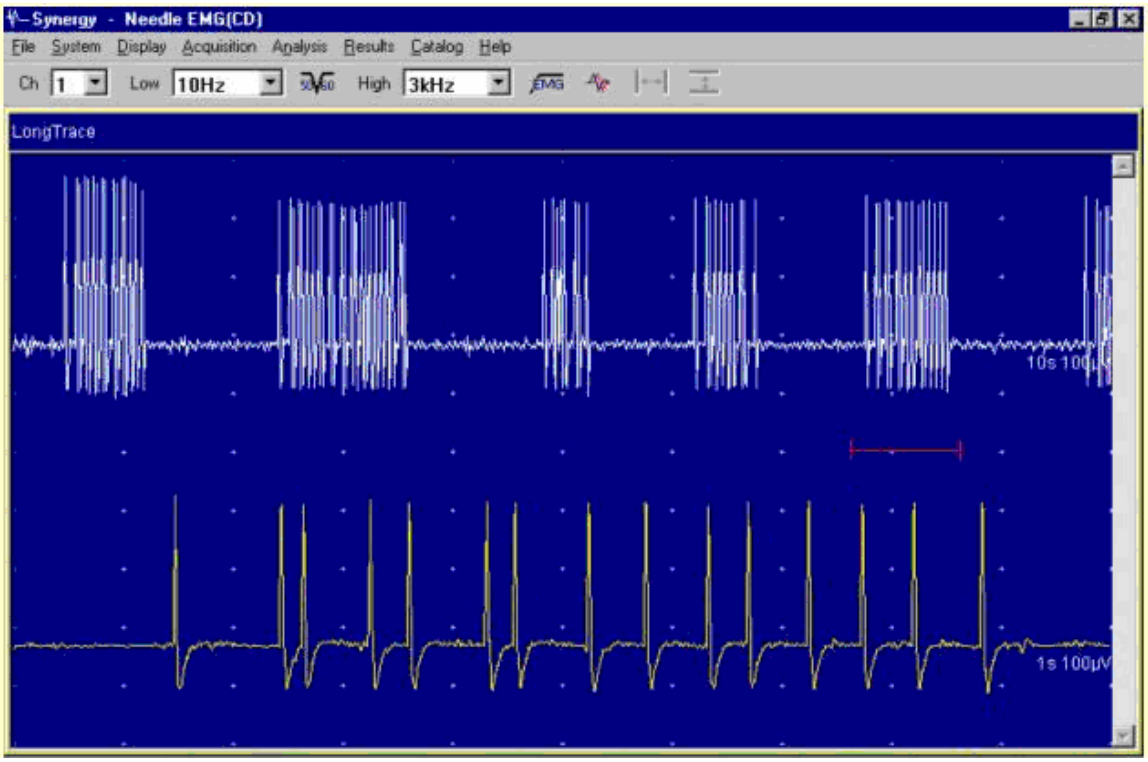
Temple PM&R
Case of the Week

67-year-old female with history of cervical cancer treated with radiation and chemotherapy 5 years ago presents with right lower extremity weakness.

MRI was conducted on the patient's lumbosacral spine

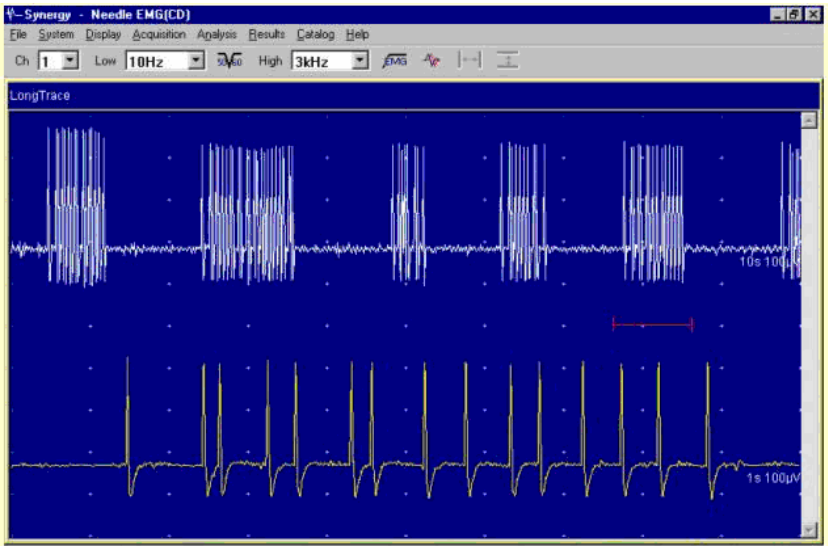
What is seen on the MRI of the spine?
What is your differential diagnosis?

EMG was conducted on the patient's right tibialis anterior, the results are shown here (similar results were also seen in rectus femoris and bicep femoris)



What is seen on this EMG?
What is your diagnosis, based on the patient's history?

This MRI scan shows a somewhat normal spine of the patient, no severe disc herniations, spine cord or bone metastasis, or other lesions are seen.



The EMG shows myokymia discharges (burst pattern), with rhythmical bursts of groups of multiple motor unit potentials.

