DOI: 10.1590/0004-282X20130105

ARTICLE

Suboccipital craniectomy with opening of the fourth ventricle and duraplasty: study of 192 cases of craniovertebral malformations

Descompressão da fossa posterior sem tonsilectomia em 192 casos de impressão basilar, malformação de Chiari e/ou siringomielia

José Alberto Gonçalves da Silva[1], Adailton Arcanjo dos Santos Jr.[2], Maria do Desterro Leiros da Costa[3], Everardo Bandeira de Almeida[4]

ABSTRACT

The prime objective in the surgical treatment of basilar impression (BI), Chiari malformation (CM), and/or syringomyelia (SM) is based on restoration of the normal cerebrospinal fluid (CSF) dynamics at the craniovertebral junction and creation of a large artificial cisterna magna, avoiding the caudal migration of the hindbrain. It is observed that a large craniectomy might facilitate an upward migration of the posterior fossa structures. There are many surgical techniques to decompress the posterior fossa; however, a gold standard approach remains unclear. The authors present the results of 192 cases of BI, CM, and SM treated between 1975 and 2008 and whose surgical treatment was characterized by a large craniectomy without tonsillectomy with the patient in the sitting position, large opening of the fourth ventricle, and duraplasty.

Key words: basilar impression, Chiari malformation, hindbrain herniation, craniovertebral decompression, syringomyelia.

RESUMO

O principal objetivo no tratamento cirúrgico da impressão basilar, malformação de Chiari e/ou siringomielia fundamenta-se na restauração da dinâmica do líquido cefalorraqueano ao nível da transição craniovertebral e criação de cisterna magna ampla. Isto é fator importante para evitar a migração caudal das estruturas da fossa posterior. A craniectomia ampla facilita a migração cranial dessas estruturas. Existem várias técnicas cirúrgicas para descomprimir a fossa posterior, mas não há evidência sobre qual a melhor.. Os autores apresentam os resultados de 192 casos de impressão basilar, malformação de Chiari e siringomielia, operados entre 1975 e 2008, nos quais o tratamento cirúrgico se baseou em ampla craniectomia com o paciente em posição sentada, sem tonsilectomia, abertura ampla do quarto ventrículo e enxerto dural.

Palavras-Chave: impressão basilar, malformação de Chiari, herniação rombencefálica, descompressão craniovertebral, siringomielia.

Basilar impression (BI), Chiari malformation (CM), and syringomyelia (SM) are the most frequent malformations at the craniovertebral junction. BI was originally described by Ackermann[1] (1790) in Cretins from the Alps. At that time, he described the small size of the posterior fossa, the elevation of the clivus, and the projection of the border of the foramen magnum into the posterior fossa. In 1857, Virchow[2] introduced the term platybasia and in 1876[3] the denomination "basilar impression."

Chiari[4,5] (1891, 1895) described four types of cerebellar anomalies. Type I was characterized by downward displacement of the cerebellar tonsils and the medial portion of the inferior cerebellar lobes, which accompanied the medulla into the cervical spinal canal. Type II showed downward displacement of the portions of the cerebellum (1891), and portions of the inferior vermis (1895), pons, medulla, and, at least, a part of the lengthened fourth ventricle, which reached the disc C4–C5, into the enlarged cervical spinal canal. In type III, the hydrocephalic cerebellum, pons, and medulla were inside a cervical meningocele (*hydroencephaloceles cerebellaris cervicalis*), through a spina bifida of the first three cervical vertebrae. In type IV, there is hypoplasia

[1]Neurosurgical Division of the Hospital Unimed, João Pessoa PB, Brazil;
[2]Neurosurgical Division of the Hospital Beneficência Portuguesa de São Paulo, São Paulo SP, Brazil;
[3]Federal University of Paraíba, João Pessoa PB, Brazil;
[4]Neuro-anesthesist, João Pessoa PB, Brazil.
Correspondence: José Alberto Gonçalves da Silva; Avenida Minas Gerais 1150; 58030-092 João Pessoa PB - Brazil; E-mail: jags1803@hotmail.com
Conflict of interest: There is no conflict of interest to declare.
Received 6 September 2012; Received in final form 9 April 2013; Accepted 16 April 2013.

of the cerebellum without herniation of cerebellar structures into the spinal canal.

