# Vertical atlantoaxial dislocation as a cause of failure of midline fixation

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ABSTRACT

We report an 11-year-old girl who had previously undergone an operation for basilar invagination involving a foramen magnum decompression and midline wire fixation. After improving initially, her neurological condition worsened again. Repeated investigations showed a firm midline craniovertebral fixation and bone fusion. However, she was found to have a vertical mobile and reducible atlantoaxial dislocation. Treatment of the vertical dislocation by lateral mass fixation resulted in lasting relief from her symptoms. Vertical instability at the atlantoaxial joints needs to be identified and appropriately treated as it may be a cause of failure of midline fixation.



## 1. Introduction

We recently discussed vertical mobile and reducible atlantoaxial dislocation as a defined clinical entity.[1] Such a form of instability can also be identified as "mobile and reducible basilar invagination". Incompetence of the lateral masses as a cause of such a dislocation has also been identified. We report an instance where a firm midline fixation procedure for basilar invagination failed. This was due to failure to identify and appropriately treat the cause and effect of the vertical mobile and reducible atlantoaxial dislocation.

## 2. Case report

An 11-year old girl had a history of progressive quadriparesis for 3 years following a fall. She had undergone surgery for "basilar invagination" by foramen magnum decompression and midline occipital bone to C2 wire fixation. Following an initial improvement after the surgery, her condition worsened.

When admitted, 14 months after the surgery, she had spastic grade-4 quadriparesis, and required support for all of her everyday activities. Investigations revealed a firm and successful midline fixation. However, a dynamic CT scan showed a vertical mobile and reducible atlantoaxial dislocation (Fig. 1). The atlantoaxial joints were in an inclined position, suggesting their incompetence.

During surgery the previously used wires and the bone in the midline were removed to facilitate exposure. The atlantoaxial joint was exposed widely after the C2 ganglion was sectioned. A bone graft was placed in the distracted joint and plate and screw fixation (screws implanted directly into the lateral masses of atlas and axis) was performed.[2–5] Additional bone graft pieces were placed in the midline over the previously operated area.

At a 32-month follow-up, the patient was ambulatory, able to perform routine activities and almost symptom-free. She was relieved of spasticity in her limbs. Investigations at this time revealed successful fixation of the vertical dislocation and fusion of the region (Fig. 2).

## 3. Discussion

Vertical mobile and reducible atlantoaxial dislocation occurs when the odontoid process moves rostrally into the foramen magnum on flexion of the head and returns to a normal position on head extension.[1] The abnormal movement results in indentation into the brainstem during the flexed-neck position. Vertical atlantoaxial dislocation as a cause of failure of the posterior midline craniovertebral fixation procedure has not been previously reported. This example presents an opportunity to evaluate and understand the pathogenesis of vertical atlantoaxial dislocation.

Differentiating patients who have vertical mobile dislocation from those who have vertical fixed dislocation or Group A basilar invagination is crucial as the treatment protocol for each of these clinical entities is specific.[2] An algorithm for the management of patients with basilar invagination was described by Menezes[6] and later modified by us.[1] The treatment of fixed vertical dislocation or Group A basilar invagination is either transoral decompression of the odontoid process and subsequent craniovertebral stabilization, which is more popularly accepted,[7,8] or atlantoaxial joint distraction, reduction and subsequent fixation.[2] However, in patients with vertical mobile and reducible atlantoaxial dislocation, fixation in the reduced position will suffice. The placement of metal spacers within the joint for the distraction of facets as advocated for patients having Group A basilar invagination is not necessary. The sectioning of the C2 ganglion provides a wide exposure of the atlantoaxial joint and allows its manipulation and fixation.

We identified a relatively inclined profile of the facets of atlas and axis to be the primary cause of Group A basilar invagination. The progressive "slip" of the facet of C2 over C1 could be the cause of "listhesis" of C1 over C2, a phenomenon that eventually results in basilar invagination.[9] In this patient, and also our previous patients, the atlantoaxial joint was markedly inclined, an anomaly that appeared to be primarily responsible for the incompetence of the lateral masses and an abnormal vertical mobility.

