Original Article

Minimally invasive posterior fossa decompression with duraplasty in Chiari malformation type I with and without syringomyelia

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ABSTRACT

Background: Posterior fossa decompression (PFD), with and without duraplasty, represents a valid treatment in Chiari malformation Type I (CM-I) with and without syringomyelia. Despite a large amount of series reported in literature, several controversies exist regarding the optimal surgical approach yet. In this study, we report our experience in the treatment of CM-I, with and without syringomyelia, highlighting how the application of some technical refinements could lead to a good outcome and a lesser rate of complications.

Methods: Twenty-six patients with CM-I, with and without syringomyelia, underwent PFD through a 3 cm × 3 cm craniectomy with the removal of the most median third of the posterior arch of C1 and duraplasty. Signs and symptoms included sensory deficits, motor deficits, neck pain, paresthesias, headache, dizziness, lower cranial nerve deficits, and urinary incontinence. Postoperative magnetic resonance (MR) was performed in all patients.

Results: Signs and symptoms improved in 76.9% of cases. Postoperative MR revealed a repositioning of cerebellar tonsils and the restoration of cerebrospinal fluid circulation. In our experience, the rate of complication was 23% (fistula, worsening of symptoms, and respiratory impairment).

Conclusion: PFD through a 3 cm × 3 cm craniectomy and the removal of the most median third of posterior arch of C1 with duraplasty represents a feasible and valid surgical alternative to treat patients with CM-I, with and without syringomyelia, achieving a good outcome and a low rate of complications.

Keywords: Cerebellar tonsils, Chiari malformation type I, Duraplasty, Posterior fossa decompression, Syringomyelia

INTRODUCTION

The pathophysiology of Chiari malformation Type I (CM-I) involves frequently the small posterior cranial fossa[8,12] with the consequent overcrowding of neural structures associated with a hyperdynamic cerebrospinal fluid (CSF) flow in craniovertebral junction (CVJ). These conditions result in an alteration of pressure gradients at CVJ with the consequent compression of brainstem.[22] However, it is likely that, in patients with CM-I, who do not present a crowded posterior fossa, other mechanisms, still unclear, can cause the impaction of the tonsils in the foramen magnum.[26] In addition, it has been largely reported that in 70% of cases CM-I is associated to syringomyelia.[2,16]

Surgical management of CM-I with and without syringomyelia is a still controversial topic in neurosurgery. Among the numerous methods proposed, the posterior fossa decompression (PFD) would seem to be the most suitable in the treatment of CM-I. PFD allows the relief of the cerebellar tonsils from the dorsal surface of the brainstem and upper cervical spinal cord, allowing physiologic and better CSF dynamics. A large amount of data demonstrated that PFD with or without removal of posterior arch of C1, and with or without duraplasty, allows an increase of volume of the posterior fossa, the repositioning of the herniated cerebellar tonsils, and the restoration of CSF circulation. However, few data exist regarding the dimension of craniectomy correlating this aspect to clinical outcome, neuroradiological findings, and rate of complications.

We performed, in 26 patients, a 3 cm × 3 cm craniectomy, with the removal of the most median third of the posterior arch of C1 and duraplasty with heterologous patches. In the light of our results and after a careful evaluation of the relevant literature, we think that this surgical strategy could represent a reliable and feasible approach to achieve a good outcome and a low rate of complications.

MATERIALS AND METHODS

Patients selection and data collection

A retrospective review of medical records of 26 patients, affected by CM-I with and without syringomyelia, undergone surgery from 2004 to 2014, was conducted. The epidemiological information, clinical presentation, duration of symptoms, radiological findings, and the clinical outcome were determined. All patients underwent brain and spine magnetic resonance (MR) study preoperatively.

Surgical technique

The patients were positioned in prone position, with the head in slight flexion and fixed with the Kees-Mayfield three-pin head holder. A midline skin incision is performed starting 1 cm above the inion to the spinous process of C2. The fascia and muscles were incised and dissected in a subperiosteal fashion until the occipital bone and the posterior arch of C1 were exposed. A 3 cm × 3 cm suboccipital craniectomy was realized. The decompression was completed through the removal of the most median third of the posterior arch of C1 beneath and contiguous to the craniectomy [Figure 1a and b]. The posterior atlanto-occipital membrane and the fibrous band of tissue covering the dura were gently dissected preserving the dural plane. Aiming to preserve the arachnoid layer, a careful Y-shaped dural incision was performed. In some cases, the dura appeared thick and required a more careful incision and dissection from the arachnoid plane. The cerebellar tonsils were dislocated downward through the foramen magnum, resulting in an obliteration of cisterna magna. In 11 of our cases, we visualized the pulsation from downward to upward of cerebellar tonsils. Duraplasty with a bovine pericardium graft was performed in 10 cases, whereas a patch of Goretex was used in 16 cases, and finally, a watertight closure was realized. The closure was reinforced by Gelfoam and a fibrin glue layer.

