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ABOUT COVER

Editorial Board Member of *World Journal of Clinical Cases*, Dr. Quach is an Associate Professor of Gastroenterology at the University of Medicine and Pharmacy at Hochiminh City, Viet Nam, where he received his MD in 1997 and his PhD in 2011. Dr. Quach has published more than 100 reviews and original papers in local and international journals. He has received several awards, including Outstanding Presentation at the Biannual Scientific Congress of Vietnamese Nationwide Medical Schools, Medal of Creativeness from the Vietnamese Central Youth League, etc. Currently, he serves as a Vice President of the Vietnam Association of Gastroenterology and Secretary General of the Vietnam Federation for Digestive Endoscopy. (L-Editor: Filipodia)

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Early reoccurrence of traumatic posterior atlantoaxial dislocation without fracture: A case report

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Abstract

BACKGROUND

In general, atlantoaxial dislocation is rare due to the stability of the C1-C2 complex. Traumatic atlantoaxial dislocations are usually anterior and accompanied by odontoid fractures. Posterior atlantoaxial dislocations are rare, and complete posterior dislocation without associated fracture is even more rare. A case of early recurrence of posterior atlantoaxial dislocation without fracture being in therapy of first closed reduction and then open reduction has not been previously reported.

CASE SUMMARY

A 45-year-old female presented with traumatic posterior atlantoaxial dislocation (TPAD) of C1-C2 without associated fractures, and Frankel Grade B spinal cord function. She was successfully managed by immediate closed reduction under skull traction. Unexpectedly, 17 d later, re-dislocation was discovered. On day 28, closed reduction was performed as before but failed. Then, open reduction and posterior internal fixation with autologous iliac bone grafts was performed. By 6 mo after surgery, atlantoaxial joint fusion was achieved, and neurological function had recovered to Frankel Grade E. At 12 mo follow-up, she had lost only 15° of cervical rotation, and atlantoaxial complex instability in joint flexing and extending were no longer observed under fluoroscopy.

CONCLUSION

Early assessment of transverse ligament is critical for TPAD without fracture avoiding re-dislocation after closed reduction.

Key Words: Reoccurrence; Traumatic posterior atlantoaxial dislocation; Without fracture; Transverse ligament; Case report

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Core Tip: Traumatic posterior atlantoaxial dislocations (TPADs) without fracture are rare. In this article, we present a 45-year-old female with early recurrence of TPAD of C1-C2 without associated fractures. The patient first underwent closed reduction and then open reduction, internal fixation, and interfacial fusion. Excellent results were achieved. In conclusion, early assessment of the condition of the transverse ligament is critical for the prevention and early discovery of the re-dislocation of TPADs.

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INTRODUCTION

Atlantoaxial dislocations are classified as anteroposterior, rotatory, central, or mixed dislocations, based on the direction and plane of dislocation^[1]. Traumatic atlantoaxial dislocations usually are anterior and accompanied by odontoid fractures^[2-5]. Posterior atlantoaxial dislocations are rare, and complete posterior dislocation without fracture is rarer still^[6-17]. Here, we present the first reported case of early recurrence of posterior atlantoaxial dislocation without fracture and the necessary treatments.

CASE PRESENTATION

Chief complaints

A 45-year-old female was admitted to our hospital with light coma and difficulty breathing 3 h after being struck in an automobile accident.

History of present illness

The patient, who was struck in the back by an automobile, was transferred from a local county clinic to our hospital 3 h after the accident. She presented with light coma, difficulty breathing, lacerations on the face and scalp and stiffness in the neck. Emergency intubation was performed, followed by computed tomography (CT) and magnetic resonance imaging (MRI) examinations. CT scans revealed posterior atlantoaxial dislocation (Figure 1). Hence, the patient was admitted to our hospital and was rescued immediately.

Physical examination

Neurological assessment showed cerebral brain stem injury and Frankel Grade B spinal cord function (only sensory function below the injury level)^[18].

Laboratory examinations

No abnormalities on preoperative examination.