Myokymia is a clinical phenomenon characterized by undulating, rippling, and wave-like movements visible on the muscle surface. It is associated with characteristic spontaneous electromyographic (EMG) activity consisting of myokymic or neuromyotonic discharges, which are spontaneously generated bursts of grouped motor unit potentials (MUPs) recurring rhythmically or semirhythmically, usually several times per second. Clinical myokymia has been reported in various disorders, including Guillain-Barré syndrome, multiple sclerosis, radiation plexopathy, brainstem tumor, and timber rattlesnake envenomation.

Diagnosis

Radiation-Induced Lumbosacral Plexopathy

Case Points

- Radiation-induced lumbosacral plexopathy is a rare phenomenon
- Motor deficits in the lower extremities commonly are bilateral (80%) and asymmetrical.
- Initial symptoms are usually at 5 years post-radiation, but can range from 1-31 years
- Weakness usually occurs distally first, then progresses proximally.
- Routine spine and pelvis radiographs and myelograms are unremarkable in lumbosacral plexopathy.
- EMG reveal myokymic discharges in most patients
- Paraspinal involvement occurs in one-half of the patients

Discussion

Lumbosacral plexopathy can result when radiation, used in the treatment of various neoplasms, is directed toward management of abdominal and pelvic malignancies.

Anatomically, the lumbosacral plexus consists of lumbar (L1-L4) and sacral (L5-S5) portions, which are connected by the lumbosacral trunk (L4-L5). The L1-L4 nerve roots transverse through the psoas muscle and then coalesce into the lumbar plexus, which then divides into anterior and posterior divisions. The first 3 nerves (iliohypogastric, ilioinguinal, and femoral) of the 7 major branches of lumbar plexus provide motor and sensory innervation to the abdominal wall. The next 3 nerves (lateral femoral cutaneous, femoral, and obturator) innervate the anteromedial thigh. The femoral nerve terminates in the saphenous nerve providing sensation along the medial aspect of the leg.

The sacral plexus also divides into anterior and posterior divisions, which further divide into various peripheral nerves, providing sensory motor innervation to posterior hip girdle, thigh, and anterior and posterior leg. The 5 main nerves are the superior gluteal, inferior gluteal, posterior femoral cutaneous, sciatic, and pudendal. The sciatic nerve divides into the common peroneal and tibial nerves in the thigh.

With prior radiation treatment and initial symptoms, a recurrent tumor may need to be distinguished from postradiation plexopathy. The median symptom-free interval for radiation-induced lumbosacral plexopathy, from treatment to the initial neurologic symptom, is **5 years**, with a range of **1-31 years**. Weakness is asymmetrical. At the height of illness, the ratio of bilateral to unilateral illness is

5:1. Acute lower extremity paralysis has been noted in a patient with cervical cancer 10 weeks after completion of radiation treatment. Patients with radiation-induced lumbosacral plexopathy most commonly present with painless weakness in 1 or both legs.

Causes of lumbosacral plexopathy not related to cancer include aortic aneurysms, diabetes mellitus (DM), obstetric procedures, trauma, and intragluteal injections. With aortic aneurysms, acute pain commonly is seen, and the resultant weakness typically worsens over 1-2 weeks and then stabilizes. A pulsatile rectal or abdominal mass also can be seen in many patients. Acute thigh pain with acute or insidious onset of weakness can result from diabetic amyotrophy and can be difficult to differentiate from the aortic aneurysms. Weakness with diabetic amyotrophy usually is noted proximally, with relative sparing of distal lower extremity muscles, compared to radiation-induced plexopathy which usually occurs distally.

The diagnosis of radiation plexopathy can be supported by diagnostic studies, such as computed tomography (CT) scanning and magnetic resonance imaging (MRI) of the pelvis. MRI is more sensitive than is CT scanning in detecting tumor recurrence.[9, 10] Enhancement of nerve roots and T2-weighted hyperintensity usually suggests tumor. Unfortunately, differentiation from tumor recurrence remains difficult. Generally, radiation plexopathy does not produce nerve enhancement. Positron emission tomography (PET) scanning with 2-[fluorine-18]-fluoro-2-deoxy-D-glucose (FDG) may be helpful in diagnosing recurrent tumor.

Electromyography (EMG) reveals **myokymic discharges** in most patients (57%) with radiation-induced lumbosacral plexopathy. Such changes occur over years; however, the absence of myokymic does not exclude radiation injury. EMG in clinically weak muscles also may reveal fibrillation potentials (ie, chronic, neurogenic motor unit changes with decreased recruitment). Paraspinal involvement occurs in 50% of cases. Compound muscle action potential (CMAP) of motor nerves may be low

Treatment: Strengthening of lower extremity muscles, use of assistive devices for ambulation (eg, cane, walker), and gait training should be prescribed for patients with weakness and proprioceptive feedback loss. Use of orthotics also may be beneficial in certain individuals with lumbosacral plexopathy. For issues of pain consider the use of nonopiate pharmacologic medications, such as tricyclic antidepressants or antiepileptic agents (eg, gabapentin, carbamazepine). The use of steroids and opiates, including methadone, can also be considered. Recent studies have show that anticoagulants, such as acenocumarol, improved the clinical symptoms of the plexopathy.

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