Traumatic Atlanto-occipital Dislocation with Long-term Survival
—Case Report—

Narihito YAMAGUCHI, Kiyonobu IKEDA, Jun ISHISE, and Junkoh YAMASHITA

Department of Neurosurgery, Kanazawa University School of Medicine, Kanazawa

Abstract

A 6-year-old boy presented with traumatic atlanto-occipital dislocation due to a traffic accident. Lateral spinal tomography showed anterior misalignment of the craniocervical junction. Magnetic resonance imaging demonstrated associated cervicomedullary injury. He underwent an occipital-cervical fusion with implantation of a spinal cord electrical stimulation unit. His consciousness level improved, but he remained quadriplegic and dependent on a respirator 2 years after the injury.

Key words: atlanto-occipital dislocation, magnetic resonance imaging, cervical spinal cord stimulation

Introduction

Traumatic atlanto-occipital dislocation (AOD) is a serious and usually fatal injury. Survival after traumatic AOD is rare. The absolute incidence of traumatic AOD is unknown because of the extremely high rate of resultant death, but review of several series of trauma victims who died at the accident scene suggests that AOD accounts for up to one-third of fatal cervical spine injuries.

The first case of traumatic AOD, in a 19-year-old male injured in a gymnastics accident, was reported in 1908. However, most survivors of this injury have been reported in the last decade, reflecting the improvement in emergency medical services. The survivors usually present with severe and persistent neurological deficits. There were three times more children than adults in a series of patients with AOD who survived over 48 hours. Most of these children were the victims of pedestrian-motor vehicle accidents, but the implications are not known.

We report a 6-year-old boy with AOD who has survived for more than 2 years with severe neurological deficits and describe his unique magnetic resonance (MR) imaging findings.

Case Report

A 6-year-old boy was struck by a car while crossing a street on a bicycle on May 9, 1992. One of the authors (J.I.) was passing by chance and examined him almost immediately after the accident. He was flaccid and apneic. Artificial respiration was begun and maintained until he was intubated in the emergency room of our hospital.

Neurological examination revealed that he was unresponsive, non-breathing, and had no voluntary or spontaneous movement of the arms and legs. His pupils were mitotic in the mid position and unreactive. He had no corneal, gag, or oculocephalic reflexes. His consciousness level was Glasgow Coma Scale 3.

Lateral radiographs of the cervical spine initially suggested AOD. Spinal tomography demonstrated AOD with abnormal separation of the occipital condyles and superior facets of the atlas. Anterior misalignment of the craniocervical junction had a Powers' ratio of 1.1 (Fig. 1). Computed
tomography (CT) of the head showed no intracranial mass lesions, but traumatic subarachnoid hemorrhage surrounding the brainstem and a hematoma in the third and fourth ventricles with enlargement of the lateral ventricles were present (Fig. 2). T₁-weighted sagittal MR imaging revealed dorsal displacement of the cervicomedullary junction and a contusional hematoma at the pontomedullary junction (Fig. 3).

Within 24 hours of injury, initial extraventricular drainage was implanted. He was taken to the intensive care unit after the operation, and his neck immobilized in the neutral position with sandbags and a firm collar. On June 2, a ventriculoperitoneal shunt was implanted. MR imaging on June 30 revealed enlargement of the fourth ventricle and a hematoma scar at the pontomedullary junction. On July 21, reconstructive repair of the AOD using a metal frame (occipito-C-1 through C-4 fusion) was combined with implantation of a fourth ventricle-peritoneal shunt and implantation of a spinal cord electrical stimulation device (PISCES set; Medtronicus, Minneapolis, Minn., U.S.A.) in the cervical epidural space (Fig. 4). Electrical stimulation was started 1 week after the

Fig. 1 Cervical spine tomogram on admission showing AOD with anterior misalignment of the craniocervical junction. The basio-dental interval is 15 mm and the Powers' ratio is 1.1.

Fig. 2 CT scan on admission showing traumatic subarachnoid hemorrhage in the cisterns surrounding the brainstem and hematoma formation in the fourth ventricle.

