Primary Obstruction of the Fourth Ventricle Outlets: Neuroendoscopic Approach and Anatomic Description

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OBJECTIVE: Primary obstruction of the foramina of Magendie and Luschka is an uncommon and still unclear cause of noncommunicating hydrocephalus. The aim of this work is the description, for the first time, of the inner aspect of these velar obstructions of the fourth ventricle outlets and the demonstration of the efficacy of neuroendoscopic treatment.

METHODS: Of 240 hydrocephalic patients treated in our institution with endoscopic third ventriculostomy, a subgroup of 10 cases with closure of the fourth ventricular outlets without associated Chiari malformation and syringomyelia was selected. In all of these cases, a transaqueductal endoscopic navigation of the fourth ventricle was performed, and the obstructed outlets were inspected. All of the clinical data and, in particular, the videotape records of endoscopic operations, as well as the cine-magnetic resonance imaging scans, were reviewed to evaluate their patency status.

RESULTS: Various degrees of stenosis were found endoscopically: restriction of the Magendie contour with thick and opaque membrane, transparent spider web-like membrane, and dense membrane with fissures acting as valves. Endoscopic third ventriculostomy was effective in almost all patients, although we noticed an unforeseen high incidence of closure of the stoma. The restored normal cerebrospinal fluid flux after ventriculocisternostomy and magendieplasty was demonstrated by comparative study of cerebrospinal fluid flow measurements by cine-magnetic resonance imaging.

CONCLUSION: This report demonstrates the effectiveness of neuroendoscopic third ventriculostomy as well as magendiestomy in cases of tetraventricular hydrocephalus attributable to primary obstruction of the outlets of the fourth ventricle and, for the first time, presents direct images of various types of outlet obstructive pathology.

KEY WORDS: Cine-magnetic resonance imaging, Foramen of Luschka, Foramen of Magendie, Hydrocephalus, Magendieplasty, Third ventriculocisternostomy

Tetraventricular hydrocephalus attributable to blockage of the fourth ventricle outlets is usually caused by either inflammatory processes (8, 11) or congenital anomalies, such as Dandy-Walker syndrome (4, 25), Chiari malformation (7, 10, 21, 27, 28), and tuberous sclerosis (9). "Idiopathic" or "primary" membranous occlusion of the fourth ventricle outlets (the foramina of Magendie and Luschka) is a less frequent and still enigmatic entity, because the reason for the 3 outlets becoming simultaneously occluded is difficult to explain. It has been suggested that, in some cases, congenital stenosis of the outlets could result in complete occlusion with late decompensation. The rationale of this evolution, however, remains unclear (26). Cine-magnetic resonance imaging (cine-MRI) dynamic studies of cerebrospinal fluid (CSF) flow have not clarified the pathophysiology of the outlet occlusion (17), and the hypothesis of membranous valves trapping the CSF flow has never been demonstrated. Neuroendoscopic third ventriculostomy and even the opening of the occluded foramen of Magendie have been proposed in recent years as novel and efficacious treatments as an alternative to traditional CSF ventricular shunts (5, 17, 19).

ABBREVIATIONS: Cine-MRI, cine-magnetic resonance imaging; **CSF**, cerebrospinal fluid; **ETV**, endoscopic third ventriculostomy; **PICA**, posteroinferior cerebellar artery

We reviewed our series of obstructed outlets of the fourth ventricle that had been explored through neuroendoscopy. In addition to presenting results of the treatment, we also report, for the first time, a description of different degrees of velar obstruction and some remarks that suggest some pathogenetic hypotheses.

PATIENTS AND METHODS

A total of 280 neuroendoscopic procedures were performed on 240 hydrocephalic patients at the Neurosurgical Department of Treviso Hospital since 1994. Among the 35 patients with tetraventricular blocked hydrocephalus, in 10 patients (8 men, 2 women; mean age, 60.6 ± 11.6 years; age range, 36–73 years) we could not identify the most common causes of this condition, such as infections, hemorrhages, or congenital malformations, such as Dandy-Walker or Chiari syndrome. Seven of these 10 patients underwent cerebral magnetic resonance imaging and cine-MRI (Magnetom Avanto, 1.5 T; Siemens AG, Munich, Germany), both before and after the neuroendoscopic treatment, to identify neuroradiological signs of fourth ventricle outlet obstruction or improvement of the hydrocephalus through restoration of normal CSF flux.

In these 10 patients, we performed a transaqueductal neuroendoscopic navigation of the fourth ventricle to explore the patency of the ventricular outlets. A flexible Codman endoscope (Codman & Shurtleff, Raynham, MA) was used, through a frontal burr hole 1.5 cm lateral to the midline and 2 cm above the coronal suture. The technical procedure is described elsewhere (18).