Vertical mobile and reducible atlantoaxial dislocation provides a unique clinical situation. As seen in the presented example, midline procedures may not always be appropriate. Addressing the incompetence of the atlantoaxial joint appears to be mandatory.

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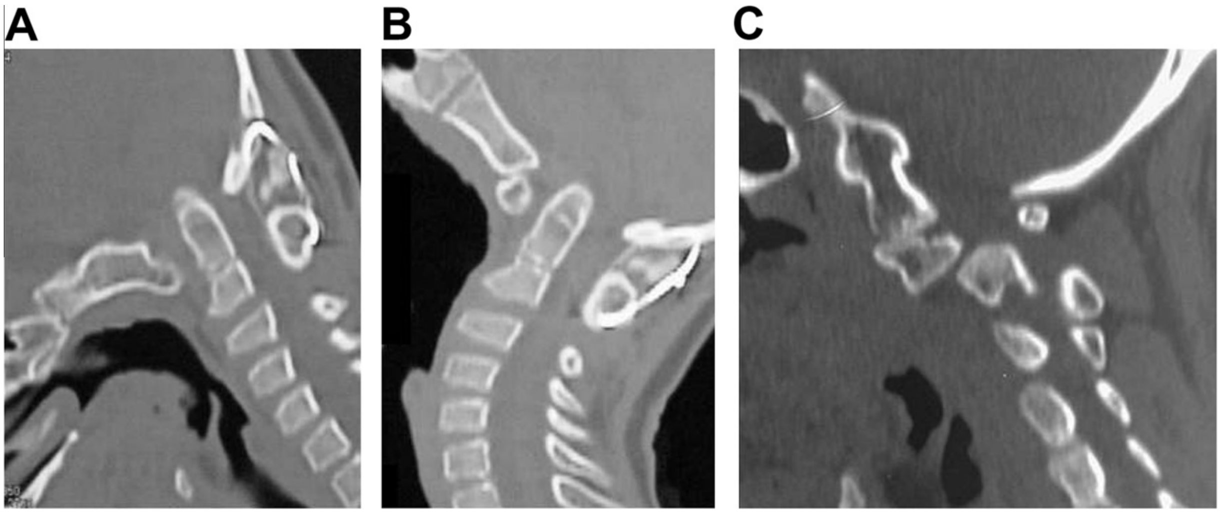


**Fig. 1.** (A) CT scan with sagittal reconstruction, with the patient's head in a flexed position, showing a firm midline fixation incorporating wires and bone graft. The odontoid process is migrated superiorly into the foramen magnum. (B) With the patient's head in an extended position, showing reduction of the vertical dislocation. (C) Sagittal section reconstruction through the lateral masses of the atlas and axis showing the atlantoaxial dislocation.

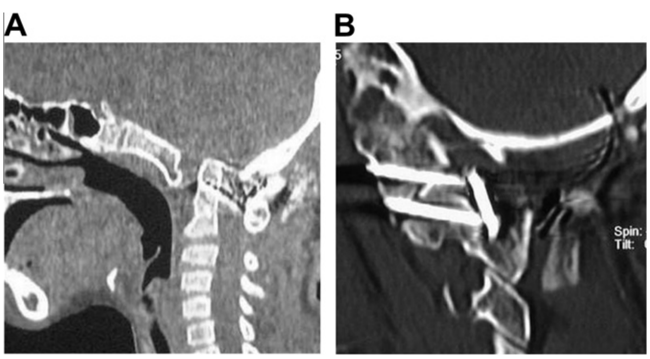


**Fig. 2.** (A) Post-operative CT scan with sagittal reconstruction showing fixation in the reduced position. (B) Sagittal section reconstruction through the lateral masses showing reduction of the dislocation and fixation of the atlantoaxial joint using the plate and screw method of fixation.

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