Associated Injuries and Mechanism of Atlanto-occipital Dislocation Caused by Trauma

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Abstract

Injuries associated with traumatic atlanto-occipital dislocation (AOD) leading to death were analyzed in 11 patients, nine injured by traffic accidents, of which five were victims of car-pedestrian accidents. On admission, unconsciousness and respiratory arrest were noted in all patients, and cardiac arrest in nine. Skull and cervical roentgenograms revealed enlargement of the retropharyngeal space due to injury of the vertebral artery or its branches in nine patients, atlanto-axial dislocation (C-1-C-2 separation) in four, and skull fracture in four. Computed tomography demonstrated subarachnoid hemorrhage (SAH) in the upper cervical and posterior fossa in nine patients, fourth ventricular hematoma in seven, and atlas fracture in three. SAH and ventricular hematoma were due to cranio-cervical injury. Other common injuries were injury of face and head excluding the mandibular region in 10 patients, mandibular fracture in three, severe chest injuries in eight, and intraperitoneal bleeding in two. The overall outcome was poor. Nine patients died within 13 hours of admission, one was diagnosed as brain dead 8 days after the accident, and the other one survived in a persistent vegetative state. Early death is probably caused by associated severe injuries, i.e. chest injuries and intraperitoneal bleeding rather than AOD. Although injury of the mandibular region is known to be associated with AOD, head, breast, and abdominal trauma may also lead to neck hyperextension-flexion in various directions. Whatever the direct cause, a distractive force to the cranio-cervical joint by hyper-extension-flexion appears to be important in the mechanism of AOD.

Key words: atlanto-occipital dislocation, atlanto-axial dislocation, trauma, dead on arrival

Introduction

Congenital anomaly or inflammation of the cranio-cervical junction rarely results in atlanto-occipital dislocation (AOD).3) However, AOD is not an uncommon occurrence in patients who die due to trauma caused by traffic accidents. Nine of 112 consecutive autopsies of victims of traffic accidents found AOD,2) and 19 cases of AOD occurred among 76 fatal injuries mainly caused by pedestrian-car accidents.1) Traffic accidents often cause multiple injuries including subarachnoid hemorrhage (SAH) in the posterior fossa, intraventricular hematoma, atlanto-axial dislocation (C-1-C-2 separation), injury of the vertebral artery, mandibular fracture, and severe chest and abdominal injuries,6,8 which frequently cause death. The mechanism causing AOD is believed to be related to mandibular fracture,3-7 but the effect of associated injuries has not been discussed in detail. The relationships between cause and effect among the associated injuries also remain uncertain.

This study analyzed a series of patients presenting with traumatic AOD and other associated injuries.
Materials and Methods

This study included 11 patients aged from 6 to 55 years (mean 30.0 yrs). Nine patients were male and two female. The cause of AOD was traffic accidents in nine cases, including car-pedestrian accidents in five. Nine patients were dead on arrival (cardiac and respiratory arrest on arrival) and the other two were hospitalized without cardiac arrest. The Glasgow Coma Scale (GCS) score was 3 for all patients (Table 1).

Results

Computed tomography (CT) of the brain was performed in nine patients, demonstrating SAH in the posterior fossa in eight patients, fourth ventricular hematoma in seven, lateral ventricular hematoma in five, diffuse brain swelling in five, intracerebral hematoma in two, and pneumocephalus in two. Bone window CT or skull roentgenography revealed skull fracture in four patients. Mandibular fracture and orbital injury were noted in three patients each on skull and facial bone roentgenograms (Table 2).

The initial roentgenograms demonstrated AOD in 10 patients, seven longitudinal and three anterior types (Table 1), enlargement of the retropharyngeal space in nine, atlanto-axial dislocation in four, and fracture of the C-3 spinous process in one. Craniocervical CT was performed in six patients, demonstrating SAH in the upper cervical area in all six, atlas fracture in three, and anterior dislocation of the occipital condyle in two (Table 2).

