Endoscopic Treatment of Hydrocephalus due to Aneurysm of the Vein of Galen – Case Report and Literature Review

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Key words
- hydrocephalus
- vein of Galen aneurysm
- neuroendoscopy
- thrombosis

Abstract

Aneurysms of the vein of Galen are uncommon vascular malformations. They are most frequently seen in infants and children, leading to heart failure and hydrocephalus. Exceptionally, they are detected in adults. Several theories have been proposed to explain hydrocephalus in these patients: obstruction of the cerebral aqueduct, impaired absorption of CSF after subarachnoid hemorrhage, passive ex-vacuo mechanism, or thrombosis of an aneurysm. Hydrocephalus has been treated mainly with cerebrospinal shunt procedures, but also direct surgery, radiosurgery and embolisation of the malformation have proved to be effective. We report the case of a partially thrombosed ectasia of the vein of Galen in a 44-year-old male, with huge hydrocephalus successfully treated with an endoscopic third ventriculostomy.

Introduction

Vein of Galen aneurysmal malformations (VGAMs) are rare intracranial congenital venous ectasia due to high blood flow associated with obstruction of a dural sinus distal to the malformation [1].

According to their angioarchitecture, two different subtypes of vein of Galen aneurysmal malformations can be distinguished. In the choroidal type, the venous pouch is fed by a complex vascular network formed by multiple choroidal arteries. This type usually causes heart failure in newborns. The mural type shows a direct arteriovenous fistula, without an interposed vascular nidus [2]. It typically occurs in infancy with macrocephaly and failure to thrive and can be associated with cardiac failure or cardiomegaly.

Although quite unusually, vein of Galen ectasias can sometimes thrombose, either spontaneously or exceptionally even after angiographic examination, owing probably to the slow blood flow through the malformation.

Symptoms depend on the entity of both blood shunt and aneurysm volume; haemodynamic impairment with consequent heart failure is more characteristic in newborns, whereas isolated hydrocephalus without cardiovascular symptoms usually occurs later.

We report the case of a patient with a partially thrombosed and calcified aneurysmal malformation of the vein of Galen who presented with symptomatic hydrocephalus at the age of 36 years and was treated with a neuroendoscopic third ventriculostomy. Although Lasjaunias and colleagues did recently suggest that this procedure offers an acceptable alternative to internal ventricular drainage after embolisation in children [3], to the best of our knowledge this is the first successfully reported case.

Case Report

A 44-year-old man was admitted to our department because of progressive paraparesis, which had lasted for about 8 years and had been worsening during the last 12 months. The patient had no history of mental retardation, but had already developed macrocephaly during childhood. He had achieved a good educational level, taking a degree in law, and had found a good position as an executive in a public office. His past medical history was unremarkable, and there was no family history of neurological disease. Eight years previously, progressive weakness of the right leg had brought the patient to neurological and neurosurgical attention. Computed tomography (CT) scan, magnetic resonance imaging (MRI) and magnetic resonance angiography revealed the presence of a VGAM, partially thrombosed with

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internal calcifications, causing three-ventricular hydrocephalus by occlusion of the aqueduct (**Figs. 1, 2**). Evan's index of ventricular dilatation at that time was about 0.79.

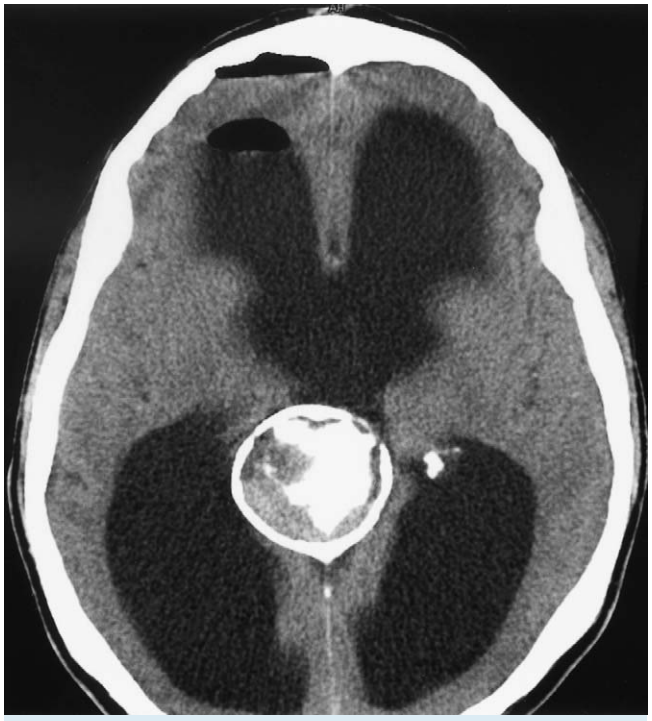

Fig. 1 Postoperative CT scan shows the malformation being partially thrombosed and calcified.

