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Treatment for Atlanto-occipital Dislocation, Vertical Atlanto-axial Dislocation, and Acute Subdural Hematoma Presenting with Out-of-hospital Cardiac Arrest: A Case Report

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Abstract

A male patient in his 50s had a head-on collision while driving. Prehospital emergency services recorded pulseless electrical activity on an electrocardiogram, and chest compressions were initiated. Before hospital arrival, return of spontaneous circulation was achieved after 17-min resuscitation during transport. His Glasgow Coma Scale score was 6, with unequal-size pupils unresponsive to light stimuli. A head computed tomography scan revealed a left acute subdural hematoma with a marked midline shift, and computed tomography of the cervical spine showed that the atlanto-occipital and atlanto-axial joint spaces were significantly widened. Initially, an emergency decompressive craniectomy for hematoma evacuation was performed, followed by posterior cervical fixation surgery in the subacute phase. After brain surgery, neurocritical care management was implemented for brain protection. Cervical spine magnetic resonance imaging revealed multiple ligament injuries at the craniovertebral junction, which confirmed the diagnosis of atlanto-occipital and atlanto-axial dislocation. On day 9, posterior fixation from the occiput to the fourth cervical vertebrae was performed. Subsequently, he was transferred to a rehabilitation hospital on day 45. No neurological sequelae were noted except for the neck rotation limitations due to the fixation surgery, and he could return to his previous job. Although craniovertebral junction ligamentous injuries are rare, they may coexist with severe traumatic brain injury. A careful reading of preoperative images focusing on the inter-joint space is important to detect craniovertebral junction ligamentous injuries in patients with traumatic brain injury inflicted with high-energy trauma.

Keywords: atlanto-occipital dislocation, atlanto-axial dislocation, traumatic brain injury, cardiac arrest, craniovertebral junction

Introduction

In head and neck trauma, the craniovertebral junction (CVJ) from the occipital bone (C0) to the axis (C2) is relatively easily injured due to the high mobility of this region. These injuries account for approximately one-third of all cervical spine injuries.[1,2] Although atlanto-occipital dislocation (AOD) and atlanto-axial dislocation (AAD) are rare, they can be fatal and should not be misdiagnosed or overlooked.[3,4] AOD can be diagnosed using several measurement methods, such as Powers' ratio, X-line method, basion-dens interval (BDI), basion-axis interval (BAI), and condyle-C1 interval (CCI), but relying on a single measurement has been considered insufficient.[1,4] Approximately 20% of AOD cases had no focal neurological findings during hospital admission, which may lead to delayed diagnosis.[4] Approximately 70% of AAD cases involve horizontal dislocations.[5] By contrast, vertical AAD is rare, accounting for approximately 1% of cervical spine injuries imaged with computed tomography (CT), but has a high mortality rate.[3] The atlanto-dental interval (ADI), the distance between the posterior surface of the atlas and the anterior surface of the odontoid process, is widely known but used in AAD of horizontal dislocation,[5] and the widespread use

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of a measurement standard to detect vertical AAD seems necessary. However, there is a risk of neurological deterioration or death following inadvertent head traction, particularly in vertical AAD.[3,6]

We experienced a case of AOD/AAD concomitant with acute subdural hematoma (ASDH). After emergency decompressive craniectomy for the ASDH, we performed posterior fixation for the AOD/AAD during the subacute phase, which enabled a safe and vigorous rehabilitation therapy, leading to a favorable functional outcome. Additionally, we discuss the diagnostic and imaging characteristics of AOD and AAD.

