

Severe Painless Cervical Spinal Stenosis In An Old Patient: To operate or not to operate?

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Citation

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Abstract

Patients with cervical stenosis usually present with cervical radiculopathy, with or without myelopathy. An 82-year-old female had a six weeks history of difficulty walking. Higher cortical functions and cranial nerves were within normal limits. Motor exam revealed mild right leg weakness and gait unsteadiness. Sensation was decreased for light touch, pinprick and proprioception. EMG of lower extremities was negative. Neck CT showed severe cervical spondylosis and spinal stenosis. Cervical CT myelography showed multilevel bulky spondylosis, degenerative disc disease and narrowing of the central canal and neural foramina. Lumbar CT myelography showed multilevel disc degeneration and bulky osteophytosis and multilevel bony and discogenic narrowing of the central canal and neural foramina. After discussion with the patient, it was decided to proceed with EMG of upper extremities; however, the patient changed her mind and refused to continue. Conclusion: The decision to proceed with surgery should be individualized and tailored to the patient's desires and needs.

INTRODUCTION

Narrowing of the lumbar spinal canal is an increasingly common problem, affecting 1 per 1000 persons older than 65 years [1]. Spinal stenosis is a degenerative condition, part of the aging process. Patients generally become symptomatic at age 50 years or older. It is estimated that 5 of every 1000 Americans older than 50 years have symptoms of spinal stenosis [2]. The degeneration of the motion segment vertebral column - the intervertebral disk and the facet joints - is believed to be the pathophysiologic mechanism involved with the development of stenosis. Degenerative changes in the spine including osteophyte formation, facet hypertrophy, bulging disks, and hypertrophy of the ligamentum flavum can result in canal or foraminal narrowing. Spondylolisthesis (the displacement of one vertebra on top of another) can further compromise the canal. On the contrary, spondylosis (bony overgrowths associated with aging of the spine) theoretically can fix the affected part of the spine and thus stop additional injury to the spinal cord caused by neck movements.

CASE REPORT

An 82-year-old female had a six weeks history of difficulty walking. She was on Terazosin for hypertension. No prior surgeries. Chews tobacco and drinks socially. No contributory family history.

Physical exam showed higher cortical functions and cranial nerves to be within normal limits. Motor exam revealed mild right leg weakness and gait unsteadiness. Sensation was decreased for light touch, pinprick and proprioception.

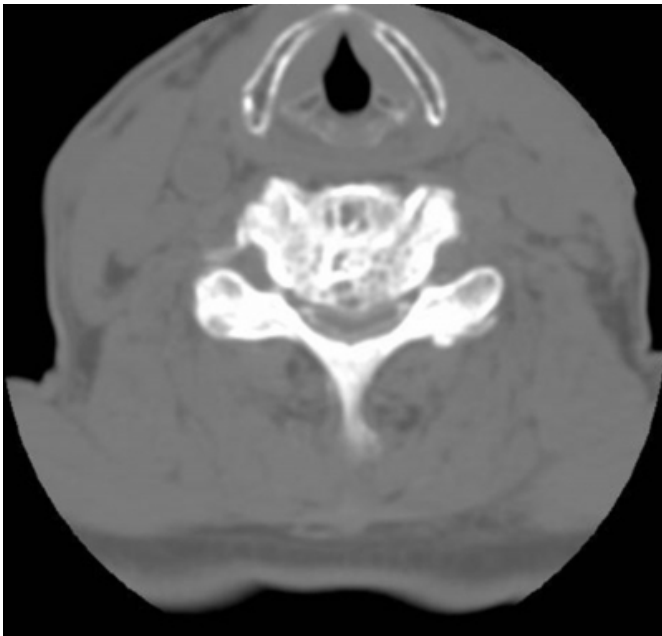
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Figure 1



Sagittal cervical CT myelogram showing disc bulging and marginal osteophytosis with severe canal stenosis and bony narrowing of the neural foramina at C4-C5 and C5-C6.

Figure 2



Transverse cervical CT myelogram showing flattening of the spinal cord.

After discussion with the patient, it was decided to proceed with EMG of upper extremities; however, the patient changed her mind and refused to proceed with investigation.