During the next 8 years the motor impairment worsened, also involving the left leg and leading to a paraparetic condition. A recent CT scan and MR imaging demonstrated a huge triventricular blocked hydrocephalus, with Evan's index averaging at the value of 0.82.

At admission, physical examination revealed macrocephaly (cranial circumference 66 cm), paraparetic gait, muscular hypertonia of the inferior limbs, polykinetic patellar and Achilles reflexes, Babinski sign bilaterally, clonus of the inferior limbs bilaterally, partial hypostenia (4/5) of the inferior limbs. The neuropsychological tests revealed a Mini-Mental State Examination (MMSE) of 24/30; Grooved Pegboard Test 133 seconds (dominant hand) and 182 seconds (non-dominant hand) [4]; Stroop test: colour naming 142 seconds, interference test 325 seconds [5], Rey Auditory Verbal Learning Test (RAVLT): 22 (trial 1–5), 7 (trial 7), 5 (trial 8) [6].

A neuroendoscopic third ventriculostomy was performed, using a flexible endoscope (Codman®, MA, USA). The posterior part of the third ventricle was inspected and showed the bulging mass of the malformation; none of the anatomic structures pertaining to the aqueduct were noticed. The anterior portion of the dilated third ventricle was translucent but the creation of the stoma proved to be of some difficulty due to the exiguous cisternal space left between the brainstem and clivus. In the end, however, a vigorous cerebrospinal flux through the stoma was obtained. The post-operative course was unremarkable, with quick mobilisation and initial improvement of inferior limb motility.

A clinical evaluation performed 4 months after the procedure confirmed the improvement of both walking and neuropsycho-