Case Report

A male patient in his 50s with a history of insomnia sustained injuries when his vehicle veered into the opposite lane at 50 km/h and collided head-on with a van. He had worn a seat belt, and the airbag had been deployed. Emergency medical services obtained an electrocardiogram at the scene, which showed pulseless electrical activity; thus, chest compressions were initiated. The estimated no-flow time, defined as the interval from cardiac arrest to cardiopulmonary resuscitation initiation, was between 3 and 9 mins, based on the approximately 6 mins that elapsed from the time of injury to the arrival of emergency medical services, followed by an additional 3 mins required to extricate him from the vehicle and start resuscitation. Additionally, a cervical collar was applied, and peripheral venous access was established during resuscitation, followed by a single intravenous administration of 1 mg of adrenaline. After 17 mins of resuscitation efforts during transport, a return of spontaneous circulation (ROSC) was achieved before arriving at the hospital. During ROSC, the vital signs were a Japan Coma Scale of III-300, gasping spontaneous respiration requiring assisted ventilation, pulse rate of 140 bpm, and blood pressure of 170/90 mmHg. The pupil sizes were 3 and 6 mm on the right and left, respectively, with no light reflex on either side. At hospital arrival, his Glasgow Coma Scale was 6 (E1V1M4), and pupil sizes were the same. The heart rate was 110 bpm, blood pressure was 148/108 mmHg, and SpO_2 was 97% at 10 l/min oxygen under assisted ventilation with a bag-valve mask. Subsequently, oral intubation was performed for airway protection, and mechanical ventilation was initiated. Physical examination revealed a contusion on the left side of the head, a subcutaneous hematoma on the right lower abdomen caused by the seat belt, and abrasions on the right forearm and left lower leg. Neither intrathoracic or intraperitoneal hemorrhage nor external bleeding was observed on ultrasound examination. Whole-body CT was performed following our institutional protocol for high-energy trauma cases.[7] Head CT showed a left ASDH with a maximum thickness of 18 mm with midline shift (Fig. 1A and B). Cervical spine CT showed significant widening of the atlanto-occipital and atlanto-axial joint spaces (Fig. 2A and B). CCI was 2.3 and 2.4 mm on the right and left (>2.0 mm) sides, respectively, and modified CCI was 3.6 and 3.0 mm on the right and left sides (>2.5 mm), respectively. BDI was 12.5 mm (>12 mm). The lateral mass index (LMI) was 4.2 and 5.8 mm on the right and left side (>2.6 mm), respectively, all of which were positive (Fig. 3A-C, E and H).[8] BAI of 6.8 mm (<12 mm), and Powers' ratio of 32.5/42.2 = 0.74 (<1) were negative, and no violation of the X-line method was observed (Fig. 3D, F and G).[8] The cervical spine has no fractures. In addition, body CT showed bilateral pulmonary contusions.

Immediately after admission, an emergency decompressive craniectomy with hematoma evacuation was performed (Fig. 1C and D). Due to the CVJ injury, surgery was performed in the supine position with the cervical collar, with utmost attention to immobilize the cervical spine. He underwent intensive care management postoperatively for severe traumatic brain injury (TBI) and post-cardiac arrest syndrome. Those included target temperature management (TTM) using an intravascular cooling catheter using Thermogard XP (Asahi Kasei Zoll Medical, Tokyo) for neuroprotection with a target temperature of 36°C for 72 h, sedation with midazolam and propofol, analgia with fentanyl, early posttraumatic seizure prophylaxis with levetiracetam, and mechanical ventilator management for $PaCO_2$ adjustment at a target from 35 to 38 mmHg.[9,10] Cervical spine was immobilized using a Philadelphia collar. The intra- and postoperative findings indicated no significant cerebral swelling during or after the craniectomy at the external decompression site. Consequently, due to concerns about infection risk and potential brain injury associated with intracranial pressure (ICP) sensor placement, we used the estimated ICP derived from transcranial Doppler assessments of the middle cerebral artery blood flow as reference for management. Sedatives were gradually tapered starting on the fourth day of hospitalization. Cervical spine magnetic resonance imaging (MRI) performed on the fifth day of hospitalization showed high signal intensity on fat-suppressed T2-weighted images in the bilateral atlanto-occipital joints, bilateral atlanto-axial joints, and posterior atlanto-occipital membrane complex (Fig. 4A-C).[4] Injury to the transverse ligament of the atlas was not apparent, and the cervical cord had no injuries. In addition, the CVJ was not fractured, and the injury pattern was primarily considered a distraction injury.

Based on these findings, we confirmed the diagnosis of AOD and AAD due to multiple ligamentous injuries of the CVJ without spine fractures. This was a case of severe TBI and cervical spine injury complicated by traumatic cardiac arrest, and the prognosis was initially poor. However, head CT findings after TTM revealed non-severe brain damage, and the possibility of a good neurological prognosis was promising. Therefore, posterior fixation from the occipital bone to the fourth cervical vertebrae with iliac crest graft