With respect to SM, Simon[6] introduced the term hydromyelia to designate the dilation of the ependymal canal by cerebrospinal fluid (CSF) and kept the term SM related to cavities that developed independent of the central canal of the spinal cord. It has been unanimously agreed, in the literature, that both are different stages of the same pathological process. However, Finlayson[7] stated that hydromyelia is considered to be a congenital disturbance due to an incomplete regression of the fetal ependymal canal, whereas SM can be congenital or acquired.

The high prevalence of BI associated with CM in the Northeast of Brazil was reported by many authors[8-12], although there is no suitable known reason for this fact.

The main objective of this paper was to analyze the surgical results obtained with the use of a large craniectomy without tonsillectomy with the patient in the sitting position in 192 cases of BI, CM, and/or SM.

METHOD

This study is based on a retrospective review of 192 patients — 117 men and 75 women with occipitovertebral malformation. One patient was excluded because the dura mater could not be technically opened during the surgery and the presence of tonsillar herniation was not possible to be determined. About 164 cases (85.8%) of BI and CM, 12 (6.2%) cases of BI only, and 15 (7.8%) cases of CM only were observed. There were 26 cases (13.5%) of SM associated with BI and/or CM and 5 other cases associated with hydrocephalus.

This study was approved by the Ethics Committee of the Hospital Unimed, João Pessoa-PB, Brazil.

One of the most important diagnostic tools was the neurological examination associated with some radiological methods.

The radiological methods were based on x-ray of the skull to analyze the Chamberlain and McGregor's lines to try to diagnose the presence of BI. MRI was not available for most of the patients, with exception of the last three cases — MRI was performed after surgery in two cases and pre- and postoperative MRI was performed in the third case.

Myelography was performed in many patients with the use of a contrast agent to analyze the level of the craniovertebral junction. In cases of CM, it may take the shape of an open C[9,13], which is not a pathognomonic sign of CM. Ventriculography may confirm CM when it depicts an elongated fourth ventricle and downward migration of the cerebellar tonsils[8,9,13].

As to the surgical technique, the patients underwent osteodural decompression without tonsillectomy with large suboccipital craniectomy associated with laminectomy varying from C1 to C3 level depending on the tonsillar herniation, dural opening in Y format, dissection of the arachnoidal adhesions, and large opening of the foramen of Magendie, and, finally, a dural grafting was performed with the use of fascia lata in 6 cases, liophilizated dura mater in 1 case, bovine pericardium in 7 seven other cases, and dura mater conserved in glycerin in 158 cases. The operation description did not mention the type of dural grafting in 19 cases and, finally, the arachnoidal membrane was not opened in 53 cases.

RESULTS

The clinical symptoms observed in the pre- and postoperative examination are summarized in Table 1, the clinical signs in Table 2, and the surgical findings in Table 3. The data of 23 (12%) patients without follow-up and 13 (6.7%) patients who died were excluded in Tables 1 and 2. All the cases were followed with clinical studies for a mean of 1 month to 32 years. The age at surgery ranged from 2 to 66 years, with a mean of 33.4 years.

The authors observed that 176 cases out of 191 patients were associated with BI, 179 cases were associated with herniation of the cerebellar tonsils as exemplified in the Figs 1 to 3, 48 cases were associated with herniation of the brainstem, 26 cases were associated with SM, 2 cases were associated with syringobulbia, and 16 patients presented a communication between the fourth ventricle and the hydromyelic cavity. As to SM and/or CM cases, the authors observed 35 cases of blockage of the foramen of Magendie. Regarding the family occur-

Table 1. Clinical symptoms observed in 155 cases of Basilar impression and Chiari malformation without tonsillectomy and syringomyelia.

