Incidence and Management of Basilar Invagination With Associated Chiari I Malformation

WFNS Spine Committee Recommendations

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Study Design. Systematic literature review plus expert opinion framed on Delphi method.

Objective. To analyze the influence of coexistent Chiari I malformation (CMI) on the management of basilar invagination (BI).

Summary of Background Data. Basilar invagination (BI) and Chiari 1 malformation (CMI) constitute the commonest anomalies of the craniovertebral junction (CVJ). Treatment becomes even more challenging for patients in whom both pathologies coexist.

Materials and Methods. Using PubMed, the authors identified 48 publications published between 2011 and 2022 concerning the incidence and management of both pathologies in combination. By means of the Delphi method, a panel of expert spine surgeons analyzed the strength of the published literature and voted statements concerning the management of BI combined with CMI.

Results. The incidence for a combination of BI with CMI is estimated between 2.4/100,000 in children and 9.6 to 19.7/100,000 in adults. BI with ventral compression of the medulla related to AAD can be treated in a single operation by sagittal realignment through C1-C2 facet joint distraction and fusion. In the event of unreducible BI, insufficient ventral decompression by C1/2 fusion alone may be overcome by adding a foramen magnum decompression to allow posterior shift of the medulla. BI patients with concomitant CMI have an undersized posterior fossa volume. This implies that surgical treatment of BI combined with CMI has either to increase posterior fossa volume or to include a posterior decompression.

Conclusion. In patients with BI, concomitant CMI is a modifier of surgical management. In BI with AAD, an additional foramen magnum decompression should be added to posterior C1-C2 realignment and fusion. In BI without AAD, whether treatment is restricted to FMD or C1/2 fusion is required on top or alternatively, demands further studies. Odontoid resections are reserved for patients with insufficient alignment after posterior surgery.

Key Words: Chiari malformation, basilar invagination, foramen magnum decompression, odontoidectomy, C1-C2 posterior fixation, C1-C2 joint manipulation

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Chiari I malformation (CMI) is the commonest malformation of the craniovertebral junction. It is defined as a tonsillar descent through a normal configured foramen magnum into the upper cervical canal. Basilar invagination (BÍ) describes a malformation characterized by the odontoid tip crossing Chamberlain´s line drawn between opisthion and hard palate. Surgical treatment options for CMI (bony decompression with and without duraplasty, with and without arachnoid opening) and BI (odontoid resection or posterior distraction, decompression without fusion or C1/2 fusion alone) have been discussed controversially for decades without reaching general consensus. Apart from both malformations as separate entities, they may also coexist in a given patient.

This paper analyzes the literature on both malformations to determine the incidence for the combination of these two disorders, to provide an overview of treatment options, and recommendations for their treatment.

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MATERIALS AND METHODS

Using PubMed, the authors reviewed the literature on BI and CMI published between 2011 and 2022 concerning their incidence, management and treatment. Separate searches for CMI and BI yielded 4578 and 755 articles each, respectively. A search for BI and CMI combined revealed 142 results. Case reports and reviews including other craniospinal anomalies were eliminated from the literature analysis. This left 48 publications dealing with different aspects of BI combined with CMI for review (Fig. 1). By means of the Delphi method, a panel of expert spine surgeons analyzed the strength of the published literature and voted statements concerning the management of BI combined with CMI. The secret voting selected from the following options: totally disagree, disagree, agree, more than agree, or totally agree.

RESULTS

Incidence

There are no data available on the incidence of BI and CMI combined. The incidence of CMI varies in different parts of the world. Data are available for western Europe with a reported incidence of 80/10,000.[1] Radiologic studies revealed that about 1% of all magnetic resonance images (MRI) covering the craniocervical junction will demonstrate CMI.[2,3] Among 1059 pediatric scoliosis patients, an incidence of 3.6% for CMI was reported.[4] Corresponding figures for adolescent scoliosis provided an incidence of 4.1% in a meta-analysis covering 4726 patients[5] and 4.2% in a retrospective analysis of 259 patients.[6] Another meta-analysis on 1863 patients with congenital scoliosis revealed an incidence for CMI of 1.72% in this subgroup.[7]

There are no data available on the incidence of BI. However, data were reported on the incidence of BI among patients with CMI revealing a rising incidence for this combination with age. Whereas an incidence of 3% for this combination was published in a study on 500 children with CMI in the United States,[8] incidences of 24.6% among 451 patients in western Europe,[9] 12% among 364 patients in the United States,[10] and 23.4% among 316 patients in China[11] were reported for adults.

