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Case Report

Contralateral Extremity Paresis and Numbness Caused by Pos-tero lateral Disc Herniation at C3–C4 in a Teenager

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1. Abstract

We report a rare case of a teenager presenting with progressive contralateral extremity paresis and numbness caused by posterolateral disc herniation at C3-C4. Intervertebral decompression and artificial disc replacement was performed. Follow-up at 2 months showed complete neuro-logic recovery. The increasing tensile stresses from the dentate ligament during flexion of the neck might be the etiology of myelopathy in this patient.

2. Introduction

Cervical disc herniation at C3–C4 accounts for an estimated 4% of all cervical disc herniations [1] and is very rare in teenagers. The presenting symptoms often appear as myelopathy, Brown-Sequard syndrome, and seldom as radiculopathy in the fewstud-ies found in the literature [2-6]. We report a rare case of C3–C4 disc herniation causing a mild left compression of the spinal cord resulting in right extremity paresis and numbness.

3. Case Report

An 18-year-old boy presented with progressive paresis and numbness of the right upper and lower extremity of 3 weeks' du-ration. Lateral radiographs revealed a greater than normal flex-ion of the neck but no instability. MRI revealed a moderate left posterolateral disc herniation at C3–C4 with mild compression of the spinal cord (**Figure 1**). High signal intensity with a length of approximately 2 cm was visible on T2-weighted MRI (**Figure 2**). Conservative treatment was instituted with a restraining col-lar limiting mobility of the cervical spine but no improvement was seen. Progressive neurologic deterioration resulted in gait disturbance and right hand clumsiness. Neurological examina-tion revealed reduced sensation to pain and temperature below the C4 dermatome and a decrease (MRC Grade 3/5) in motor function in the right extremity. Physical examination revealed positive Hoffman and Babinski signs in the right limb. Because of progressive neurologic deterioration and ineffective conserva-tive treatment, intervertebral decompression and artificial disc replacement were performed. Follow-up at 2 months revealed normal motor strength and sensation in the right extremity. Postoperative radiographs demonstrated good realignment of the cervical spine (**Figure 3**).

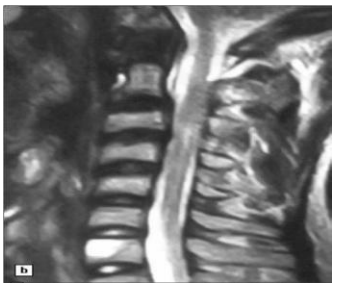

Figure 1: MRI revealed a moderate left posterolateral disc herniation at C3–C4 with mild compression of the spinal cord.

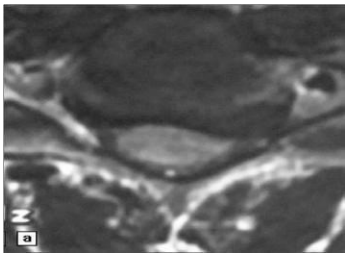

Figure 2: High signal intensity with a length of approximately 2 cm was visible on T2-weighted MRI.

Figure 3: Postoperative radiographs demonstrated good realignment of the cervical spine.

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4. Discussion

Cervical disc herniation of C3–C4 usually presents with quite different symptoms from the more commonly recognized lower cervical disc herniation [5]. As an unusual presentation of my-elopathy, we report a rare case of C3–C4 left disc herniation re-sulting in right extremity dysfunction.

In this patient, it was very unusual that the mild compression of the spinal cord led to such severe neurological defects and that the left compression caused right extremity dysfunction. The theory of mechanical compression in which a herniated disc causes compression of the cord, leading to local tissue ischemia, injury, and neurological impairment certainly fails to explain the entire clinical presentation. Although the role of dentate ligaments in the etiology of myelopathy remains questionable [7-10], the theory of tensile stresses from the dentate ligaments appears to explain the clinical findings in this case well. First, the dentate ligament fibers run mediolaterally along the spinal cord with the dural attachments anchored by the dural root sleeves and dural ligaments and these are relatively fixed [11]. Tensile stresses transmitted to the spinal cord from the dura via the den-tate ligaments may occur from a disc herniation as a result of displacing the spinal cord dorsally with less displacement of the dural attachments. Second, inferred from the clinical symptoms, the site of neurological injury was in the right corticospinal and left spinothalamic tracts. Both are located in the lateral columns of the spinal cord. Dentate ligament fibers also originate from the lateral columns. Therefore, the corticospinal and spinothalamic tracts should be most vulnerable because of lateral pulling by the dentate ligaments. Third, the arch of cervical flexion in this case was apparently larger than normal. Stretching on neural tissues by the dentate ligaments can be greatly amplified during a larger cervical flexion and can exceed the material properties of the tissue, leading to tissue disruption and transient or permanent neurological injury [12]. Last, the unbalanced tensile stresses transmitted to the spinal cord from the dura via the dentate liga-ments generated by the non-centered disc herniation might be responsible for the asymmetric nerve damage seen in this case.

Removal of the herniated disc means removal of the tensile stresses transmitted to the spinal cord from the dura via the dentate ligaments, and early decompression through an anterior surgical approach is crucial to obtain good results.

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