Hemothorax was noted in five patients, pneumothorax in three, subcutaneous emphysema in three, rib fracture in two, and scapular fracture in two. Intraperitoneal bleeding was seen in two patients and pelvic fracture in three. Femur fracture was noted in three patients and tibial fracture in two (Table 2).

Nine patients died within 13 hours of admission and one patient was diagnosed as brain dead on the 8th day (Case 10). The other patient survived in a vegetative state on a respirator for 20 months after the accident (Case 11) (Table 1).

Representative Cases

Case 5: A 7-year-old boy was struck and dragged by an automobile. He suffered cardiorespiratory arrest and was resuscitated at the accident site. He was hospitalized 27 minutes later. His GCS was 3. He had subcutaneous hematomas of the face, cervical region, and left femur. Roentgenograms showed the longitudinal type of AOD and separation of the posterior elements of the C-1 and C-2 vertebrae (Fig. 1), multiple fractures of the C-1 lamina, occipital bone, facial bones, and left femur. Head CT demonstrated diffuse brain swelling. Cervical post-contrast CT showed hemorrhage surrounding the upper cervical cord and in the right lateral neck at the C-1 and C-2 vertebral levels (Fig. 2). He died 4

Table 1 Summary of clinical courses

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Cause of injury</th>
<th>Finding on admission</th>
<th>Type of AOD</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>GCS</td>
<td>Respiration</td>
<td>Pulsation*</td>
</tr>
<tr>
<td>1</td>
<td>55</td>
<td>F</td>
<td>TA (pedestrian)</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>F</td>
<td>TA (in a car)</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>M</td>
<td>TA (in a car)</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>31</td>
<td>M</td>
<td>TA (pedestrian)</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>7</td>
<td>M</td>
<td>TA (pedestrian)</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>21</td>
<td>M</td>
<td>TA (riding a motor cycle)</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>49</td>
<td>M</td>
<td>TA (in a car)</td>
<td>3</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>22</td>
<td>M</td>
<td>fall</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>6</td>
<td>M</td>
<td>TA (pedestrian)</td>
<td>3</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>54</td>
<td>M</td>
<td>accident while working</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11</td>
<td>7</td>
<td>M</td>
<td>TA (pedestrian)</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Pulsation of the femoral artery. anterior: anterior displacement of occiput, longitudinal: axial separation of occiput and atlas, TA: traffic accident, +: positive, -: negative.
hours after hospitalization following tracheostomy and open cardiac massage.

**Case 9:** A 6-year-old boy was hit by a car and thrown 8 m from the collision site onto the roadway. He was hospitalized 17 minutes after the accident. His GCS was 3 and his pupils were fully dilated. No respiration was noted but some weak pulsations were present. Oral intubation was immediately performed. Lacerations in the forehead and right parietal scalp, bloody cerebrospinal fluid rhinorrhea, and a subcutaneous hematoma in the left lower extremity were noted. Cervical roentgenograms showed AOD of

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the longitudinal type and atlas fracture with enlarged retropharyngeal space (Fig. 3). Head and neck CT demonstrated SAH in the posterior fossa and upper cervical area (Fig. 4). A cervical swelling not noted on arrival gradually enlarged, and he died 13 hours after admission.

**Case 10:** A 54-year-old male caught his face between an electric-light pole and a machine while working on a moving truck. His neck was distracted. He showed cardiac and respiratory arrest on arrival 20 minutes after the accident but was resuscitated. He had subcutaneous hematomas in the bilateral cheeks. Head and neck roentgenograms revealed AOD of the longitudinal type with an enlarged retropharyngeal space and bilateral mandibular fractures. CT showed severe SAH predominantly in the posterior fossa and diffuse brain swelling with ventricular hemorrhage. He died 21 days after admission.

**Case 11:** A 7-year-old boy was struck by an automobile. He showed cardiac and respiratory ar-
rest on arrival with non-reactive dilated pupils. He had contusions in the mid-frontal scalp, a subcutaneous swelling in the nuchal region, and an open femur fracture. His pulsation was restored following cardiopulmonary resuscitation with oral intubation. Cervical roentgenograms showed AOD of the anterior type (Fig. 5 left). Head and cervical CT revealed bilateral cerebral edema, slight ventricular hematomas, subarachnoid clots around the lower brain stem and upper cervical cord, and anterior dislocation of the occipital condyle (Fig. 6).

