

A case of delayed radiation-induced lumbosacral radiculoplexopathy after 30 years

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Abstract

Lumbosacral radiculoplexopathy in colon cancer treatment is a very rare but serious complication after radiation. We report here a 66-year-old man with slowly progressive lower limb weakness which arose 30 years after local radiation treatment for colon cancer. Electrophysiological studies revealed signs of denervation confined to the lower limbs. Other causes were excluded by clinical presentation, serological, cerebrospinal fluid and imaging studies. This case shows that delayed radiation-induced lumbosacral radiculoplexopathy can occur 30 years after the initial treatment.

INTRODUCTION

With improved long-term cancer survival, chronic or delayed complications of treatments become a medical and public health concern. Radiation therapy is a common adjunctive modality used in the treatment of colorectal cancer after surgical ablation. Radiation-induced peripheral neuropathy is one of the most serious complications as the resultant disability may have considerable impact on quality of life of cancer survivors. However, till to-date, radiation-induced neuropathies have not received much attention among the health care professionals.^{1,2} Its occurrence is thought to be rare, but the incidence is increasing due to growing number of long-term cancer survivors. We present here a patient in whom lumbosacral radiculoplexopathy developed 30 years after irradiation for descending colon cancer.

CASE REPORT

A 66-year-old man was referred for progressive leg weakness over a year. The weakness began as a foot drop in the right leg, and progressed to the left leg a month later. The patient had no complaint in the upper extremities. He has past history of colon cancer 31 years ago, that was treated with surgery and radiotherapy. However, the total dose of radiation of the initial treatment is unknown, as the treatment was given long time ago at another hospital. There was recurrence of the colorectal cancer two years earlier which was treated successfully with surgery.

On examination, there was a radiation burn scar in the middle and lower abdomen. On

neurologic examination, there was mild atrophy of the leg muscles. For muscle power, there was marked feet weakness with dorsiflexion (Medical Research Council Grade 1), plantar flexion (Grade 4), eversion (Grade 2), and inversion (Grade 2). The power of the other muscles in the legs were grade 4 in abduction and extension of the hip and flexion and extension of the knee and grade 3 in internal rotation of the hip. Deep tendon reflexes were absent at the both knees and ankles. Sensory examination was normal. In upper extremities, muscle strength and tendon reflexes were normal. Laboratory studies including blood chemistry, serological screening for autoantibodies, and cerebrospinal fluid analysis revealed no abnormalities. Magnetic resonance imaging (MRI) at the onset of weakness showed atrophy of lower lumbosacral paraspinal muscles, subcutaneous tissues and fatty changes in the bone marrow of the whole lumbar vertebral bodies without any evidence of tumor recurrence or metastasis (Figure 1). The first electrophysiological study, which was performed as the symptoms were confined to the right lower extremity, disclosed a widespread denervation changes in the right lower limb and right lumbosacral paraspinal muscles. At that time, the study did not include the left lower limb. The second study was performed a year later. It showed slowed motor and sensory conduction in the legs. There were marked reductions in amplitude of compound muscle action potentials in the peroneal nerves and sensory nerve action potentials in the sural nerves. H-reflexes were absent and F-wave latencies in the peroneal and



Figure 1: Lumbosacral T₂-weighted MRI in sagittal view with fatty changes in the vertebral bodies (white arrows), with subcutaneous atrophy (gray arrows), without cancer recurrence.

tibial nerves were prolonged. There were no abnormalities of motor and sensory conduction in the upper limbs. Needle electromyography showed denervation potentials; decreased recruitment; and long-duration, high-amplitude motor unit potentials in peroneal, tibial, and femoral nerve innervated muscles and lumbosacral paraspinal muscles both sides.

He was slightly better after physiotherapy for 3 months. Positron emission tomography-computerized tomography performed 14 months after the onset of leg weakness, showed no evidence of cancer recurrence. The weakness subsequently stabilized without further deterioration.

DISCUSSION

The main unusual feature of this case is the development of progressive lumbosacral radiculoplexopathy 30 years after therapeutic radiotherapy. There was no evidence of cancer recurrence. This case shows that the diagnosis of radiation-induced neuropathy should be considered in patients presenting with lower extremity weakness or pain even up to 30 years after radiation therapy. The course of the radiation-induced neuropathy is one of steady progression or stabilization in 90% of patients, although cases of improvement have been reported.³

Although radiotherapy has shown benefit in reducing cancer recurrence and improves survival, it has associated risks. Radiation-induced radiculoplexopathy is one of the complications associated with cancer treatment that is uncommon and can be challenging in diagnosis and treatment. Diagnosis of radiation-induced neuropathy require the exclusion of tumor recurrence, pre-existing neuropathies, chemotherapy, or other unrelated cause of neuropathy. In the case of lumbosacral radiculoplexopathy, other unrelated etiological causes include amyotrophic lateral sclerosis, diabetes mellitus, immune-mediated neuropathies, or leptomeningeal metastases or cauda equina infiltration from other tumors.^{5,6} We excluded the other diagnosis with the serological, electrophysiological, imaging tests, and cerebrospinal fluid analysis. Our patient is unusual in the long delay of 30 years before development of radiation-induced lumbosacral radiculoplexopathy. Al-Benna *et al.* has reported a brachial neuropathy 22 years after radiation therapy⁷ and Hsia *et al.* has reported a post-irradiation polyradiculopathy 24 years after radiation and chemotherapy for Hodgkin's disease.⁸

The mechanism of radiation-induced lumbosacral radiculoplexopathy remains uncertain.

The radiation effects to the lumbosacral plexus correlate to the cumulative dose, technique, concomitant disease, and pharmacotherapy administered. Injury can occur with local total doses of 4,000-6,000 cGy and with doses greater than 2 Gy per fraction.⁹ Size and localization of the irradiated area also play a crucial role. Adverse effects of radiotherapy to the nerve can be subdivided into early and late effects. Early effects occur two days after nerve irradiation and include bioelectrical alterations, enzyme changes, abnormal microtubule assembly and altered vascular permeability. The late effects occur between one year and decades post-irradiation and they can be split into two phases. The first phase includes changes in electrophysiology and histochemistry of neurons and glial cells and the second phase includes fibrosis of the tissue surrounding the nerves. Indirect ischemic damage due to microvascular injury also harms neurons and glial cells. The major damage caused in neurons, DNA damage, takes place immediately at the instant of ionization. As the mitotic rate of these cells is very slow, the damage is normally not expressed until the cell attempt to divide. Therefore, this damage is latent for periods ranging from days to decades and radiation-induced neuropathy can develop at any time after the treatments.⁷ Recently, there is increased use of intensity-modulated radiotherapy for treatment of many pelvic malignancies, this could prevent “dose dumping”, with less damage to the normal tissue including the lumbosacral plexus.¹

This case shows that radiation-induced lumbosacral radiculoplexopathy can occur after a prolonged delay of 30 years.

DISCLOSURE

Conflict of Interest: None

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