Postoperative Shingles Mimicking Recurrent Radiculopathy after Anterior Cervical Diskectomy and Fusion

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Abstract

Study Design Case report and review of literature.

Objective To report the case of a 67-year-old woman who developed delayed onset (6 months) of symptomatic shingles after cervical nerve root decompression in a previously symptomatic dermatome.

Methods The patient's clinic course and outcomes were retrospectively reviewed. The study required no outside funding. The study authors have no financial interest in any of the products or techniques discussed.

Results The patient received definitive treatment for shingles once the zoster form rash manifested. The patient, however, developed postherpetic neuralgia and remained symptomatic at her 2-year postoperative visit.

Conclusions Although shingles is a common disease state affecting patients in the fifth and sixth decades of life, it is rarely seen in the setting of cervical nerve root decompression. This case demonstrates the need to include shingles on the differential diagnosis of recurrent neurogenic pain after anterior cervical decompression and fusion.

Keywords
- anterior cervical diskectomy
- shingles
- radiculopathy

Introduction

A 67-year-old woman initially presented in 2009 for evaluation of left upper extremity and axial cervical pain. Her past medical history was significant for stage IIIb colon adenocarcinoma that had been in remission since 2007. The patient was subsequently referred to our practice after failing to respond to 12 months of nonoperative treatment consisting of anti-inflammatory medications, physical therapy, neuromodulatory medications, and an epidural steroid injection, which gave the patient several weeks of moderate relief.

Case Report

Upon our initial examination, the patient reported a history of continued left upper extremity pain, weakness, numbness, and tingling as well as several recent falls, changes in balance, and decreased fine motor coordination and dexterity. Physical examination was notable for decreased strength graded as 4/5 in her left wrist flexors, biceps, intrinsics, and triceps. Sensitivity to light touch was diminished in the radial aspect of the left forearm as well as thumb, index, and long finger consistent with C6 and C7 dermatomes. Also notable from examination was unilateral left-sided hyperreflexia graded as 3+ at her biceps, triceps, and brachioradialis as well as a positive Hoffman sign of the left upper extremity. No other abnormal upper motor neuron signs were present in the right upper extremity or bilateral lower extremities.

Imaging, including plain films and magnetic resonance imaging (MRI), were obtained demonstrating C5–C6 and C6–C7 spondylosis with corresponding central and neuroforaminal cervical stenosis. Based on her history, physical examination,

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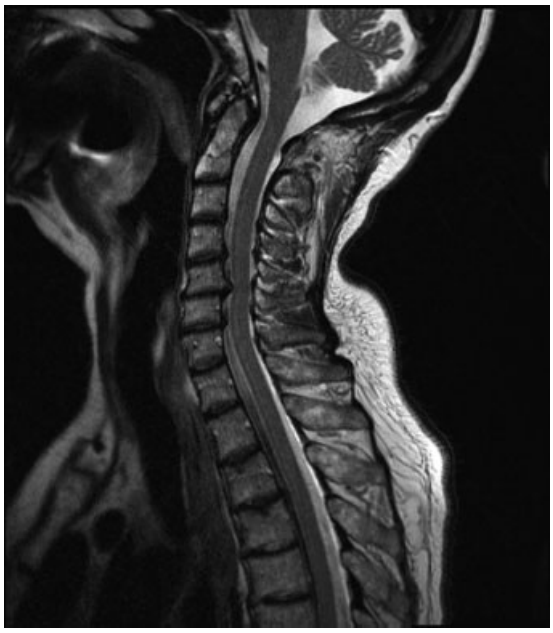

Fig. 1 Preoperative T2-weighted sagittal magnetic resonance imaging clearly demonstrating a large disk-osteophyte complex at the C5–C6 level.