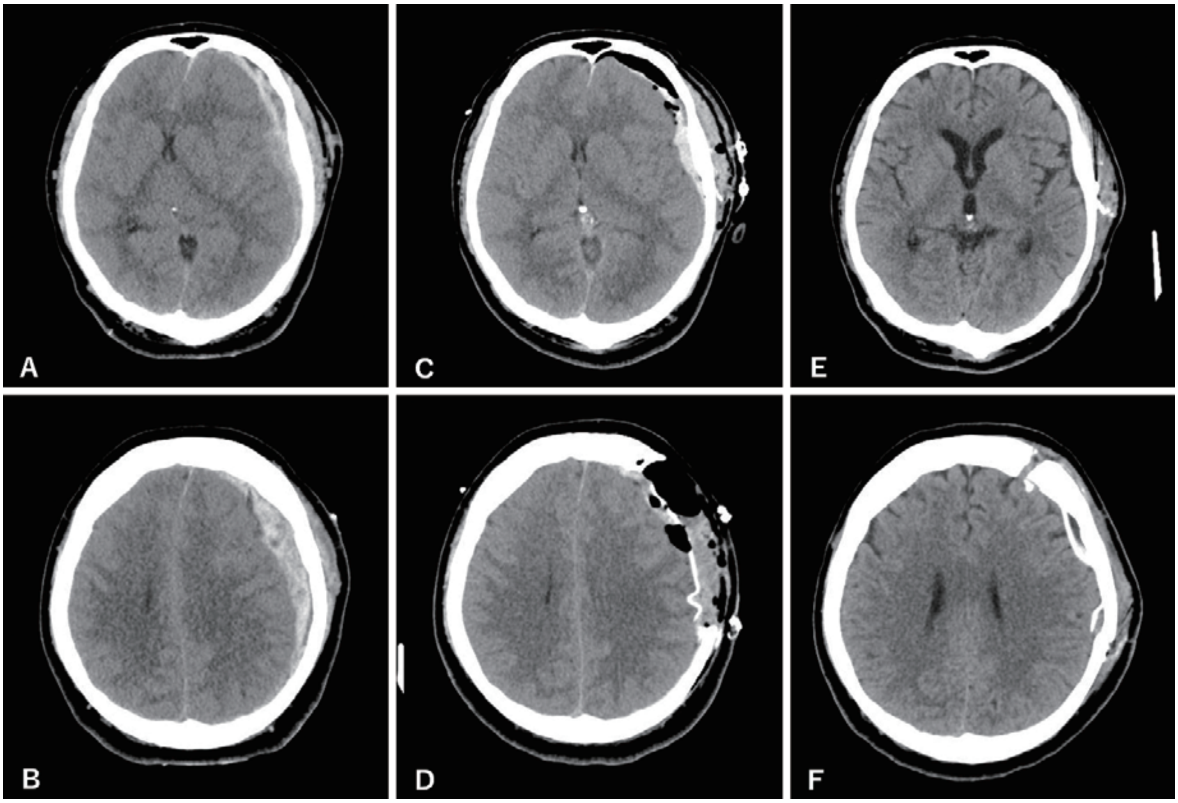

Fig. 1 Head CT scan at arrival and postoperatively. A, B: Head CT showing a left acute subdural hematoma at arrival. C, D: Head CT after decompressive craniectomy for hematoma removal. E, F: Head CT after cranioplasty using autologous.
CT: computed tomography

was performed on the ninth day of hospitalization (Fig. 2 C-G). On the eighth day of hospitalization, tracheostomy was performed simultaneously with the posterior fixation surgery to prevent aspiration although he could obey simple commands. He was weaned off the mechanical ventilator on the 10th day of hospitalization. On the 12th day of hospitalization, he was discharged from the intensive care unit to the ward. Cranioplasty was performed on the 30th day (Fig. 1E and F), and the tracheostomy tube was removed on the 39th day. On the 45th day, he was transferred to a rehabilitation hospital, and his neurological prognosis at 6 months after the injury was rated as modified Rankin Scale (mRS) of 1 and Glasgow Outcome Scale (GOS) of good recovery, allowing the patient to return to his previous job. All participants provided consent for this report.

Discussion

Among cervical spine injuries, AAD has a frequency of 2.7%, whereas AOD is even rarer at approximately 1%.[2] Previously, AOD had a mortality rate of up to 100%,[11] but survival rates have been improving due to the advancement of intensive care medicine. Nevertheless, many cases resulted in death before hospital arrival, and survivors were frequently left with neurological sequelae.[11] AOD was also reported as the cause of 6%-8% of traffic accident fatalities.[4] Similarly, for AAD, many patients die before arriving at the emergency department.[6]

