

MSK Case No. 16, March 2007

Presenting Symptom(s): neck, upper back, and left arm pain

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Case Prepared by: Jennifer Baima, MD; Zacharia Isaac, MD

Affiliations: Brigham and Women's Hospital; Spaulding Rehabilitation Hospital

Disclosures: J Baima, None; Z Isaac, None

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Appropriate Audience: Residents and practicing physicians

Learning Objectives: After completing this educational activity, participant will be able to: (1) Formulate a differential diagnosis for the immunosuppressed patient with radicular upper limb pain; (2) Correlate imaging findings with the patient's clinical presentation; (3) Understand and diagnose infectious/ inflammatory radiculopathy.

Estimated Level of Difficulty: Intermediate

History

A fifty-two year old man with a history of psoriatic arthritis on methotrexate and remicade presents to clinic with greater than four weeks of neck pain, upper back pain, and severe left upper limb pain. He complains of pain in his posterior thorax that radiates into the bilateral neck and posterior shoulders, the left superomedial scapula, posterolateral left upper arm, ulnar forearm, and medial two and a half digits (digits IV and V). He describes the pain as aching, burning, and stabbing with associated numbness. Although his severe pain had been intermittent, it is now occurring at rest and at night. Reaching above his head exacerbates his pain. There are no mitigating factors identified. There are no associated changes in the patient's gait, bowel, or bladder.

1. Prior to continuing, please develop a differential diagnosis and list each possible diagnosis in order of likelihood.
2. Is there any additional information regarding the clinical history that might be helpful in clarifying your differential list or changing its order of priority?

Commentary I

Differential diagnosis at the time of presentation includes traumatic, degenerative, infectious, neoplastic, and metabolic causes. If the patient had a traumatic injury, he may have suffered damage to the spinal cord, C8 or T1 nerve root, lower trunk brachial plexus, or peripheral nerve. The patient could have left C7-T1 foraminal compression from disc herniation or spine arthropathy. In the cervical spine, spondylosis is a more common cause

of foraminal compression than disc herniation.¹ The patient did not have a known history of diabetes, but his radicular pain could occur as a result of diabetic radiculopathy.

Since he is on remicade, a TNF-alpha inhibitor, he would be at risk for infectious etiologies including but not limited to epidural abscess, varicella zoster, and Lyme disease. Axial back pain is the most common complaint in patients with pyogenic spondylitis.² Neoplastic causes include intramedullary, intradural, or extradural spinal cord tumors. Although he is only fifty-two years-old, the patient reported night pain and rest pain, which may be red flags for neoplasia. Other common complaints in patients presenting with spinal tumors include spontaneous onset of symptoms, aching character of symptom manifestation, and symptoms provoked by standing and walking. In one study, the incidence of spine tumors presenting to musculoskeletal physiatrists was 0.69% in two academic multidisciplinary spine centers.³

Additional information that would further elucidate the diagnosis includes any prior history of trauma, infection, or cancer. We should inquire if the patient had any international travel or exposure to tick bites. It would be helpful to further clarify the time course leading up to the development of severe radicular pain. We could further narrow our differential by asking if the patient noticed any associated weakness or gait disorder.

History, continued

Approximately four weeks prior to presentation, he was diving into a pool and struck another person with his head. He felt that his neck “crunched” in a flexed position at that time. The patient experienced immediate-onset neck pain and left upper extremity pain without associated weakness. About ten days later, the patient began having symptoms of left upper extremity weakness. He sought chiropractic treatment and had about six such treatments prior to presentation. He presented to his primary care physician and an MRI was ordered before referral to physiatry. This was the first imaging study since the patient’s injury.

1. If necessary, revise your differential diagnosis based on the additional clinical history.
2. On which details of the physical examination should you focus at this point?

Commentary II

Given that there is associated weakness, it would be important to examine the patient for muscle atrophy and quantify muscle strength. Skin should be inspected for rashes. Cervical range of motion should be assessed. Neck and axilla should be palpated for lymphadenopathy. Cervical spinous processes and paraspinal musculature should be palpated for tenderness and deformity. Neurologic exam should be performed to look for signs of myelopathy. MRI findings should be correlated with the patient’s physical exam. Given that the patient had a traumatic injury, this is the most likely cause of his symptoms at this time.