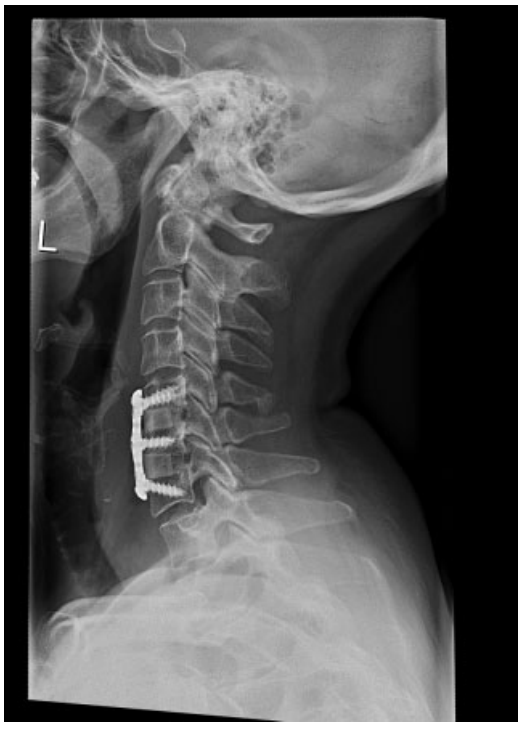

Fig. 3 Postoperative lateral standing cervical radiograph demonstrating uncomplicated two-level anterior cervical decompression and fusion at the C5–C6, C6–C7 level.

and imaging studies, the patient was diagnosed with cervical spondylotic myeloradiculopathy (►**Fig. 1**, ►**Fig. 2**). Her symptoms were recalcitrant to thorough nonoperative treatment, and she underwent surgical management with anterior cervical decompression and fusion at C5–C6 and C6–C7. Her radicular symptoms completely resolved and her myelopathic symptoms improved significantly in the immediate postoperative period. The patient was discharged from the hospital on postoperative day 1. Plain radiographs demonstrated acceptable instrumentation position and alignment without evidence of complications (►**Fig. 3**). By her 3-month examination, the patient had complete resolution of axial, radicular, and myelopathic symptoms. Left upper extremity examination demonstrated intact 5/5 strength in all muscle groups, intact sensation to light touch, and 2-point discrimination as well as a negative Hoffman sign and symmetric reflexes.

At her 6-month evaluation, the patient presented stating that 3 weeks earlier, she began experiencing a severe exacerbation of radicular symptoms in her left upper extremity. Upon

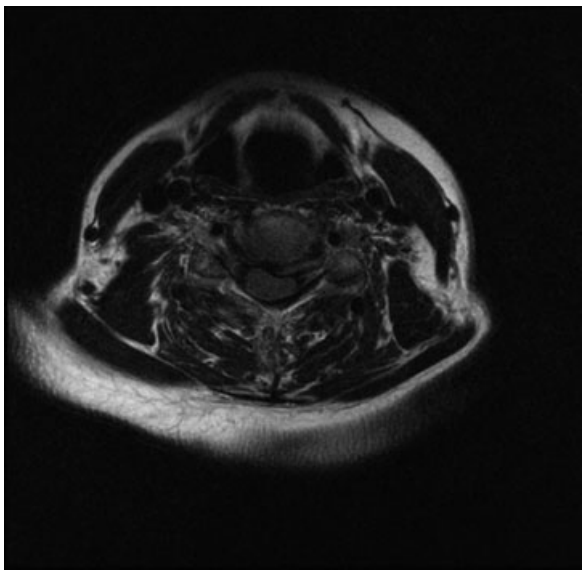

Fig. 2 Preoperative T2-weighted axial magnetic resonance imaging demonstrating a left-sided disk herniation with resultant foraminal stenosis at C5–C6 impinging upon the left C6 nerve root.

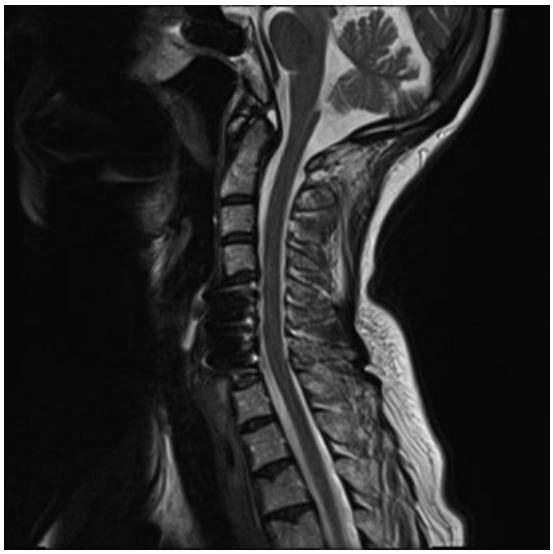

Fig. 4 T2-weighted sagittal magnetic resonance imaging performed 6 months postoperatively after patient presented with recurrent symptoms. Note that no persistent and/or recurrent stenosis apparent at the C5–C6, C6–C7 level.