We reviewed both the videotape records of neuroendoscopic operations and the cine-MRI of our patients to describe the patency status of the fourth ventricle outlets, and we tried to correlate it with the preoperative clinical data.

The mean follow-up time after the treatment was 47.7 ± 48.7 months (range, 2 months to 12 years). Two patients were lost to follow-up.

RESULTS

Clinical Data

Eight patients came to our attention after a mean period of 10.8 ± 8.5 months (range, 2–24 months) from the onset of normal-pressure hydrocephalus–like symptoms. In 2 patients, the presentation was subacute (10 days), with symptoms of intracranial hypertension (Table 1). One patient (Patient 3) complained of gait difficulties, ideomotor slowdown, and incontinence (Hakim triad), suggesting the diagnosis of normal-pressure hydrocephalus.

Preoperative Neuroimaging

Computed tomographic scanning and magnetic resonance imaging, performed in 7 patients, revealed an obstructive tetraventricular hydrocephalus, with downward displacement of the third ventricle floor (4 patients), obliteration of the retrocerebellar CSF space (6 patients), and slight displacement of the cerebellar tonsils without herniation (2 patients). In 4 patients, the interpeduncular and prepontine cisterns were significantly compressed and reduced in volume by the abnormally dilated fourth ventricle (Fig. 1). Cine-MRI was performed in 8 patients:

Patient no.	Age (y)/sex	Symptoms	Duration of symptoms	Procedure	Follow-up	Clinical outcome
		TABLE 1. Clinical features of patients[a]				
1	53/M	Ideomotor slowdown, gait difficulties, depression	5 mo	ETV	12 y	Improved; worsened after 12 y; new III versus improved
2	49/F	Dizziness, gait difficulties, memory impairment	2 y	ETV	7 y	Improved
3	70/M	Gait difficulties, ideomotor slowdown, incontinence	2 mo	ETV	6 y	Improved; worsened after 3 y; 2007 VP shunt
4	73/M	Gait difficulties, ideomotor slowdown, memory impairment	6 mo	ETV, aqueductoplasty	6 y	Improved; after 4 y, ischemic stroke leading to vegetative status
5	64/M	Gait difficulties, incontinence	10 d	ETV	Lost to F/U	
6	36/M	Dizziness, visual impairment, headache, vomiting	10 d	ETV	2 mo; lost to F/U	Improved
7	65/F	Cervical pain, headache, gait difficulties, dizziness, nausea, vomiting	2 y	Magendieplasty	34 mo	Improved
8	69/M	Gait difficulties, memory impairment	6 mo	ETV	15 mo	Improved
9	69/M	Gait difficulties, incontinence, memory impairment	1 y	ETV	5 mo	Improved; worsened after 2 mo; new III versus improved
10	58/M	Gait difficulties	8 mo	ETV	2 mo	Improved

[a] ETV, endoscopic third ventriculocisternostomy; VP, ventriculoperitoneal; F/U, follow-up; III, third ventriculostomy.

FIGURE 1. Preoperative magnetic resonance imaging (MRI) scans showing a huge dilation of the fourth ventricle. A, Patient 7. T1-weighted sagittal MRI scan, showing downward displacement of the third ventricle floor, obliteration of the retrocerebellar cerebrospinal fluid (CSF) space, and reduc-tion of the interpeduncular and prepontine cisterns. B, Patient 8. T2-weighted sagittal MRI scan showing that the CSF flux is clear through the cerebral aqueduct. C, Patient 2. T1-weighted sagittal MRI scan showing that the funnel-shaped aqueduct is wide open.

in 3 patients (37.5%), cine-MRI showed a complete absence of CSF flux at the obex level; in 2 patients (25%), the CSF flux was uncertain; and in 3 patients (37.5%), the flux at the obex appeared normal. Closure of the fourth ventricle outlets was suggested by the cine-MRI study in 62.5% of cases. In all cases the aqueduct appeared wide, with a good CSF flux at cine-MRI and a distorted shape resembling an upside-down funnel.

In 1 patient (Patient 4), an aqueductal stenosis was diagnosed. Since the anatomic structure of the aqueduct was favorable, an aqueductoplasty was performed. The subsequent exploration of the fourth ventricle revealed a membranous obstruction of the outlets. Therefore, in this case with double obstruction, an endo-scopic third ventriculostomy (ETV) was performed.

