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Radiation-Induced Brachial Plexopathy: MR and Clinical Findings

Brian C. Bowen, Ashok Verma, Alfred H. Brandon, and Jeffery A. Fiedler

Summary: A 54-year-old man had a slowly progressive bilateral brachial plexopathy 17 months after surgery and radiation therapy for a stage IV supraglottic carcinoma. MR imaging at presentation showed a symmetric pattern of parascalene and inter-scalene hyperintense signal on T2-weighted images and after contrast enhancement. Although hyperintense signal has been more often associated with recurrent tumor than with delayed radiation injury or fibrosis, the location and pattern of the signal abnormalities suggested a diagnosis of radiation-induced plexopathy. This diagnosis was confirmed by the relative stability of the neurologic and MR findings 30 months after treatment.

Index terms: Brachial plexus; Iatrogenic disease or disorder; Therapeutic radiology, complications

Delayed radiation injury to the brachial plexus is one of the common causes of nontraumatic brachial plexopathy in previously treated cancer patients (1, 2). While the clinical history and physical findings can help differentiate plexopathy caused by radiation therapy from that caused by tumor infiltration, the two clinical syndromes may overlap (3) and an imaging examination, usually magnetic resonance (MR) imaging, is often requested to clarify the diagnosis (4–6). The utility of MR imaging is in part based on results showing that neck or axillary areas of delayed radiation injury or fibrosis are usually isointense or hypointense relative to muscle on T2-weighted images, thus distinguishing them from tumor infiltration, which is hyperintense (5, 7). Recently, however, Thyagarajan et al (6) reported that patients with plexopathy caused by radiation injury had hyperintense signal in or near the plexus on T2-weighted images as frequently as did patients with tumor infiltration. Almost all of the patients described in the latter study, though, had a history of breast cancer or lymphoma, and there were no cases of previously treated head and neck cancer. We describe a patient who had previously undergone resection of a supraglot-

tic carcinoma and dissection of both sides of the neck in whom bilateral brachial plexopathy developed approximately 9 months after radiation therapy. Although there was hyperintense signal on T2-weighted images and corresponding contrast enhancement on T1-weighted images at the base of the neck, as may occur with recurrent tumor or infection, the location and distribution of the signal abnormalities and lack of a mass effect suggested the final diagnosis of radiation injury.

Case Report

A 54-year-old man with supraglottic carcinoma (T2, N2c, M0; stage IV) underwent a supraglottic laryngectomy with right and left lateral neck dissections. On each side of the neck, one lymph node (level III, internal jugular chain) of 27 was positive for tumor cells. After surgery, the patient completed a 42-day course of fractionated (180 cGy) radiation therapy. Nominally, a total dose of 5940 cGy was delivered to each side of the neck, sparing the spinal cord, and 4500 cGy to the supraclavicular areas.

Approximately 9 months after completing radiation therapy, the patient first noticed progressive numbness of the left hand and paresthesias of both hands. A few months later, he had painless weakness of the left shoulder muscles. A neurologic examination 17 months after radiation therapy revealed a mild asymmetric (left more than right) atrophy and weakness of the supraspinatus, infraspinatus, and deltoid muscles. Touch and pinprick sensations were diminished on the left thumb and index finger and on the lateral aspect of the left forearm. Deep tendon reflexes were absent in the upper extremities and normal in the lower extremities. Clinical myelopathy or Horner syndrome was absent. Complete blood count, sedimentation rate, blood chemistry profile, and urinalysis were all normal. Concentric needle electromyography showed evidence of active denervation and some reinnervation of bilateral deltoid and biceps muscles, and of the left triceps, extensor digitorum communis, flexor carpi ulnaris, and first dorsal interosseus muscles. The left deltoid and triceps showed myokymic repetitive discharges. A follow-up clinical examination 20 months after radiation therapy re-

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vealed mild progression of bilateral neurologic deficits in the C-5 to C-7 distributions. These deficits stabilized and were unchanged at 24 and 30 months after radiation therapy. As in the initial examination, deep tendon reflexes in the upper extremities were absent.