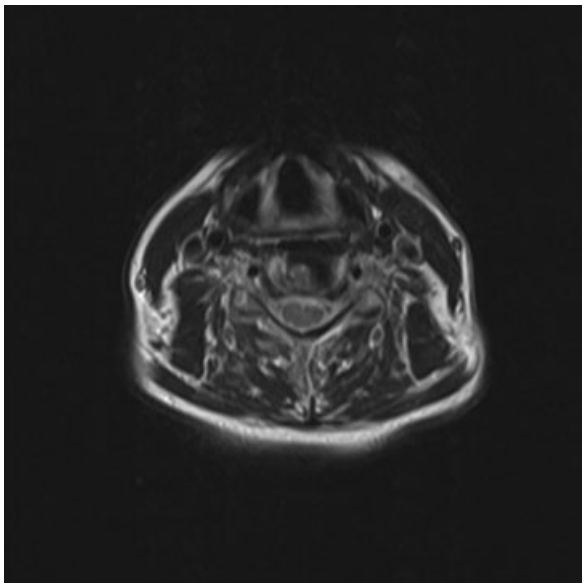

Fig. 5 T2-weighted axial magnetic resonance imaging at the C5–C6 level confirming no persistent and/or recurrent stenosis impinging upon the affected left C6 nerve root.

examination her symptoms were consistent with a left C6 radiculopathy. Repeat cervical radiographs and cervical MRI (►**Fig. 4**, ►**Fig. 5**) failed to demonstrate recurrent stenosis or pseudarthrosis and showed no evidence of adjacent-level changes. An electromyography test (EMG) was ordered and the patient was scheduled for further follow-up exams. Shortly after this examination, the patient developed an erythematous vesicular rash extending from the left shoulder to the first and second digits (►**Fig. 6**, ►**Fig. 7**) and the EMG was not performed due to the presence of this pathognomonic rash. She was diagnosed with herpes zoster (HZ) in a left C6 nerve root distribution. She was started on valacyclovir and topical steroids, and she reported progressive reduction in symptoms.

The patient is now 1 year post–cervical spine surgery and despite several courses of antiviral medications, steroids, and

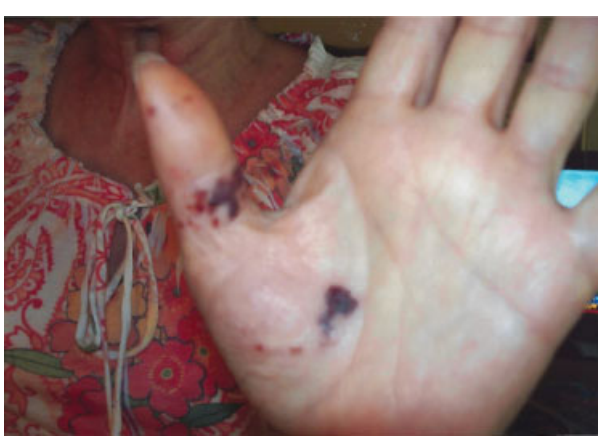

Fig. 6 Clinical photograph of patient's proximal left upper extremity demonstrating early dermatitis herpetiformis vesicular rash with erythematous macular base in a C6 dermatome.

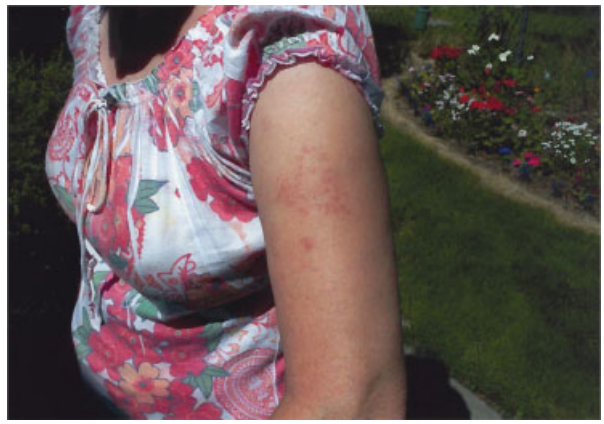

Fig. 7 Clinical photograph demonstrating patient's ventral left upper extremity, with late phase dermatitis herpetiformis lesions, characterized by scaled papules in a C6 dermatome.

