incorrectly attributed to other potential causes, such as arthritis and fibromyalgia. Pain, reflex changes, and atrophy were concentrated in the lower plexus (C8–T1) distribution in 75% of patients, whereas the remaining 25% of patients had global signs of involvement of the entire (C5–T1) plexus.

When brachial plexopathy is due to tumor, lymphedema is relatively infrequent (15%). It is most often seen in the setting of prior radiotherapy or axillary lymph node dissection. Involvement of the sympathetic trunk or ganglia near the upper thoracic (particularly T1) vertebrae may produce unilateral Horner's syndrome, which has been identified in approximately 23% of patients. When Horner's syndrome is present, concomitant epidural disease can be identified in 32% of patients. In general, the presence of Horner's syndrome, serratus anterior or rhomboid muscle weakness, or pain consistent with vertebral involvement should raise the clinical suspicion of epidural extension.



**Figure 18–2.** Brachial plexopathy. Computed tomography scan of patient with non-Hodgkin's lymphoma showing a huge shoulder and chest wall mass displacing mediastinal structures and invading the left brachial plexus (arrows).

## LUMBOSACRAL PLEXOPATHY

### Anatomy

The lumbosacral plexus forms from the ventral rami of the L1–S2 nerve roots and is anatomically divided into lumbar and sacral portions. The lumbar plexus forms within the iliacus muscle lateral to the L1–L4 vertebrae and then courses posterolaterally, just anterior to the iliac wing.

The *lumbar plexus* consists of *anterior* and *posterior divisions*. The anterior division gives rise to the *iliohypogastric*, *ilioinguinal*, and *genitofemoral nerves* (L1–L2), which carry sensory fibers from the skin of the lower abdomen, upper thigh, and lateral genitalia, and the *obturator nerve* (L2–L4), which provides motor supply to the adductors and gracilis muscles. The posterior division of the lumbar plexus divides into the *iliohypogastric* and *lateral femoral cutaneous nerves* (L2–L3), which provide sensation to the lateral hip and thigh, and to the *femoral nerve* (L2–L3), which carries motor fibers to the psoas, iliacus, sartorius, and quadriceps muscles and provides sensory fibers to the skin of the anterior thigh and medial upper leg.

The *sacral plexus* arises from the ventral rami of S1–S4 and runs lateral to the border of the sacrum, penetrating the sciatic notch. A portion of the lower lumbar plexus (L4–L5) connects to the sacral plexus via the lumbosacral trunk, which runs over the sacral ala to join the upper sacral plexus within the true pelvis. The *anterior division* of the sacral plexus provides motor supply to the gemelli, quadratus femoris, obturator internus, and hamstrings; the remaining components continue as the *tibial nerve* (L4–S3), which provides motor supply to the foot plantar flexors and intrinsic muscles and sensation to the heel and sole. The *posterior division* continues as the *common peroneal nerve* (L4–S2) to the peroneal muscles, the tibialis anterior, extensor digitorum, and extensor hallucis muscles and carries sensory fibers from the lateral leg and dorsal foot and toes. The posterior division also gives rise to the *superior* (L4–S1) and *inferior* (L5–S2) *gluteal nerves*, which supply the gluteus muscles. The *sciatic nerve*, which contains components of the common peroneal and tibial nerves, is part of the posterior division; the *posterior femoral cutaneous nerve* (S1–S3), which follows the course of the sciatic nerve as a separate bundle, carries sensory fibers from skin of the pos-



terior thigh from the buttock to the knee. The *pudendal nerve* (S2–S4), along with many smaller nerve bundles, supplies the pelvic floor and genital musculature, as well as the external anal and urethral sphincters. This nerve also carries afferent sensory fibers from the perineum (Truex and Carpenter, 1969).