Imaging examinations

CT scans revealed posterior atlantoaxial dislocation and no evidence of fracture. The odontoid process was situated anterolateral to the anterior arch of C1 with facet joint dislocation of C1-C2. The atlanto-occipital joint, body and posterior arch of C2, occipital condyles, and the remainder of the visualized cervical spine appeared normal. A T2W MRI midsagittal view of the cervical spine demonstrated no cord compression or intramedullary cord signal abnormality at the level of the atlantoaxial dislocation, with bending in the cervical spinal curvature. On a T1W MRI coronal view of the cervical spine, the membrana tectoria/apical and transverse ligaments at the lateral attachment of the odontoid and the superior transverse ligament were visibly torn (Figure 1).

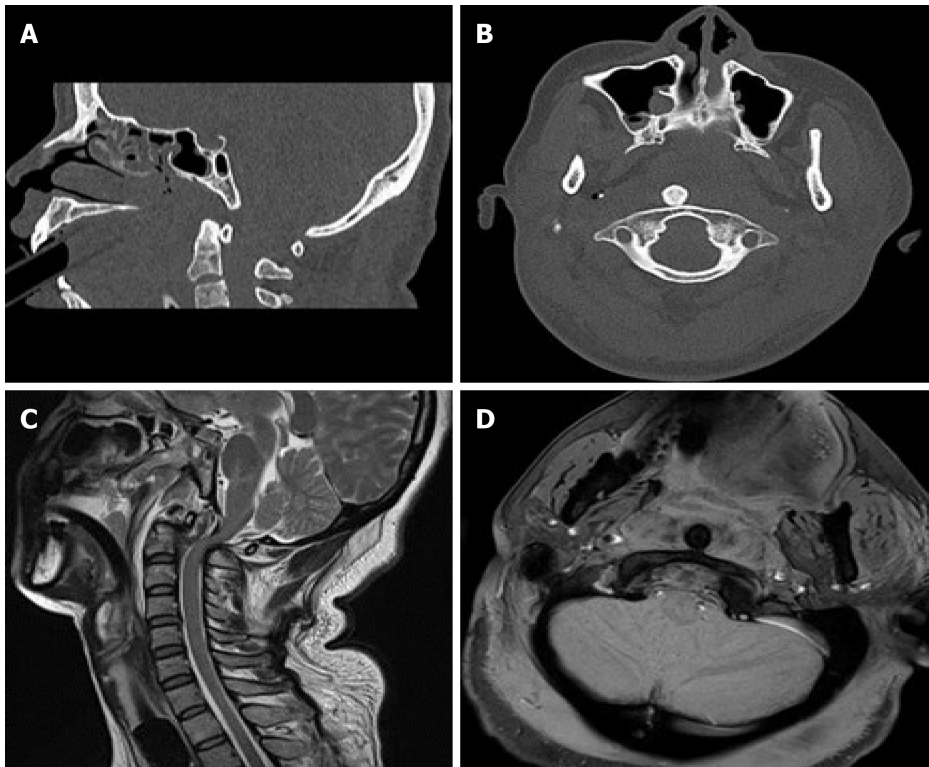


Figure 1 Radiographs on admission. A and B: Computed tomography scans of midsagittal and coronal sections showing posterior atlantoaxial dislocation without fracture; C and D: T2W magnetic resonance imaging (MRI) midsagittal and T1W MRI coronal views showing signal changes with no cord edema but transverse ligament rupture.

FINAL DIAGNOSIS

Traumatic posterior atlantoaxial dislocation (TPAD) without associated fracture but with neurological deficit.