Likewise, a rising incidence for CMI was found with age among patients with BI. Whereas this combination was evident in 50% of 2200 children with BI,[12] this figure rose to 94.2% in adults among 69 BI patients in western Europe.[13] Differentiating pediatric and adult BI patients according to presence or absence of atlantoaxial dislocation according to Goel's classification of 2011,[14] showed an incidence of 24.7% for a concomitant CMI among 510 patients with[15] compared with 82.5% of 63 patients without atlantoaxial dislocation[16] in India, indicating that the vast majority of BI patients demonstrated atlantoaxial dislocation in that region. In contrast, a series of 82 BI patients from western Europe reported no atlantoaxial dislocation in 40.2%, indicating a lower rate for BI with atlantoaxial dislocation in this region.[17]

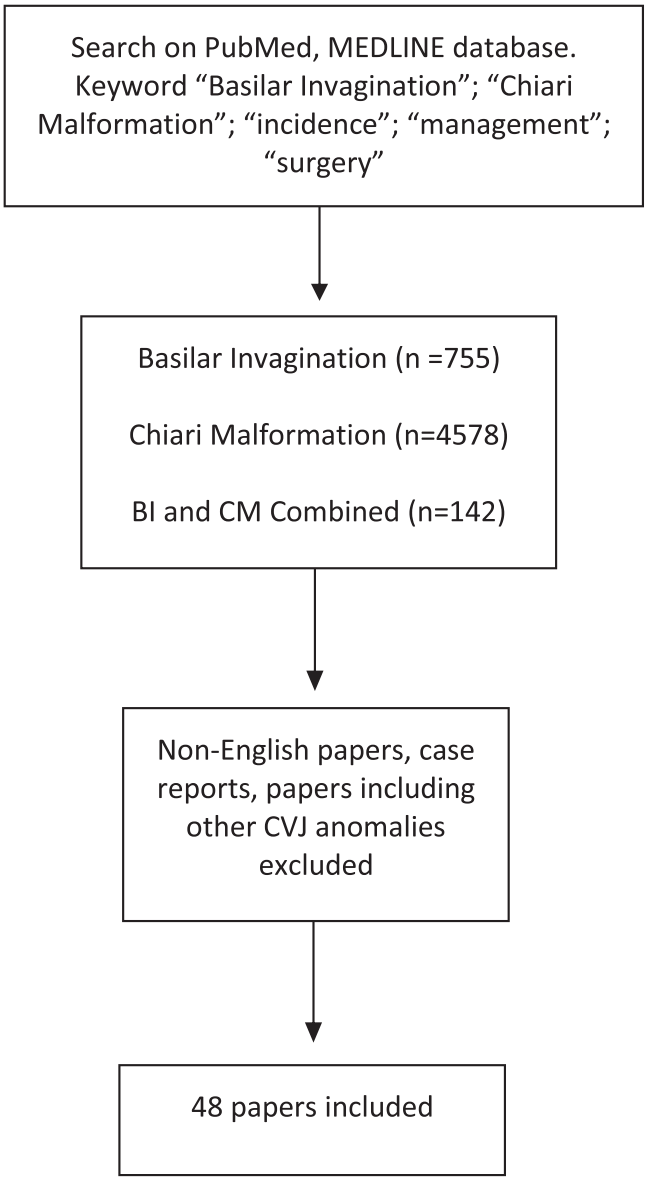

Figure 1. Flowchart for the literature search on Chiari malformation associated with basilar invagination.

On the basis of the data presented above, the incidence for a combination of BI and CMI can be expected to vary between different parts of the world. Furthermore, it appears that the incidence of this combination rises with age. Children with CMI may develop BI as well as those with BI a CMI later in life. The incidence for a combination of BI with CMI may be estimated between 2.4/100,000 in children and 9.6 to 19.7/100,000 in adults.