Symptoms	Cases/ number	%	R	%	A	%	U	%
Headache	90	58	80	88.8	3	3.3	7	7.7
Neck pain	56	36.1	54	96.4	–	–	2	3.5
Nuchal rigidity	82	52.9	75	91.4	–	–	7	8.5
Diplopia	37	23.8	32	86.4	–	–	5	13.5
Rhinolalia	76	49	1	1.3	–	–	75	98.6
Dysphagia	74	47.7	70	94.5	–	–	4	5.4
Dysarthria	25	16.1	22	88	–	–	3	12
Nasal reflux	47	30.3	40	85.1	–	–	7	14.8
Vertigo	83	53.5	75	90.3	2	2.4	6	7.2
Limb weakness	96	61.9	59	61.4	27	28.1	10	10.4
Paresthesia in face	26	16.7	24	92.3	–	–	2	7.6
Limb paresthesia	60	30.7	39	65	9	15	7	61.6
Sexual troubles	59	30	20	33.8	2	3.3	37	62.7
Legs collapse	54	34.8	53	98.1	–	–	1	1.8
Anhidrosis	1	0.6	–	–	–	–	1	100
Hyperhidrosis	3	1.9	–	–	–	–	3	100
Syringomyelic pain	4	2.5	3	7.5	–	–	1	25
Seizures	8	5.1	3	37.5	–	–	5	62.5

R: regressed; A: amelioration; U: unchanged; BI: basilar impression; CM: Chiari malformation; SM: syringomyelia.

Table 2. Clinical signs observed in 155 cases of Basilar impression and Chiari malformation without tonsillectomy.

Signs	Cases/ number	%	R	%	A	%	U	%
Papilledema	3	1.9	3	100	–	–	–	–
Lesion of the Vth nerve	66	42.5	50	75.7	–	–	16	24.2
Lesion of the VIth nerve	14	9	10	71.4	–	–	4	20.5
Facial spasm	5	3.2	3	60	–	–	2	40
Lesion of the XIth nerve	75	48.3	34	45.3	3		33	4
Lesion of the XIIth nerve	26	16.7	13	50	–	–	13	50
Abolition of gag and palatal reflexes	78	50.3	21	26.9	–	–	57	73
Soft palate paresis	25	16.1	18	72	–	–	7	28
Nystagmus	65	41.9	26	40	–	–	39	60
Cerebellar–vestibular disturbance	54	34.8	48	88.8	–	–	6	11.1
Limbs hypotony	55	35.4	40	85.1	–	–	15	27.2
Spasticity	47	30.3	27	57.4	10	21.2	10	21.2
Limb paresis	84	54.1	50	59.2	20	12.9	14	9
Hyperreflexia	90	50	6	6.6	3	3.3	81	90
Hoffman sign	42	27	17	40.4	–	–	25	59.5
Babinski sign	27	17.4	14	51.8	–	–	13	40.1
Unsteady gait	62	40	31	50	14	25.5	10	16.1
Hypopallesthesia	102	65.8	2	1.9	–	–	100	98
Disturbance of superficial sensation	51	32.9	28	54.9	3	5.8	20	39.2
Syringomyelic dissociation	23	14	7	30.4	4	17.3	12	52.1
Fasciculation	3	1.9	1	33.3	–	–	2	66.6
Atrophy	13	8.3	–	–	1	7.6	12	92.3
Claude Bernard-Horner syndrome	3	1.9	1	33.3	–	–	2	66.6

R: regressed; A: amelioration; U: unchanged; BI: basilar impression; CM: Chiari malformation; SM: syringomyelia.

Table 3. Surgical findings observed in 191 cases of Basilar impression and Chiari malformation without tonsillectomy and syringomyelia.

Findings	Cases/number	%
Thinning of the occipital bone	80	41.8
Thickening of the occipital bone	64	33.5
Thickening of dura mater and atlanto-occipital ligament	37	19.3
Pulseless dura mater	32	16.7
Arachnoiditis	112	50.6
Blockage of the foramen of Magendie	35	18.3
Brainstem herniation	48	25.1
Tonsillar herniation1	79	93.7
Tonsillar cyst	2	1.0
Communication of the fourth ventricle and hydromyelic cyst	16	8.3
Vascular network anomaly	83	43.4
Syringobulbia	2	1.0
Syringomyelia	26	13.6
BI + CM	164	85.8
Isolated BI	12	6.2
Isolated CM	15	7.8
Hydrocephalus	5	2.6

BI: basilar impression; CM: Chiari malformation; SM: syringomyelia.

rence of BI, the authors found three couples of relatives in the work: brother and sister, two sisters, and mother and son.

The downward displacement of the herniated tonsils varied from the level of the end of C1 to the end of C4, being 26 cases of the caudal migration in C1, 43 cases in C1 and C2, 13 cases in C1 and C3, 27 cases in C2, 27 cases in C2 and C3, 6 cases in C3, and, finally, 8 cases in the level of C3 and C4.

