

Case reports

Unusual mechanism of lethal cervical spinal cord injury in a case of atlanto-axial diastasis

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Summary. A case of traumatic cervical spinal cord injury due to congenital craniocervical abnormalities is described. The autopsy revealed evidence of hyperextension of the neck due to frontal impact, congenital occipitalization of the atlas accompanied by basilar invagination of the odontoid process and atlanto-axial diastasis. The subsequent narrowing of the spinal canal led to a predisposition for this unusual mechanism of spinal cord injury.

Key words: Traffic accident – Spinal cord injury – Cervical vertebrae – Atlantoaxial joint – Basilar impression

Zusammenfassung. Ein Fall einer traumatischen Halsrückennmarkverletzung bei einem Mann mit kongenitalen craniocervikalen Fehlbildungen wird beschrieben. Die Autopsie zeigte Zeichen der Hyperextension des Halses infolge eines Frontalanpralls, eine angeborene Occipitalisation des Atlas, begleitet von einer basalen Invagination des Dens und einer atlanto-axialen Diastase. Die nachfolgende Einengung des Spinalkanals führte zu einer Prädisposition für diesen ungewöhnlichen Mechanismus einer Verletzung des Rückenmarks.

Schlüsselwörter: Verkehrsunfall – Rückenmarksverletzung – Zervikalwirbel – Atlanto-occipitales Gelenk – Basiläre Impression

Introduction

It is generally accepted that basilar invagination of the odontoid process and atlanto-axial diastasis have occasionally resulted from occipitalization (assimilation) of the atlas [6, 7, 9, 10, 11]. In these cases, traumatic cervical spinal cord injury has probably been caused by hyperflexion of the neck dependent on the location of the odontoid process [11].

In this paper, we describe a case of lethal spinal cord injury due to hyperextension of the cervical area, in which occipitalization of the atlas accompanied by basilar invagination and atlanto-axial diastasis was observed as a predisposition to spinal cord injury.

Case report

A 65-year-old man was struck by a vehicle while crossing the road on a bicycle and was taken to hospital. On arrival, the patient was semicomatous, and was intubated by the emergency personnel. Shortly afterwards cardiopulmonary arrest occurred for several minutes. The cardiovascular system was restored by resuscitation but spontaneous respiration was not elicited. His general condition did not improve and the man died of multiple organ failure 48 days after the accident.

Postmortem examination

External examination of the body revealed *brevicollis* as compared with stature (Fig. 1) and small excoriations in the frontal and occipital regions. Internal examination revealed the following: the brain was moderately swollen with subdural hemorrhage at the right parietal region and there was a contusion on the inferior surface of the right temporal region.

Sectioning of the brain revealed lamellar necrosis around the parieto-occipital sulcus and near the boundary between the regions perfused by the anterior and middle cerebral arteries. At the base of skull, the odontoid process of the axis protruded out of the foramen magnum and the vertebral canal was reduced in size (Fig. 2).

An X-ray of the cervical vertebrae revealed that the occipital bone was fused to the atlas and the atlanto-dental distance (ADD) was widened. Furthermore, the odontoid process protruded upward and flexed backward so that the vertebral canal flexed forward at the level of the odontoid process (Fig. 3). Longitudinal sectioning of the cervical vertebrae showed that the atlas was fused to