Treatment Options

For the management of patients with a combination of BI and CMI, each individual patient demands a detailed clinical and radiologic analysis to identify those components requiring treatment. Symptoms may be related to:

Ventral compression of the medulla from the odontoid peg, which may be caused by two mechanisms: atlantoaxial dislocation, that is, the odontoid crossing McRae´s line,[18] or craniocervical kyphosis in the absence of atlantoaxial dislocation which is commonly associated with assimilation of the atlas to the occiput.[17]
Posterior compression of the medulla by cerebellar tonsils, that is, CMI.
Craniocervical instability.
Syringomyelia.
Hydrocephalus.
Arachnoiditis at the foramen magnum.

All these mechanisms may contribute to clinical symptoms in a given patient in any combination.[17,19] For each of these different aspects various surgical treatment strategies have been advocated.

Ventral Compression

For patients with ventral compression of the medulla related to atlantoaxial dislocation representing group A BI according to Goel,[14] ventral decompression used to be obtained by odontoid resection through a transoral[20] or transnasal[21] approach. Alternatively, ventral decompression may be achieved by sagittal realignment with distraction of the C1/2 facet joints from a posterior approach.[22,23] As all patients with atlantoaxial dislocation have to be considered unstable and require a posterior C1/2 fusion, combining C1/2 facet joint distraction and fusion in a single posterior procedure has gained increasing international acceptance making odontoid resections obsolete for this type of invagination. It is generally agreed, that the occiput should be spared in the fusion construct for this type of invagination unless the atlas is assimilated to the occiput or anatomic variations of the vertebral artery make screw placement in C1 too hazardous.[24]

To achieve a ventral decompression in patients without atlantoaxial dislocation, that is, group B BI according to Goel,[14] is more demanding. Distraction of C1/2 facet joints with C1/2 fusion alone may not lead to a sufficient ventral decompression in this type of invagination, which often is referred to as unreducible, although it has been claimed that C1/2 fusion alone represents sufficient therapy for this type as well.[16] The problem of insufficient ventral decompression by C1/2 distraction alone may be overcome by adding a foramen magnum decompression and/or a fusion in slight retroflexion including the occiput, to allow a posterior shift of the medulla.[17,25] If these maneuvers fail, a transoral or transnasal resection of the odontoid-clivus complex may be considered as a second step.[20]

Posterior Compression

Posterior compression of the medulla by cerebellar tonsils, that is, CMI, is a result of a tonsillar descent in response to a reduced posterior fossa volume as described first by Nyland and Krogness in 1978.[26] It is general consensus that a foramen magnum decompression represents the treatment of choice for CMI. Regarding decompression techniques for CMI, however, no international consensus exists. It is agreed that a decompression should involve a small medial suboccipital craniectomy and a limited C1 laminectomy.[27,28] Whether the decompression should also involve a duraplasty, arachnoid opening and dissection or coagulation of cerebellar tonsils, however, is controversial.

CMI patients with concomitant BI have also been shown to have a undersized posterior fossa volume.[14,29–32] However, whether the presence of CMI in patients with BI requires a foramen magnum decompression has not been widely discussed or studied in the literature. The reduced volume of the posterior fossa in BI with CMI implies, that surgical treatment should either increase posterior fossa volume or include a posterior decompression in this setting. The classifications for BI by Menezes *et al.*[20] in 1980 and a first classification by Goel *et al.*[33] in 1998 recommended foramen magnum decompressions for patients with an associated CMI. The Goel classification of 2011 points out that posterior fossa volume tends to be reduced in group B invagination in particular,[14] which was supported by further studies subsequently.[29–32] A posterior decompression on top of realignment and C1/C2 fusion has also been advocated for patients with group A BI and CMI.[34]

In contrast, it has been proposed recently, that a sole C1/C2 fusion may be sufficient for posterior decompression claiming that the tonsillar descent is a reaction to C1/2 instability and thus reversible by C1/C12 fusion.[35,36] However, this view is highly controversial and not widely supported.[24,37] It is known that CMI secondary to intracranial mass lesions, certain arteriovenous malformations or low-pressure syndromes related to spinal loss of CSF is reversible by treating the cause of CMI. It has also been established, that cerebellar tonsils will ascend in CMI, if cerebrospinal fluid (CSF) flow at the foramen magnum has been established with surgery.[38,39] However, whether C1/C2 fusion alone may have the same effect opening CSF pathways by increasing posterior fossa volume has not been studied on a larger scale so far.