Neck traction of 1 kg was performed to stabilize the occipito-atlantal joint. After this procedure, the joint was markedly elongated (Fig. 5 right). Traction was discontinued, and his head and neck were immobilized with sandbags and towels. His pupils were 1.5 mm in diameter on the next day and slowly reactive. He was responsive to deep painful stimuli on the 3rd day after admission. His blood pressure was normalized on the 5th day. Tracheostomy was performed on the 6th day. Facial movements in his nose wings were noted on the 9th day. Head CT on the 15th day demonstrated ventricular dilatation, bifrontal subdural effusion, and low density areas in the bilateral basal ganglia. These findings were compatible with severe cerebral hypoxia (Fig. 7). Magnetic resonance (MR) imaging demonstrated cord compression at the craniocervical junction with posteriorly dislocated dens, and an epidural hematoma in the vicinity of the occipital condyle (Fig. 8).
Fusion from the occiput to C-3 was performed 4 weeks after the accident using a metal material. After surgery, spontaneous respiration was restored to several times per minute, necessitating use of a respirator. He remains in a vegetative state for 20 months after the injury.

Discussion

Severe injuries associated with AOD were probably the cause of death in Cases 1-4, 6, and 7. In contrast, Cases 9-11 had no other severe injuries except to the cervicocranial region, and survived longer.

Atlanto-axial dislocation occurred in four patients. The cranium, atlas, and axis constitute a single junctional unit, and the tectorial membrane and alar ligaments are important anatomical structures in the unit. Transection of these produces instability of the junctional unit. This observation suggests that C-1-C-2 separation was frequent in patients with AOD caused by trauma.

Trauma to the mandibular region is believed to be related to AOD, and occurred in five of our patients (fractures in 3 and mandibular lacerating wounds without fracture in 2). Four of the other six patients had skull fracture (including basal fracture with cerebrospinal fluid rhinorrhea). Ten patients had some wounds of the face and head other than in the mandibular region. The mandibular bone is closer to the cervicocranial junction than the other regions of face and head, so direct force on the mandibular region could cause AOD more easily. A direct force on the head and face is probably also related to AOD. Forces acting on the chest and abdominal area could cause marked hyperextension and hyperflexion of the neck in all directions to produce a distractive force on the neck, so resulting in AOD.

Case 10 had only mandibular fractures, and so these simple injuries caused AOD. This patient had SAH in the upper cervical cord and posterior fossa, and hematomas of the fourth and lateral ventricles due to craniocervical injury. The other patients had SAH, predominantly in the posterior fossa. No patient had lateral ventricular hematoma without fourth ventricular hematoma and SAH in the posterior fossa. SAH was more common than fourth ventricular hematoma, which was more frequent than lateral ventricular hematoma. These observations indicate that ventricular hematoma and SAH were due to injury of the craniocervical junction.

Eight of 11 patients had severe head injuries, including cerebral contusion and skull fracture, but no intracranial epidural or subdural hematoma was associated with AOD. This may be due to hypotension or heart failure as discussed later.

Enlargement of the retropharyngeal space was frequently associated with AOD. CT revealed neck swelling due to injury of the vertebral artery or its branch in Case 5 (Fig. 2). Remarkable enlargement of retropharyngeal hematoma causes neck swelling, but such marked neck swelling was not common in patients with AOD. The extent of hematoma may have been limited by hypotension or heart failure secondary to AOD, chest injury, traumatic shock, etc. We found that Case 9 demonstrated gradual enlargement of cervical hematoma after normalization of blood pressure. Schneider and Schemm pointed out the importance of vascular insufficiency of the brain stem due to vertebral artery injury, rather than direct brain stem injury. We emphasize that such vertebral injuries may have severe consequences.

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