The vascular network anomalies in 83 cases were characterized by the absence of the posterior inferior cerebellar artery (PICA) on the right side and another case on the left side, absence of both PICAS in four cases, a left hypoplastic PICA in two cases, a left missing PICA and a right large PICA in four cases, a left and right hypoplastic PICA in two cases, a left hypoplastic and a right large PICA in two cases, absence of the right PICA and a left hypoplastic PICA in one case, and, finally, looping sign of both PICAS in 66 cases.

DISCUSSION

Many theories have been presented in an attempt to explain the origin of CM. Experimental models, using vitamin A as a teratogen agent in hamsters, were used to try to explain the genesis of craniovertebral malformations. Marin-Padilla[14] has demonstrated that CM I may be essentially a primary para-axial mesodermal insufficiency occurring after the closure of the neural folds. According to that theory, a small posterior fossa induced by an underdeveloped occipital bone would be the prime factor in the formation of the hindbrain herniation. The cerebellum grows more rapidly after birth than small posterior fossa, resulting in a disproportion between the developing hindbrain and the posterior fossa, and, consequently, a caudal migration of the cerebellar tonsils into the cervical canal is produced[14].

The neurological examination was one of the most important tools to make the diagnosis of BI, CM, and SM, as summarized in Table 1. One of the most uncommon signs in BI was fasciculation as described by Raupp et al.[15] The authors emphasized the compression-ischemic etiology and ruled that Pozo et al. described one similar associated with BI. We have observed three patients with fasciculation in our present paper and four other cases in a recently published paper[16].

A comparative study of symptoms in isolated cases of BI and CM showed that in BI the association of motor and

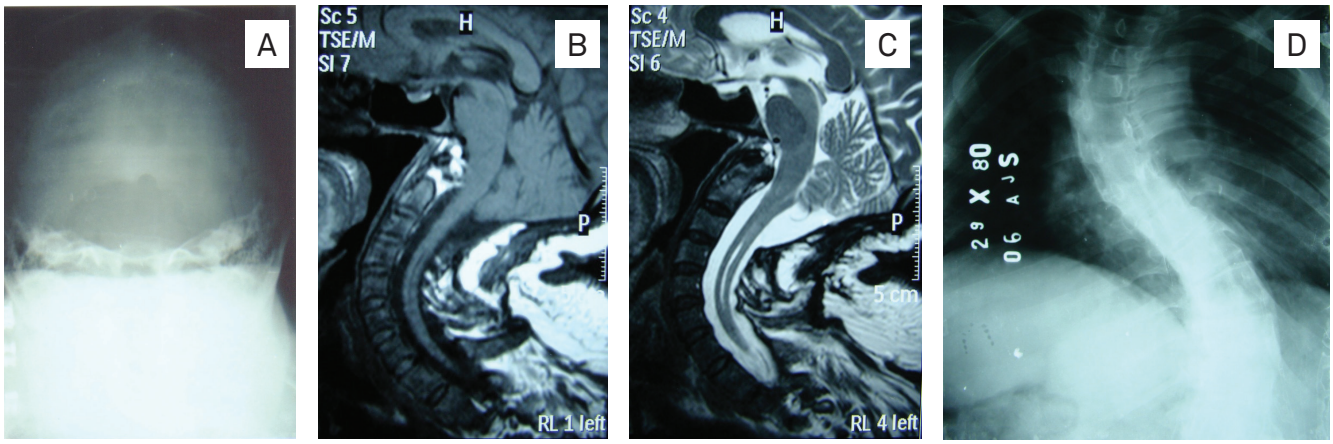

Fig 1. (A) Skull x-ray showing large craniectomy of the posterior fossa. (B) Preoperative MRI depicting hindbrain herniation and enlargement of the spinal canal. (C) Postoperative MRI showing a large artificial cisterna magna. (D) X-ray depicting thoracic and lumbar escoliosis.