Neuroendoscopic Findings

We reviewed the video-recorded transaqueductal neuroen-doscopic navigation of the fourth ventricle of 9 of the 10 patients with idiopathic tetraventricular hydrocephalus, identifying dif-ferent degrees of both stenosis of Magendie's foramen and velar obstruction. The recorded video of Patient 6 was missing. Among these patients, in 5 cases, both Luschka's foramina were verified, whereas, in 2 patients, only the left Luschka's foramen could be observed; in 2 patients, the lateral outlets were not explored, mainly owing to the overabundant plexi entirely fill-ing the lateral recesses. Usually, the left foramen of Luschka was easier to explore, compared with the right one, because the trajectory of the scope, which is introduced through a right frontal burr hole, is directed from the right to the left.

The sizes of velar obstructed Magendie's foramina were found to be small in all cases, whereas the lateral foramina showed a significant enlargement, proportional to the fourth ventricle dilation (Fig. 2). We tried to define the size of the Magendie's foramen, comparing it with the hypoglossal trig-ona and the diameter of the posteroinferior cerebellar arteries (PICAs). Although this method offers only a relative measure, we based our results also on our previous in vivo observations

of the fourth ventricle structures (20). In 6 cases, the foramen was extremely small, and in 1 of these cases (Patient 5), the minute foramen was kept open only by a loop of the left PICA, which surrounded a slight fissure through the membrane.

The entity of the foramen velar obstruction was quite vari-able (Fig. 3). Our findings are summarized in Table 2.

In 3 patients (Patients 5, 8, and 9), we found evidence of a pos-sible inflammation of the fourth ventricle plexi, which were coated
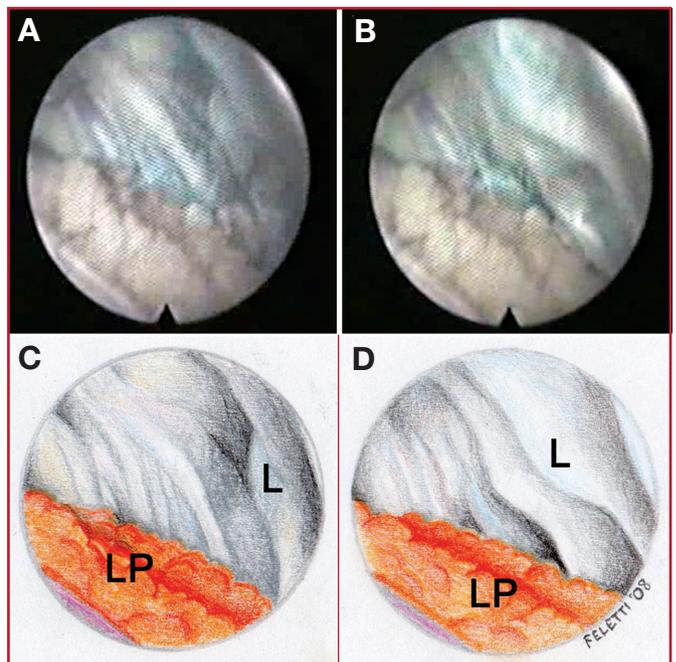
FIGURE 2. Enlarged left lateral recess. A and B, endoscopic appearance; C and D, corresponding drawings. The foramen of Luschka (L) is occluded by a thick membrane, which is pushed outward (A and C) and inward (B and D) by the CSF flux. LP, left plexus.
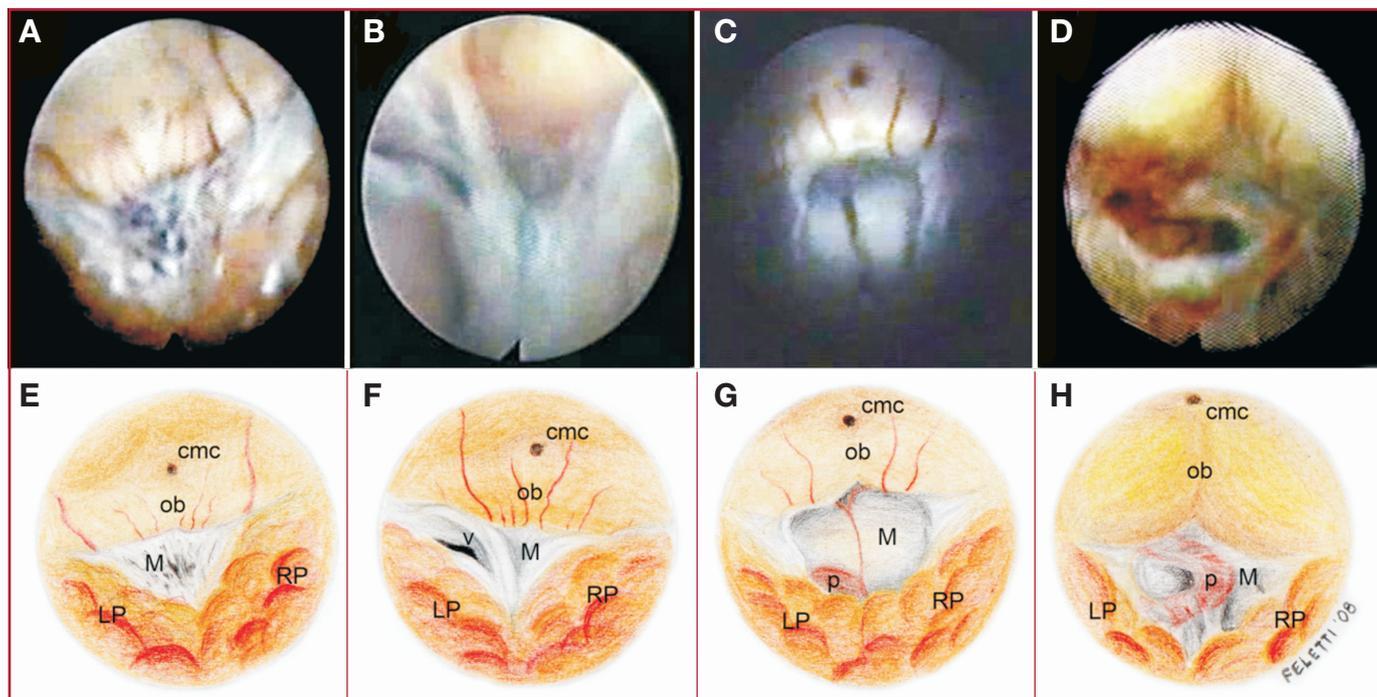
FIGURE 3. *Different types of membranous occlusion of the foramen of Magendie. A–D, endoscopic appearance; E–H, corresponding drawings. A, thick and opaque membrane; it can be more or less uniform, but it is usually discontinuous. B, thick membrane with a fissure, acting with a valve mechanism. C, thin and transparent membrane, with spider web–like architecture; the posteroinferior cerebellar arteries (PICAs) are well visible behind the* *obstruction. D, the membrane is thick and discontinuous, and a loop of the left PICA contributes to the blockage of the foramen and creates a small fissure through the membrane. LP, left plexus; RP, right plexus; M, foramen of Magendie; ob, obex; cmc, centromedullary canal; v, valve-like mechanism of the membrane; p, PICA.*