MR imaging of the brachial plexus was done at 17 months (Fig 1A–C) and 30 months (Fig 1D–F) after radiation therapy. Similar findings were observed in both studies. On precontrast T1-weighted images, the anterior and middle scalene muscles showed confluent, intermediate signal intensity (Fig 1A). On T2-weighted images, bilateral, approximately symmetric bands of hyperintense signal (relative to muscle signal in general) were observed along the posterior and lateral margins of the anterior scalene muscles at the base of the neck (Fig 1B). The posterior portion of each band encompassed the region between the anterior and middle scalene muscles traversed by the upper brachial plexus (Fig 1G). On the postcontrast T1-weighted images (Fig 1C and D), there was a band of enhancement along the periphery of the anterior scalene, including the brachial plexus region, which mimicked the hyperintense band seen on the T2-weighted image. There was no adenopathy and no evidence of compression or displacement of adjacent muscles, vessels, or the overlying fat. The enhancing band was contiguous with the anterior scalene muscle to the level of its insertion on the anterior first rib (Fig 1E and F). In the axilla, the brachial plexus and adjacent soft tissues showed no abnormal enhancement. The cervical spinal cord had normal contour and signal intensity, and no advanced degenerative changes of the cervical spine were evident.

Discussion

The C-5 to C-7 roots of the brachial plexus course between the anterior and middle scalene muscles (Fig 1G). The C-5 and C-6 roots unite along the lateral border of the anterior scalene to form the upper trunk. C-7 forms the major portion of the middle trunk. The C-8 and T-1 roots, located posterior to the anterior scalene, form the lower trunk. The brachial plexus exits the neck by passing through the scalene triangle, accompanied by the subclavian artery and ensheathed in a sleeve-like diverticulum of the prevertebral fascia (8).

Injury to the brachial plexus after radiation therapy is associated with three distinct clinical syndromes: classic delayed, progressive radiation injury or radiation fibrosis (9, 10); reversible or transient plexopathy (11); and acute ischemic plexopathy (12). Brachial plexopathy caused by radiation fibrosis is the most common of these syndromes. Its frequency, which has been estimated at 10% to 70% in older studies (3) and 5% to 9% more recently (13), de-

pends on several factors, including total dose (1), fractionation dose, and premorbid state of the irradiated nerves (10, 13). Kori et al (1) found that radiation fibrosis is unlikely to occur with total doses of less than 6000 cGy. Our patient nominally received approximately 6000 cGy to each side of the neck, and symptoms of plexopathy developed 9 months later. Depending on the extent of overlap of the lateral neck and supraclavicular radiation fields, higher doses (up to 7500 cGy) could have been delivered to the lower portion of the neck along/near the field borders (unpublished calculations).

The clinical findings associated with radiation fibrosis often differ from those of plexopathy caused by tumor infiltration. In 50% to 80% of patients with classic radiation injury, plexopathy is relatively painless (1–3, 6). Kori et al (1) and others (9) found that neurologic signs, as in our patient, were predominantly in an upper trunk distribution, involving C-5, C-6, or C-7 roots. Weakness usually involved shoulder abduction and arm flexors. This presentation is in contrast to the typical findings for plexopathy resulting from tumor infiltration, in which symptoms of severe pain, muscle weakness in a C-8 to T-1 (lower plexus) distribution, and frequently Horner syndrome, predominate (1, 2, 9). Some investigators, though, have not found a relation between the distribution of plexopathy and its pathogenesis (2, 6, 13). Electromyographic findings of myokymic discharges in limb muscles, as recorded in our patient, are associated with radiation plexopathy and not tumor infiltration, but are not pathognomonic (2).

A few studies (7, 14) have described the MR findings in cases of fibrosis or scarring in the neck following surgery and radiation treatment for malignant neoplasms, and in these studies no history of brachial plexopathy was given. Glazer et al (7) and others (5) found that fibrosis was isointense to hypointense relative to muscle on T2-weighted images and not masslike, while tumor was hyperintense. Som et al (14), however, noted that vascularized scar in the postoperative neck, with or without radiation, was hyperintense on T2-weighted images, enhanced with contrast material, and mimicked tumor. They concluded that dense fibrous scar with lower signal intensity than tumor on MR images, as described by Glazer et al (7), is relatively rare.