Follow-up and outcomes

Patients' clinical examinations were compared with their preoperative examinations in the 1st, 6th, and 12th months after surgery. A postoperative MR imaging study was performed at each follow-up control [Figures 2a and b, 3a and b]. In each, control was determined the outcome (improved, no change, or worse).
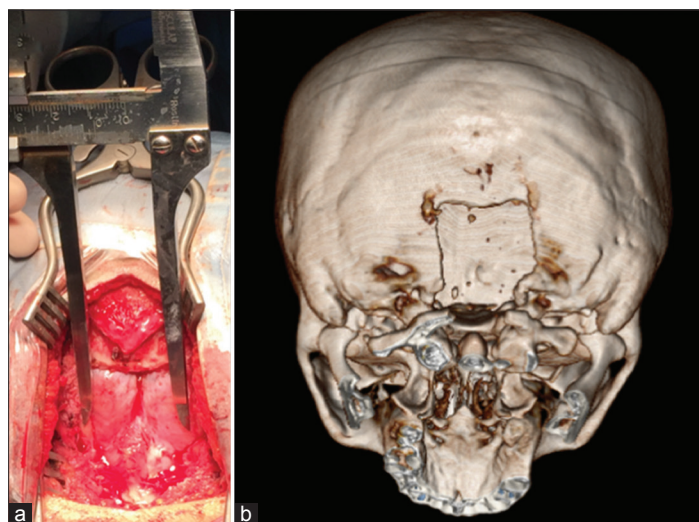

Figure 1: (a) The image shows the 3 cm × 3 cm craniectomy and the removal of the most median third of the posterior arch of C1, (b) Three-dimensional computed tomography scan reconstruction demonstrates the suboccipital craniectomy and removal of the most median third of posterior arch of C1.
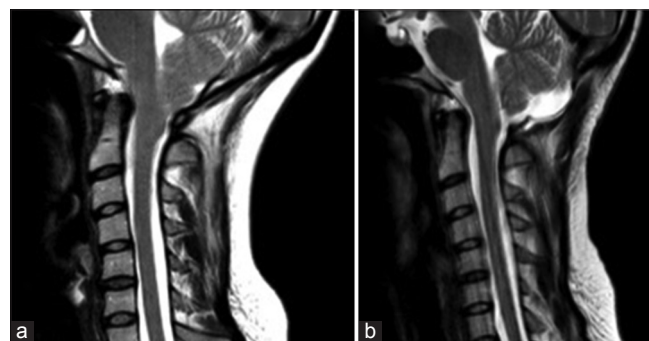

Figure 2: (a) T2 magnetic resonance (MR) weighted images showing the descent of cerebellar tonsils through the foramen magnum and the compression of the medulla oblongata, (b) T2-weighted postoperative MR in sagittal plane demonstrates the repositioning of cerebellar tonsils and the enlargement of subarachnoid spaces of posterior cranial fossa.

Table 1: Summary of nosological, clinical, and neuroradiological data of patients with Chiari malformation type I without associated syringomyelia.

