

Nerve Transfer for Elbow Flexion in Radiation-Induced Brachial Plexopathy: A Case Report

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Abstract Radiation-induced brachial plexopathy is an uncommon but devastating late complication seen in patients receiving radiation therapy to the chest wall and axilla. Treatment options are unfortunately limited. We report a case of a 59-year-old woman treated with radiation therapy for breast cancer 12 years earlier, who presented with loss of elbow flexion and marked shoulder weakness. Electromyogram and intraoperative stimulation of the musculocutaneous nerve branches were consistent with a proximal motor nerve conduction block. Microsurgical transfer of median and ulnar nerve fascicles to the biceps and brachialis branches of the musculocutaneous nerve, respectively, were performed. The patient recovered MRC grade 4/5 elbow flexion after surgery. The characteristics of this disorder and surgical treatment options are reviewed.

Keywords Nerve transfer · Radiation brachial plexopathy · Fascicular transfer

Introduction

Radiation therapy directed at the chest, axillary region, thoracic outlet, or neck can cause neurotoxicity, known as radiation-induced brachial plexopathy (RIBP) [18, 29–31, 37]. The radiation dose, treatment technique, and concomitant use of chemotherapy all demonstrate association with the development of radiation injury to the brachial plexus. RIBP can be difficult to differentiate clinically from neoplastic

brachial plexopathy, neuritis, and compressive neuropathy [17]. A survey of the literature reveals that various medical therapies, transdermal electric nerve stimulation, dorsal column stimulators, neurolysis, and neurolysis with omentoplasty have been used as treatments for RIBP with limited success [4, 17, 20, 21, 25, 37].

We report a case of RIBP occurring 12 years after external beam radiation for breast cancer. Given the predominant motor complaints, including loss of elbow flexion and evidence of proximal conduction block, the patient appeared to be an excellent candidate for nerve transfer. This patient is presented to discuss treatment options, including microsurgical reconstruction with nerve transfers.

Case Report

A 59-year-old right-hand-dominant woman underwent treatment of T2N1M0 right breast cancer by right modified radical mastectomy, excision of lymph nodes, and combination chemotherapy in 1990. In 1991, she was treated with 48 Gy of external beam radiation divided into 38 treatments over 4 weeks, hyperthermia, and radium bead implants to the right chest wall and axilla. She had hyperbaric treatments for problems with delayed wound healing. In December of 1992, she had a reconstruction with a double pedicle tram flap. Over several years, she progressively noted marked neurological dysfunction in the right upper extremity. She initially sought treatment for neurological dysfunction 3 years earlier. She saw a neurologist, and EMG studies and a PET scan were obtained to rule out a neoplastic process, and a diagnosis of radiation neuritis was made. She complained of constant tingling and numbness in her right arm and the median nerve distribution of the

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right hand and palm. She did not have any active right elbow flexion, had very little movement at the right shoulder, and was frequently dropping things from her right hand. She also had muscle cramping and increased pain in the right arm with activity. She awoke at night with right shoulder, neck, and rib pain. The pain is only occasional and at its worse 3–4/10 and has been manageable without continual pain medication. She noticed swelling in her right arm but not in her hand.

On physical examination, she had no active right elbow flexion or shoulder abduction, adduction, and internal or external rotation. The accessory nerve remained functional with intact shrugging. She had moderate contracture of her shoulder joint with abduction limited to approximately 70–80°. There was surprisingly little atrophy of these muscles. There was mild tenderness to percussion over the scalene muscles on the right side but no clearly palpable scarring or pain induced by mild passive shoulder movement. Hand function was intact bilaterally. Moving and static two-point discrimination was within normal limits in the median and ulnar nerve distribution in the left hand and in the median nerve distribution in the right hand. There was no sensation in the distribution of the lateral antebrachial cutaneous nerve (LABC), the terminal sensory branch of the musculocutaneous nerve. Right upper extremity reflexes were absent. Pressure over the median nerve in the right proximal forearm produced some increased paresthesia in the median nerve distribution of the right hand. There were no other significant findings.

Nerve conduction studies and an electromyogram were performed, which revealed good function in the supra- and infraspinatus muscles but absent responses in the right biceps or deltoid. The lateral antebrachial cutaneous nerve response was absent, and a right axillary response was unobtainable. Electromyogram demonstrated isolated small motor units recordable from the deltoid, biceps, and brachioradialis at rest.