The MR findings in two series of patients with

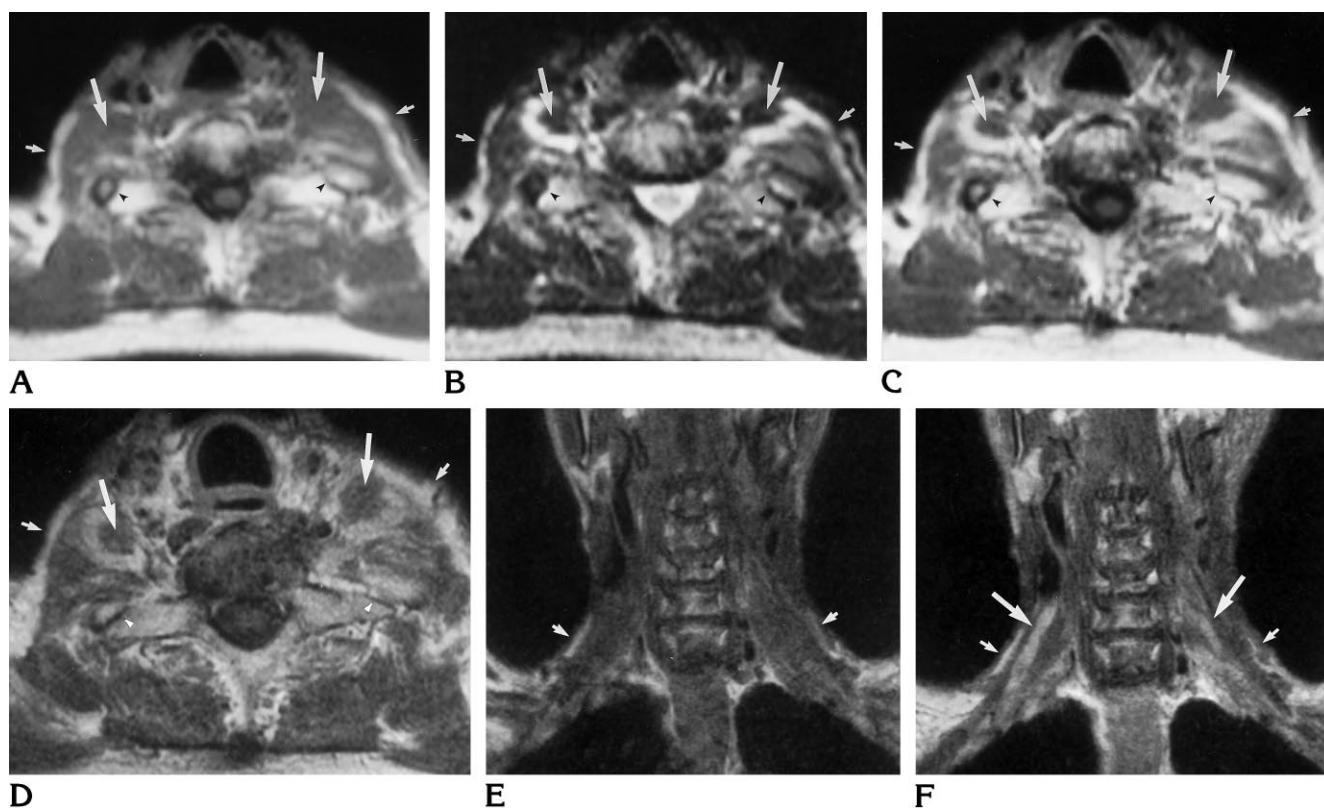


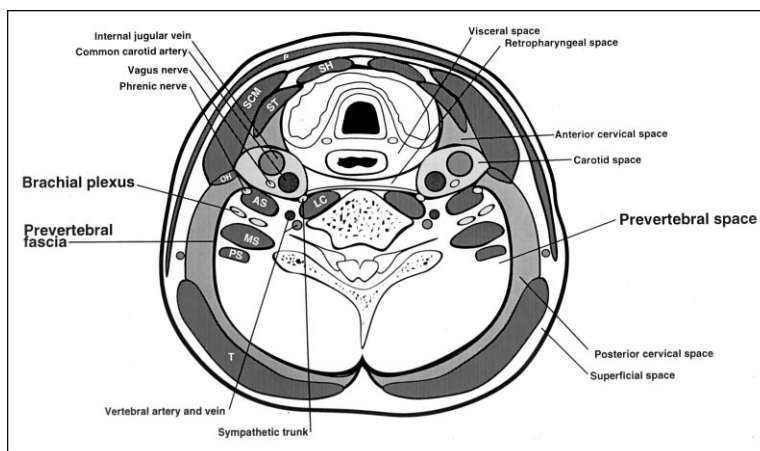
Fig 1. Fifty-four-year-old man with supraglottic carcinoma who underwent bilateral neck dissections. The left internal jugular vein is absent.

A, T1-weighted (800/16/2 [repetition time/echo time/excitations]; matrix, 210×256) and B, T2-weighted (2100/80/2; matrix, 192×256) axial MR images at the C-7 to T-1 level of the brachial plexus 17 months after radiation therapy. In A, the region of the scalene muscles (*large arrows*) shows intermediate, uniform signal intensity with overlying fat (*small arrows*). In B, there is a band of hyperintense signal along the margin of the anterior scalene muscles (*large arrows*). A portion of each first rib is seen (*arrowheads*).

C, A contrast-enhanced T1-weighted MR image at the same level shows a thick rim of enhancement lateral and posterior to the anterior scalene muscles (*large arrows*) in the same distribution as the abnormal signal in B.