Age (y) sex	Preoperative symptoms	Neuroradiological findings	Associated pathologies	Outcome	Complications
40, M	Headache, cervical pain	Cerebellar tonsils descent below Mc Rae line: 11 mm	Hydrocephalus	Improved	
22, F	Headache	Cerebellar tonsils descent below Mc Rae line: 7 mm		Improved	
38, F	Headache, paresthesias	Cerebellar tonsils descent below Mc Rae line: 17 mm		No change	
60, M	Cervical pain	Cerebellar tonsils descent below Mc Rae line: 10 mm		Improved	
27, M	Sensory and motor deficits	Cerebellar tonsils descent below Mc Rae line: 17 mm	Hydrocephalus	No change	
30, F	Headache, sensory and motor deficits	Cerebellar tonsils descent below Mc Rae line: 15 mm		Improved	
35, M	Headache, cervical pain, sensory and motor deficits, dizziness	Cerebellar tonsils descent below Mc Rae line: 16 mm		Improved	
26, F	Sensory and motor deficits, paresthesias	Cerebellar tonsils descent below Mc Rae line: 11 mm	Hydrocephalus	Worse	Worsening of preoperative symptoms
57, M	Sensory and motor deficits, paresthesias	Cerebellar tonsils descent below Mc Rae line: 16 mm		Improved	
15, M	Cervical pain, paresthesias	Cerebellar tonsils descent below Mc Rae line: 13 mm	Hydrocephalus	Improved	
21, F	Headache, cervical pain	Cerebellar tonsils descent below Mc Rae line: 8 mm		Improved	
36, M	Headache, cervical pain, sensory deficits, paresthesias	Cerebellar tonsils descent below Mc Rae line: 14 mm		Improved	
28, M	Cervical pain, sensory and motor deficits	Cerebellar tonsils descent below Mc Rae line: 13 mm		Improved	
17, M	Headache, cervical pain	Cerebellar tonsils descent below Mc Rae line: 5 mm		Improved	
28, F	Cervical pain, paresthesias	Cerebellar tonsils descent below Mc Rae line: 8 mm		Improved	

RESULTS

The study included 26 patients, 12 males and 14 females. The mean age at surgery was 35.8 years. The patients were divided into two groups. The former is made up of CM-I patients without syringomyelia, while the second is from patients with CM-I associated with syringomyelia. The characteristics of both series are summarized in Tables 1 and 2.

The first series included patients with CM-I without associated syringomyelia. This series included 15 patients, aged between 15 and 60 years. Symptoms at presentation are listed in Table 1. The most frequent presenting symptoms were cervical pain (9 patients, 60%), headache (8 patients, 53.3%), paresthesias (8 patients, 53.3%), sensory deficits (7 patients, 46%), motor deficits (6 patients, 40%), and dizziness (1 patient, 6.6%). The mean tonsillar descent measured from the rim of the foramen magnum was 12.1 mm (range 5–17 mm). In 4 patients (26.6%), CM-I was associated with hydrocephalus. There were no long-term neurological or surgical complications. There were no deaths. Mean postoperative follow-up was 27.5 months (range 5–72 months). At follow-up, 12 patients (80%) demonstrated a complete resolution of symptoms and 3 patients (20%) demonstrated a partial resolution of clinical signs and symptoms. Postoperative studies also showed a reduction of hydrocephalus in all cases.

The second series consisted of patients affected by CM-I with associated syringomyelia. This series included 11 patients, aged between 16 and 67 years. Symptoms at presentation are listed in Table 2. Preoperative symptoms were sensory deficits in all patients, motor deficits (9 patients, 81.8%), paresthesias (7 patients, 63.6%), cervical pain (7 patients, 63.6%), headache (4 patients, 36.3%), lower cranial nerve deficits (3 patients, 27.2%), and bladder incontinence (2 patients, 18.1%). The mean tonsillar descent, measured from the rim of the foramen magnum, was 13.2 mm (range 5–18.3 mm). In 2 patients (18.1%), CM-I was also associated with hydrocephalus and a patient (9%) additionally presented scoliosis and tethered cord. One patient (9%) developed a CSF leakage. There were no long-term neurological complications. There were no deaths. Mean postoperative follow-up was 27.5 months (range 5–72). At follow-up considered, 8 patients (72.7%) demonstrated a complete resolution of symptoms. Of the remaining 3 patients (27.2%), one patient showed a worsening of the preoperative symptoms, while another patient revealed the appearance of paresthesias and respiratory impairment. The third patient presented no change in the clinical picture. Postoperative studies showed a moderate reduction of hydrocephalus in all cases.

Progressive reduction in syrinx volume was observed in 8 patients (72.7%) [Figure 3a and b], while it remained unchanged in the remaining 3 cases (27.2%).

DISCUSSION

CM-I includes a group of entities of congenital or acquired etiology that has in common the caudal displacement of the cerebellar

Table 2: Summary of nosological, clinical, and neuroradiological data of patients with Chiari malformation type I with associated syringomyelia.