Exploration and neurolysis were recommended to decompress any fibrotic constriction to improve her pain, sensation, and motor weakness. She elected to proceed with surgical management to explore and release the musculocutaneous, axillary, and median nerves and perform a neurolysis. If no function with electrical stimulation is noted, then nerve transfer reconstruction would proceed if the muscles looked healthy, suggesting innervation, or a Steindler flexorplasty if the biceps and brachialis muscles appeared unhealthy and chronically denervated. Although a Steindler flexorplasty is not the most powerful primary muscle transfer for elbow flexion, in the patient with brachial plexus palsy, more favorable and stronger muscles such as the latissimus [6] and pectoralis major may not be available for transfer, as in this patient. In this patient population, a Steindler flexorplasty has demonstrated very

satisfactory outcomes [5, 24]. Preoperatively, she was also noted to have slight but notable contraction of her deltoid muscle suggestive of recovery. Therefore, exploration and reinnervation of the deltoid muscle was not recommended at this time. Upon exploration, the biceps muscle appeared normal, and her right musculocutaneous and median nerves were therefore examined through a longitudinal incision along the brachial sulcus on the medial aspect of the upper arm. All scar tissue was released, and intraoperative nerve stimulation of the biceps branch produced marked supination and elbow flexion, while stimulation of the brachialis branch gave strong elbow flexion. Based on these findings, no reconstructive procedures were performed, and her incision was closed. In the recovery room when the patient was fully awake, however, her elbow flexion palsy persisted.

As proximal conduction block was postulated, the patient was returned to the operating room the same day to proceed with nerve transfer reconstruction with the double fascicular transfer technique using median and ulnar nerve fascicles as donors [22]. Internal neurolysis and fascicular dissection of the median and ulnar nerves was performed at the level of the musculocutaneous nerve motor branches to dissect their individual motor fascicles. The biceps muscle was reinnervated by transferring an expendable flexor digitorum sublimis fascicle from the median nerve to the biceps branch of the musculocutaneous nerve. The brachialis muscle was reinnervated by the transfer of an expendable flexor carpi ulnaris fascicle of the ulnar nerve (Figs. 1 and 2). Satisfactory residual motor function in both the median and ulnar nerves was verified by stimulation of the entire nerve excluding the selected donor fascicles and noting good hand and wrist movement.

The patient remained in an arm sling for 1 week postoperatively and then only for comfort thereafter, as

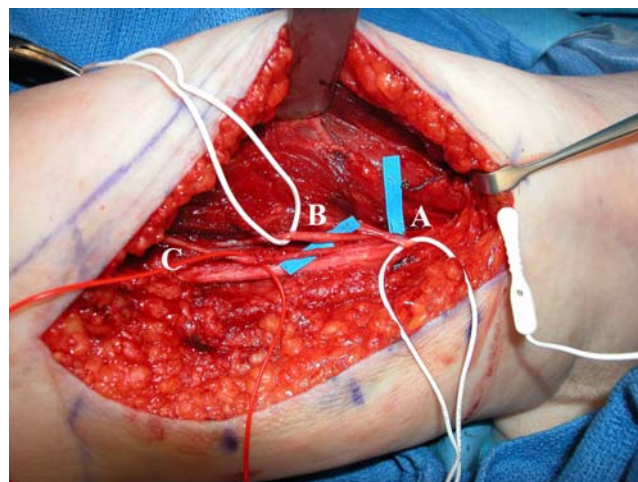


Figure 1 Intraoperative dissection of biceps (A), LABC (B), and brachialis (C) branches of the musculocutaneous nerve.

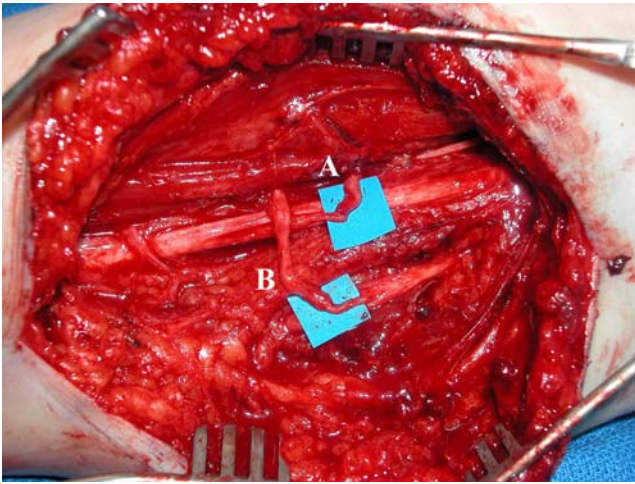


Figure 2 Transfer of FDS fascicle of median nerve to biceps branch (A) and FCU fascicle of ulnar nerve to brachialis branch (B) of the musculocutaneous nerve.

the nerve transfers were completed with no tension throughout the full range of elbow motion. The patient resumed supervised physical therapy immediately to maintain mobility of her shoulder, elbow, and hand. Once she had demonstrated evidence of muscle reinnervation, strengthening and motor reeducation was instituted to facilitate cortical remapping and more spontaneous activation of target muscles. This involves repeated stimulation of donor motor nerve branches and visual and proprioceptive feedback to further develop and reinforce the new motor pathways. At her 6-month postoperative visit, she had evidence of reinnervation of her elbow flexors with detectable voluntary muscle contraction, and at her last follow-up after 2 years, she had recovered MRC grade 4/5 elbow flexion strength. Physical examination of hand function demonstrated intact FDS function to all digits and FCU wrist function, illustrative of the redundancy of innervation to these structures. No functional donor morbidity was noted as she maintained good hand function, sensation, and strength, and she no longer complained of pain. She remained very pleased with the improvement of her extremity function and her overall outcome.

