Case Report

Pathology of the spinal cord damaged by ossification of the posterior longitudinal ligament associated with spinal cord injury

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A 63-year-old male became quadriplegic after spinal injury associated with ossification of the posterior longitudinal ligament of the cervical spine and died 4 years later. A postmortem examination of the cervical spinal cord showed various unfavorable pathological changes accounting for severe myelopathy.

Keywords: pathology; spinal cord; injury; ossification

Introduction

Ossification of the posterior longitudinal ligament (OPLL) is a major cause of spinal canal stenosis in Japan.^{1,2,3} The spinal cord, being in a narrowed spinal canal in a patient with OPLL, is easily damaged by head and neck trauma resulting in various neurological deficits.^{4,5} The pathological findings in spinal cords damaged by OPLL vary from minimal cord compression to the development of a crescent-shaped deformity with total neuronal loss and necrotic cavitation.⁶ Although OPLL has been considered to enhance the destruction of the spinal cord, traumatic extra-axial force is one of the major factors for acute spinal cord deterioration, resulting in paralysis in patients with asymptomatic OPLL.

We report an autopsy study of quadriplegia caused by a large mixed-type OPLL associated with spinal cord injury.

Case report

A 63 year old male truck driver was admitted to the hospital after sustaining a severe neck injury in a motor vehicle accident. Neurological examination showed quadriplegia and sensory loss below a C6 level. His previous medical history revealed only a left ulnar nerve palsy due to fracture of his left elbow 7 years prior to this admission. Plain films and computerized tomography (CT) of the cervical spine showed severe canal stenosis due to extensive mixed-type OPLL from C2–C7. Magnetic resonance imaging (MRI) revealed marked cord compression due to this ossified mass (Figure 1). After 4-year medical

treatment, the patient had a fatal episode of sepsis secondary to a urinary tract infection.

Postmortem study of the cervical spinal cord showed various pathological changes. The posterior aspect of the cervical spinal vertebrae was covered by a bulging ossified mass from C2–T1 (Figure 2). This mass involved the dura from C4–C7, and deformity of the spinal cord was apparent from C4–C7 and most severe at the level of C5/6. The anteroposterior diameter of the spinal cord at C6 was compressed to 2 mm. Normal structures of the spinal cord at this level showed widespread destruction with complete neuronal loss and replacement of the gray matter, posterior columns and posterior subarachnoid space by a proliferation of aberrant peripheral nerve bundles (APNB). There was a cavity formation from C5–C7 in the gray matter. While matter was also destroyed except for the anterior column. In addition, there was also discoloration of the posterior column reflecting secondary ascending degeneration from C2–C4. Destruction of the pyramidal tract observed below C7 was considered to be a secondary descending degeneration (Figure 3).

Discussion

OPLL usually enlarges gradually. Therefore, the patient with OPLL often has a long asymptomatic period, although the spinal cord is histologically deformed with degeneration of neurons. Frequently, there is sizable discrepancy between the size of OPLL, the degree of cord compression, and symptoms of the patient.^{7,8} In these cases, one must recognize that even though symptoms may be minimal, the compressed spinal cord in a tight canal is very vulnerable to trauma. The severely compressed cord in the large

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mixed-type OPLL, given direct mechanical extra-axial stress, will be further compromised by contusion of the spinal cord.

Figure 1 (a) Cervical plain lateral X-ray showing huge extensive mixed-type OPLL from C2 to C6. (b) Sagittal MRI disclosing severely compressed cervical spinal cord by OPLL

Spinal cord pathology of OPLL has been reported in a small number of Japanese patients.^{1,3,9,10,11} Characteristic histological changes in the previous literature include gray matter destruction with neuronal loss, white matter destruction with demyelination sparing anterior columns, cavity formation in the gray matter and secondary degeneration of the lateral and posterior columns. Although the spinal cord damage in our case was enhanced by the injury, the comparatively preserved anterior columns despite severe destruction of gray matter and the posterior and lateral columns, together with morphological changes such as a flattening of the spinal cord with deformity of the anterior horn and decreasing the area of the cord, mimics the histological deterioration by OPLL. The majority of these abnormal histological findings are thought to be caused by mechanical compression and resulting secondary circulatory

Figure 2 Posterior aspect of the cervical vertebrae at autopsy showing large mixed-type OPLL bulging into the spinal canal with involving the dura mater

impairment. The gray matter is known as a watershed zone of the parenchyma,¹² and easily destroyed by both mechanical and ischemic changes resulting in a necrotic cavity. Prominent proliferating tangles of

peripheral nerve bundles mainly occupied this collapsed gray matter and posterior subarachnoid space along the posterior roots predominantly at the C6 segment. APNB mimicking amputation

Figure 3 (a–h) Cross-section at the spinal cord segments from C2–T1. The cross sectional shape of the spinal cord was triangular from the C5–C6 segments. The anteroposterior diameter was 2mm at the most severely affected C6 segment. The anterior horn was deformed by compression by OPLL from the C4–T1 segments. The entire transverse plane of the cord was damaged severely, except for small parts of the anterior columns at the C6 segment. Aberrant peripheral nerve bundles were numerous, mainly in the collapsed gray matter and subarachnoid space. Secondary ascending degeneration of the posterior columns from C2–C4 segments and descending degeneration of the lateral pyramidal tracts from C7–T1 segments, were also encountered. (Original magnification $\times 5$, hematoxylin-eosin)

neuroma is consistent with a proliferation of Schwann cells forming myelin associated with damaged axons. This change may occasionally be seen in spinal cord regeneration after injury, spondylosis, and syringomyelia.¹³ Thus replacement of gray matter by APNB reflects chronic degeneration and regeneration, and implies impairment of neurological function.

Morphologically, the present case showed a triangular deformity at the C5 and C6 segments with a decreased area of parenchyma. Yu *et al*¹⁴ reported that considerable improvement was generally achieved after therapy in patients with a boomerang-shaped cord cross-section shown by CT-myelography, but not in patients with a triangular shape. Kobayashi¹⁵ also found that patients with triangular-shaped cord cross-sections had poor functional progress. Fujiwara *et al*¹⁶ suggested that a transverse area of 30 mm² was the critical point below which functional impairment of the spinal cord became irreversible. Okada *et al*¹⁷ also reported that the transverse area determined by MRI was closely correlated with postoperative prognosis. Our case supports all of these clinical datas, and indicates the morphological analysis as well as pathological examination is important to determine the prognosis.

We conclude in this postmortem study that the spinal cord deformed by OPLL is vulnerable to extra-axial force such as trauma, and the morphological and pathological changes well explains quadriplegia without neurological recovery of this patient.

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