Age (y) sex	Preoperative symptoms	Neuroradiological findings	Associated pathologies	Outcome	Complications
51, F	Headache, cervical pain, sensory and motor deficits	Cerebellar tonsils descent below Mc Rae line: 16 mm, syringomyelia		Improved; Resolution of syringomyelia	
62, M	Headache, cervical pain, sensory and motor deficits, paresthesias	Cerebellar tonsils descent below Mc Rae line: 12 mm syringomyelia		Improved; Reduction of syringomyelia	
59, F	Headache, cervical pain, sensory deficits, paresthesias	Cerebellar tonsils descent below Mc Rae line: 15 mm syringomyelia		Improved; Reduction of syringomyelia	
18, M	Sensory and motor deficits, paresthesias, dizziness	Cerebellar tonsils descent below Mc Rae line: 16 mm syringomyelia		Improved; Resolution of syringomyelia	
67, F	Cervical pain, sensory and motor deficits, dizziness, lower cranial nerve deficits	Cerebellar tonsils descent below Mc Rae line: 14 mm syringomyelia		Worse; Syrinx volume unchanged	Respiratory impairment, paresthesias
40, F	Sensory and motor deficits, paresthesias, lower cranial nerve deficits	Cerebellar tonsils descent below Mc Rae line: 12 mm syringomyelia	Hydrocephalus	Improved; Resolution of syringomyelia	
36, F	Sensory and motor deficits, paresthesias	Cerebellar tonsils descent below Mc Rae line: 11 mm syringomyelia		Improved; Resolution of syringomyelia	
47, F	Sensory and motor deficits, paresthesias	Cerebellar tonsils descent below Mc Rae line: 16 mm syringomyelia		Improved; Reduction of syringomyelia	
16, F	Cervical pain, sensory and motor deficits, bladder incontinence	Cerebellar tonsils descent below Mc Rae line: 12 mm syringomyelia	Hydrocephalus, scoliosis, tethered cord	Worse; Syrinx volume unchanged	Worsening of preoperative symptoms
32, F	Headache, cervical pain, sensory deficits, dizziness, paresthesias, lower cranial nerve deficits, bladder incontinence	Cerebellar tonsils descent below Mc Rae line: 11 mm syringomyelia		No change; Minimal reduction of syrinx volume	Fistula
22, F	Cervical pain, sensory and motor deficits	Cerebellar tonsils descent below Mc Rae line: 15 mm syringomyelia		Improved; Reduction of syringomyelia	

Figure 3: (a) T2 magnetic resonance (MR) weighted images showing the descent of cerebellar tonsils through the foramen magnum and T2–T9 syringomyelia, (b) T2 MR weighted 6-month postoperative picture reveals the decrease in size of syringomyelia.

tonsils through the foramen magnum into the cervical canal. This may be due to the underdevelopment of occipital somites from paraxial mesoderm. As a result, a small posterior fossa is developed predisposing to a downward herniation of its contents, i.e., cerebellar tonsils migrating to cervical spinal canal. In 50%–76% of patients, the malformation is associated with hydromyelic cavitation of the spinal cord and medulla oblongata.[2,16] In these cases, it has been postulated that obstruction of the outlets of the fourth ventricle, with diversion of the CSF pulse wave into the central canal or the foramen magnum, may cause a pressure gradient between the intracranial and intraspinal compartments, pulling in fluid from the fourth ventricle into the central canal.[29] CM-I usually presents after the second or third decade of life. Patients with CM-I present a wide range of signs and symptoms as headache and cervical pain. Weakness and lower extremity spasticity are also frequent, associated with disorders related to myelopathy (motor and sensory losses, hypo- and hyper-reflexia, and Babinski response), cerebellar (ataxia), and brainstem dysfunctions (respiratory irregularities, nystagmus, and lower cranial nerve dysfunction). Upper limbs deficit associated with

signs of syringomyelia (thermic and pain dissociation, and hand muscle atrophy) is described in patients with syringomyelia. Few studies have been performed on the histopathological alterations present within the cerebellar tonsils of patients with CM-I.[27] Purkinje cells loss and reactive gliosis were demonstrated to be the most frequent findings and their genesis was connected to local trauma depending on cerebellar tonsils herniation.[27]