AOD is classified using the Traynelis classification: type I, anterior dislocation; type II, vertical dislocation; and type III, posterior dislocation.[4] The appropriate measurement parameters differ for each type.[4] For type II, BDI and CCI are sometimes considered suitable; however, CCI and modified CCI are reported to have high sensitivity and specificity.[8] Using CCI, AOD is diagnosed when the measurement is >2.0 mm. In contrast with modified CCI, the diagnosis is made when the distance between the condyle and the C1 lateral mass socket on axial images is >2.5 mm. Anterior dislocation, accounting for most cases of AAD, is often defined by ADI enlargement.[5] However, diagnosis using ADI alone can be challenging for vertical AAD, such as in the present case. Instead, LMI, defined as the distance between the first and second cervical vertebral facet joints, is used to diagnose vertical AAD if LMI >2.6 mm.[12] However, MRI is recommended as an adjunct for diagnosis. These measurement parameters were also useful in case, and subsequent MRI confirmed ligamentous injuries. Nevertheless, obtaining an MRI during the initial treatment phase is often challenging, making knowledge of

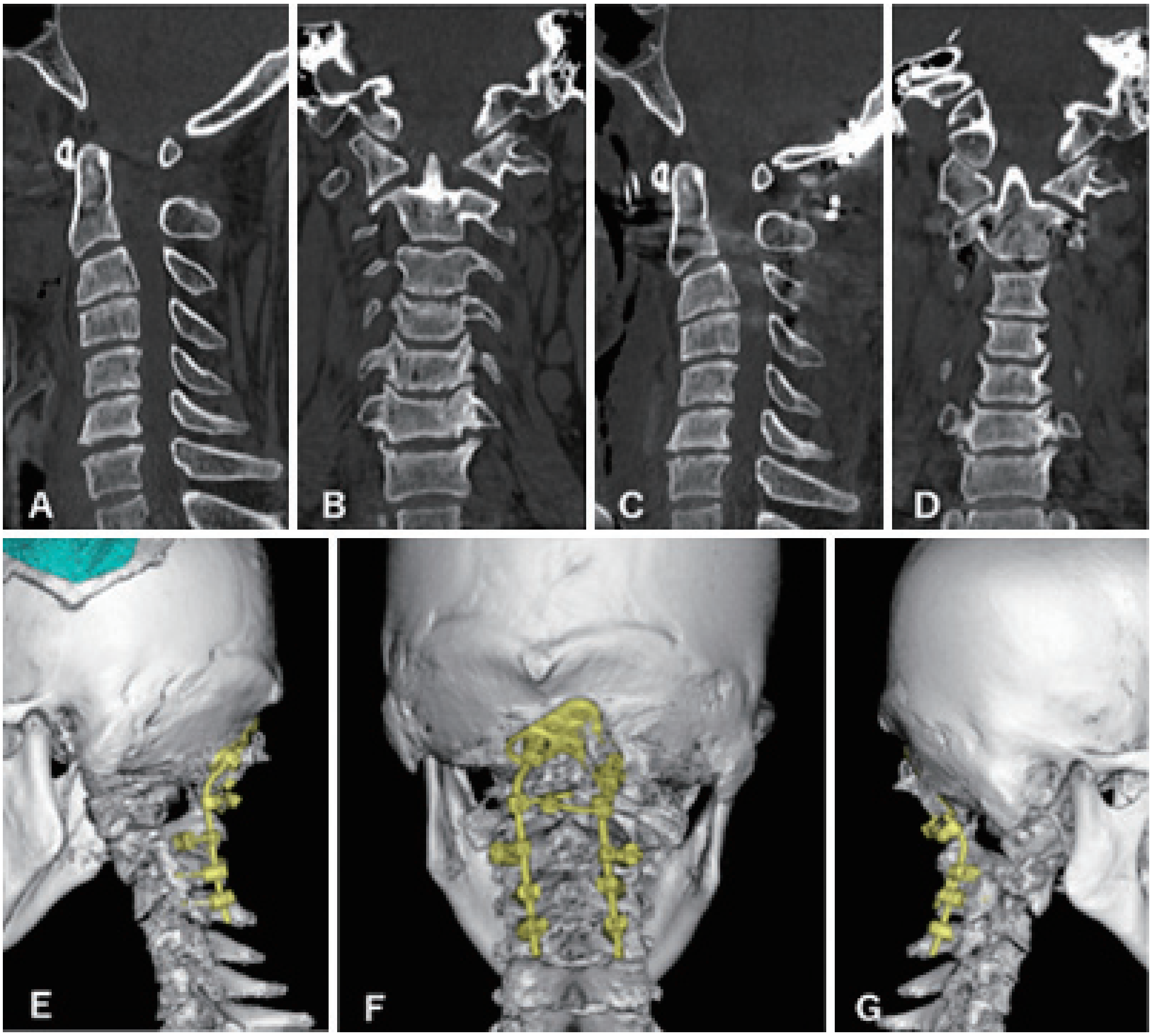

Fig. 2 Cervical spine CT at arrival and postoperatively. A, B: Cervical spine CT at arrival showing AOD/AAD (A: sagittal, B: coronal). Details are described in Fig. 3. C, D: Cervical spine CT after posterior spinal fusion (C: sagittal, D: coronal). E, F, G: 3D of cervical spine CT after a posterior spinal fusion.
AAD: atlanto-axial dislocation; AOD: atlanto-occipital dislocation; CT: computed tomography

diagnostic tools in CT necessary for those involved in early trauma care.

In moderate to severe TBI, GCS motor score and pupillary reaction have been reliable predictors of neurological outcomes.[13] Moreover, similar associations have been suggested in cases of severe TBI following cardiac arrest.[14] In contrast, the present case is considered a rare instance with a favorable neurological prognosis, with mRS of 1 and GOS of good recovery, despite the absence of bilateral pupil reaction from the initial treatment phase. However, ICP monitoring might have been performed for optimal neurocritical care based on the guidelines for severe TBI.[9,10] Although determining the definitive cause of cardiac arrest was challenging in the present case, we suspected that AOD and/or AAD rather than the TBI might have been the cause based on the imaging findings of the TBI. Although thorough evaluation for cardiac function physical examination has not been possible, fixation surgery was performed uneventfully in the subacute phase when the general condition was stable as symptomatic AOD or AAD that led to cardiac arrest.

The present case showed the necessity for proactively suspecting the presence of ligamentous CVJ injuries based on the initial CT findings and not being solely focused on severe TBI. Consistent management from continued cervical spine protection to fixation surgery was maintained, leading to a favorable outcome. Particularly in patients with high-energy trauma accompanied by CVJ injuries, treatment and management of coexisting life-threatening injuries of other locations may take priority. Nevertheless,