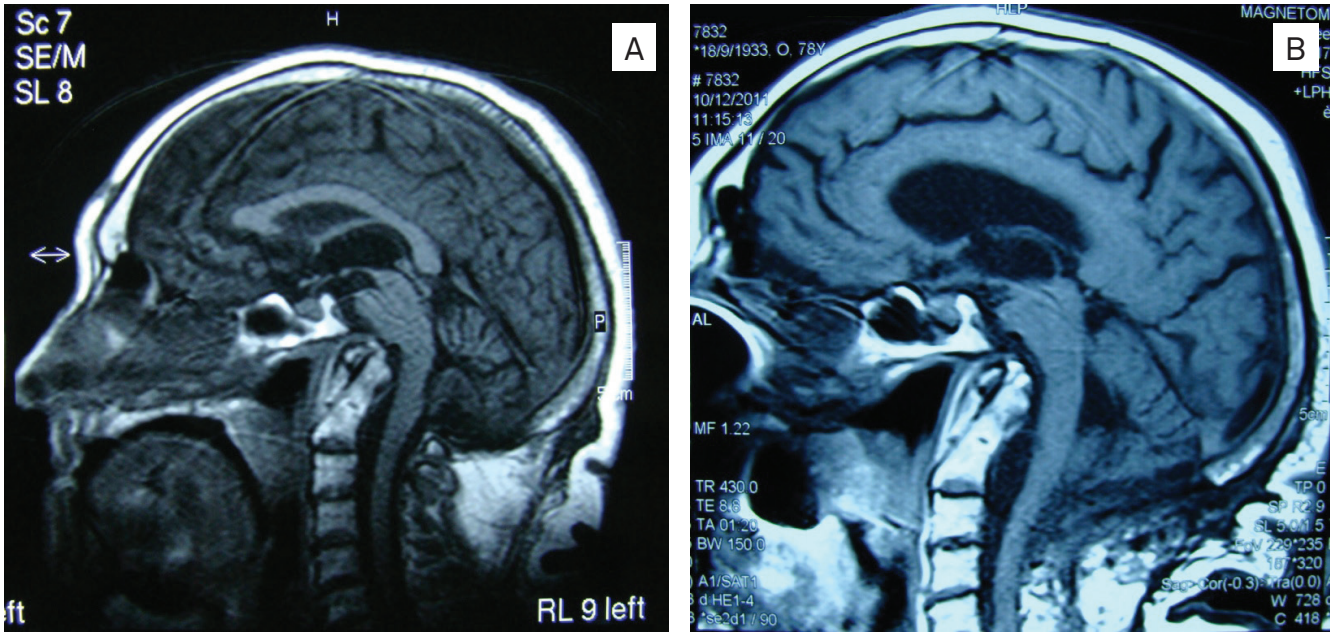

Fig 2. (A) Preoperative MRI showing tonsillar herniation and anterior compression of the brainstem. (B) Postoperative MRI depicting upward migration of hindbrain and no compression of the brainstem.

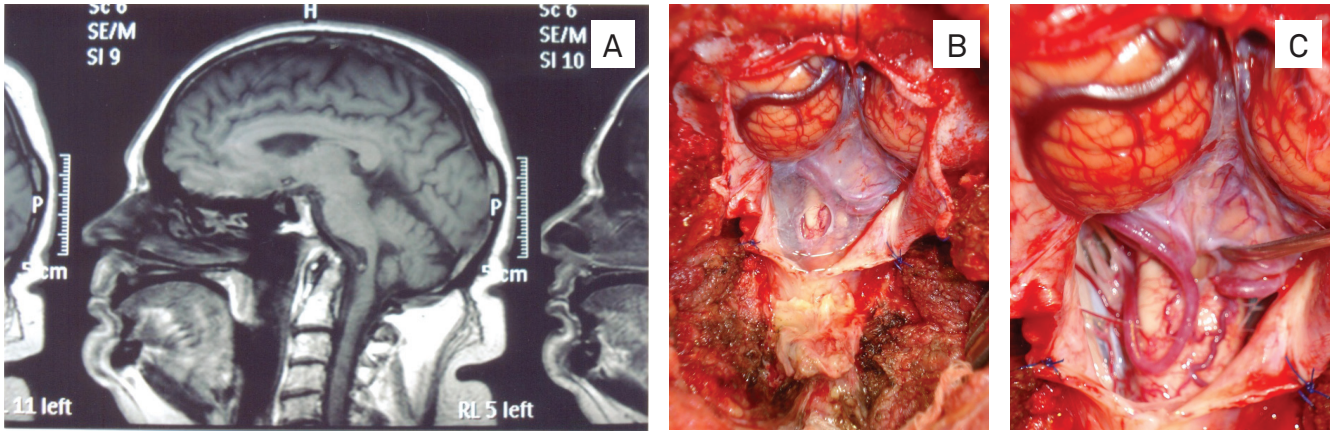

Fig 3. (A) Postoperative MRI showing basilar impression with compression of the brainstem. (B) Arachnoid adhesions of the posterior fossa. (C) A large opening of the fourth ventricle and looping sign of both posterior inferior cerebellar arteries.

