

Sudden Quadriplegia after Acute Cervical Disc Herniation

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ABSTRACT: Background: Acute neurological deterioration secondary to cervical disc herniation not related to external trauma is very rare, with only six published reports to date. In most cases, acute symptoms were due to progression of disc herniation in the presence of pre-existing spinal canal stenosis. **Case report:** A 42-year-old man developed weakness and numbness in his arms and legs immediately following a sneeze. On physical examination he had upper motor neuron signs that progressed over a few hours to a complete C5 quadriplegia. An emergent magnetic resonance imaging study revealed a massive C4/5 disc herniation. He underwent emergency anterior cervical discectomy and fusion. Postoperatively, the patient remained quadriplegic. Eighteen days later, while receiving rehabilitation therapy, he expired secondary to a pulmonary embolus. Autopsy confirmed complete surgical decompression of the spinal cord. **Conclusions:** Our case demonstrates that acute quadriplegia secondary to cervical disc herniation may occur without a history of myelopathy or spinal canal stenosis after an event as benign as a sneeze.

RÉSUMÉ: Quadriplégie subite suite à une hernie discale cervicale aiguë. Introduction: Il est très rare d'observer une atteinte neurologique aiguë secondaire à une hernie discale cervicale qui ne soit pas reliée à un traumatisme externe. Seulement six cas ont été publiés. Dans la plupart des cas, les symptômes aigus étaient dus à la progression de la hernie en présence d'une sténose préexistante du canal spinal. **Observation:** Un homme de 42 ans a développé de la faiblesse et de l'engourdissement dans ses bras et ses jambes immédiatement après avoir éternué. À l'examen physique, il présentait des signes d'atteinte du neurone moteur supérieur qui ont évolué en quelques heures vers une quadriplégie C5 complète. L'IRM a montré une hernie discale C4-C5 massive. Il a subi d'urgence une discectomie cervicale antérieure avec fusion. Le patient est demeuré quadriplégique et il est décédé 18 jours plus tard d'une embolie pulmonaire au moment d'un traitement de réadaptation. L'autopsie a confirmé la décompression complète de la moelle épinière. **Conclusions:** Ce cas illustre qu'une quadriplégie aiguë secondaire à une hernie discale cervicale peut survenir sans histoire de myélopathie ou de sténose du canal spinal suite à un événement aussi banal qu'un éternuement.

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In the absence of severe external trauma, spinal cord injury attributable to disc herniation most often occurs within the thoracic spine.¹ Within the cervical spine, nontraumatic quadriplegia may occur secondary to myelitis,² infection,³ tumor,⁴ vascular compromise,⁵ spondylotic myelopathy,⁶ and fibrocartilagenous embolism.⁷ We report a rare case of acute complete quadriplegia resulting from a cervical disc herniation without severe external trauma in a patient with no previous evidence of canal stenosis.

CASE REPORT

A 42-year-old male was brushing his teeth when he sneezed. He immediately felt pain in his neck and numbness in his left arm. Within a few minutes, both legs and arms became weak and he had difficulty walking. He was brought to the emergency room of a community

hospital by his wife. The past medical history was significant for Buerger's disease (thromboangiitis obliterans) with claudication in the extremities; however, these symptoms had largely subsided after the patient quit smoking. There was no past history of radiculopathy or myelopathy.

By the time of transfer to a neurosurgical facility, approximately eight hours after the initial events, neurological examination revealed a

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Figure: Magnetic resonance images at first presentation. Axial T1 weighted image (A) shows a large centrally herniated C4/5 disc fragment (arrow). Sagittal proton density (B) and T2-weighted (C) images show the disc fragment compressing the spinal cord to an hour glass shape.

flaccid quadriplegia. There was no sensory function below the C4 dermatome and motor function in the extremities was limited to shoulder shrug (motor power of the deltoids and below was 0/5 bilaterally). There was complete loss of muscle tone in the extremities and deep tendon reflexes were absent. Plantars were mute. There was no sacral sensory sparing. Anal wink and bulbocavernosus reflex were absent. The patient had a mean arterial pressure greater than 85 mm Hg with volume support alone.