TREATMENT

Under general anesthesia (GA), closed reduction under skull traction was attempted immediately, aided by fluoroscopic viewing and neurological monitoring according to the Wong three-step method as follows^[11]. During the distraction phase, traction was applied in slight flexion to keep the C1 ring opposed to the posterior odontoid process. While maintaining 10° flexion, traction was increased from 3.0 to 5.0 kg at a rate of 0.5 kg every 15 min. During the realignment phase, the C1 ring had slipped back over the odontoid but was displaced forward because of the slightly excessive flexion angle of traction. During the release phase, traction was modified to slight extension, allowing the C1 ring to come closer to the anterior odontoid process. Then traction was released and a cervical collar was applied. Fluoroscopy imaging demonstrated that the odontoid process was reset in the osseous-ligamentous ring of C1, and lateral X-ray of the cervical spine showed an atlantodental interval (ADI) of 11 mm and atlantoaxial interval (AAI) of 32 mm. CT of coronal and midsagittal sections of the cervical spine revealed that the odontoid process returned to the C1 ring, but the lateral atlantodental space remained asymmetric (the right side was smaller than the left side; [Figure 2](#)).

On day 2, the patient recovered consciousness but her respiration was blocked. Thus, a tracheotomy was performed. On days 3, 7, and 12, X-rays were taken and re-dislocation was not observed. Her neural function recovered to Frankel Grade C by day 10. However, her mental state deteriorated with obvious loss of muscle strength in the limbs on day 17. Re-dislocation was confirmed by X-ray and CT scans ([Figure 3](#)).

Due to the patient's cerebral brain stem injury and poor respiration, reduction of the repeated dislocation was not possible until 28 d after the first reduction. Under GA, closed reduction was attempted as before but failed even with a traction weight of 10.0 kg. With the patient in the prone position under 10.0 kg traction, the facet joints of C1-C2 and C2-C3 were exposed, and as expected, the apical, alar, cruciate, posterior



Figure 2 Radiographs after closed reduction. A: Lateral X-ray showing 11 mm atlantodental interval and 32 mm atlantoaxial interval of C1-C2; B and C: Computed tomography scans of coronal and midsagittal sections showing return of the odontoid process into the C1 ring with asymmetric lateral atlantodental spaces.



Figure 3 Radiographs after re-dislocation. A: Lateral X-ray showing complete posterior dislocation of the atlantoaxial joint; B: Computed tomography scan of coronal section; C: Three-dimensional reconstruction showing C1-C2 re-dislocation without any associated fractures.

longitudinal, and transverse ligaments were torn. The spinous process of C2 was pulled back gently with a towel clamp, and the posterior arch of C1 was nudged forward by the surgeon's thumb. A snapping sound indicated successful reduction without odontoidectomy and was confirmed fluoroscopically. Then traction was released. Under fluoroscopy, a cervical fixation system with a 3.5-mm-diameter pedicular screw (Tianjin New Medical Devices Co., Ltd., Tianjin, China) was applied in the C1-C2 pedicles, and interfacial fusion was performed using autologous iliac bone grafts (Figure 4). The cervical spine was immobilized using a rigid cervical collar for 1 mo postoperatively.

OUTCOME AND FOLLOW-UP

Rehabilitation therapy was started the first day after surgery, and the follow-up period was 12 mo. The patient's neurological function recovered to Frankel Grade C by day 60 and Grade D by day 90. By 6 mo after surgery, atlantoaxial joint fusion was achieved, and neurological function had recovered to Frankel Grade E. After 12 mo, lateral X-ray of the cervical spine showed a normal atlantoaxial complex, with perfect positioning of the pedicular screws and good bone fusion. A T2W MRI midsagittal view of the cervical spine revealed that curvature had returned to normal without cord edema at the C1-C2 Level. A T1W MRI coronal view showed that the odontoid process had returned to the C1 ring (Figure 5). The patient had lost only 15° of cervical rotation, and atlantoaxial complex instability in joint flexing and extending were no longer observed under fluoroscopy. Moreover, an open mouth view with manual cervical traction showed no distraction at the C1-C2 joint.

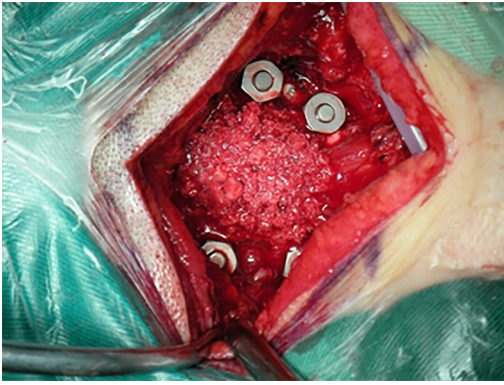


Figure 4 A cervical fixation system with four C1-C2 pedicular screws and autologous iliac bone grafts.