### Pathophysiology

Tumors commonly invade the lumbosacral plexus by direct extension from pelvic primary neoplasms and less frequently by secondary extension from metastases to regional pelvic lymph nodes, the sacrum or iliac wings, or lumbar vertebrae (Fig. 18–3). The most commonly reported tumors producing lumbosacral plexopathy include carcinomas of the colon and rectosigmoid, gynecologic malignancies, retroperitoneal sarcomas, and lymphomas (Jaeckle et al., 1985; Pettigrew et al., 1984). Plexopathy is part of the original tumor presentation in approximately 15% of patients. In general, bulky tumors within the pelvis compress and invade the plexus directly. On occasion, tumor tracks along the connective tissue and epineurium of the nerve trunks (Ebner et al., 1990). This tendency to infiltrate along the nerve may explain why some patients with findings of diffuse plexopathy do not have radiographically demonstrable mass le-

sions and why in some circumstances the location of the pelvic tumor seems unrelated to the principal site of neurologic involvement.

### Clinical Presentation

Tumors invading the lumbosacral plexus typically produce clinical syndromes based on their level of involvement. Clinical patterns of plexus involvement include the upper plexus (L1–L4), the lumbosacral trunk (L4–L5), and the lower plexus (S1–S4). Plexopathy is usually unilateral, although bilateral findings are present in 25% of patients. Patients typically present with leg pain, which is often followed weeks to months later by progressive leg numbness and weakness. The pain is typically severe, constant, dull, and aching and may have sharp or cramping superimposed local, referred, or radicular components. Pain is often worse when the patient becomes supine; usually the patient has difficulty finding a comfortable position. Additional pain that worsens with movement or weight bearing generally implies nearby bony invasion. Involvement of the iliopsoas muscle may force the patient to assume a position in bed with the hips and knees flexed, similar to that seen with meningeal inflammation. The pain may worsen following a bowel movement or be exacerbated by a rigorous neuro-



**Figure 18–3.** Lumbosacral plexopathy. Computed tomography scan showing large left pelvic node metastases from melanoma that caused severe pain and weakness in L4–L5 distribution.

logic examination. Pain is so common (98%) that its absence should raise a red flag regarding this diagnosis.

Symptomatic weakness and sensory complaints eventually develop in 60% of patients. In a series of 85 cancer patients with lumbosacral plexopathy and radiographic or surgical evidence of tumor in the region of the plexus (Jaekle et al., 1985), objective leg weakness was identified in 86%, sensory loss in 73%, focal reflex loss in 64%, and ipsilateral leg edema in 47% of patients. Patients typically had tenderness over the sciatic notch, and straight leg raising tests often reproduced their pain. Clinical dysfunction of the lower plexus is most common and is most commonly seen with colorectal tumors. A dysesthetic syndrome ("hot dry foot") has been described in as many as one-third of patients (Dalmau et al., 1989) due to sympathetic plexus involvement within the pelvis. Incontinence and impotence are typically absent unless bilateral plexopathy is present.

Tumors deep in the pelvis, in particular those accompanying cervical carcinomas and tumors of the rectosigmoid region, present with numbness and dysesthesias in the perianal area and perineum. These patients may have a palpable rectal mass, decreased anal sphincter tone, and sensory loss of the perineum from involvement of the lower sacral plexus, located anterior to the piriformis muscle. The sensory symptoms often occur early and are followed later by lower extremity pain and weakness.

#### **DIFFERENTIAL DIAGNOSIS: PLEXOPATHY IN THE CANCER PATIENT**

##### **Radiation Plexopathy**

Because the timing of onset of tumor plexopathy following the original cancer diagnosis overlaps with the typical time frame for radiation plexopathy, the clinician is often confronted with the difficult task of distinguishing these two entities in previously treated patients. Complicating this issue, these conditions may coexist simultaneously in a given patient, which may in part explain why patients whose tumor responds to treatment may not have clinical improvement. The single most valuable diagnostic clinical parameter suggestive of tumor plexopathy is the demonstration of persistent or recurrent tumor in the general region of the plexus (e.g., in the axilla or lung

apex), as determined by clinical examination, magnetic resonance imaging (MRI), and/or computed tomography imaging (CT) (Thyagarajan et al., 1995). Radiation plexopathy is implicated if there is evidence of radiation-induced changes in skin, bone or soft tissues and if myokymic discharges are found on electromyography (EMG), which are unusual findings in tumor plexopathy.