TABLE 2. Endoscopic features[a]

Patient no.	Size of foramen of Magendie	Membrane	View of foramen of Luschka	Lateral ventricle plexi	Fourth ventricle plexi	Difficult ETV
1	Small	Thick, opaque, with dense, fibrous branches	Left	Normal	Normal	No
2	Very small	Dense, discontinuous, with fissures and laciniae, pulsating	Both	Normal	Normal	No
3	Small	Thick, opaque, with dense fibrous branches	None	Normal	Normal	No
4	Very small	Thick, opaque, with dense, fibrous branches	Left	Normal	Normal	No
5	Very small	Thick, discontinuous owing to a PICA's loop, not pulsating	None	Normal	Arachnoiditis	Yes
6[b]	?	?	?	?	?	?
7	Small	Spider web–like, bulging down, pulsating	Both	Normal	Normal	Magendieplasty
8	Very small	Thick, opaque, with dense, fibrous branches	Both	Normal	Slight inflammation	No
9	Very small	Thick, opaque, uniform, with dense, fibrous branches	Both	Normal	Arachnoiditis	No
10	Very small	Thick, opaque, with dense, fibrous branches	Both	Normal	Normal	No

[a] ETV, endoscopic third ventriculostomy; PICA, posteroinferior cerebellar artery.
[b] The video recording of the endoscopic procedure performed on this patient was missing.

by overabundant arachnoid without any clinical sign or related disease. This peculiar feature was particularly marked in Patient 9, in whom the plexi were almost opaque and had a marbled appearance owing to the fourth ventricular arachnoiditis (Fig. 4).