sensory disturbances is the most frequent, while in CM cases, the cerebellar and vestibular signs predominate with the involvement of the lower cranial nerves[9,16].

Regarding the surgical technique, all 192 patients of our work were operated in the sitting position. Gardner and Goodall[17] used this type of position for the surgical treatment of 13 out of 17 patients with CM.

Because of the small size of the posterior fossa caused by BI and/or CM, especially when both anomalies are present, the authors performed a large craniectomy that extends cranially to the transverse sinuses, frequently situated lower than those in normal people, and laterally to 3–4 cm from the midline. The large opening is to try to increase the volume of the posterior fossa and to create a large artificial cisterna magna, which is commonly absent in cases of hindbrain herniation and may facilitate an upward migration of the herniated portions of the cerebellum and brainstem.

Batzdorf[18] and Duddy and Williams[19] admitted that a small craniectomy, on the other side, has been emphasized to avoid the "slump" of the hindbrain into the craniectomy. This slump would perpetuate the craniospinal pressure dissociation at the craniovertebral junction and could be the main cause of poor long-term clinical results[20].

Duddy and Williams[19], however, using a small craniectomy, revealed a frequent downward migration of the cerebellum and brainstem and pointed that as a cause for poor results. These authors observed a downward migration of the posterior fossa structures in 53% of their patients, whereas no change was noticed in 41% and cranial migration of the hindbrain was observed in only one patient.

Sahuquillo et al.[21] compared the results obtained in 10 cases in which a small craniectomy was performed with other 10 patients that underwent an extensive craniectomy. An upward migration of the cerebellum and brainstem was observed in all of the last patients, while in those patients in which a small craniectomy was performed, there was a downward migration in seven of them.

On the other side, Gonçalves da Silva et al.[16] using a large craniectomy with the patient in the sitting position, tonsillectomy, large opening of the fourth ventricle, and duraplasty with creation of a large artificial cisterna magna in 104 cases of BI, CM, and/or SM observed a significant upward migration of the posterior fossa structures.

The volume of the posterior fossa is reduced in cases of BI and/or CM as first described by Ackermann[1]. In recent times, however, some authors[22,23] using x-ray, computed tomography, and MRI have also demonstrated that the posterior fossa volume, in the presence of BI and CM, is smaller than those observed in normal people. Milhorat et al.[24] verified a decrease of 13.4 mL in the total volume of the posterior fossa and 40% (10.8 mL) in the CSF volume of this region.

As to the cerebellar tonsils, the majority of the neurosurgeons[10,12,18,21-26] leave them intact, performing just the opening of the fourth ventricle. We used such technique in 191 cases. In one patient the dura mater was not opened .

Other surgeons[11,17,26-28] recommend the dissection of the arachnoid adherences of the tonsils and vessels and opening of the fourth ventricle. They recommend tonsillectomy as a protection against slump, eliminating the compressive effect over the medulla and spinal cord, as described by Williams[20]. It also contributes to the creation of a large artificial cisterna magna, which can facilitate an upward migration of the hindbrain structures, as we recently published a report of 104 cases[16] in whom a large artificial cisterna magna was created and an upward migration of the posterior fossa structures was depicted by the postoperative MRI. Batzdorf[27] recommends lightly diathermying of the pia mater over the surface of the tonsils. Williams[20], however, admitted that creating an artificial cisterna magna is more efficient than a substantial removal of the cerebellar tonsils, causing no morbidity after the evacuation.

Iskandar et al.[29] reported on five pediatric cases of SM without hindbrain herniation. All patients improved after undergoing posterior fossa decompression. Kyoshima et al.[30] described four similar cases with a good recovery of the patients after the decompressive operation. The authors named the cisterna magna filled by the tonsils as "tight cisterna magna" and designated Iskandar's description as "Chiari O malformation".