Physical Examination

On physical exam, there was atrophy of the first dorsal interossei and the abductor pollicis brevis. All other muscles demonstrated normal bulk and tone. The patient had no rashes. There was limited cervical range of motion in forward flexion, right lateral flexion, and cervical extension. There was no palpable lymphadenopathy. There was tenderness to palpation over the cervical spinous processes and paraspinal musculature. There was no palpable step-off deformity. There were symmetric radial pulses and capillary refill of the fingers was intact. Manual muscle testing revealed intact strength in the right upper extremity and bilateral lower extremities. Evaluation of the left upper extremity yielded weakness that was most profound in the finger abductors, thumb abductors, finger flexors, and wrist extensors. There was mild weakness of the left elbow flexors and extensors. Sensory exam demonstrated intact sensation in the right upper extremity and bilateral lower extremities. However, the left upper extremity had decreased light touch and pin prick sensation in the medial arm and digits III through V.

1. At this point, review your differential diagnosis and revise as appropriate.

2. Are there additional observations on physical examination that might be helpful in narrowing your differential list?

Commentary III

The patient has weakness in muscles innervated by median, ulnar, and possibly radial nerves (wrist extensors). A lesion of the medial cord could produce these symptoms if the wrist extensor weakness is from the extensor carpi ulnaris only. Moving proximally in the brachial plexus, a C8 lesion would produce weakness in the dorsal interossei, abductor pollicis brevis, abductor digit minimi, lumbricals, flexor digitorum profundus and superficialis, and extensor carpi ulnaris. Impairment in these muscles could explain the patient's distribution of weakness. The patient's traumatic neck flexion injury would more likely produce a spinal cord or root level injury than a medial cord injury. Medial cord injury usually results from traction on the upper limb during extension. If we are concerned about a medial cord lesion, it may help to perform a pulmonary exam on the patient. Upper lung lesions can result in lower trunk or medial cord compromise. At this point, it is important to check for central spinal canal compromise by checking Hoffman's, wrist clonus, deep tendon reflexes, and tandem gait pattern. Presence of upper motor neuron signs would indicate the need for more urgent intervention.

Physical Examination, continued

Upper motor neuron signs were absent and reflexes were intact and symmetric. Percussion of the neurovascular complex at the left Erb's point reproduced left trapezius and arm pain. Tinel's test at both elbows caused hand paresthesias in an ulnar distribution. Upper extremity root tension signs of the median and radial nerves were negative. Cranial nerves II-XII were intact bilaterally. Gait exam revealed normal heel-walking, toe-walking, and tandem gait.

1. If necessary, revise your differential diagnosis based on the additional physical findings.

Clinical Impression

There are no upper motor neuron signs to suggest central spinal canal impingement. At this point, it appears that the patient has a C8 radiculopathy. He has subjective pain in a C8 distribution and objective findings of atrophy in muscles that are innervated by this myotome. Review of the patient's diagnostic imaging will likely yield a final diagnosis. We would expect to see degenerative spine arthritis leading to compromise of the nerve root, a common cause of radiculopathy in the cervical spine.¹ Of note, the patient has a history of psoriatic arthritis. He did not have any prior inflammatory arthritis of his cervical spine, and his disease was well controlled on remicade.

1. What diagnostic tests would you order at this time?

Commentary IV

MRI performed at four weeks post-injury showed degenerative changes including disc desiccation and loss of disc height at multiple cervical levels. There was right-sided foraminal narrowing at C3-C4, bilateral foraminal narrowing at C4-C5, and bilateral foraminal narrowing at C5-C6 with moderate central canal stenosis. There was right greater than left foraminal narrowing at C6-C7, and disc narrowing and dessication **at C7-T1 without significant foraminal narrowing or central canal stenosis**. Calibur and signal characteristics of the spinal cord were within normal limits. Muscle strain injury was visible in the left spinalis cervicus, multifidus, semispinalis capitus, and to a lesser extent in the splenius capitus.

Vertebral level	Nerve root	Right foramen	Left foramen	Central canal
C2-C3	C3	No stenosis	No stenosis	No stenosis
C3-C4	C4	Severe stenosis	Mild stenosis	Mild stenosis
C4-C5	C5	Severe stenosis	Severe stenosis	Severe stenosis
C5-C6	C6	Severe stenosis	Severe stenosis	Severe stenosis
C6-C7	C7	Severe stenosis	Moderate stenosis	Moderate central
C7-T1	C8	No stenosis	No stenosis	Mild stenosis

We are surprised to learn that the patient does not have evidence of C7-T1 foraminal stenosis compromising the left C8 nerve root. He does appear to have foraminal stenosis at C7 which could produce most of the above symptoms. However, this stenosis appears worse on the right than the left. The patient's symptoms do not correlate with this MRI.

As such, we elect to perform further diagnostic testing. An MRI with gadolinium is ordered as the patient may have a lesion not visualized on noncontrast imaging. The patient appears to be overall worsening since the original insult. Perhaps this lesion increased in size to cause the patient's clinical exacerbation. An EMG was also ordered to locate the lesion. Since we do not see a lesion at C8, the patient may have compromise of the medial cord of his brachial plexus.