Neuroendoscopic Treatment and Outcome

All patients underwent a neuroendoscopic procedure. In 9 patients, an ETV was performed. Only in 1 case (Patient 5) did the ETV procedure present some difficulties, owing to the
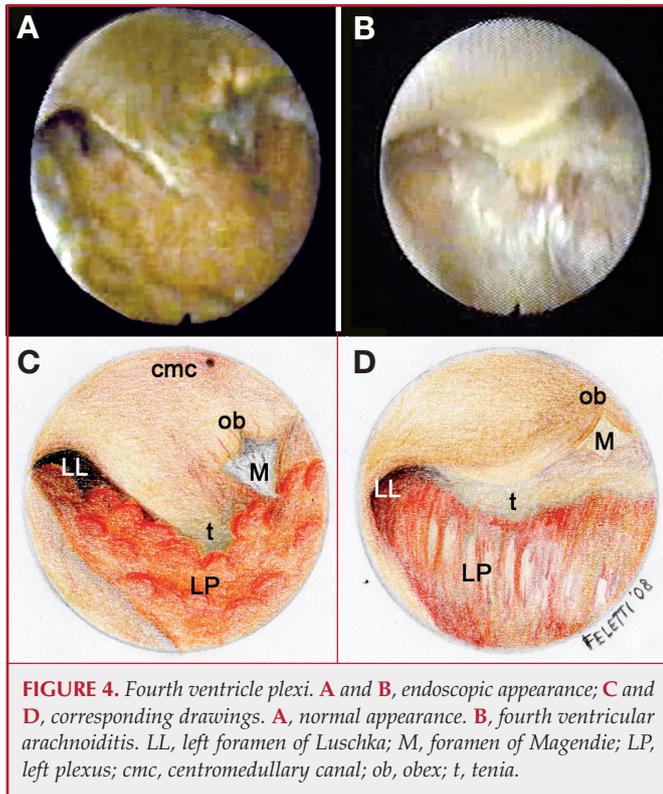
FIGURE 4. *Fourth ventricle plexi.* **A** *and* **B**, *endoscopic appearance;* **C** *and* **D**, *corresponding drawings.* **A**, *normal appearance.* **B**, *fourth ventricular arachnoiditis. LL, left foramen of Luschka; M, foramen of Magendie; LP, left plexus; cmc, centromedullary canal; ob, obex; t, tenia.*

reduction of the interpeduncular prepontine cistern. All patients improved rapidly during the first weeks after the operation, without any infective complications or neurological deficits. Long-term outcomes were good, with maintenance of full independence in the activities of daily living for 7 patients. Two patients complained of a clinical worsening (Patient 1 after 12 years and Patient 9 after 2 months). In both cases, there was closure of the stoma requiring a new successful and lasting ETV (mean follow-up, 4.5 ± 2.1 months). Patient 3, who had suspected normal-pressure hydrocephalus, worsened after 3 years and required a ventriculoperitoneal shunt, which resolved his symptoms.

In 1 case (Patient 7), we opted for a magendieplasty (i.e., the endoscopic opening of the foramen of Magendie). The patient improved rapidly, with complete headache relief. All neurological symptoms resolved in less than 8 weeks.

CSF flow through the ventricular and cisternal system was studied before and after the neuroendoscopic procedure with the use of cine-MRI: the comparison of flux measurements confirmed that CSF flux was restored after the ventriculocisternostomy and the opening of the foramen of Magendie.

DISCUSSION

In 1877, Hilton (13) first described at autopsy a case of hydrocephalus secondary to primitive outlet occlusion of the fourth ventricle with clear signs of intracranial hypertension. Sub-sequently, other authors reported single cases of primary fourth ventricular midline outlet obstruction. We now add 10 new cases of primary membranous obstruction of the fourth ventricle outlets in patients older than 15 years to the 22 cases previously reported in the literature (Table 3).

Etiology and Pathogenesis

After the first description by Magendie, anatomists were divided for a century on this issue, often ascribing the autopsy report of the foramen of Magendie to an artifact resulting from clumsy manipulations of a membrane between the velum medullaris inferior and the obex. Only the founding of neurosurgery by Walter Dandy at the beginning of the 20th century brought the debate to an end, as it was demonstrated *in corpore vivo* that the outlets of the fourth ventricle do, indeed, exist.

However, the variability and even the possible absence of the fourth ventricle outlets are well known. In an autopsy study, Barr (3) found that, in 3% of cases, the area of the foramen of Magendie was only 1 mm², and, in 1 specimen, the foramen was totally absent. Two percent to 3% of brains had a membrane, sometimes partially fenestrated, at the level of Magendie's foramen, although the cerebellar vermis was normal. As a result, Magendie's foramen was partially or totally obstructed in up to 6% of cases. Conversely, Luschka's foramina were consistently present in all specimens (3).

Even taking into account such anatomic variety, the sequence of pathological events leading to the primary occlusion of the fourth ventricle foramina is difficult to explain. A pathophysiological explanation based on the anatomic variations of Magendie's foramen and the development of semipermeable membranes obstructing the foramen has been suggested (26). The subcritical rates of CSF flux would maintain a clinical tolerance until either a variation in brain compliance with age or a decrease of the membrane's permeability could tilt the balance and cause of an upstream ventricular dilation. Such progressive fibrosis could also gradually impair the permeability, but there is no clear explanation for the acute onset of symptoms in some patients (26).