Fig. 3 T₁-weighted sagittal MR image on admission demonstrating a contusional hematoma in the pontomedullary junction and dorsal displacement of the cervicomedullary junction.

Fig. 4 Postoperative cervical spine radiograph showing the occipital-cervical fusion construction and the implanted electrode for stimulation in the extradural space at the C-2 to C-4 levels.
operation.

No neurological improvement was observed in the following 4 months. However, early in December pursuing eye movement was recognized. In March 1993, he began to respond to orders for eye opening or closing, and show feelings of joy and anger on his face. In May 1994, he was totally quadriplegic, and required gastric tube feeding and respiration. Examination of the awake patient found total absence of pharyngeal sensation, inability to swallow, no activity in the trapezius or sternomastoid muscles, and a flaccid tongue.

Discussion

Traynelis et al. suggested that there are three specific types of AOD: Type I involves anterior displacement of the occiput with respect to the atlas; Type II is primarily a longitudinal distraction with separation of the occiput from the atlas; and Type III is posterior displacement of the occiput with respect to the atlas.

Several different radiological criteria have been developed, frequently specific to a particular subtype of AOD. The BC/AO ratio proposed by Powers et al. has been observed in the 2 years since injury.

AOD is more common in children than adults. The craniovertebral junction of children does appear to be inherently less stable, possibly due to the small occipital condyles and an almost horizontal plane of articulation between the cranium and the atlas.

The extremely high incidence of mortality associated with AOD is probably the result of medullary injury. Medullary injury may occur as a direct result of the injury as in our patient or due to an extradural hematoma. Less severe brainstem insults may cause cardiopulmonary instability leading to bradycardia, irregular respirations, or apnea.

The use of traction in the initial management of patients with AOD is controversial, as neurological structures may be further injured by traction. In most cases, traction results in longitudinal distraction, and is therefore not indicated for patients whose spinal column is in good alignment and with primary longitudinal dislocation (Type II).

Traction may be indicated in anterior (Type I) or posterior (Type III) displacement to realign the bone structures and decompress the brainstem or spinal cord.

AOD limited to ligamentous tear without bony fracture may achieve partial healing, resulting in chronic instability if treated only by simple external immobilization. Therefore, definitive treatment by posterior fusion of the occiput to the upper cervical spine should be undertaken once the patient has been adequately stabilized.

Fusion has been successfully accompanied with wire and/or autogenous bone graft.

Recently, cervical spinal cord stimulation has been used to treat patients with a prolonged period of unconsciousness with some success, although the actual mechanism raising the consciousness level has not been clarified. We implanted a spinal cord electrical stimulation unit 10 weeks after the injury and started stimulation 1 week later. At 11 months after the injury (8 months after beginning stimulation), we recognized an obvious increase in the patient’s consciousness level, although the relationship between the treatment and increased consciousness was unclear. However, his extremities have been flaccid without contraction and no decubitus has been observed in the 2 years since injury.
Beneficial effects have been achieved by direct and/or indirect action of cervical spinal cord stimulation on the spinal cord and brainstem in patients with spastic hemiparesis or peripheral vascular disease.

Two years postinjury, he was totally quadriplegic, requiring tube feeding and continuous respirator support. His physical state is like the locked-in syndrome with little chance of recovery. Modern emergency resuscitation and medical care techniques may lead to an increase in such patients, and this presents serious social and medico-ethical problems.

References


2) Blackwood NJ: Atlanto-occipital dislocation. A case of fracture of the atlas and axis, and forward dislocation of the occiput on the spinal column, life being maintained for thirty-four hours and forty minutes by artificial respiration, during which a laminectomy was performed upon the third cervical vertebra. Ann Surg 47: 654–658, 1908

3) Bools JC, Rose BS: Traumatic atlantooccipital dislocation: Two cases with survival. AJNR 7: 901–904, 1986

4) Bracale GC, Selvetella L, Mirabile F: Our experience with spinal cord stimulation (SCS) in peripheral vascular disease. PACE 12: 695–697, 1989


Address reprint requests to: N. Yamaguchi, M.D., Department of Neurosurgery, Asanogawa General Hospital, 83 Naka, Kosaka-machi, Kanazawa 920, Japan.