Test Results

Further work-up including NCV/ EMG ensued to determine the cause of the patient's symptoms .

NCV findings:

- Sensory nerve action potentials:
 - Left median and right ulnar SNAPs showed mild decrease in amplitudes and prolonged distal latencies.
 - Right median SNAP showed mild decrease in amplitude with normal latency.
 - Left ulnar SNAP showed mild prolongation of distal latency.
 - Left radial SNAP was normal.
- Motor nerve action potentials:
 - Right median CMAP showed mild prolongation of distal latency with normal amplitude and forearm conduction velocity.
 - Left median, both ulnar, and left radial CMAPs were normal.
- F waves:
 - Both median and right ulnar F waves were mildly prolonged.
 - Left ulnar F wave was normal.

EMG Findings:

- Large amplitude, long duration, polyphasic potentials with reduced recruitment were seen in the left abductor pollicis brevis, abductor digiti minimi, extensor digitorum communis, extensor indicis, flexor carpi ulnaris, and flexor digitorum profundus muscles.
- Positive sharp waves and fibrillation potentials were seen in the left abductor pollicis brevis, abductor digiti minimi, extensor digitorum comunis, extensor indicis, flexor carpi ulnaris, and flexor digitorum profundus muscles.

- Left deltoid, biceps, and triceps muscles were normal.

Overall, the patient had abnormalities in both arms. However, right-sided findings appear chronic and unrelated. On the left, the patient had abnormal median and ulnar sensory nerve abnormalities. Abnormal motor units, fibrillations, and positive sharp waves were seen in C8-innervated muscles.

1. If necessary, augment or change the clinical impression based on the test results.

Commentary V

From this EMG, it does appear that the patient has a C8 radiculopathy. All of the above abnormal muscles share this innervation. Extensor digitorum and extensor indicis are innervated by the posterior interosseus nerve, a branch of the radial nerve. The radial nerve branches off the posterior cord of the plexus. Thus, we have ruled out a medial cord lesion and appear to have a C8 radiculopathy with no associated mechanical compression. Perhaps the patient has a metabolic or infectious cause of radiculopathy producing his symptoms.

1. What additional diagnostic testing would you order?

Commentary VI

An ESR and fasting blood glucose may give us further insight into infectious and metabolic causes of this patient's radicular pain. The patient denies any fever or chills. However, he is on an immunosuppressant medication and may have an atypical presentation of infection. An elevated ESR is often found when epidural abscess is the cause of radicular pain and weakness.⁴ This patient's ESR was 16. The expected range of ESR in patients with pyogenic spondylitis is 40- 90 mm/ hr.⁵ Viral infection is not an uncommon cause of pain. Varicella zoster virus infection can present with radicular pain as the only initial manifestation.⁶ Patients usually progress to have a rash. Immunosuppressed patients are more likely to be susceptible to zoster.⁷ The most common metabolic cause of neuropathy, diabetes, could be responsible for this patient's radicular pain. Diabetic neuropathy classically presents as bilateral sensory loss in a stocking pattern in the lower limbs. This patient did not have any lower limb complaints, and pain was his presenting problem. Diabetes can also cause radiculopathy. Diabetic radiculoplexopathy, also called diabetic amyotrophy, typically involves asymmetric pain and weakness of the lower limbs. This condition may occur in the cervical region. Usually, these patients develop unilateral sensorimotor neuropathy predominant in the hands and forearms.⁸ Our patient did present in this manner, but he did not have an elevated blood glucose level.

Test Results, continued

While awaiting further diagnostic tests, the patient developed a target lesion over left anterior thigh consistent with erythema migrans. He was evaluated in the Emergency Department and a head CT revealed no acute intracranial abnormality. Laboratory data included elevated CSF protein and CSF pleocytosis. Western blot revealed significant levels of IgM, IgG, and IgA antibody to *B. burgdorferi*, but no significant immunoblot results for IgG. These results were interpreted as consistent with early Lyme infection.

1. What is the impact of the additional test results on the final diagnosis?
2. Considering all the data from the history, physical examination and laboratory studies, what is/are your final diagnostic impression(s)?

Final Diagnostic Impression

The patient has a C8 Lyme radiculopathy.

Commentary VII

The patient has clinical and electromyographic evidence of C8 nerve root injury. He has a positive Lyme titer. The constellation of erythema migrans, serologic evidence of Lyme disease, and concurrent evidence of C8 radiculopathy without foraminal compression at C7-T1 on MRI yields the final diagnosis.

1. What treatment would you now initiate for this patient?

Commentary VIII

The patient's remicade and methotrexate were discontinued. He was treated with a four week course of intravenous ceftriaxone with resolution of symptoms.