Another possible etiopathogenetic hypothesis stems from an observation made by Carpentier et al. (5), who reported that, after a third ventriculostomy for a tetraventricular hydrocephalus, flow rate studies showed the appearance of spontaneous, although minimal, physiological flow through the foramina of Magendie and Luschka, which had not been detected in preoperative studies. Hence, they concluded that a temporary adherence of Luschka's membrane to the dura led to a functional obstruction of the fourth ventricle outlets. This mechanism of functional blockage may be quite similar to the outlet occlusion that is observed in the Chiari malformation: displacement of the cerebellar tonsils leads the outlets to face the dura, diminishing the physiological CSF flux and causing dilation of the fourth ventricle. As a consequence of high intraventricular CSF pressure, the contact between the foramina of Magendie and Luschka, respectively, with the suboccipital and petrosal dura gives rise to a definitive arachnoidal membranous organization, with disappearance of the CSF flux. An ETV is then effec-

TABLE 3. Review of the literature on primary membranous obstruction of the fourth ventricle outlets in the adult (age, >15 years)[a]

Series (ref. no.)	No. of patients	Age (y)/sex	Symptoms and signs	Treatment	Out-come	Follow-up
Hilton, 1877 (13)	1	34/M	Headache, gait disturbance, vomiting	None Ò(autopsy)	Dead	
Coleman and Troland, 1948 (6)	1	17/M	Headache	Suboccipital craniectomy		
Holland and Graham, 1958 (14)	1	31/F	Blurred vision, headache, nausea, weakness of right leg	Suboccipital craniectomy	Dead	
Amacher and Page, 1971 (2)	1	21/F	Headache, vomiting, nausea	Suboccipital craniectomy + VC shunt	Good	1 y
Yoshioka et al., 1985 (29)	1 2	34 68	Blurred vision, occipital pain Gait disturbance, urine incontinence, slurred speech	Suboccipital craniectomy + VP shunt Suboccipital craniectomy + VA shunt	Good Stable	1 y
Rifkinson-Mann et al., 1987 (26)	1 2	42/M 52/M	Hemiparesis, right homonymous hemianopsia, headache, vomiting Headache, vomiting, blurred vision	Suboccipital craniectomy Suboccipital craniectomy	Good Good	1 y 1 y
Aesch et al., 1991 (1)	1	35/M	Ataxia, headache, nausea	VP shunt	Good	2 mo
Osaka et al., 1995 (24)	1	20/F	Headache, nausea, papilledema	Suboccipital craniectomy	Good	
Hashish et al., 1999 (12)	1 2	35/F 68/F	Headache, nausea, gait disturbance Headache, gait disturbance, memory impairment, dismetria, pyramidalism	Suboccipital craniectomy + VC shunt Suboccipital craniectomy + VC shunt	Good Good	7 y 5 y
Huang et al., 2001 (15)	1	15/F	Headache, nausea, vomiting, amenorrhea	Suboccipital craniectomy	Good	14 mo
Carpentier et al., 2001 (5)	1	58/F	Visual impairment with maximum head rotation, dizziness, headache, nausea, vomiting, gait disturbance	ETV	Good	3 y
Karachi et al., 2003 (17)	1 2 3	21/F 53/F 68/M	Headache, vomiting, papilledema Vertigo, nausea Gait disturbance, sphincteric disorders, impairment of higher functions	ETV ETV ETV	Good Good Good	26 mo 24 mo 58 mo
Mohanty et al., 2008 (23)	5			ETV	Good	4.2 y (mean)

[a] VC, ventriculocisternal; VP, ventriculoperitoneal; VA, ventriculoatrial; ETV, endoscopic third ventriculocisternostomy.

tive to keep the intraventricular pressure equal to the subarachnoid space pressure. Consequently, this pathophysiological phenomenon can explain acute and reversible symptoms and the spontaneous functional reopening of the foramina after ETV (5).

In our series, we observed, in 3 cases, the presence of arachnoid tissue over the choroidal plexi of the fourth ventricle, which were the plexi of the normal lateral ventricles (Fig. 4). This feature can suggest a subclinical inflammation of the plexi of the fourth ventricle, leading to an overabundant arachnoidi-

tis and involving the outlets of the ventricle, forming a membrane. In this context, the foramina may be prone to occlusion.

