CASE REPORT

Cervical Spine Stenosis Progressing to Acute Cord Compression

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Introduction

Cervical spine stenosis is a common presentation in the primary care office practice. We discuss a case presenting with an altered mental status masking an underlying unstable cervical spine with myelopathy.

Case Description

Our patient is a 56-year-old woman with a past medical history of Hepatitis C, cirrhosis, recurrent urinary tract infections, major depression, and chronic back pain, as well as on opiates, who presented for an outpatient evaluation for the first time with new bilateral upper extremity numbness for four weeks. She reported a long-standing history of neck pain, and a MRI of the cervical spine performed a year prior showed 3-4mm grade I anterolisthesis of C3 on C4. Other than chronic neck pain, she did not previously complain of neurological symptoms until this presentation. Her medication list included hydromorphone, cyclobenzaprine, zolpidem, citalopram, and Vitamin D.

To rule out a contributing metabolic neuropathy, laboratory testing included negative inflammatory markers, normal electrolytes, normal TSH, negative HIV, normal hemoglobin A1c, borderline vitamin B12 with normal methyl malonic acid level, and normal serum protein electrophoresis. Hepatitis C status was confirmed positive. Given her new symptoms of numbness, a repeat MRI of the cervical spine was performed, which noted a 4mm C3 on C4 anterolisthesis, mildly worsening C3-C4 canal stenosis, and a subtle T2 signal increase in the right aspect of the cord at C3-C4, which was interpreted as representing either vasogenic edema versus myelomalacia. As no other mass or cavitary lesions were seen on MRI, her negative laboratory evaluation and focal nature of her symptoms, cervical myelopathy was believed to be the underlying problem.

With the patient's clinical presentation and new MRI findings, she was referred for a neurosurgical evaluation. However, she did not keep the neurosurgical appointment due to increasing weakness, somnolence, and falls. She then presented to our primary care office 2 days later, mentally altered, with slurred speech and inability to stand needing wheelchair assistance. She admitted to taking more than usual opiate doses due to neck pain. Given her manifestation, she was transported via paramedics for hospitalization.

During initial evaluation in the emergency room, patient was mentally altered exhibiting somnolence with poor attention span. Falls were believed to be due to her altered mental status. CT scan of the head was negative for acute pathology. Hydromorphone use likely contributed to her altered mentation. Ammonia level was mildly elevated to 43 micromoles/L and hepatic encephalopathy was also thought to be a contributing factor. Urinalysis was suggestive of a urinary tract infection. MRI of the brain did not show any evidence of a cerebrovascular accident. Patient recovered her mental status to baseline within a day after withholding opiates and on levofloxacin therapy. Ammonia level normalized to 20 micromoles/L. With improvement in her mentation, she could comply with physical examination. She exhibited persistent motor weakness in bilateral lower extremities, in addition to neck pain with decreased neck range of motion, bilateral arm numbness extending distally in the hand, and difficulty with writing. New MRI of the cervical spine now revealed a 6mm vertebral anterolisthesis of C3 on C4 with moderate cord compression with myelomalacia. Neurosurgery consultation was obtained and determined that the patient was having a severe circumferential cervical instability with progressive myelopathy and impending paralysis. Patient received emergent anterior C3-C4 cervical decompression, discectomy, and fusion. This was followed by posterior cervical C3-C4 decompressive laminectomy and fusion of C3-C6. Post-surgical stabilization, she was transferred to a skilled nursing facility.

Since then, the patient has demonstrated ability to ambulate with a cane and makes clinic visits. Long-term benefit from our treatment intervention remains to be seen.

Discussion

Cervical spine stenosis is a common health condition with an estimated prevalence of about 4.9% in the general population and 6.8% in those 50 years of age or older.[1] Presentation ranges from asymptomatic incidental findings and neck pain to more severe forms of radiculopathy from compression of nerve roots in the neural foramina to the most severe form, cervical myelopathy from cord compression.

Cervical spondylosis or osteoarthritis of the cervical spine is caused by degeneration of intervertebral discs, and osteophyte bone formation with normal aging. Cervical myelopathy or injury to the spinal cord can result from cervical spondylosis, subluxation of vertebrae in the sagittal plane as seen in our patient. Congenital narrowing of spinal canal is a predisposing factor. Symptoms for C3-C5 compressive myelopathy are arm weakness, sensory loss, loss of manual dexterity, and overall arm clumsiness. Damage at C5-C8 level causes spastic changes and proprioceptive changes in the legs leading to loss of balance and falls.[2]

The natural history of cervical spondylosis with conservative non-operative intervention is not well-described. In patients with symptomatic myelopathy, it has been shown that 20% to 62% will deteriorate at 3 to 6 years, but no specific disease or patient-related characteristics can predict this. In patients with asymptomatic myelopathy, the development of symptoms is estimated around 8% within one year and 23% within four years.[3]

Mild symptoms are best treated with conservative care with immobilization of cervical spine with the head in the neutral or slightly flexed position. With moderate to severe symptoms, conservative care only helps 30% to 50% of patients, and surgery is generally considered the choice of treatment. Role of surgery on the natural history of cervical myelopathy has been questioned and is best reserved for brief duration of symptoms and mild neurologic deficits.[2]

Apart from pain control, our patient reported noncompliance to conservative care recommended to her early in the course of her disease prior to our evaluation. Symptomatic deterioration was witnessed within a month of establishing care with us. With an unstable cervical spine during hospitalization, surgical intervention was possibly the therapy of choice. It is unknown whether her symptoms progressed as the natural course of the disease leading to lower extremity motor instability and falls, or if falls from encephalopathy mediated further cord injury leading to neurologic deterioration.

Prescription opiate overdose is a national epidemic, accounting for 67.8% of all opiate related emergency room visits in 2010 with over a half of these patients needing hospitalization.[4] In the same year, opiate analgesics accounted for 60% of all drug overdose deaths in the United States.[5] In our patient, severity of neck pain, feeling of hopelessness due to her functional status, and poor social support possibly led to the overdose. Suboptimal drug metabolism from liver cirrhosis possibly contributed.

Identifying deteriorating motor function involving the lower extremities posed a challenge. This was masked with confounding clinical comorbidities that may potentially precipitate altered mentation and motor weakness including opiate intoxication, encephalopathy from hepatitis C cirrhosis, and a urinary tract infection. Persistent motor deficits despite improvement of mentation and with known history of cervical myelopathy led to further evaluation and eventual surgical intervention.

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