Not only the cerebellar tonsils but also the brainstem and cerebellum have a tendency to migrate downward in the sitting position. The authors observed, during the surgery, herniation of the cerebellar tonsils in 179 (93.7%) patients and downward migration of the brainstem in 48 (25.1%).

SM was observed in 26 (13.6%) patients, of which there was communication between the fourth ventricle and the hydromyelic cyst in 16 (8.3%). Syringobulbia was detected in only two (1%) patients. Blockage of the foramen of Magendie was observed in 35 (18.3%) patients, mainly caused by the presence of a dense membrane or adhesions between the tonsils. Gardner and Goodall[17] described in many cases obstruction of the foramen of Magendie by a membrane representing an unperforated rhombic roof.

The vascular network anomalies in 83 (43.4%) cases were characterized by the missing of the PICA on the right side and another case on the left side, absence of both PICAS in 4 cases, a left hypoplastic PICA in 2 cases, a left missing PICA and a large right in 4 cases, a left large PICA and a right hypoplastic in 2 cases, a left hypoplastic and a right large PICA in 2 cases, absence of the right PICA and a left hypoplastic in 1 case, and, finally, looping sign on the PICAS in 66 cases.

As to postoperative complications, transient respiratory distress syndrome was observed in seven patients, which resulted in death in four of them. In six cases of CSF fistula, four were transient, one associated with hydrocephalus underwent a ventriculoperitoneal shunt with complete closure of the CSF leakage, and, finally, one patient died due to meningitis. The authors observed one patient with

hypertensive cerebral hemorrhage who died. A case of brachial diplegia caused by incomplete laminectomy of C1 and compression of the spinal cord was observed and the patient underwent reoperation and presented total recovery after two months. The authors detected other complications such as hiccups in two cases — from these one lasted one day and the other two days — seven cases of hallucination lasted one to eight days, pneumonia in two patients, and, finally, superficial wound infections in three cases with complete recovery of all of them.

The postoperative mortality was 6.7% (13 patients). Seven patients had sudden respiratory distress and apnea, two had a hypertensive cerebral hemorrhage, one had a bilateral subdural hematoma with recurrence provoked by overdrainage of the ventriculoperitoneal valve, one had a massive gastrointestinal bleeding, and, finally, two cases with CSF fistula associated with severe meningitis.

Despite the lack of modern diagnostic tools and presence of intensive care unit just for few patients (34.8%), the positive clinical outcome of the majority of the cases can almost entirely justify the author's approach of this kind of neurosurgical disorder.

ACKNOWLEDGMENTS

This paper is dedicated to Clemente Augusto de Brito Pereira, MD, PhD, São Paulo.