DISCUSSION

The pathophysiology of spinal stenosis is related to cord dysfunction elicited by a combination of mechanical compression and degenerative instability. With aging, the intervertebral disk degenerates and collapses, leading to spur formation. This most commonly occurs at C5-6 and C6-7. A relative decrease in spinal motion occurs at these levels with a concomitant increase in spinal motion at C3-4 and C4-5.

The resultant degeneration and abnormal motion lead to instability with anterolisthesis or retrolisthesis (subluxation of vertebral bodies out of the normal cervical alignment). Therefore, the cord tends to be compressed from spur formation at C5-6 and C6-7 and compressed from listhesis at C3-4 and C4-5. Often, this is accompanied by posterior canal compromise from ligamentum flavum hypertrophy.

The cord is subject to further injury from repetitive dynamic injury during normal neck movements. These static and dynamic compressive forces on the cord lead to spinal cord injury and the clinical myelopathic syndrome.

Stenosis of the cervical and thoracic spine may result in myelopathy from cord compression, while stenosis in the lumbosacral region often results in radicular pain or neurogenic claudication (radiating leg pain associated with walking that is relieved by rest).

Cervical stenosis may occur at a very slow or very fast rate [3] and consequently clinical manifestation of the disease varies. There was no complaint of pain in our case despite the moderate to severe cervical canal stenosis and neural foramina narrowing and mild to moderate lumbar canal stenosis shown on CT myelography. The sagittal or anteroposterior cervical spinal canal measurements at the disk level were 9.49 mm for C3-C4, 6.74 mm for C4-C4 and 4.56 mm for C5-C6. The patient's main complaint was difficulty walking. This clinical manifestation of spinal stenosis is the result of compressive pressure applied by the narrowing spinal canal on the spinal cord and the neural foramina on the nerve roots. Its degree of expression depends on the diameters of bony frame and neural structures. If the slow narrowing of the spinal canal and neural foramina is accompanied by degenerative changes that lead to atrophy of the spinal cord and nerve roots and natural fixation of the cervical spine, the patient may experience only mild radicular pain or symptoms of myelopathy. Pain is also a subjective feeling where psychological factors play an important role in pain perception.

Our patient presented with symptoms of myelopathy (spastic gait) with no symptoms of radiculopathy (radiating pain) due to slow progression of spinal canal stenosis and the concomitant degenerative processes in the vertebral column and spinal cord and nerves.

Cervical stenosis progresses in as many as one third of affected individuals. Among the symptoms are a propensity for initial deterioration, followed by a period of stability (may require years), and subsequent progression of myelopathy.

The natural history of lumbar spinal stenosis is not well understood. A slow progression appears to occur in all affected individuals. Even with significant narrowing, such persons are very unlikely to develop an acute cauda equina syndrome in the absence of significant disk herniation.

Management of spinal stenosis is aimed toward symptomatic relief and prevention of neurologic sequelae. Conservative measures provide temporary relief but remain an important adjunct in the overall treatment algorithm preceding surgical decompression. Generally, surgery is indicated when the signs and symptoms correlate with the radiologic evidence of spinal stenosis. Surgery is recommended when significant radiculopathy, myelopathy (cervicothoracic), neurogenic claudication (lumbar), or incapacitating pain is present.

Moreover, anesthesia in older patients carries big cardiovascular and cognitive risks. Cognitive deterioration after general narcosis may be severe and permanent especially after long exposure to inhaled anesthetic, halothane [4]. Desired benefits from surgery should be weighed against expected risks from anesthesia and sound

judgment should be employed. The decision to proceed with surgery should be individualized and tailored to the patient's desires and needs.

CONCLUSION

Clinical manifestations of spinal stenosis may not correlate with the radiologic findings on CT myelography. Patients with cord compression that develops very slowly may not experience pain. It is up to the surgeon and the patient to decide if surgery is required. Usually, if the patient's quality of life is compromised because of pain and there are no effective or acceptable nonsurgical treatments, it is reasonable to recommend surgery. However, if the old patient's symptoms are not causing much distress, he/she may leave this life before even disease causes debilitating symptoms. The choice of surgical procedure and the decision to fuse the spine should be individualized for optimal outcome.

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