Craniocervical Instability

How to define craniocervical instability in the context of BI and CMI is another controversial topic. Although occiput to C1 (C0-C1) fusions by transcondylar screws have been advocated for BI,[40] it is generally believed that C0/C1 instability is not a feature of BI. A recent study identified alterations of condylar height and length as well as alterations of the C1 lateral mass in group B BI leading to flat-tilt condylar joints in these patients.[41] However, whether these morphologic changes represent C0/C1 instability remains to be determined. Currently, craniocervical instability is considered to involve the C1/C2 segment primarily in this context.[17]

Atlantoaxial dislocation representing group A BI clearly constitutes C1/C2 instability. But how should C1/C2 instability be defined in the absence of this dislocation, that is, group B BI.[30] Can we consider all pa-

tients with group B BI to be stable? Recently, attempts were made to study C1/C2 facets to solve this issue.[42,43] According to these studies, the C1 facet may be in line with the C2 facet or displaced either anteriorly or posteriorly. Whereas anterior displacement of the C1 facet will lead to a widening of the atlantodental interval clearly indicating C1/C2 instability,[9] the relevance of posterior displacements is less clear. A posterior displacement may just indicate mobility of this segment. It has been shown that up to 10% of CMI patients without BI or normal controls may demonstrate a posterior C1 facet displacement without any clinical significance even after foramen magnum decompressions of the CMI.[9] Other groups consider a posterior displacement of the C1 facet as a proof of instability and describe C1/C2 instabilities even in the absence of any C1/C2 facet malalignment.[44] Additional aspects may be analyzed in these patients such as the orientation of the C1/C2 facet joints. Whereas the normal anatomic position of the C1/C2 facet joints is horizontal, an anterior tilt of C1/C2 facet joints may indicate instability as well as an opening of facet joints in functional CT scans obtained in flexion and extension. Thus, functional CT studies may be used to identify patients with BI requiring C1/C2 fusion.[17] Furthermore, clinical signs such as nystagmus, double vision, or ataxia associated with certain head positions or movements may be interpreted to indicate C1/C2 instability in this context.[17] However, these aspects have not been studied on a larger scale to provide a thorough basis for a general recommendation.

Syringomyelia

In the context of BI and CMI, syringomyelia is caused by CSF flow obstruction at the foramen magnum. Foramen magnum decompression with establishment of a free CSF passage at this level is considered sufficient to reduce the size and clinical effects of syringomyelia in the long term. Additional measures such as shunting a syrinx are not recommended for first line treatment[27,28] and are even questionable for patients requiring revision after a failed foramen magnum decompression.[45]

Hydrocephalus

Symptomatic hydrocephalus is a rare feature of CMI or BI but will require treatment before any other surgical procedure for BI and CMI if present.[46]

Foramen Magnum Arachnoiditis

The relevance of arachnoid changes in CMI, which may vary from fibrotic changes to adhesions with neural structures to even severe arachnopathies as a result of arachnoiditis, is another controversial issue. Whereas a number of groups reported arachnoid pathologies of varying degree in CMI,[46–49] the relevance of these findings on clinical signs before surgery or postoperative outcomes has not been widely recognized. A major problem is the difficulty to diagnose arachnoid pathologies on preoperative images or with intraoperative ultrasound unless they are severe and the result of a meningitis, a hemorrhage or previous intradural surgeries. In such severe cases, the blood supply to the medulla is compromised as a result of arteries and veins embedded in thick arachnoid scars.[45,50] It follows that severe arachnopathies may cause vascular damage to the medulla as an additional pathomechanism independent from compression, instability, or CSF flow obstruction. Whether less profound arachnoid changes may have a clinical impact in CMI as well, has been proposed[46,47] but has not gained wide spread acceptance, particularly not from neurosurgeons favouring bony decompressions without dura opening. Comparing patients with CMI with and without additional BI revealed no differences for associated arachnoid pathologies[17] suggesting that arachnoid pathologies may play a similar role in patients with BI and CMI as in CMI without BI.

The statements produced by consensus conference are summarized on Table 1.