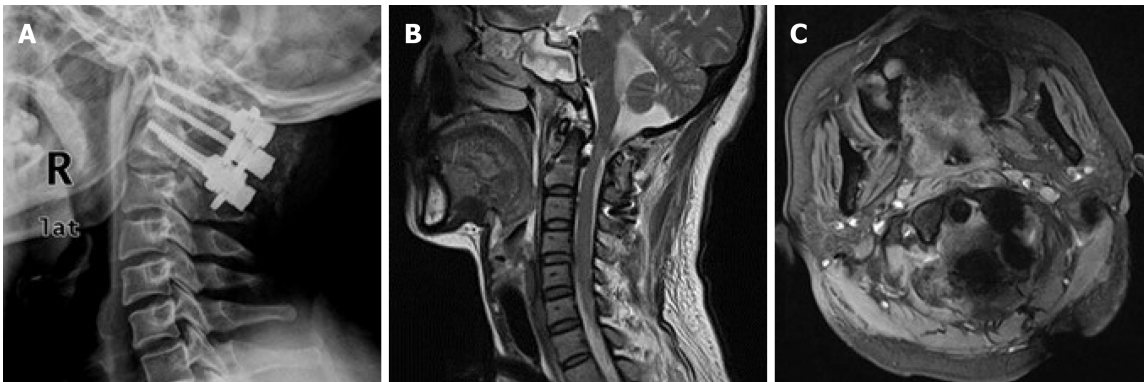


Figure 5 Radiographs 12 mo after internal fixation. A: Lateral X-ray showing normal atlantoaxial complex, perfect positioning of pedicular screws, and good bone fusion; B: T2W magnetic resonance imaging (MRI) midsagittal; C: T1W MRI coronal views showing normal cervical spinal curvature and positioning of the odontoid process in the C1 ring without cord edema.

DISCUSSION

In general, atlantoaxial dislocation is rare due to the stability of the C1-C2 complex. The odontoid process interlocks within the osseous-ligamentous ring formed ventrally by the anterior arch of the atlas and dorsally by the transverse ligament. Anterior movement of the axis vertebra (C2) is restricted by the anterior arch of the atlas when the odontoid is normal. Posterior movement is restricted by the cruciate ligaments, especially the transverse ligament, and rotatory movement is restricted by the alar ligaments^[19,20]. Silbergeld *et al*^[21] reported that posterior displacements of 5-10 mm can develop after isolated transverse ligament rupture, whereas further separation of C1 and C2 without fracture requires disruption of the apical, alar, cruciate, and posterior longitudinal ligaments. Therefore, only trauma tends to cause enough force to induce posterior dislocation, and the injury pattern is consistent with a high-grade hyperextension mechanism, as described by Haralson and Boyd^[6]. This theory is supported by the fact that most previously reported cases of posterior atlantoaxial dislocation as well as the present case resulted from road traffic accidents^[15].

All previously reported cases had either mild or no neurological deficit related to C1-C2 dislocation^[6-17,22]. It seems that if the initial distraction of the atlantoaxial complex does not cause a neurological deficit, then a high degree of displacement can be tolerated in this region due to the large spinal canal^[6]. Tucker *et al*^[23] demonstrated that posterior dislocation without odontoid fracture reduced the canal area by only 36%, which avoids injury to the cord. Wong *et al*^[11] proposed that the extra free space also allows a margin of error during reduction. Because patients seen in clinical practice likely represent those who avoided severe distraction injury to the spinal cord, suspicion of dislocation is warranted, especially in patients who have concomitant head injuries and altered mental state. In contrast to previous reports, the neurological deficit in the present case was severe with an unknown mechanism, but in addition to cervical spinal cord injury, cerebral brain stem damage cannot be ruled out as a possible cause.