Final Discussion

Lyme disease is a common entity in the Northeast region. In 2004, there were 6.7 cases of Lyme disease per 100,000 people in the US.⁹ The typical rash seen in Lyme disease occurs in about 90% of patients within one month of infection but may not be observed prior to clinical presentation of early disseminated Lyme disease. This rash is usually found near the axilla, inguinal region, behind the knees, or at the belt line. It is typically asymptomatic and spreads over several days to reveal a central area of clearing.

Our patient had clinical evidence of Lyme neuropathy prior to developing erythema migrans. Classically, erythema migrans occurs before other manifestations of Lyme disease. Since early disseminated Lyme disease may also occur in the first month of infection, the radiculopathy may present prior to, concurrently with, or after the rash. Unfortunately, there is insufficient evidence on the temporal course of erythema migrans in disseminated Lyme disease since adults often miss the tick bite and routine screening is discouraged.

Screening for Lyme disease is not recommended because of a high incidence of false positive results. For patients who have clinical evidence suggestive of Lyme disease, ELISA should be performed. Relapsing fever, syphilis, lupus, and other inflammatory conditions can yield a positive test. If the ELISA is negative, there is no need for further testing and it is unlikely that the patient has Lyme disease. If this is positive, a Western blot should be done to confirm the results.⁹

Reported neurologic findings in early disseminated Lyme disease include motor, sensory, or mixed radiculopathy, lumbosacral or brachial plexopathy, mononeuropathy or mononeuropathy multiplex, cranial neuropathy, and polyneuropathy. Radicular symptoms are often severe and occur in a dermatomal pattern with or without motor or reflex changes. In the United States, it is twice as common to have cranial nerve findings as compared to radicular symptoms.¹⁰

Electrodiagnostic testing in Lyme neuropathy usually reveals axonal polyneuropathy. However, some patients may have evidence of demyelinating disease as well. Nerve biopsies demonstrate Wallerian degeneration and evidence of regeneration. There are thought to be two forms, one acute and one chronic. The acute form presents as a severe axonal radiculoneuropathy that resolves spontaneously. The chronic form, although milder in symptoms and on electrodiagnostic testing, often does not improve unless the patient is treated.¹¹

Treatment includes oral doxycycline, amoxicillin, or cefuroxime. If the patient has disseminated Lyme disease as indicated by cardiac or neurologic findings, intravenous cephalosporins or penicillins are recommended. Most patients recover completely with a four week course of these antibiotics. Some patients require a second four-week course for recurrent symptoms. There has been no demonstrated benefit to longer courses of antibiotics.⁹

Bibliography

1. Radhakrishnan K, Litchy WJ, O'Fallon WM, Kurland LT, "Epidemiology of cervical radiculopathy. A population-based study from Rochester, Minnesota, 1976 through 1990." *Brain* 1994 Apr; 117(Pt2): 325-35.
2. An HS and Seldomridge JA, "Spinal Infections," *Clinical Orthopaedics and Related Research* 2006 Mar; 444: 27-33
3. Slipman CW, Patel RK, Botwin K et al, "Epidemiology of Spine Tumors Presenting to Musculoskeletal Physiatrists," *Arch Phys Med Rehabil* 2003 April; 84: 492-495
4. Tang HJ, Ling HJ, Liu YC, and Li CM, "Spinal epidural abscess--experience with 46 patients and evaluation of prognostic factors." *J Infect.* 2002 Aug;45(2):76-81.
5. Carragee EJ, Kim D, van der Vlugt T, and Vittum D, "The clinical use of erythrocyte sedimentation rate in pyogenic vertebral osteomyelitis." *Spine.* 1997 Sep 15;22(18):2089-93.
6. Kost RG, Straus SE, "Postherpetic neuralgia--pathogenesis, treatment, and prevention." *N Engl J Med* 1996 Jul 4;335(1):32-42.
7. Galil K, Choo PW, Donahue JG, Platt R, "The sequelae of herpes zoster. *Arch Intern Med* 1997 Jun 9;157(11):1209-13.
8. Katz JS, Saperstein DS, Wolfe G, Nations SP, Alkersam H, Amato AA, and Barohn RJ, "Cervicobrachial Involvement in Diabetic Radiculoplexopathy." *Muscle and Nerve* 2001 (24): 794-798.
9. www.cdc.gov
10. Halperin JJ, "Lyme disease and the Peripheral Nervous System," *Muscle and Nerve* Aug 2003: 133-143.
11. Logigian EL and Steere AC, "Clinical and electrophysiologic findings in chronic neuropathy of Lyme disease." *Neurology* 1992 (42): 303-311.