Diagnosis

Although tetraventricular hydrocephalus is easily diagnosed with neuroimaging, the presence of a membranous obstacle at the level of the foramen of Magendie can be difficult to detect by magnetic resonance imaging and cine-MRI. The most reliable explanation of the low predictive value of cine-MRI in cases of

fourth ventricular obstruction is that clarified by Karachi et al. (17). Whereas the thin diameter of the cerebral aqueduct allows a clear identification of CSF flow in the rostrocaudal axis, the flow measurements in the fourth ventricle are less reliable, because it is a larger structure, it has 3 outlets, and the foramina of Luschka are often dilated and intussuscepted into the basal cisterns; therefore, the flux is more turbulent than through the aqueduct. Moreover, the dilated fourth ventricle compresses the cisterna magna, making the measurement more difficult.

In our series, we were able to detect the obstructive nature of the hydrocephalus in 3 of 8 cases studied with cine-MRI, while, in another 2 cases, the flux at the obex was uncertain. This means that cine-MRI has a sensitivity of 62.5% in our study.

This is one of the reasons motivating the exploration of the fourth ventricle whenever it is safely possible, i.e., when the cerebral aqueduct is widely patent. This can be considered the only way to conclusively diagnose a membranous obstruction of the outlets of the fourth ventricle while minimizing invasiveness but ensuring absolute sensitivity, as demonstrated in previous experience (18–20). Although the well-known morbidity of aqueductoplasty has often been described in the literature, complications of fibroscopic navigation through a nonstenotic aqueduct, in contrast to what common sense might suggest, do not cause neurological deficits (24), as confirmed by our experience.

Membrane Morphology

We observed different types of velar blockage of the foramen of Magendie, and we tried to group them into categories, according to the consistency of the membrane, the grade of tightness of its net, and its aptitude to float with the CSF flux (Fig. 3). When the foramina of Luschka were explored (12 of 18 foramina), they appeared to be closed by a membrane similar to that blocking the foramen of Magendie (Fig. 2). In all cases, the lateral recesses were broadly engorged, proportionally to the dilation of the fourth ventricle, as reported in similar cases (16, 17).

As the structure of the membranes is so uneven, it can likely explain the varying severity of symptoms experienced by the patients. Two patients (Patients 5 and 6) presented with an acute onset of symptoms. Unfortunately, the video recording of the endoscopic procedure performed on Patient 6 was missing. However, we noticed that Patient 5 presented with a wide-mesh net-like membrane covering the foramen of Magendie, suggesting a possible correlation between the length of symptoms, the structure of the membranes, and the completeness of Magendie's obstruction. We hypothesize that patients with a congenital, subtotal membranous obstruction of Magendie's foramen have a greater tolerance of outflow reduction and usually present with chronic symptoms. On the contrary, patients with less obstructing membranes are less accustomed to endure CSF flux decreases, and either the slightest increase of CSF production or a small enhancement of the membrane fibrosis can lead to acute decompensation.

Treatment

Excluding the case reported by Hilton (13) in 1877, the patients before 2001 were treated with suboccipital craniec-

tomy and surgical excision of the membranes, either alone (6, 14, 15, 24, 26) or associated with ventriculocisternal shunts (2, 12) or ventriculoperitoneal or ventriculoatrial shunts (29). Authors have generally reported good outcomes. Aesch et al. (1), in 1991, reported the case of a patient treated exclusively with a ventriculoperitoneal shunt with good results.

Mohanty et al. (22) reported on 3 patients with acquired fourth ventricle outlet obstruction who underwent ETV with good postoperative results. Mohanty et al. (23) recently reported a series of 22 cases of fourth ventricle outlet obstruction from various etiologies treated with ETV; in 5 of the patients, no underlying etiology for the obstruction was detected. However, the first case of primary obstructed outlets successfully treated by ETV was that described by Carpentier et al. (5) in 2001. Karachi et al. (17), in 2003, added 3 more cases of primary obstruction of the fourth ventricular outlets that were successfully treated with ETV.

Our experience confirms that neuroendoscopy is a safe and effective procedure to restore the CSF flux in patients with membranous obstruction of the fourth ventricle and that it should be considered the treatment of choice in such cases. Moreover, as the cerebral aqueduct is widely patent, a direct inspection of the fourth ventricle is feasible, leading to the direct detection of membranous obstruction.

Even in cases of aqueductal stenosis, a membranous blockage of the foramen of Magendie may develop, as in our Patient 4. A possible explanation could be that the absence of a valid CSF flux favored the occlusion of a stenotic foramen of Magendie, although nothing could be said about Luschka's foramina. This incidental observation, although limited to a single case, could be an argument against the performance of aqueductoplasty alone.

We performed ETV in 9 cases, with an immediate success rate of 100%. The procedure proved to be quite difficult in only 1 case, because of the reduction of the interpeduncular prepontine cistern owing to fourth ventricle dilation.