Emergent magnetic resonance imaging (MRI) demonstrated a very large C4/5 disc herniation as shown in Figure 1 (A, B and C). There was minimal evidence of degenerative change in the rest of the cervical spine. The patient underwent immediate anterior cervical discectomy (under microscopic visualization) and fusion with xenograft. Intraoperatively, the posterior longitudinal ligament was found to be ruptured and the thecal sac appeared indented by two large fragments of extruded nucleus pulposus. A complete epidural decompression was performed. There were no intraoperative complications.

Postoperatively, the patient's neurological deficits did not improve. He was subsequently transferred to an inpatient rehabilitation center. Eighteen days postoperatively, he suddenly collapsed and expired. Autopsy showed the cause of death to be a massive pulmonary embolism. Complete surgical decompression of the spinal cord was confirmed. Unfortunately, a post-operative MRI was not performed. Furthermore, due to the many years that have elapsed since the case, histological sections of the spinal cord were not available.

DISCUSSION

Cervical spinal cord injury may result from a central soft disc herniation or minor cervical trauma in a patient with spondylosis and pre-existing compromise of the spinal canal.⁸ The present case is unusual because of the acute severity of the symptoms and the lack of any pre-existing canal stenosis. We distinguish our case from those acute disc herniations resulting from overt external trauma. Such trauma usually results in high impact injury to the spine with momentary large forces causing a

breakdown of its structural integrity. In our case, the precipitating factor for the disc herniation was a force as benign as a sneeze.

To our knowledge, there have been only six published reports of acute progressive myelopathy resulting from cervical disc herniation in the absence of external trauma.⁸⁻¹³ All patients had improvement in function or resolution of symptoms after surgical treatment, with the exception of the case reported by Suzuki et al.¹¹ Although ruptured cervical discs most commonly occur at the C5/6 level,¹¹ myelopathy was attributed to a C6/7 disc herniation in all but one of the previous reports. Warabi et al¹³ described a patient with nontraumatic progressive myelopathy and multilevel cervical disc herniations; the C4/5 disc was thought to be responsible for the acute symptoms in that case.

In general, compressive myelopathy by central disc herniation shows a stepwise neurological deterioration.¹² This suggests that the pathophysiology involves not only direct spinal cord damage resulting from compression but also vascular compromise, secondary to diminished blood supply to the anterior spinal artery and its branches.¹¹ Patients with severe cervical stenosis may have longstanding myelopathy prior to sudden neurological deterioration. Acute vascular compromise may lead to insufficient perfusion in regions of the spinal cord already in a critical need for blood supply. The symptoms are believed to rapidly disappear after surgical decompression.¹² However, several reports describe incomplete neurological recovery and permanent deficits.^{8,10-12}

In the present case, dorsal column function (proprioception) was lost. Therefore, we believe that infarction in the distribution of the anterior spinal artery alone would not explain the neurological dysfunction and that acute, severe spinal cord compression is the most likely pathophysiology.

Our patient had a history of Buerger's disease (thromboangiitis obliterans), an inflammatory occlusive vascular

disorder involving small- and medium-sized arteries and veins in the distal upper and lower extremities. However, it is unlikely that this condition contributed to the patient's spinal cord ischemia. Although the autopsy showed some atherosclerotic coronary disease, there was no evidence of a systemic vasculitis.

In all of the previously reported cases, patients had some degree of pre-existing spinal stenosis,¹⁰⁻¹³ or had clinical symptoms (neck pain, lower extremity paresthesias, incontinence) in the hours or days before the development of progressive weakness. This may have afforded a larger "window of opportunity" for timely diagnosis and management. In our case, the patient presented with a complete spinal cord injury and, despite timely intervention, the outcome was poor. Presumably the clinical outcome is related to both the duration and the severity of the presenting neurological dysfunction.

The phenomenon of acute cervical disc herniation resulting in quadriplegia without any severe external trauma is exceedingly rare. This case demonstrates that a large central soft disc herniation may result in rapid progression of severe neurological deficits, even in previously healthy patients with no prior symptoms of myelopathy or evidence of cervical stenosis. Our patient had no recovery of neurological function despite early referral to a spine specialist and prompt diagnostic and therapeutic intervention.

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