Although the pathogenesis of CM-I is still mattered of debate, the posterior fossa volume mismatch is the leading cause of CM-I. Marin-Padilla showed that CM-I may be caused by a mesodermal insufficiency occurring after closure of the neural folds.[24] According to this hypothesis, a small posterior cranial fossa may be an essential reason in the hindbrain hernia formation.[13] This event leads to herniation of the tonsils below the foramen magnum. However, it is widely accepted that CM-I depends on a premature stenosis of spheno-occipital synchondrosis with transformation of posterior cranial fossa in a narrow funnel shape without statistically significant differences of anterior-posterior diameter of the foramen magnum between CM-I and control group.[16] Morphometric studies demonstrated that the disproportion of posterior cranial fossa is due attributable to basioccipital hypoplasia.[25] In addition, it should be considered that CM-I is characterized by occurrence of frequent and concomitant severe adhesions between the dura, arachnoid, and neural tissue. This arachnoid scarring may cause itself clinical symptoms, which are independent of brain stem compression and syringomyelia, acting as an additional factor for impaired CSF flow.[13]

MR represents the optimal tool in the diagnosis since it is noninvasive and has a good correlation with clinical findings. Cine flow MR studies have recently been introduced into clinical use and have gained importance in decision-making of the ideal surgical procedure.[15] The use of intraoperative imaging for Chiari I malformation is usually limited by the CSF flow dynamics across the foramen magnum which significantly change when the patient is positioned for surgery.[26] Computed tomography gives data in axial, sagittal, and coronal sections and makes achievable three-dimensional reconstructions, for a more correct knowledge of the anatomy of the CVJ.

Indications for surgery include the presence of neurological symptoms, their progression, and/or headache caused by herniation of the cerebellar tonsils and significantly deteriorating the patients' quality of life. However, nowadays, there is still considerable controversy about the optimal surgical procedure for the treatment of CM-I with or without syringomyelia. In cases characterized by symptoms clearly attributable to syringomyelia, shunting of the syringomyelic cavity is indicated, based on fluid diversion in the subarachnoid space or into extracavitary locations (peritoneal and pleural cavities). However, in these cases, treated with shunting procedures, the cause is not removed and the pathogenetic mechanism persists. PFD with and without cervical laminectomy is the preferred treatment option that allowing a satisfactory CSF flow restoration and tonsillar repositioning.[1,7]

The goal of decompression is the improvement of symptoms and the reduction of CSF pressure at the CVJ. The surgical approach may be limited to a merely extradural decompression with lysis of extradural adhesions. It may consist of the opening of the outer layer of dura mater (dura splitting),[6,9] performing transverse microincisions of the outer layer of the dura mater[13] or achieving duraplasty with or without lysis of adhesions around the cerebellar tonsils, and reducing in size of the latter through coagulation or partial tonsillectomy.[4,19,23]

Despite several series have been published about the choice of approach in terms of PFD, the dimension of craniectomy still represents matter of debate, due to few data exist in literature. The optimal extent of bony removal varies from surgeon to surgeon. A reduced craniectomy could cause an inadequate decompression, while a large craniectomy might theoretically allow an abnormal dural distention and the descent of the cerebellar tonsils through the bony defect. Sindou *et al.* analyzed the clinical outcome of a series of 44 patients affected by CM-I with and without syringomyelia.[28] Their technique consisted of a large craniectomy extended to the occipital condyles on either side to achieve an optimal decompression of cerebellar tonsils, with dural opening and preservation of arachnoid membrane. They reported an improvement of the Karnofsky score in 83% and 80% of patients, respectively, with CM-I alone and with associated syringomyelia.[28] As well, Chotai *et al.* demonstrated a good rate of symptoms improvement performing a large craniectomy, stressing the importance to create a new artificial cisterna magna.[9] On the other hand, Klekamp *et al.* referred that large craniectomy is correlated to a worse outcome than smaller craniectomy.[17] However, smaller decompressions are linked with few rate of complications.[6,9] As well, the removal of the posterior arch of C1 during PFD has to consider. Few authors focused on this aspect.[28] In particular, two studies showed encouraging both clinical and radiological results, (90% improvements), performing a PFD with C1 arch resection.[9]

The objective of the craniectomy should be to restore the volume of the cisterna magna and decompress the brainstem. An excessively wide craniectomy could cause cerebellar herniation into spinal canal and compression on brainstem, obstruction of the liquoral circulation, and formation of arachnoidal scarring. In the light of these observations, in our series, we performed a suboccipital 3 cm × 3 cm^2 craniectomy combined with the removal of the most median third of the posterior arch of C1, below and contiguous to the craniectomy. This phase allows us a sufficient decompression of the posterior cranial fossa and the restoration of CSF flow at CVJ.