In 3 cases, the third ventriculostomy belatedly failed (after 2 months, 3 years, and 12 years). We decided to try a second ETV in 2 of these cases, whereas we opted for a ventriculoperitoneal shunt in the third case, to comply with the choice of the patient and his family. Therefore, the failure rate of ETV in these patients is significantly higher than in other groups of patients, but in agreement with that reported by others (23).

In 1 case, we endoscopically opened the membranous obstruction of the foramen of Magendie (magendieplasty), with complete restoration of CSF flux and symptom resolution. The technique has been described elsewhere (18). This procedure is indicated when the anterior cisterns are compressed and there are indications that ETV would be more difficult or prone to reocclusion. It implies the presence of a large and translucent spider web–like membrane covering the foramen of Magendie with the PICAs clearly visible.

CONCLUSION

Primary membranous obstruction of Magendie's and Luschka's foramina is a rare and unclear cause of tetraventricular

hydrocephalus. Using direct endoscopic observation via transaqueductal navigation of the fourth ventricle, we verified that the area of the foramen of Magendie is almost always undersized, and we demonstrated that these membranes are often discontinuous and their structure can be very variable. Conversely, the lateral recesses are inclined to enlarge.

These membranes become functionally obstructive, either because there is a change in their permeability or because of their anatomic alignment up against the dura when intraventricular hypertension cannot be compensated by flow adaptation through the foramina. In some cases, the appearance of a previously unnoticed chronic inflammation of the fourth ventricle choroidal plexi may explain the development of the velar barrier.

ETV provides an effective treatment, as it restores pressure equilibrium between the extra- and intraventricular spaces, although a high rate of failure is predictable in these cases; a second ETV is often effective, however. Magendieplasty is indicated only in very selected cases.

Disclosure

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COMMENTS

Longatti et al. have presented a good clinical study on a rare occurrence such as the primary obstruction of the outlets of the fourth ventricle causing tetraventricular hydrocephalus. Such a pathological entity accounts for how tetraventricular hydrocephalus, too often attributed to a primary or acquired deficit of the cerebrospinal fluid desorption, might be the result of an obstruction of the fourth ventricle outflow. In such cases, the endoscopic third ventriculostomy (ETV) could be a valid treatment of the hydrocephalus, avoiding the implant of a ventriculoperitoneal shunt. Furthermore, the authors attempted to provide some tips on how to preoperatively anticipate the possibility of such occurrence in a subset of tetrahydrocephalic patients, which might be quite useful in foreseeing which patients would gain benefit of the ETV alone or associated with the magendieplasty.

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This report of primary obstruction of the outflow of the fourth ventricle is a relatively large series of a very uncommon pathological entity. One would predict that ETV would be successful and, as the authors results demonstrate, it was. The relatively high rate of late occlusion may be caused by the same pathological process that

caused the original obstruction, such as an inflammation around the basal cisterns. The diagnosis of this pathological entity may be more difficult than is presented here because cine phase contrast magnetic resonance imaging may not be so sensitive to this finding. Other, newer high resolution T2-weighted imaging sequences, such as fast imaging employing steady-state acquisition, may be more sensitive. This is report is most useful in reminding the clinician that fourth ventricular outflow can occur and not to assume that panventricular hydrocephalus is communicating. Shunting such a patient would be unnecessary.

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The authors have presented a comprehensive study of patients presenting with tetraventricular hydrocephalus on the basis of fourth ventricular outlet obstruction. This condition is rare and to find 10 patients at any single institution is impressive. Then, to have all 10 patients treated by endoscopic means and to document their treatment with video recordings is unique. I commend the authors on their attention to detail, their excellent review of the literature, and their demonstration of the efficacy of ETV for this condition. This series underscores several important endoscopic principles: 1) ETV is a safe and effective treatment for all forms of noncommunicating hydrocephalus where the obstruction is distal to the anterior third ventricle; 2) exploration of the fourth ventricle should be discouraged as it does not contribute to the outcome and may be associated with serious complications, thankfully not seen in this series; and 3) rigid endoscopes provide a far superior view than flexible ones, and it may be this restriction that accounts for the failure to treat these patients with opening of the fourth ventricle outlet foramina. It would certainly seem more physiologically appropriate to restore normal anatomy as the first line of surgical management. However, the images provided by the authors clearly demonstrate the inability to define anatomical structures. I would contend that, given their obvious endoscopic experience, there would have been a higher number of patients treated with foraminoplasty if rigid endoscopy was used to better define the anatomy. Of course, the downside to such a procedure would be the need, in some cases, to make another burr hole for the ETV if the rigid endoscope showed inappropriate anatomy for a foraminoplasty. Although my personal treatment paradigm for these patients was always ETV, this series confirms that exploration of the fourth ventricle, at least with a flexible scope, is probably a waste of time.

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