References

1. Ackermann JF. Über die Kretinen, eine besondere Menschenabart in den Alpen. Gotha, in der Ettingerschen Buchhandlung, 1790.
2. Virchow R. Untersuchungen über die Entwicklung des Schädelgrundes in gesunden krankhaften Zustand und über den Einfluss Derselben auf Schädelform, Gesichtsbildung und Gehirnbau. Berlin, 1857.
3. Virchow R. Beiträge zur physischen Anthropologie der Deutschen mit besonderer Berücksichtigung der Friesen. Buchdruckerei der königlichen Akademie der Wissenschaften, G. Vogt, Berlin, 1876.
4. Chiari H. Über Veränderungen des Kleinhirns infolge von Hydrocephalie des Grosshirns. Dtsch med Wschr 1891;17:1172-1175.
5. Chiari H. Über Veränderungen des Kleinhirns, des Pons und der Medulla Oblongata in Folge von congenitaler Hydrocephalie des Grosshirns. Dtsch Akd Wissensch 1895;63:71-125.
6. Simon Th. Beiträge zur Pathologie und pathologischen Anatomie des Central-Nervensystem. Arch Psychiat Nervenkr 1875;5:108-163.
7. Finlayson AI. Syringomyelia and related conditions. In: Baker AB, Baker LH (Eds). Clinical Neurology, vol 3, Philadelphia 1981:1-17.
8. Canelas HM, Zaclis J, Tenuto RA. Contribuição ao estudo das malformações occipito-cervicais, particularmente da impressão basilar. Arq Neuropsiquiatr 1952;10:407-476.
9. Caetano de Barros M, Farias W, Ataíde L, et al. Basilar impression and Arnold-Chiari malformation. J Neurol Neurosurg Psychiatry 1968;31:595-605.
10. Carneiro GS Filho. Tratamento cirúrgico-circunferencial da invaginação basilar. Tese, Recife, 2001.
11. Arruda JAM, Costa CMC, de Tella Jr OI. Results of the treatment of syringomyelia associated with Chiari malformation: analysis of 60 cases. Arq Neuropsiquiatr 2004;62:237-244.
12. Botelho RV, Bittencourt LRA, Rotta JM, et al. The effects of posterior fossa decompressive surgery in adult patients with Chiari malformation and sleep apnea. J Neurosurg 2010;112:800-807.
13. Malis LI. The myeligraphic examination of the foramen magnum. Radiology 1958;70:196-221.
14. Marín-Padilla M. Cephalic axial skeletal-neural dysraphic disorders: embryology and pathology. Can J Neurol Sci 1991;18:153-169.
15. Raupp E, Lisboa L, Fontanari J. Fasciculações e impressão basilar. Estudo a propósito de um caso. Ver Brás Neurol 1986;22:99-101.
16. Gonçalves da Silva JA, dos Santos Jr. AA, Melo LRS, et al. Posterior fossa decompression with tonsillectomy in 104 cases of basilar impression, Chiari malformation and/or syringomyelia. Arq Neuropsiquiatr 2011;69:817-823.
17. Gardner WJ, Goodall RJ. The surgical treatment of Arnold–Chiari malformation in adults. An explanation of its mechanism and importance of encephalography in diagnosis. J Neurosurg 1950;3: 199-206.
18. Batzdorf U. Syringomyelia: current concepts in diagnosis and treatment. Baltimore: Williams & Wilkins, 1991.
19. Duddy MJ, Williams B. Hindbrain migration after decompression for hindbrain hernia: a quantitative assessment using MRI. Brit J Neurosurg 1991;5:141-152.
20. Williams B. Surgery for hindbrain related syringomyelia. In: Advances and technical standards in neurosurgery, vol 20, Berlin Springer Verag 1993:108-164.
21. Sahuquillo J, Rubio E, Poca MA, et al. Posterior fossa reconstruction: a surgical technique for the treatment of Chiari I malformation and Chiari I/syringomyelia complex-preliminary results and magnetic resonance imaging quantitative assessment of hindbrain migration. Neurosurgery 1994;35:874-885.
22. Nyland H, Krogness KG. Size of posterior fossa in Chiari type I malformation in adults. Acta Neurocirurgica 1978;40:233-242.
23. Heiss JD, Patronas N, DeVroom HL, et al. Elucidating the pathophysiology of syringomyelia. J Neurosurg 1999;91:553-562.
24. Milhorat TH, Chou MW, Trinidad EM, et al. Chiari I malformation redefined: clinical and radiographic findings for364 symptomatic patients. Neurosurgery 1999;44:1005-1017.
25. Romero FR, Pereira CAB. Suboccipital craniectomy with or without duraplasty. Wat is the best choice in patients with Chiari type I malformation. Arq Neuropsiquiatr 2010;68:623-626.
26. Taricco MA. Tratamento cirúrgico da siringomielia associada à malformação de Chiari tipo I. Tese, FMUSP, São Paulo, 1994.
27. Batzdorf U. Chiari I malformation with syringomyelia. Evaluation of surgical therapy by magnetic resonance imaging. J Neurosurg 1988;68:726-730.
28. Raftopoulos C, Sanchez A, Matos C, et al. Hydrosyringomyelia-Chiari I complex. Prospective evaluation of a modified foramen magnum decompression procedure: preliminary results. Surg Neurol 1993; 39;163-169.
29. Iskandar BJ, Hedlund GL, Grabb PA, et al. The resolution of syringohydromyelia without hindbrain herniation after posterior fossa decompression. J Neurosurg 1998;89:212-216.
30. Kyoshima K, Kuroyanagi T, Oya F, et al. Syringomyelia without hindbrain herniation: tight Cisterna magna. Report of four cases and a review of the literature. J Neurosurg 2002(Spine 2)96:239-249.