Published series reports clinical improvement rates after surgical treatment from 71% to 100%[10,20] and syringomyelia resolution rates from 52% to 91%.[18,20] Our results seem to be aligned. The incidence of clinical improvement of our patients was 76.9% (72.7% and 80% in patients with and without syringomyelia, respectively). In literature, the reported rate of complications is between 3% and

40%.[1,7,15] In our experience, the rate of complications was 23% (20% in the first series and 27.2% in the second one). Compared to other series,[1,7,15] our data demonstrate a good postoperative outcome and, overall, a low incidence of complications.

Fenestration of the arachnoid plane is still mattered of debate; some authors claim that preservation of arachnoidal layer, likewise dura mater, is associated to lower risk of CSF-related complication and is a safe and effective surgical option, mainly when no evidence of arachnoiditis or obstruction of the outflow at the level of foramen of Magendie occurs.[20,26] It is our opinion that opening of the arachnoid exposes the subarachnoid space to cellular debris that increases the risk of adhesive arachnoiditis, aseptic meningitis, and CSF leak. Conversely, others argue the need to explore the arachnoid flat and identify arachnoid strands and veils, potential obstacles to the ascent of cerebellar tonsils and to the reestablishment of normal CSF flow.[4] Several authors[14,19,23] emphasized the usefulness and the utility of cerebellar tonsils reduction to restore the CSF pathway. Guyotat *et al.* supported the clinical outcome improvement with tonsils resection, especially in patients with syringomyelia.[14] For other authors, it is enough to remove herniated cerebellar tonsils from the cervical canal through atlanto-occipital interspace without perform any craniectomy or C1 laminectomy; this route could be considered a safe procedure, as a matter of fact that in CM-I cerebellar tonsils have often a sclerotic or an atrophic histopathologic pattern.[19] We believe that a tailored surgical technique is necessary, with tonsils reduction/tonsillectomy, only, if an optimal intraoperative restore of CSF flow is not achieved. The need to create a wide cisterna magna to allow the cerebellar tonsils ascent is widely reported in literature.[8,12]

Another critical issue is the duraplasty. Some authors published that PFD with duraplasty is more effective than PFD alone.[1,4,15] A systematic review of literature[11] showed a higher reoperation rate in patients who underwent PFD alone compared with PFDD group, conversely, CSF-related complications were more frequent in PFDD, but the difference was not statistically significant. This study highlighted, however, that clinical outcome is similar in both groups.[11] A meta-analysis was recently conducted to compare the validity of PFD with duraplasty and PFD in treating patients with CM-I. This study confirmed that the decrease in syringomyelia was better for patients treated with PFDD than for patients treated with PFD alone.[5] In line with these data, our number two series, although numerically limited, has shown, in all cases, a decrease in size of the syringomyelic cavity.

The choice of dural substitute is a critical topic. Attenello *et al.* compared the use of pericranium autograft and synthetic expanded polytetrafluoroethylene (ePTFE) in pediatric patients. Both dural grafts were associated to a great rate of clinical improvement and a minimal incidence of complications; ePTFE provided an earlier improvement in syringomyelia than autologous graft, without differences in absolute incidence.[3] Other authors compared unsutured and suturable dural substitutes, obtaining similar

clinical outcome and rates of complication.[30] In another study by Lee *et al.*, the use of porcine and bovine dural grafts was matched; the rate of pseudomeningocele was higher in porcine ones.[21] In our institution, we always realized a duraplasty using bovine pericardium or Gore-Tex (a synthetic polytetrafluoroethylene graft), performing a watertight closure to obtain a wide space around cerebellar tonsils.

CONCLUSION

Although CM-I physiopathology is still unclear, signs and symptoms could be attributed to a CSF flow impairment due to an overcrowding of posterior fossa structures. The main goal of surgical treatment is to restore a normal outflow at CVJ. Our treatment option is a suboccipital 3 × 3 craniectomy, the removal of the median part of C1 arch, and duraplasty with bovine dural graft or Gore-Tex patch. We can consider satisfactory our clinical results and our rate of complications, even more if compared with the data reported in literature. This technique can be considered a valid and safe choice for the treatment of CM-I.

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Conflicts of interest

There are no conflicts of interest.

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