D-F, MR images of the brachial plexus 30 months after radiation therapy. Contrast-enhanced T1-weighted axial MR image (900/16/2; matrix, 400×512) at the C-7 to T-1 level (D) shows that enhancement along the lateral and posterior margins of the anterior scalene muscles (*large arrows*) is similar in thickness and extent to that shown in C. The superficial fat (*small arrows*) and portions of the first ribs (*arrowheads*) are again shown. E and F are T1-weighted coronal MR images (900/16/1; matrix, 440×512) through the region of the scalene muscles before (E) and after (F) contrast enhancement. In F, the enhancement along the margins of the anterior scalene muscle appears as linear bands (*large arrows*) deep to the subcutaneous fat (*small arrows*).

G, Axial diagram of anatomy of lower portion of neck, at the level of the thyroid gland, shows the location of the brachial plexus bounded by the prevertebral fascia. Layers (*darker lines*) of the deep cervical fascia ensheath the fascial spaces, indicated on the right, except for the superficial space, which has an outer margin formed by the superficial cervical fascia. Representations of the prevertebral space vary (16). The muscles shown are the platysma (P), sternohyoid (SH), sternothyroid (ST), sternocleidomastoid (SCM), inferior belly of the omohyoid (OH), anterior scalene (AS), middle scalene (MS), posterior scalene (PS), longus colli (LC), and trapezius (T).



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brachial plexopathy and clinically suspected radiation fibrosis or malignant tumor have also been described (5, 6). Most of the patients had a history of breast carcinoma or lymphoma, and the signal abnormalities described were in the axilla. In the series of 25 patients reported by Bilbey et al (5), three patients had postradiation fibrosis of the plexus; and abnormal tissue contiguous with or surrounding the brachial plexus was hypointense on T2-weighted images. Thyagarajan et al (6) found that hyperintense signal on T2-weighted images in or near the brachial plexus, occurred as frequently in patients with delayed radiation injury/fibrosis as in those with tumor infiltration. In the 21 patients in that series who had proved radiation fibrosis, the median interval between radiation therapy (5000 cGy) and the development of plexopathy was 110 months (range, 7 to 570 months). The time intervals for symptom development and imaging follow-up in our patient were within the ranges reported by Thyagarajan and colleagues.

The signal abnormalities seen in our case (Fig 1A–F) most likely resulted from radiation treatment alone or from the combination of radiation and surgical dissection. Lateral neck dissection alone, and consequent vascularized scar (14), could not account for the signal abnormalities along the posterior margin of the anterior scalene muscles, because the scalene group and the brachial plexus are enclosed by the prevertebral fascia (Fig 1G). The dissection, in which the lymph nodes at levels II, III, and IV were removed, did not breach the prevertebral fascia (15). The rimlike pattern around the anterior scalene muscles, with lack of mass effect, resembles a fasciitis; however, the anterior and middle scalene muscles do not have an intervening layer of deep cervical fascia (8, 16). Focal tumor recurrence or infection would be unlikely to produce the observed pattern, although perineural tumor spread without mass effect might.

Radiation is known to cause a connective tissue response ranging from edema and acute inflammation early to chronic inflammation, progressive fibrosis, and neovascularization later (17). These pathologic changes could account for the signal abnormalities along and between the scalene muscles. Whether the lateral neck dissections hastened and contributed to the postradiation changes is conjectural; however, Cheng and Schulz (18) found that ex-

tensive connective tissue fibrosis and associated peripheral nerve injury occur earlier (a few months to a few years compared with many years) and/or at lower radiation doses (5000 to 6000 cGy compared with 8000 to 10 000 cGy or more) when the peripheral nerve is situated in or near tissue subjected to dissection before radiation therapy. Peripheral nerve injury, with associated brachial plexopathy, results from encasement and constriction of the nerve trunk by fibrous tissue and/or disruption of vascular supply to the nerve (9, 10). The combination of radiation and surgical dissection best explains both the MR findings and the onset of plexopathy 9 months after treatment.

In conclusion, radiation fibrosis or tumor infiltration in the postoperative neck may have similar signal characteristics. Consequently, other MR imaging features become important in determining the cause of plexopathy in patients with treated head and neck cancer. The features that suggest radiation-induced plexopathy include bilaterality and approximate symmetry when both sides of the neck have been treated; a curvilinear contour located posterior and lateral to the anterior scalene muscle, coinciding with the distribution of the brachial plexus; the absence of a focal mass or displacement of adjacent structures; and stability of the findings on serial studies. Thus, the morphologic features of the signal abnormalities, rather than the hyperintensity, hypointensity, or enhancement per se, are the key findings that favor a diagnosis of radiation plexopathy over recurrent tumor or infection.

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