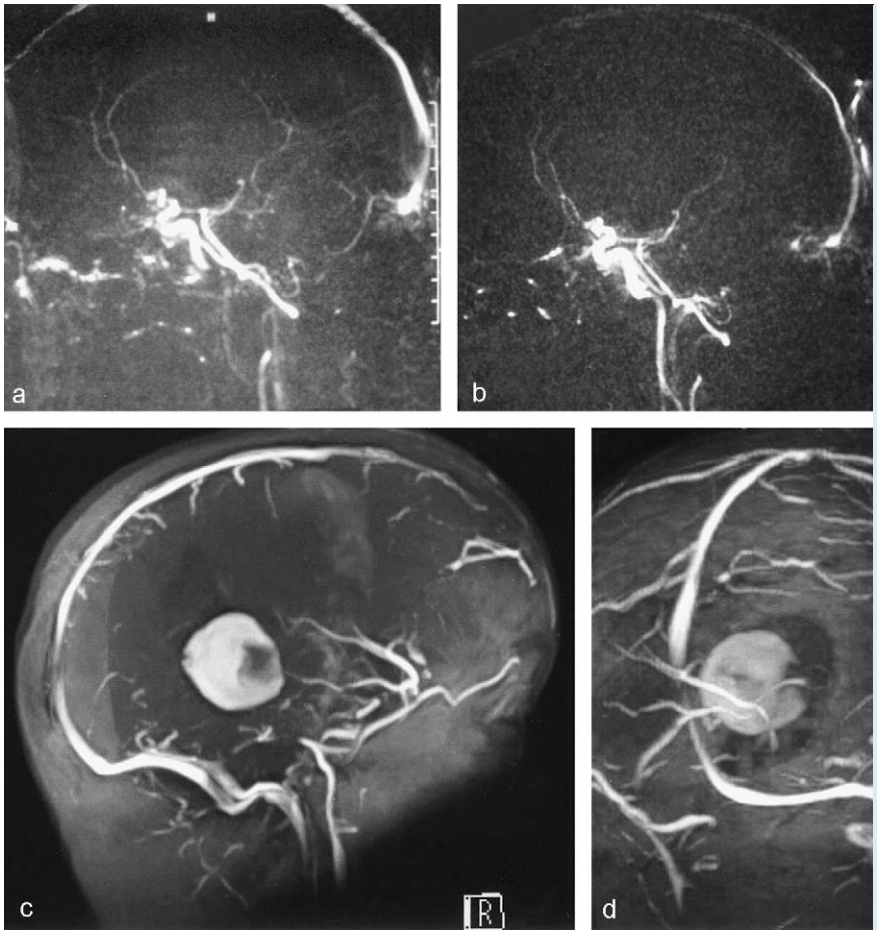

Fig. 2 **a** and **b**: Phase-contrast MRI angiography, velocity encoding (VENC) 10–60; neither incoming blood flux towards the aneurysm nor flux inside the malformation are detectable. **c** and **d**: MRI venography, time-of-flight (TOFF) 2D; venous drainage from the aneurysm and straight sinus are barely visible. These findings confirm the thrombosis of the malformation.

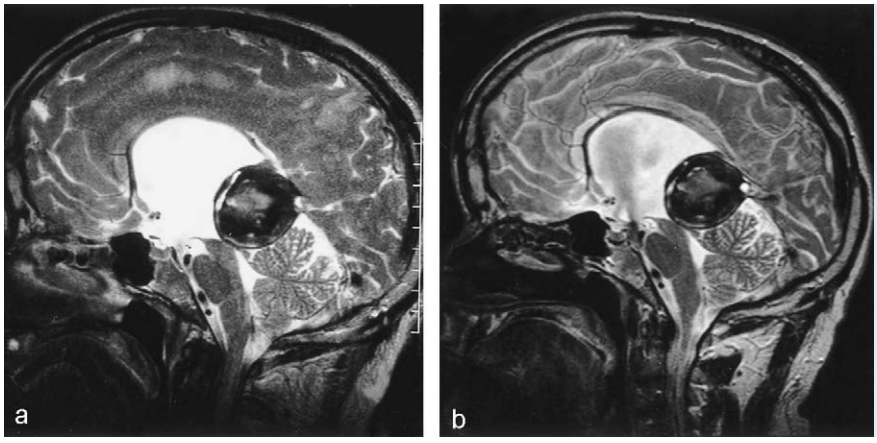

Fig. 3 **a** Preoperative T_2-weighted sagittal MRI, showing the mass effect exerted by the ectasia of the vein of Galen. The vascular malformation occludes *aditus aquaeducti* pushing down the *colliculi* resulting in blocked hydrocephalus. **b** Postoperative T_2-weighted sagittal MRI confirms the good CSF flow through the stoma on the floor of the third ventricle without a significant variation of the ventricular diameters.

logical tests, with an MMSE of 29/30, Grooved Pegboard Test 108 seconds (dominant hand) and 191 seconds (non-dominant hand), Stroop test 63 seconds (colour naming), 101 seconds (interference test), Rey Auditory Verbal Learning Test: 22 (trial 1–5), 9 (trial 7), 8 (trial 8). A new MRI demonstrated a good flow through the stoma of the third ventricle, with an Evan's index of 0.78 (**Fig. 3**).

Discussion

The vein of Galen is a short midline venous structure resulting from the confluence of the two internal cerebral veins and the basal vein of Rosenthal. It represents the caudal relic of the median prosencephalic vein, a centrally located vessel that drains the choroid plexus. Malformations of the Galenic vein are believed to result from a dysembryogenic event involving the cerebral vasculature between 6 and 11 weeks of gestation [7].

VGAMs can have different manifestations, in relation to the entity of blood shunt that they determine, or to their mass effect. The shunt from cerebral arteries into a dilated vein of Galen can lead to an increased cardiac output and finally to heart failure. This scenario is more frequent in newborns. In infants and toddlers, obstruction of the aqueduct of Sylvius with consequent hydrocephalus is prevalent.

Although neonates with a malformation of the vein of Galen leading to congestive heart failure still have high mortality rates (40–50%), the prognosis for children harbouring these lesions has significantly improved during the last 15 years. Due to advances in neuroradiological interventional techniques, it is now possible to obliterate the malformation with lower morbidity and mortality than for surgical treatment [8–11]. Valavanis and Yasargil have been pioneers in the study and treatment of these vascular malformations, from the neuroradiological and the surgical sides, respectively. In particular, Yasargil provided one of the most widely used classification systems for vein of Galen malformations according to the morphology and arterial supply pattern [12–15].