The syndromes of brachial and lumbosacral plexopathy due to irradiation have been reasonably well described (Kori et al., 1981; Pierce et al., 1992; Powell et al., 1990; Sheldon et al., 1987; Basso-Ricci et al., 1980; Bagley et al., 1978; Harper et al., 1989; Killer and Hess, 1990; Mondrup et al., 1990; Thomas et al., 1985; Olsen et al., 1990; Ashenhurst et al., 1977). The incidence was probably higher in patients treated in the 1960s with telecobalt therapy. Recently, a long-term (34 years) follow-up study of 71 breast cancer patients who received a calculated dose of 57 Gy in 16 to 17 fractions over 3 to 4 weeks to the axilla, supraclavicular area, and parasternal lymph nodes, reported that late-stage progressive plexopathy was common. Eleven of the 12 patients alive at the time of follow-up had paralysis of the limb (Johansson et al., 2000).

The frequency of radiation plexopathy in treated patients is approximately 1.8% to 4.9% (Pierce et al., 1992; Powell et al., 1990; Sheldon et al., 1987). Sheldon et al. (1987) reported this complication in 2% of patients with stage III breast cancer treated with 4.5 to 5 Gy to the breast and regional lymphatics at a median follow-up period of 65 months (5 years). In other series, the interval between administration of radiotherapy and the development of plexopathy ranged from 3 months to 14 years, with a median of 1.5 years, and was most frequent at tissue doses greater than 50 Gy (Kori et al., 1981; Killer and Hess, 1990).

Radiation plexopathy probably has a multifactorial etiology. Cavanaugh (1968) studied the effects of radiation and crush injury of rat sciatic nerve at doses between 200 and 2000 R. At doses above 1000 R, there was failure of cellular proliferation and development of pathologic changes in Schwann cells, endoneurial fibroblasts, and vascular and perineural cells. In another physiologic study, severe delayed damage of the anterior and posterior nerve roots was observed following 35 Gy administered to the lumbar region in rodents (Bradley et al., 1977). In humans, radiation plexopathy is presumably caused by tissue

fibrosis with retraction of nerve trunks, direct toxic effects on axons, and radiologic effects involving the vasa nervorum, producing microinfarction of nerve axons (Greenfield and Stark, 1948).

In the clinical setting, several points can help to distinguish radiation-induced from tumor-induced plexopathy (Table 18–1). Radiation plexopathy usually presents with causalgic dysesthesias in the arm, often accompanied by lymphedema. In contrast, only a small percentage of patients with tumor plexopathy will present with dysesthesias, and lymphedema is less frequent. In tumor plexopathy, the presenting feature is often severe unrelenting pain; in contrast, pain usually develops late in the course of radiation plexopathy and is less often the major symptom. These two entities can sometimes be distinguished by the level of plexus involvement: Radiation plexopathy usually affects the upper (77%) or entire (23%) plexus; in tumor plexopathy, the lower plexus (75%) or the entire plexus (25%) appears to be more frequently affected (Kori et al., 1981; Mondrup et al., 1990). However, recent studies have shown that the neurologic level of plexus involvement can be more variable than noticed in these earlier reports (Boyaciyan et al., 1996). Sparing of the lower plexus from radiation damage afforded by intervening bony and soft tissues has been postulated as a reason for the more frequent occurrence of upper plexus involvement with radiation, but seems questionable based on studies of tissue dosimetry and observations of associated radionecrosis of the clavicle or ribs in these patients

(Pierce et al., 1992). Many patients with radiation-induced plexopathy also have skin changes, including telangiectasia, atrophy, and diffuse induration; radiographs may show other associated radiation changes, including typical changes in the apex of the lung, radionecrosis of regional bony structures, and pericardial fibrosis.