Discussion

Approximately 30% of all patients with cancer receive radiation therapy [14]. Radiation-induced brachial plexopathy is defined as neurologic impairment of a transient or permanent nature as a sequela to radiation therapy. The mechanism is believed to be a combination of localized ischemia and failure of cellular proliferation but is incompletely understood. This results in fibrosis of the neural and perineural soft tissue secondary to microvascular

insufficiency, leading to entrapment of nerve fibers [7, 15, 29, 37–39, 44]. In addition, nerve ischemia may lead to ischemic demyelination or metabolic disruption of axonal electrical properties, thus resulting in conduction block that is commonly observed in RIBP [7, 34–36].

The incidence of radiation-induced brachial plexopathy is 1.2% of women irradiated for breast cancer. This incidence, now significantly declining, is directly related to the use of a three-field technique versus tangential fields alone, a total radiation dose >50 Gy, and the use of adjuvant chemotherapy [31]. The three-field technique includes radiation of the axillary/supraclavicular area as the third field as compared to the standard tangential or two-field technique and signifies a greater dose of irradiation. The incidence of brachial plexopathy was 4.5% among patients receiving chemotherapy, compared with 0.6% when chemotherapy was not used [29, 31]. The reason for this is not completely clear, but it is thought that chemotherapy might enhance the radiation-induced effect on nerve tissue and thereby diminish the latency period of presentation. The dose-response curve of RIBP has been recently characterized by Johansson and colleagues by retrospective analysis of 150 breast cancer patients [14]. They have recommended an increased awareness of the long latency period and wide spectrum of different side effects of treatment because doses believed safe at 5 years may result in serious late side effects beyond the 5-year period with any treatment protocol. They discouraged any tendency toward a return to the use of fewer, larger fractionation schedules for economical reasons in patients with potentially curative disease.

Symptoms of RIBP begin from 6 months to 20 years after radiotherapy (median time 1.5 years) [8]. The main presenting symptoms most commonly include sensory changes, such as numbness, paresthesia, and dysesthesia, edema, and motor weakness of the upper extremity. Only a minority of patients will complain of significant pain, which is usually localized to the shoulder and proximal arm. The patient in this report therefore demonstrated a classic presentation and evolution of symptoms. Muscle paralysis associated with RIBP is thought by most to be an axonal injury with muscle denervation. This report emphasizes that at least a significant component of the nerve injury is a proximal conduction block at the level of the brachial plexus. A conduction block or neurapraxia implies that the axon itself is intact and therefore, the target muscle is essentially normal. The natural history of RIBP is variable, as Pierce et al. reported that 80% of their RIBP patients improved spontaneously, and only 20% suffered from progressive deterioration [31]. However, observations of all other investigators suggest that one third of RIBP patients deteriorate rapidly, while the other two thirds remain stable for years [16, 18].

The main differential diagnosis of RIBP is a neoplastic process. The pattern of neurological involvement can be used to determine the likely etiology of a brachial plexopathy following mastectomy: either tumor recurrence, RIBP, or a radiation-induced neoplasm. Because the exposure to radiation is usually diffuse and the injury global, very proximal or distal signs are less common in RIBP. Very focal presentations are also less common and would suggest a focal process such as a tumor [13, 37]. Diagnostic imaging with modalities such as CT, MRI, and PET has been used to differentiate RIBP from a neoplastic process. MRI is the modality of choice for evaluation of the brachial plexus, and RIBP-associated fibrosis appears as a diffusely thickened plexus with signal intensity similar to skeletal muscle [2, 11, 32, 40, 45]. In contrast, a neoplastic process would be identified by the presence of a focal mass. Finally, PET imaging may reveal malignant etiologies of brachial plexopathy with increased uptake of 18 fluoro-2-deoxy-D-glucose [1]. Diagnostic imaging in this case report included a PET scan, which ruled out a neoplastic process prior to her referral to our institution.

