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Is atlantoaxial instability the cause of Chiari malformation? Outcome analysis of 65 patients treated by atlantoaxial fixation.

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Author information

Abstract

OBJECT: Understanding that atlantoaxial instability is the cause of Chiari malformation (CM), the author treated 65 patients using atlantoaxial stabilization. The results are analyzed.

METHODS: Cases of CM treated using atlantoaxial fixation during the period from January 2010 to November 2013 were reviewed and analyzed. Surgery was aimed at segmental arthrodesis.

RESULTS: The author treated 65 patients with CM in the defined study period. Fifty-five patients had associated syringomyelia. Forty-six patients had associated basilar invagination. Thirty-seven patients had both basilar invagination and syringomyelia. Three patients had been treated earlier using foramen magnum decompression and duraplasty. According to the extent of their functional capabilities, patients were divided into 5 clinical grades. On the basis of the type of facet alignment and atlantoaxial instability, the patients were divided into 3 groups. Type I dislocation (17 patients) was anterior atlantoaxial instability wherein the facet of the atlas was dislocated anterior to the facet of the axis. Type II dislocation (31 patients) was posterior atlantoaxial instability wherein the facet of the atlas was dislocated posterior to the facet of the axis. Type III dislocation (17 patients) was the absence of demonstrable facet alignment and was labeled as "central" atlantoaxial dislocation. In 18 patients, dynamic images showed vertical, mobile and at-least partially reducible atlantoaxial dislocation. All patients were treated with atlantoaxial plate and screw fixation using techniques described in 1994 and 2004. Foramen magnum decompression or syrinx manipulation was not performed in any patient. Occipital bone and subaxial spinal elements were not included in the fixation construct. One patient died, and death occurred in the immediate postoperative phase and was related to a vertebral artery injury incurred during the operation. One patient had persistent symptoms. In the rest of the patients there was gratifying clinical improvement. More remarkably, in 7 patients, the symptoms of lower cranial nerve paresis improved. No patient worsened in their neurological function after surgery. Reductions in the size of the syrinx and regression of the CM were observed in 6 of 11 cases in which postoperative MRI was possible. During the follow-up period, there was no delayed worsening of neurological function or symptoms in any patient. Sixty-three patients improved after surgery, and the improvement was sustained during the average follow-up period of 18 months.

CONCLUSIONS:

On the basis of outcomes in this study, it appears that the pathogenesis of CM with or without associated basilar invagination and/or syringomyelia is primarily related to atlantoaxial instability. The data suggest that the surgical treatment in these cases should be directed toward atlantoaxial stabilization and segmental arthrodesis. Except in cases in which there is assimilation of the atlas, inclusion of the occipital bone is neither indicated nor provides optimum stability. Foramen magnum decompression is not necessary and may be counter-effective in the long run.

KEYWORDS: CM = Chiari malformation; CN = cranial nerve; Chiari malformation; JOA = Japanese Orthopaedic Association; VAS = visual analog scale; atlantoaxial dislocation; atlantoaxial fixation; basilar invagination; cervical; foramen magnum decompression; syringomyelia

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