DISCUSSION

The variations on incidences for CMI in different parts of the world and the rate of BI patients developing CMI as well as CMI patients developing BI with ongoing age illustrate that calculations of incidences for malformations of the craniocervical junction in one part of the world may not be valid for others and that they need to be differentiated according to age. The incidences for the combination of BI with CMI calculated in this paper between 2.4/100,000 for children and up to 19.7/100,000 in adults are estimates. Few centers in the world have gathered experience with BI or CMI based on several hundred patients. Calculating incidences from small patient numbers may be misleading. As an example, a Brazilian study on 20 adult CMI patients undergoing surgery reported concomitant BI in 80%.[51]

The results of the literature analysis presented above illustrate the complexity of BI with and without CMI. As several pathomechanisms may play a role in a given patient it appears illogical to assume that one type of surgery will be applicable to all patients with BI and CMI. Each patient has to be analyzed individually with detailed radiologic and clinical examinations. Patients with BI and CMI may demonstrate a ventral compression of the medulla. In that case, a ventral decompression by some form of posterior realignment should be considered. Others may present without ventral compression so that no ventral decompression is necessary. Likewise, BI patients may be observed with no posterior medullary compression related to CMI. Consequently, they will not require a posterior decompression unless treatment of a ventral compression requires posterior shifting of the medulla.

The concept to reverse a posterior medullary compression related to CMI by C1/C2 fusion alone remains controversial and is not supported by the great majority of surgeons working in this field. In the setting of BI, CMI may develop as a consequence of a reduced

TABLE 1. WFNS Spine Recommendations on Incidence and Management and Management of Combined Basilar Invagination and Chiari Malformation

Statement 1: (50% totally agreed; 50% more than agreed)
Incidence
The incidence of BI and CMI varies in different parts of the world: in Western Europe and United States it can be estimated at 8/1,00,000
The incidence for this combination increases with age: children with CMI may develop a basilar invagination in adulthood and vice versa
Statement 2: (50% totally agreed; 30% more than agreed; 20% agreed; 10% disagreed)
Surgery
Treatment of patients with a combination of BI and CMI should include appropriate posterior fossa decompression
Statement 3: (60% totally agreed, 30% more than agreed; 10% agreed)
Modifiers
Chiari I malformation is a modifier in the treatment algorithm for basilar invagination

posterior fossa volume. Thus, the pathomechanism is no different compared with CMI patients without BI. The gold standard for treatment of CMI is a foramen magnum decompression. Aims of the decompression are to provide space for neural structures and to establish a free CSF pathway in this region. To what extent treatment of BI by C1/C2 fusion alone can achieve the same effect by increasing posterior fossa volume and providing unobstructed CSF flow at the foramen magnum, has not been sufficiently studied so far and remains to be established. With the experience gained so far, BI patients with CMI should undergo a foramen magnum decompression as part of their surgical management.

CONCLUSION

The incidence for a combination of BI and CMI varies between different parts of the world, but generally rises with age. As several pathomechanisms play a role, it appears illogical to assume that one type of surgery will serve all patients with BI and CMI alike. Each patient should be analyzed individually with detailed radiologic and clinical examinations. In patients with BI, concomitant CMI is a modifier of surgical management. BI patients with atlantoaxial dislocation should undergo posterior realignment and C1-C2 fusion as first line of treatment. With concomitant CMI, an additional foramen magnum decompression should be added. In BI without atlantoaxial dislocation, concomitant CMI is particularly common. Whether treatment in this subgroup can be restricted to foramem magnum decompression or C1/2 fusion is required on top or alternatively, demands further studies. Odontoid resections may be reserved for patients with insufficient alignment after posterior surgery.

➢ Key Points

- ❑ The incidence of BI and CMI combined varies in different parts of the world: in Western Europe and United States it can be estimated at 8/100,000 in adults.
- ❑ The incidence of BI and CMI combined increases with age: children with CM type 1 may develop a basilar invagination in adulthood and vice versa.
- ❑ For BI with atlantoaxial dislocation leading to ventral compression of the medulla, posterior realignment with C1/C2 distraction and fusion should be considered as first line of treatment.
- ❑ For BI without atlantoaxial dislocation, ventral compression related to craniocervical kyphosis may not be reducable by C1/2 joint distraction sufficiently. An osteoligamental foramen magnum decompression may allow posterior shifting of the medulla in this subgroup.
- ❑ Chiari malformation is a modifier in the treatment algorithm for basilar invagination: management of BI and CM1 combined should include a posterior fossa decompression.

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