On electrodiagnostic studies, 90% of all patients with either neoplastic or radiation plexopathy will have nerve conduction abnormalities. Myokymic discharge (abnormal spontaneous discharges accompanied by wavelike muscle quivering) is present in 63% of patients with radiation plexopathy compared with 4% of those with a brachial plexus neoplasm [3, 12, 19]. Nerve conduction studies reveal no significant differences between RIBP and neoplastic disorders of the brachial plexus [35]. Roth et al. suggested the presence of prolonged conduction block in RIBP and its association with myokymic discharges [34–36]. This was further emphasized by Esteban and Traba who found conduction block in all of their radiation plexopathy cases [7]. According to electrophysiologic studies reported by Boyaciyani et al., there is some degree of segmental demyelination in brachial plexus fibers of asymptomatic patients following radiation therapy [3].

Various treatments have been used for RIBP. Early physical therapy is important to prevent lymphedema, atrophy, and frozen shoulder [37]. Neuropathic pain may respond to medical therapy, including tricyclics, anti-arrhythmics, anti-convulsants, nonsteroidal anti-inflammatory drugs, and steroids [17]. Some degree of muscle function recovery following the use of anticoagulant agents has been reported in RIBP, raising the possibility that nerve ischemia may play a role in its pathogenesis [10]. Transcutaneous electrical nerve stimulation, dorsal column stimulator therapy, and dorsal root entry zone lesions can be considered for intractable cases of chronic pain [17, 33, 37].

Surgical treatment options have centered around the attempt to release fibrotic tissue to eliminate mechanical constriction of the plexus and the surrounding vasculature.

Operations have been described to decompress the brachial plexus and revascularize nerves and surrounding tissues, but there is no treatment which will reliably reverse or change the natural history of RIBP. Toward that end, neurolysis with or without placement of an omental or latissimus dorsi flap as a source of well-perfused tissue is advocated by some [4, 20, 21, 25]. Dissection alone can lead to some relief of pain in a minority of patients. However, success rates vary in the literature, and while some have noted improvement in motor function and pain, others have reported deterioration as a result of such surgery [16, 26]. In this particular case report, we were surprised to find the target muscles in such healthy condition and were very optimistic after neurolysis and noting strong muscle contraction after electrical stimulation. It was felt that this may be adequate treatment, and the patient was therefore awakened. However, the persistent palsy of the musculocutaneous nerve confirmed a conduction block even though innervation to the muscles remained intact.

To our knowledge, there is no report in the literature involving nerve transfers for the treatment of RIBP. The reinnervation of the biceps by nerve grafting or nerve transfers from outside the brachial plexus was originally reserved for brachial plexus injuries, including avulsion of C5 and C6 roots [28]. This case of RIBP presented a unique situation of a proximal conduction block of the musculocutaneous nerve at the level of the brachial plexus. Active elbow flexion was prevented, but muscle innervation and viability of the neuromuscular junction was maintained despite the several-year history of progressive loss of motor function, not unlike a central neurological lesion. In the case of a traumatic brachial plexus injury, wallerian degeneration occurs distally with target muscle denervation and eventual degeneration of the motor endplate, which prohibits the possibility of reinnervation with nerve transfers or graft reconstruction after approximately 1 year [23]. The experimental literature has demonstrated inferior recovery of muscle function with longer time periods of denervation [9]. In the case reported, there was no muscle denervation or atrophy prior to the nerve transfer procedure, which optimizes the recovery of the neuromuscular junction and thus muscle function by minimizing the period of muscle denervation.

A number of different nerve transfer procedures can be chosen based on the availability of expendable donor motor nerve branches [27, 41, 42]. We have recently described the double fascicular transfer from the median and ulnar nerves to the biceps and brachialis branches of the musculocutaneous nerve, and this has become our transfer procedure of choice when good hand function is preserved or recovered [22]. This technique allows the transfer to regenerating motor axons very close to the neuromuscular junction of

the elbow flexors, which minimizes the distance and time required for the regenerating front to reach the target muscle. As such, even patients with late presentation following injury remain candidates for this procedure. Reinnervation of both the biceps and brachialis muscles permits the recovery of strong elbow flexion, as the brachialis is the primary flexor of the elbow, while the biceps is a primary supinator and secondary flexor. Donor morbidity is avoided as redundant and expendable wrist and finger flexors are selected for transfer and verified by direct intraoperative stimulation of the remaining nerve fascicles. Other donor nerves such as the thoracodorsal and medial pectoral nerves can also be used and expected to recover satisfactory elbow flexion, but with greater donor morbidity and longer time requirement for reinnervation and may also require the use of nerve grafts [43].

Conclusion

Differential diagnosis of brachial plexopathy after radiation therapy includes RIBP, neuritis, and neoplastic invasion. Optimal treatment is multimodal and includes early physical therapy, medical therapy for neuropathic pain, and operative treatment for select patients. Restoration of motor function by nerve transfers is feasible and may be attempted in those patients with RIBP resulting in significant upper extremity weakness secondary to conduction block.

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