Differently from the paediatric age group, VGAMs are very unusual in adults, probably because such malformations are frequently symptomatic and consequently detected within infancy.

Furthermore, VGAMs may spontaneously thrombose and calcify, although this is a rare event. A round calcified rim in the pineal region has been considered pathognomonic for an aneurysm of the vein of Galen [16,17]. This calcification occurs in approximately 14% of patients harbouring these malformations [18]. In the literature, a total of 45 cases of spontaneous thrombosis of VGAM have been reported to date (**Table 1**) [19–28].

The reasons and the mechanisms determining this event are not well understood. Probably the main cause of spontaneous thrombosis of a VGAM is slow blood flow and obstructed venous outflow due to compression by a haematoma or post-haemorrhagic oedema, vascular spasm and regressive changes in the vessel walls [25]. Collins et al. reported a case of an association between thrombosis and recurrent aseptic meningitis [29].

Some authors have suggested a correlation between thrombosis and ventricular peritoneal shunt positioning [30–32]. Spontaneous thrombosis has also been reported to occur shortly after cerebral angiography [24], suggesting possible effects of the angiographic contrast media on the thrombotic process, i.e. slow flow or stasis of contrast media in the venous malformation may determine some degree of venous outflow obstruction [21,22,27]. Indeed, specific features of contrast media have been indicated as responsible for thrombotic events in VGAMs [33–36]. Other authors have emphasised increased flow pressure and turbulence on the venous side, leading to progressive myointimal proliferation of the vein of Galen, resulting in gradual venous thrombosis [37]. Moreover, thrombosis of the dural sinuses and deep venous system has frequently been reported in association with VGAMs, and could represent an additional causative factor. Other reported risk factors for thrombosis of the vein of Galen, independently from the presence of a vascular malformation, are use of oral contraceptives [38], postpartum status [39] and sickle cell anaemia [40].

Thrombosis of these aneurysms may be progressive [41] and often occurs during gestation or during the perinatal period [42]. The initial symptoms are often those stemming from hydrocephalus; sometimes seizure may be the initial symptom, particularly when an immediate thrombotic closure of the malformation occurs [24]. It is not clear how thrombosis influences the outcome, but it has been hypothesised that it may protect patients from developing congestive heart failure [43].

We reviewed all the reported cases of vein of Galen malformations in adults and, more specifically, those which determined hydrocephalus (**Table 2**).

The development of hydrocephalus as a late manifestation of vein of Galen malformations in adult patients may be due to obstruction of the cerebral aqueduct as a direct or indirect effect of the aneurysmal mass; communicating hydrocephalus after a subarachnoid haemorrhage [66]; a dual mechanism (aqueduct obstruction and defective absorption of CSF) [77]; passive ex