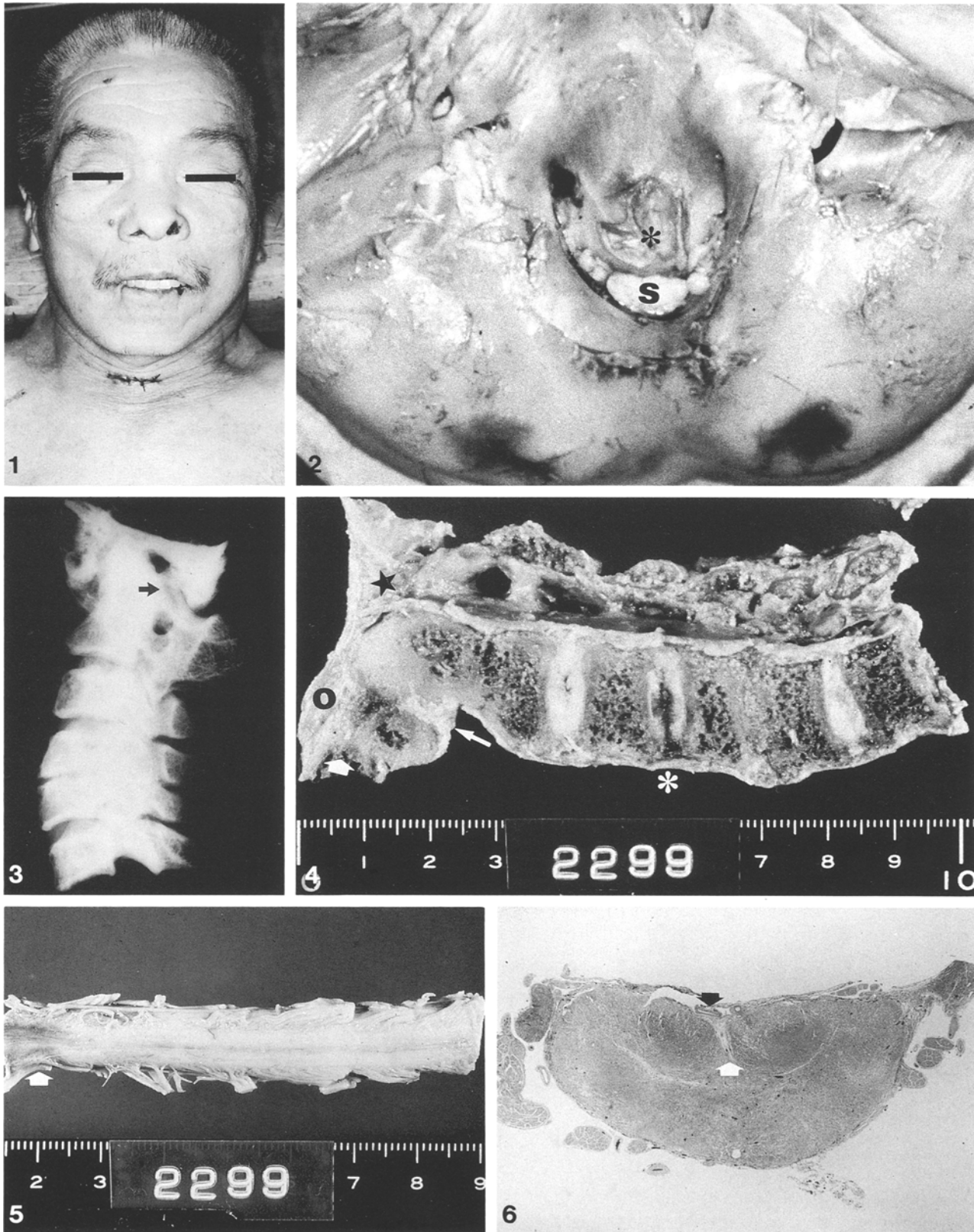


Fig. 1. The characteristic brevicollis in victim of congenital occipitalization of the atlas

Fig. 2. Compressed rostral spinal cord (s) and protruded odontoid process (*) at the base of skull

Fig. 3. X-ray phot of cervical vertebrae extirpated from the occipital bone to C6 at autopsy. The occipital bone is fused to the atlas

and the cervical vertebral canal is flexed at the level of the odontoid process of the axis (*arrow*)