3,600 mg of gabapentin daily in divided doses, the patient has persistent symptoms of postherpetic neuralgia (PHN). Due to her persistent symptoms, an EMG was obtained at her 9-month follow-up that showed no active denervation or residual cervical radiculopathy in the C6 distribution. Her motor strength has returned to normal.

Discussion

This case report is the first to discuss the development of late (6 months) shingles outbreak after cervical nerve root decompression in a previously symptomatic dermatome. Due to the absence of a clinically apparent rash at her 6-month evaluation, HZ was not initially considered as a possible etiology of this patient's symptom. There was no prior record of HZ in her medical history. Immediate evaluation for recurrent radicular symptoms focused on ruling out the presence of pseudarthrosis, implant failure, recurrent neuroforaminal stenosis, or infection. Flexion/extension radiographs as well as MRI failed to demonstrate instrumentation failure, lucency about the implants, adjacent level stenosis, recurrent neuroforaminal stenosis, or a discernible diskitis/infection.

The varicella zoster virus (VZV) is the progenitor of both varicella (chicken pox) and HZ (shingles). Varicella commonly affects most individuals in their first or second decade of life. Typical symptoms include exudative pharyngitis, fever, myalgia, and a macular rash progressing to a diffuse vesicular rash. Once the initial VZV has abated, the VZV remains dormant within the dorsal root ganglia. Although the exact mechanism of activation is unknown, most outbreaks occur during periods of relative decrease in cell-mediated VSV immunity.[1] Identifiable risk factors include psychosocial stress, malignancy, recent illness, traumatic events, and surgery.[2]

HZ has been shown in several studies to affect between 20 and 30% of the general population with most cases appearing after the fifth decade of life at a rate of 1% per year.[3,4] Immunocompromised patients see an increased risk of 80

to 90% over immunocompetent patients of like age.[4] Development of HZ carries significant morbidity, and the literature has shown that one in five patients will develop long-term sequelae.[5]

Clinical presentation of HZ begins with neuropathic pain, itching, burning, and dysesthesias followed by the onset of a classic unilateral dermatomal rash. The rash is described as an erythematous base, initially maculopapular eruptions then progressing to vesicular eruptions that typically resolve within several weeks of prodromal symptoms. The development of chronic pain symptoms after HZ is referred to as PHN, and it carries the highest rate of postinfection morbidity and long-term sequelae. Ocular involvement also conveys severe morbidity if not recognized and treated immediately. Despite the presence of these sequelae, reactivation of HZ after primary activation remains low due to increased VZV antibodies.

Cervical spinal dermatomal involvement in HZ outbreaks has been noted to be as high as 15%.[6] Trunk involvement, including one to three separate dermatomes of the thoracic or lumbar spine, make up 60% of spinal involvement. Hata et al conducted a large cohort study and described 17 specific disease states in which there was a 1.8- to 8.4-fold increased risk of HZ development compared with the general population.[2] Disk herniation was described in this study as an independent risk factor for primary activation or reactivation of VZV leading to HZ.

Diagnosis of HZ remains largely clinical in nature and depends on the observed identification of a clinically apparent rash as well as classic prodromal symptoms. In patients where the diagnosis of HZ is unclear, serologic studies are available. HZ may be detected through serum or cerebrospinal fluid polymerase chain reaction DNA analysis in an acute idiopathic setting. Limitations of polymerase chain reaction testing are based on the sometime chronic nature of VZV, which may produce false-positive results if the disease state has been present greater than 2 weeks. Vesicular lesion biopsy may also be used to identify VZV through culture virus isolation or rapid direct fluorescent antibody testing. Fluid culture is sensitive and specific but can take several days to return results. Direct fluorescent antibody sensitivity and specificity are highly dependent on the presence of active lesions.