The lumbosacral plexus is also subject to radiation-induced injury. The median onset of this complication is approximately 5 years (range, 1 month to 31 years) (Thomas et al., 1985). Another series found an earlier onset of 12 months (range, 1 month to 156 months) (Ashenhurst et al., 1977). The incidence appears to be higher with larger doses of radiation, but has been reported at doses as low as 17 Gy.

The clinical features of radiation plexopathy overlap with tumor plexopathy. Patients with radiation plexopathy usually present with weakness (60%), numbness, or paresthesias (50%) (Thomas et al., 1985). As in radiation brachial plexopathy, pain at presentation is also uncommon (10%) in patients with radiation lumbosacral plexopathy and frequent (98%) in patients with recurrent tumor. Later in the course of lumbosacral plexopathy due to radiation, pain develops in 50% of patients. Weakness is often bilateral in radiation plexopathy in contrast to tumor plexopathy, in which more than 75% of patients primarily have unilateral involvement. The distribution of plexopathy is not as useful a distinguishing point as it is in brachial plexopathy; both conditions most commonly affect the lower (L5–S1) portion. The presence of a rectal mass supports tumor

**Table 18–1.** Differential Diagnosis of Radiation Plexopathy Versus Recurrent Tumor Plexopathy

	<i>Tumor</i>	<i>Radiation</i>
Presentation	Pain	Paresthesias, weakness
Pain	Common, severe	Occasional
Edema	Occasional	Common
Plexus involvement		
Brachial	Usually lower plexus	Upper or whole plexus
Lumbosacral	Lower plexus, unilateral	Commonly bilateral
Horner's syndrome	Common	Infrequent
Local tissue necrosis	Infrequent	Common
Rectal mass lumbosacral plexopathy (LSP)	Common	Unusual
Myokymia (EMG)	Unusual	Common
Nerve enhancement (MRI)	Common	Unusual

recurrence; sphincteric dysfunction is slightly more common with radiation-induced plexopathy. Leg edema occurs with approximately equal frequency in the two conditions. The progression of neurologic dysfunction is generally slower in radiation plexopathy than with tumor recurrence.

Electromyography appears to be helpful in distinguishing between these two entities. Approximately 60% of patients with radiation-induced plexopathy will exhibit myokymia, which is rarely seen in metastatic plexopathy (Roth et al., 1988). Neuroimaging with CT (Moskovic et al., 1992) or with MRI can be of help in distinguishing tumor recurrence from radiation injury. Magnetic resonance imaging appears to be more sensitive than CT in detecting tumor recurrence (Taylor et al., 1997).

The treatment of radiation-induced plexopathy is symptomatic. Most patients have a slow but steady progressive loss of motor function and require supportive therapy.

#### **Additional Causes of Plexopathy in Cancer Patients**

Plexopathy following chemotherapy appears to be associated primarily with regional intra-arterial administration (Kahn et al., 1989). Although rare, plexopathy has been reported as a paraneoplastic disorder and on occasion may be steroid responsive (Lachance et al., 1991). When making a diagnosis, the physician must also consider conditions affecting the plexus that occur in noncancer patients, including aortic aneurysms (Chapman et al., 1964); diabetic amyotrophy (Chokroverty et al., 1977), vasculitis, and other rare conditions (Chad and Bradley, 1987). Idiopathic or postinfectious brachial neuritis can produce symptoms very similar to those of metastatic plexopathy, although the onset is usually more acute and often follows a history of viral infection or immunization. Because patients with metastatic systemic cancer often have more than one site of nervous system involvement, the possibility of concomitant leptomeningeal or epidural metastases must be considered, which can have a similar clinical appearance.