Table 1 Literature review: spontaneous thrombosis in vein of Galen malformations

Reference	Number of cases	Sex, Age	Symptoms and signs	Hypothesised cause of thrombosis	Calcification	Treatment of malformation	Outcome	Follow-up
Heinz ER et al. [42]	1	?, 6 months	Hydrocephalus	Pre- or perinatal thrombosis		Surgical removal		
Weir BK et al. [44]	1	?, 7 months	Hydrocephalus			Surgical removal		
Jamieson KG et al. [41]	1		Hydrocephalus			Surgical removal		
Siqueira EB et al. [18]	1	F, 30 years	Progressive spasticity, hydrocephalus	?	Yes	Surgical removal	Significant improvement	4 years
Lazar ML et al. [16]	1	F, 11 months	Craniomegaly, vomiting, somnolence, tense fontanelle, hydrocephalus	?	Yes	Surgical removal	Improved	10 months
Copty M et al. [45]	1	F, 45 years	Progressive paraparesis, headaches, hydrocephalus	?	Yes	Aneurysm removal	Stable	10 months
Six EG et al. [46]	1	M, 3.5 years	Ataxia, lethargy, fever, vomiting, irritability, hydrocephalus	?	No	Thrombus resection	Good	
Carson LV et al. [47]	1	?, 37 years	Macrocephaly, headaches, vomiting, slow thought		Yes	Clipping of feeding vessels		
Dean DF et al. [48]	1	M, 3 years	Macrocephaly, hydrocephalus, bulging fontanelle, motor and mental retardation		Yes	Clot removal	Improved	2 years
Diebler C et al. [49]	3	2–l5 months						
Skirkhoda A et al. [50]	2?	3 months	Microcephaly		Yes	Clipping of feeding vessels	Good	
de Morais JV et al. [51]	1	F, 8 months	Increasing head circumference, mild mental retardation, vomiting, unconsciousness, hydrocephalus	?	Yes	Surgical removal	Improved	1 year
Olin MS et al. [43]	1	F, 4.5 years	Lethargy, hydrocephalus	7	Yes	None	Improved	?
Di RoccoC et al. [52]	1	M, 9 years	Abrupt mental deterioration, hydrocephalus	?	Yes	None	Good	
Whitaker JB et al. [27]	1							
Gangemi M et al. [53]	1?							
Chapman S et al. [53]	1	M, 3.5 months		?	Yes			
Mancuso P et al. [54]	1	F, 5 months	Fever, seizures, hydrocephalus, intracranial hypertension	Superior sagittal sinus thrombosis	No	None	Good	
Collins JJ et al. [29]	1	M, 20 years	Severe headache, fever, meningismus, sudden hemisensory loss	Benign recurrent aseptic meningitis	No	None	Good	1 year
Hanigan WC et al. [55]	1	F, 5 days	2 episodes of intracranial haemorrhage, irritability, cranial sutures separated, tense fontanel, head circumference 44 cm	Haemorrhage	No	None	Profoundly retarded	16 months
Beltramello A et al. [19]	2							
Hurst RW et al. [22]	2	M, 4 months M, neonate	Increased head circumference, hydrocephalus Hydrocephalus and increased head circumference at 6 months of age	? ?	No No	None None	Good Good	3 years 11 months
Roszkowski M et al. [56]	1	?, 8 years		?	No	Aneurysm removal		
Lasjaunias P et al. [9]	4	?	?	?	?	?	1 neurologically normal	?
Kim BK et al. [57]	1	F, l3 years	Sudden headache	?	No	None	Improved	3 weeks
Sehulman H et al. [58]	1	?, 2 years		?	Yes		Normal development	5 years
Chiang V et al. [59]	1	F, neonate	None	?	No	None	Excellent	14 months
Nikas DC et al. [25]	2						Excellent	
Konus OL et al. [24]	1	M, 5 years	Seizures 12 hours after AGF, macrocephaly, mental retardation	Angiography	No	None	Stable	2 years
Konovalov AN et al. [23]	2	F, 9 years F, 5 years	Left hand feeble, shuffling left foot, deteriorating memory Late walking, bizarre gaze	? ?	No No	Aneurysm removal Aneurysm removal	Improved Improved	10 days 2 weeks
Tawk RG et al. [30]	1	M, 4 months	Irritability, anorexia, vomiting, macrocrania	VP shunt	No	None	Neurologically intact	17 years
Kuzeyli K et al. [31]	1	M, 6 months	Nausea, vomiting, irritability, stupor episodes, seizures, macrocrania	VP shunt	No	None	Excellent	7 years
Sasidharan CK et al. [32]	1	M, 4days	Increased head circumference, sutural diastasis, wide open fontanels	VP shunt	No	None	Spastic diplegia, mental retardation	16 years
Marques RM et al. [28]	1	M, 65 years	Generalised tonic-clonic seizures	?	No	None	Improved	?
Vijayaraghavan SB et al. [26]	1	Fetus 21 gestational weeks	/	?	No	None	Dead	/

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Table 2 Literature review: vein of Galen malformations causing hydrocephalus in adults (>16 years)

