

Commentary on: “Spontaneous Ankylosis of Occiput to C2 following Closed Traction and Halo Treatment of Atlantoaxial Rotary Fixation”

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The authors present a unique case of an 11-year-old child with spontaneous fusion of his occiput to C1–C2 following traction, manipulative reduction, and halo immobilization for refractory atlantoaxial rotary fixation.

The child initially presented with torticollis, neck pain, and headaches and underwent numerous chiropractor manipulations over 9 months with alleviation of his neck pain and headaches. However, his torticollis persisted; thus, he was referred to an orthopedic surgeon where initial workup included magnetic resonance imaging, magnetic resonance angiography, and computed tomography (CT) imaging. This revealed a Fielding and Hawkins type II rotatory fixation, with remodeling changes involving the left C2 articular facet. The treatment options of surgical versus nonsurgical reduction were discussed with the family, and they decided for nonsurgical reduction and immobilization. Thus, the child was placed in traction and had improvement but not complete resolution of his rotational deformity. Subsequently, an attempt was made to preserve the traction reduction with halo vest immobilization for 3 months. At his 6-month follow-up, the patient demonstrated mild residual torticollis. CT imaging was obtained to provide further clarity on bony anatomy and showed spontaneous fusion from the occiput to C2.

Because most of the head and neck rotation occurs at the occiput–C1–C2 integrated functional complex, it is desirable to try to preserve the motion levels around the craniocervical junction as much as possible. Therefore, nonsurgical management is the clearly recommended first line of treatment using halter-type traction and reduction followed by Minerva or halo bracing for 3 months. However, if reduction is not achieved and/or there is persistent atlantoaxial rotary fixation after 3 months of bracing, surgical reduction and internal fixation fusion for

management is the recommended treatment with the hope of limiting the surgical reduction and fusion efforts to the C1–C2 segments. In a retrospective case review, we found instability patterns following nonoperative care of very young children with ligamentous injuries involving the craniocervical junction to be unpredictable with spontaneous ossification not a regular occurrence.¹

I agree with the authors' management of this child and would have recommended a similar approach even with the child's very delayed presentation.² I suspect that the child had extension of his fusion from occiput to the atlantoaxial motion segments secondary to the recurrent trauma of the craniocervical articular surfaces and surrounding soft tissues over the course of numerous chiropractor neck manipulations and/or prolonged traction periods, which eventually led to focal joint inflammation and subsequent spontaneous fusion. Based on our previous surgical experience in operating on the craniocervical junction of the very young patient, surgical reduction and fusion targeting the atlantoaxial segment would not have predictably prevented extension of the fusion to the occipitocranial junction. However, with the advent of innovative magnetic resonance imaging, one may be able to predict who will have extension of the fusion by highlighting increased signal of the occiput–C1–C2 complex ligaments. For example, Landi et al found increased signal in the alar, capsular, and transverse ligaments in two patients with Fielding and Hawkins type II rotatory fixation using short T1 inversion recovery (STIR) sequences.³ Overall, the management of refractory atlantoaxial rotary fixation in children remains challenging and frustrating. The one remedy in absence of a decisive evidence-based treatment is to clearly present all treatment options to the patient and their family to invite a shared decision-making approach for outcomes like the occurrence of a spontaneous fusion.

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