Fig. 4. Longitudinal section of the cervical vertebrae showing fusion of the occipito-atlantal and atlanto-axial joints without mobility (*large and small arrow*) and flexion of the cervical vertebral canal at the level of the odontoid process of the axis (☆). The intervertebral disc of C3-C4 is fissured (*). O: occipital bone

the occipital bone and the atlanto-axial joint was dislocated without mobility, the intervertebral disc of C3–C4 was fissured and the vertebral bodies of C4 and C5 were compressed (Fig. 4). The anterior surface of the tectorial membrane in the cervical canal protruded into the canal because of the extended odontoid process, so that the upper cervical spinal cord was flattened. As a result the 1st and 2nd neural segments showed colliquation and necrosis, but there was no contusion (Fig. 5).

Histological examination of the cervical spinal cord showed that the 2nd neural segment was compressed and oval in form, the anterior median fissure was deviated and the lumen of the spinal artery was stenosed (Fig. 6). The parenchyma suggested necrosis, ischemia and demyelination and the lateral funiculus showed degeneration. The lungs, liver and kidneys demonstrated the characteristic findings of multiple organ failure.

Discussion

We describe a case in which a man with atlanto-axial diastasis sustained a head trauma and subsequently died of an unusual cervical spinal cord injury. The occiput-atlas-axis complex joint participates in rotary movement of the head, and congenital morphological alteration has often caused various clinical and radiological manifestations, particularly when associated with preceding trauma [2, 3, 8, 9, 10, 11]. The transverse ligament originates from the posterior surface of the vertical odontoid process and is attached to the anterior arch of the atlas, thereby preventing backward movement. Burwood [4] reported that occipitalization of the atlas occurs in 0.08%–2.75% of the population [6, 11], and this malformation is accompanied by atlanto-axial diastasis and basilar invagination in 60% of the cases [6].

The mechanism of these complications has been considered as follows: when occipitalization of the atlas occurs (the lateral mass of the atlas is the origin of the transverse ligament and where it adheres to the occipital bone), the result is instability which appears to provoke atlanto-axial dislocation with the simultaneous upward projection of the odontoid process to form a basilar invagination [9]. The odontoid process is moved and tends to extend backwards by dislocation so that the vertebral canal is reduced in size and the cervical spinal cord sustains damage [9].

The atlanto-axial dislocation was diagnosed by X-radiography and revealed that the atlanto-dental distance

(ADD) was more than 2mm [5, 9, 11]. However, Aboulker et al. [1], advocated that even if the ADD was more than 2 mm, diastasis was present if the atlanto-axial joint did not have mobility, as in this case.

In the case of atlanto-axial dislocation, when the cervical region is flexed, the atlas slides forward causing the cervical spinal cord to be compressed between the posterior arch of the atlas and the odontoid process of the axis. But in the case of atlanto-axial diastasis, the vertebral canal is reduced not by a sliding of the atlas but because the odontoid process is located near the cervical canal due to extension and upward deviation.

Therefore the transitional zone between the bulbus and cervical spinal cord is compressed by the upper portion of the odontoid process when the cervical region is flexed [7, 9]. However, the longitudinal section of the cervical vertebrae revealed a fissure at the intervertebral disc that was provoked by hyperextension (Fig. 4). It contradicts the cause of death in atlanto-axial diastasis as generally stated.

It has been concluded that the victim sustained a frontal impact, the cervical region was hyperextended causing the cervical spinal cord to suffer commotion, resulting in edema which further compressed the cervical spinal cord into the narrowed cervical vertebral canal. The resulting vascular insufficiency caused the necrosis and colliquation. Finally, multiple organ failure syndrome occurred resulting in death.

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Fig. 5. The upper level of cervical spinal cord showing necrosis and colliquation (*arrow*)

Fig. 6. Histological findings of a section of the 2nd neural segment showing deviation of the anterior median fissure (*white arrow*) and stenosis of the lumen of the spinal artery (*black arrow*) (Hematoxylin-Eosin staining, $\times 5.5$)