Reference	Sex, age (years)	Associated lesions	Treatment of hydrocephalus	Outcome	Follow up
Mylonas C et al. [60]	F, 55	/	VP shunt	Improved	?
Mayberg MR et al. [61]	M, 64	Dural AVM, straight sinus thrombosis	VP shunt	Improved	?
Lewis Al et al. [62]	M, 66 F, 63	Dural fistula Dural fistula	VP shunt; embolisation, RS VP shunt; embolisation, RS	Improved Improved	2 years ?
Pun KK et al. [63]	Not available	/	VP shunt	Improved	Not available
O'Reilly GV et al. [64]	M, 56	/	Oral dexamethasone	Improved	?
Rosenfeld JV et al. [65]	F, 57	/	External drainage, VP shunt	Improved	2 years
Askenazy HM et al. [66]	M, 39	Not available	Not available	Not available	Not available
Agee OF et al. [67]	M, 37	/	VA shunt	Improved	?
Hernesniemi J [68]	M, 46	/	Surgery, occipital catheter	Improved	3 years
Kleindienst A et al. [69]	M, 48	/	Shunt	Improved	?
Abe T et al. [70]	F, 19	/	VP shunt	Improved	9 years
Shin M et al. [71]	F, 64	Dural AV fistula. SAH	VP shunt, RS, RT on dural AV fistula	Improved	1 year
Ribeiro VT et al. [72]	F, 18	Not available	Not available	Not available	Not available
Amacher Al et al. [73]	F, 27	/	Torkildsen shunt	Dead	?
Strowitzki M et al. [74]	F, 27	SAH	External drainage. VP shunt	Initial improvement, dead 1 year after (bleeding)	?
Siqueira EB et al. [18]	F, 30	/	Surgery	Improved	4 years
Lasjaunias P et al. [10]	?, 25 ?, 18	/ /	Embolisation Proposed for embolisation	Stable Lost to follow-up	? /
Tomsick TA et al. [75]	F, 25	/	Embolisation, RT, shunt	Improved	3 years
Wong KN et al. [76]	M, 38	/	VP shunt, embolisation, surgery	Initial improvement, vegetative status (haemorrhage)	?

vacuo hydrocephalus; thrombosis of an aneurysm, and high venous pressure. Most authors believe that mechanical obstruction of the mesencephalic aqueduct and high venous pressure with consequent impaired CSF absorption are the primary causes of the development of hydrocephalus [78].

The most frequently adopted treatment of hydrocephalus in adult patients affected with VGAM consists in ventriculoperitoneal shunting. In a few cases, it has been reported that treatment of the aneurysm itself through embolisation and radiosurgery was followed by a reduction in volume of the malformation and, consequently, a regression of the hydrocephalus. Hernesniemi noted that surgical excision of the malformation associated with an occipital catheter in a 46-year-old male, was followed by regression of hydrocephalus [68] Agee reported a case of a patient successfully treated with a ventriculoatrial shunt.

To the best of our knowledge, this is the first case of hydrocephalus due to an aneurysm of vein of Galen treated with an endoscopic third ventriculostomy, the rationale of the treatment being the aqueductal blockage of the CSF.

Some technical difficulties should be taken into account. They depend mainly on the pathological variations in the posterior fossa caused by a relevant mass in the pineal region, pushing the brainstem forward and so reducing or even occluding the prepontine cisterns. The basilar artery becomes very close to the clivus and therefore little, if any, space is available in the floor of the third ventricle to perform a third ventriculostomy. In our case we preferred to transversally open a stoma in the dorsum sellae, using it as a reference and support point, to avoid any dangerous contact with the basilar artery.

Endoscopic third ventriculostomy is considered the standard treatment of blocked hydrocephalus because of its well-known advantages compared to traditional shunts. It seems to minimise the risks related to quick, substantial reduction of CSF pressure, i.e., a rise in differential pressure between the ventricle and the vascular system and potential volume growth of the vascular malformation, with a higher risk of rupture [78, 79]. In fact, third ventriculostomy brings intracranial pressures back to normal levels within in a few days, avoiding overdrainage and hypotension. Thus, high venous pressure should be a specific indication to third ventriculostomy in patients affected by hydrocephalus associated with such vascular malformations.

Conclusion

Vein of Galen ectasias are not frequent, and they are unusually detected in adulthood. Occasionally they can thrombose. They can determine hydrocephalus, either owing to the mass effect on the cerebral aqueduct or because they bleed with consequent impairment of CSF reabsorption. Third ventriculostomy is a safe, relatively simple treatment for non-communicating hydrocephalus; it allows normalisation of cerebral pressures while avoiding abrupt hyperdrainage and diminishing the risk of rupture of the vascular malformation.

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