Possible reduction techniques include closed manipulation under skull traction and open decompression and reduction. Controversy persists regarding whether internal fixation of such injury after closed reduction. Among the 12 previously reported cases in the English literature, seven were successfully treated initially with closed reduction *via* manipulation and skull traction^[6-8,10,11,14,15]. However, stability was achieved without internal fixation in only four cases^[7,8,14,15]. We propose that assessment of the transverse ligament is critical when planning treatment, because an intact transverse ligament may sufficiently stabilize the atlantoaxial complex after reduction of the odontoid peg into the osseoligamentous ring^[6,14]. Zhen *et al*^[17] reported that transverse ligament rupture in cases of anterior atlantoaxial dislocation without fracture can be diagnosed indirectly when radiographs show an ADI ≥ 5 mm. Dickman *et al*^[24] reported that such ligamentous injuries can be directly seen on MRI. A compromised transverse ligament on MRI strongly suggests that internal fixation will be necessary^[15,22]. Most commonly, posterior surgical fusion is necessary due to continuing C1-C2 instability or incomplete reduction. In fact, three of the previous seven cases of successful closed reduction eventually underwent posterior fusion and internal fixation^[6,10,11]. The choices for posterior fixation included wiring techniques and screw constructs^[25,26]. The use of C1-C2 pedicle screws is recommended because they afford greater stiffness and are associated with a high fusion rate without postoperative halo vest immobilization. In our patient, dislocation reoccurred after the first closed reduction, and because the transverse ligament was torn, upper cervical spine stability could only be restored *via* open reduction and posterior stabilization.

Possible reasons for re-dislocation soon after closed reduction in this case include the following: First and most importantly, the C1-C2 complex was quite unstable due to complete rupture of the transverse ligament and disruption of the apical, alar, cruciate, and posterior longitudinal ligaments. This was verified by an ADI of 11 mm and AAI of 32 mm on lateral X-ray after closed reduction. Second, the cervical collar does not sufficiently immobilize C1-C2 and may even increase the AAI, thereby increasing the risk of re-dislocation. Third, excessive extension of the neck during tracheotomy operation or care also could have played a role in the re-dislocation. Finally, other inappropriate movements of cervical vertebra during nasal feeding, repositioning in the bed, and positioning for radiographic examination may have contributed to the re-dislocation.

We have the following recommendations based on treatment outcomes in this case. (1) Early MRI assessment of the transverse ligament after closed reduction is essential, and if the transverse ligament is compromised, internal fixation should be performed as soon as possible. (2) Reliable external fixation should be applied after closed reduction, using devices such as the Halo-vest rack and head-neck-chest brace or plaster, but not a cervical collar. (3) During intubation or tracheotomy, fiber-optic intubation with cervical immobilization is preferred to minimize neck extension. (4) Radiographic examination should be performed immediately if the patient's mental state or muscle strength in the limbs quickly deteriorates soon after reduction, because these conditions may indicate spinal cord injury resulting from re-dislocation. And (5) Closed reduction should be attempted as soon as possible after re-dislocation. In this case, closed reduction was not attempted immediately following re-dislocation, and failure of the delayed closed reduction was likely due to scar tissue adhesions around the atlantoaxial joint and contracture of the anterior longitudinal ligament and longus capitis and longus colli muscles on both sides.

Closed reduction under fluoroscopic guidance with continuous spinal cord monitoring is preferred for repairing any atlantoaxial dislocation. However, determining the appropriate amount of traction can be difficult, and over-distraction of the C1-C2 complex is often fatal^[27]. Alternatively, the flexion/extension maneuver, which is used for reinserting the odontoid process into the osteoligamentous ring of the atlas, poses a risk of injury to the spinal cord. Thus, extreme caution must be exercised when repairing any atlantoaxial dislocation.

CONCLUSION

TPAD without fracture with complete rupture of the transverse ligament can easily reoccur after closed reduction. Early assessment of transverse ligament is critical for TPAD without fracture avoiding re-dislocation.

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