

Congenital malformations of occipital vertebrae as cause of instability and degeneration of atlanto-axial joints

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Introduction

The articular complex of the craniocervical junction (CCJ) is formed by five synovial joints: two atlanto-occipital, and three atlanto-axial (one anterior and two lateral). Their intrinsic characteristics and architecture provide the motion of the skull and the upper cervical spine and the ideal distribution of forces applied to that segment^(1,2). Morphological disturbances secondary to congenital malformations can provoke chronic instability and biomechanical overload. To recognize bone configuration, ligamentar attachments and the more prevalent anomalies, it becomes important to understand the signals of joint functional damage.

Method

Two radiologists have, retrospectively, analyzed 2T MRI of 58 patients (25 male, 33 female; mean age: 44.1 year, SD: 16.1) selected by the presence of craniometric and morphological malformations of the CCJ and 23 control subjects (10 male, 13 female, mean age 55.5 year, SD: 11.8). Images were acquired at neutral position for cervical spine and the techniques included T1 spin echo (time of echo-TE: 12 to 16ms; time of repetition-TR: 520 to 650ms) and T2 fast spin echo was also chosen as fluid sensitive sequence (TE: 126 to 128ms; TR: 3900 to 7300ms) multiplanar views. The slice thickness was within 3 to 6mm and reconstruction matrix varied from 256x256 to 320x252.

Anomalies defined as manifestations of the occipital vertebrae (atlanto-occipital assimilation, basiocciput hypoplasia, occipital condyle hypoplasia and odontoid deformities)⁽³⁾ were identified and correlated to signals of instability and synovial degenerative changes at atlanto-axial joints. Anterior atlanto-axial space wider than 3mm was defined as subluxation. The articular compromise was divided in mild (intra-articular liquid with normal space)(Figure 1) and severe (irregular space with low subcondral signal with normal or narrowing of space width)(Figure 2). Fischer tests were applied and *p* value below 0.05 was considered significant.

Results

Basiocciput hypoplasia was identified in 44 patients (75.9%), occipital condyle hypoplasia in 11 (18.9%) and odontoid deformities in 4 (6.9%). Atlanto-occipital assimilation was present in 26 patients (44.8%), 17 (65.4%) presented bilateral articular fusion and 9 (34.6%), unilateral. Atlanto-axial subluxation was noticed in 12 patients (20.7%) and 11 of those (91.6%) presented at least one of the manifestations of the occipital vertebrae (Figure 3). 26 patients (44.8%) have demonstrated atlanto-axial articular damage, 17 (65.4%) severe degenerative signals, and 9 (34.6%) mild. It was significantly correlated to atlanto-occipital subluxation (*p*=0.0001).

Although the mean age of the control group was significantly higher than that of the patients, atlanto-axial degenerative disease was present in only 3 controls (13.0%). None of the control individuals presented atlanto-axial subluxation.

Discussion

The articular relationships at the CCJ are maintained by the skeletal shape combined with intrinsic stabilizing structure formed by the transverse and alar ligaments. Congenital malformations are associated to ligament laxity that can lead to cronical instability⁽⁴⁾. Biomechanical articular overload harms the synovial coverage and can promote precece degeneration. The knowledge of the normal anatomy and the skeletal abnormalities of the CCJ is necessary to understand the hole of ligamentary function and the consequences of biomechanical compromise to articular degenerative disease.

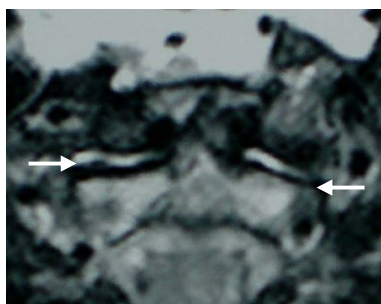


Figure 1: Mild articular compromise. T2WI showing intra-articular liquid (arrows) at atlanto-axial joints.

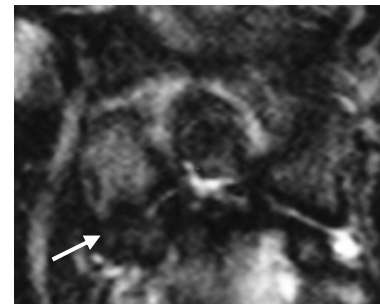


Figure 2: Severe atlanto-axial degeneration. T2WI with irregular subcondral sclerosis and narrowed space (arrow).

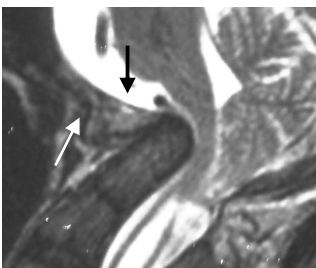


Figure 3: Atlanto-axial subluxation. T2 sagittal image with enlargement of the anterior atlanto-axial space (black arrow) and atlanto-occipital assimilation (white arrow). The transverse ligament was not individualized for this patient.

References

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