The treatment goal for HZ outbreak involves early reduction of symptoms. A multimodal therapeutic approach is the recommended treatment including antivirals, oral and topical analgesics, as well as the addition of neuromodulatory medications. Although frequently used, there are conflicting studies as to the role and effectiveness of oral corticosteroid use. Consensus data suggests judicious, limited courses of oral corticosteroid.[5]

Antiviral therapy is recommended in the patient with acute shingles, ideally initiated within 72 hours of initial symptom development. The 72-hour window remains optimal for treatment initiation, but in the case of an individual whom continues to actively produce new vesicular lesions, antiviral therapy is still indicated.[7] Several agents are commercially available in the United States including acyclovir, valacyclovir, and famciclovir. Duration of treatment depends upon agent of choice; however, most regimens last 5 to 7 days. It is hypothesized that early antiviral treatment decreases viral shedding, thus decreasing the duration and severity of the primary outbreak. Unfortunately, antiviral treatment has not been shown to decrease the incidence of PHN.[5,7]

Low-dose gabapentin in conjunction with antiviral treatment has been shown to be effective in reducing acute neuropathic pain. It is noted in several sources that gabapentin is rarely initiated in conjunction with antiviral therapy unless the patient was initially treated by a dermatogist.[5] Tricyclic antidepressants such as amitriptyline have a limited role in treatment of HZ but may be used in the setting of chronic PHN.

With the introduction of the VZV vaccination in 1995, prevention appears to be the next step in reducing HZ outbreaks as well as the morbidity associated with HZ especially in the setting of PHN. A large placebo-controlled, double-blinded clinical trial was published in the *New England Journal of Medicine* in 2005, in which patients $>$ 60 years of age were given placebo or zoster vaccination. The results of this study demonstrated a reduction in the incidence of HZ by 51.3% among vaccinated patients compared with the placebo control. Those who received the vaccination but still developed HZ had a truncated clinical course and a greater than 60% reduction in the incidence of PHN.[7]

Although our patient had no prior history of HZ, she had multiple risk factors predisposing her for a primary activation including age greater than 50 years, history of malignancy, and recent cervical surgery. It has been reported that a standard anterior cervical decompression has a direct affect upon the dorsal root ganglion; however, the development of HZ symptoms along the same dermatome suggests otherwise. Several case reports from the otolaryngology literature described a primary HZ activation after trigeminal nerve microvascular decompression as described by Mansour et al,[8] where they described a case of confirmed HZ at postoperative day 3, along the maxillary branch of the trigeminal nerve.

This is the first case report of a patient who developed shingles in the same distribution of a decompressed nerve root late in the postoperative time course. Our review of the current literature demonstrated several cases of perioperative HZ cervical radiculopathy. These cases noted symptoms prior to or in the immediate postoperative period. The timing of our patient's presentation appears unique in that HZ symptoms developed 6 months after cervical nerve root decompression in a previously symptomatic dermatome. This case demonstrates the need to included HZ on the differential diagnosis of recurrent cervical radiculitis in the extended period after anterior cervical decompression and fusion.

Disclosures
Jason T. Montgomery, none
Brandon D. Lawrence, none
Darrel S. Brodke, none
Alpesh A. Patel, none

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Editorial Perspective

EBSJ wishes to thank the authors of the case report as well as the commentators for reminding us as clinicians of the complexities of our central nervous system and that radicular symptoms may indeed be multifactorial in origin. The article is very helpful in presenting our readership with recommended management in terms of diagnostic thinking as well as practical clinical care questions for postviral neuropathy, such as instituting antiviral therapy and short use of anti-inflammatory therapy as well as considering anticonvulsant medications. Our commentators provided a very helpful perspective on differential diagnosis of radicular pain and emphasized the important of differentiating between various types of pain, with burning pain and mechanical allodynia being more typical for a postherpetic neuralgia and a radiculopathy having a more stabbing or electric quality. *EBSJ* would add Parsonage-Turner syndrome (or idiopathic neuralgic amyotrophy, historically also referred to as brachial plexus neuropathy) to this discussion as a differential diagnosis. This condition most commonly affects a shoulder girdle distribution but may extend to other nerve groups as well and combines severe pain with a delayed onset of motor and sensory changes. MRI of the affected brachial plexus with neurography and electromyography may assist in the diagnosis. Especially needle EMG can be very helpful in identifying the axonal disruption of this inflammatory process.[1]

In general, when spine patients with neurologic presentations fail to make expected recovery, and especially when they worsen despite our best intentions and applications of care, it seems advisable to proceed early on with a comprehensive and mindful reevaluation of affected patients to clear the air and then proceed with the most directed treatment pathway. Presenting the patients and their families with realistic outlooks for recovery, which can range from weeks to years and in some cases may leave lasting deficits and symptoms for some of the mentioned conditions, in an open fashion would seem to be a helpful element of the care process as well.

Reference

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