#### **DIAGNOSIS**

The clinical diagnosis of metastatic plexopathy is best confirmed by MRI or CT scanning of the appropriate areas. Magnetic resonance imaging provides the best

anatomic detail and is usually a preferred procedure, when available. MRI has been shown to be more sensitive than CT in the identification of tumor plexopathy (Taylor et al., 1997; Qayyum et al., 2000; Thyagarajan et al., 1995). Diagnosis can be difficult if the scan does not show a mass lesion. In these instances, one must maintain a high clinical suspicion of tumor recurrence and consider repeating the imaging procedures at further neurologic progression. Although CT or MRI scans may identify tumor recurrence, the radiographic appearance can be difficult to interpret. Increased  $T_2$  intensity within nerve trunks with or without enhancement has been identified in patients meeting clinical criteria for radiation plexopathy, findings considered more typical for tumor plexopathy; and fibrotic masses may also mimic local tumor recurrence (Wouter van Es et al., 1997). A careful evaluation by an experienced radiologist may be helpful, as detection of a pattern of  $T_2$  abnormalities and enhancement and an associated mass effect may provide provisional bias toward one of these diagnoses.

The presence of a local or regional tumor recurrence, by clinical examination or imaging, supports the diagnosis of tumor plexopathy. In one study, the absence of a palpable axillary mass was inversely diagnostic as 95% of patients with CT evidence of brachial plexus tumor have a palpable axillary mass (Moskovic et al., 1992). In a recent study, positron emission tomography (PET) was utilized to evaluate the brachial plexopathy in 19 breast carcinoma patients with plexopathy; 14 had abnormal 18-fluorodeoxyglucose uptake within the involved plexus. However, the specificity and sensitivity of PET for tumor plexopathy have not been elucidated (Ahmad et al., 1999).

Electromyography can be useful in delineating the distribution and extent of denervation. It occasionally reveals more extensive findings than would have been predicted clinically. The main clinical usefulness appears to be in distinguishing radiation plexopathy from neoplastic involvement, as stated above, and occasionally in supplementing the radiographic findings when planning treatment fields. Electromyography in experienced hands can be used to differentiate plexopathy from other entities, such as neoplastic meningitis, radiculopathy from epidural tumor, and neuropathy.

#### **TREATMENT**

Treatment of cancer-related plexopathy is largely palliative and symptom directed. Treatment generally in-



volves radiation to the involved field and pain control measures. Consultation with pain management specialists is recommended. Studies have shown that pain is often poorly controlled in patients with plexopathy. Proper pain control often requires multimodality approaches, including a clear understanding of the proper use of opiate analgesics, continuous infusion pumps, local and regional blocks, sympathectomy, rhizotomy, or other specialized procedures that are generally outside the normal experience of radiation and medical oncologists. In appropriate settings, specific chemotherapy for the underlying neoplasm is warranted, and tumor response may be associated with improvement of pain and other symptoms (particularly in responsive tumors such as lymphomas). Occasional relief of chronic pain has been achieved with plexus dissection and neurolysis (Sundaresan and DiGiacinto, 1987). Dysesthesias and causalgia are resistant to therapy, but may respond to transcutaneous nerve stimulation, tricyclic antidepressants, anticonvulsants such as gabapentin, carbamazepine or phenytoin, or regional nerve or sympathetic ganglion blocks. Often, opiate analgesics are required. Lymphedema may be treated with compressive devices and elevation.

Unfortunately, the reported results of treatment are largely disappointing. In brachial plexopathy, radiation therapy to the involved plexus relieved pain in only 46% of cases (Kori et al., 1981). Treatment of tumor in the lumbosacral plexus produced improvement in pain in only 15% of patients reassessed at 1 month from treatment; objective improvement in strength occurred in only 17% and objective reduction in tumor size in only 28% (Jaekle et al., 1985). In another study of 28 patients with carcinomatous lumbosacral plexus neuropathy, radiation produced improvement in 85% but only a 29% objective response rate (Ampil, 1986). The best subjective/objective treatment result was observed with a total dose of at least 30 Gy. In most cases, the duration of response has been brief, and the relatively short survival of patients with tumor plexopathy suggests that its occurrence represents a late-stage complication of malignancy.

Plexopathy patients are particularly prone to painful contractures, compressive neuropathies, pressure ulcerations, respiratory and urinary tract infections, joint subluxations, and deep venous thromboses. Preventative measures, including adequate pain management, initiation of physical rehabilitation, and assessment of home equipment needs may have

a significant impact on preservation of quality of life in these patients.

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