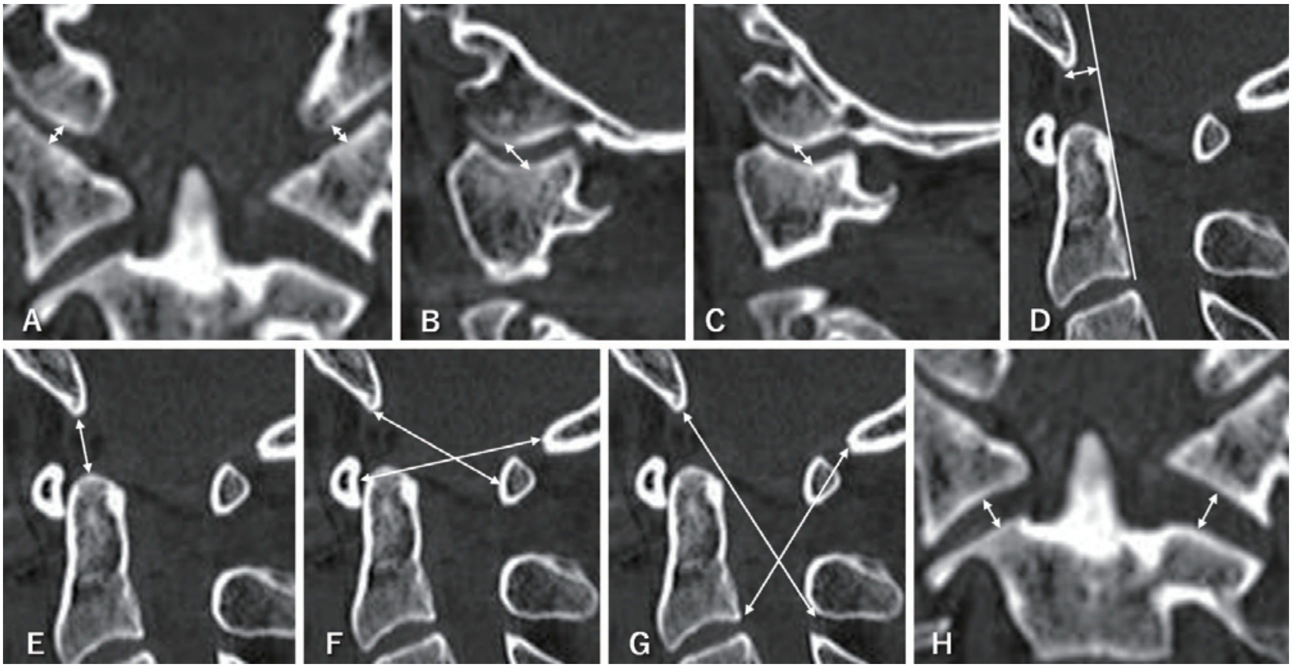

Fig. 3 Measurements to determine the presence of AOD/AAD in the cervical spine CT on arrival. A: CCI was 2.3 and 2.4 mm on the right and left, respectively. The right (B) and left (C) modified CCIs were 3.6 and 3.0 mm, respectively. D: BDI was 12.5 mm. E: BAI was 6.8 mm. F: The Powers' ratio was 0.74 (32.5/42.2). G: X-line method was described. These lines were crossed and normal. H: LMI was 4.2 and 5.8 mm on the right and left, respectively.
AAD: atlanto-axial dislocation; AOD: atlanto-occipital dislocation; BAI: basion-axis interval; BDI: basion-dens interval; CCI: condyle-C1 interval; CT: computed tomography; LMI: lateral mass index

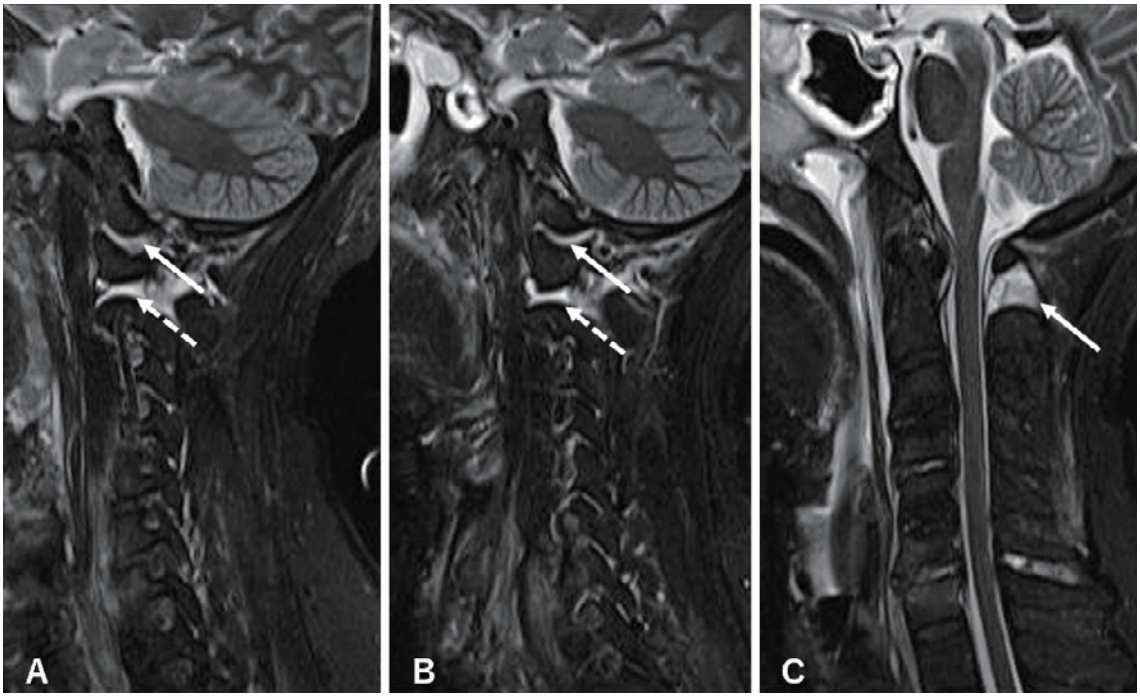

Fig. 4 Preoperative cervical spine MRI. MRI with fat-suppressed T2-weighted sequence. A: High intensity between C0 and C1 on the right side. The dashed arrow shows high intensity between C1 and C2 on the right side, indicating ligament injuries. B: This was a similar finding on the left side as in A. C: Injury of the posterior atlanto-occipital membrane complex.
MRI: magnetic resonance imaging

from the initial treatment phase until the completion of definitive care, these patients should be managed with the awareness of the CVJ injury, including patient positioning, ward management, and regular imaging follow-ups.

Conflicts of Interest Disclosure

All authors have no conflict of interest.

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