

Brain herniations into arachnoid granulations: about 68 cases in 38 patients and review of the literature

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Abstract

Introduction Brain herniations (BH) into arachnoid granulations (AG) in dural venous sinuses and calvarium have rarely been reported in the literature.

Methods MRIs of 38 patients with BH into AG (BHAG) were retrospectively analyzed. Locations of BHAG, gyrus/lobe of the herniated brain, parenchymal abnormalities of the BH, and clinical and radiological conditions with raised intracranial pressure were recorded.

Results Sixty-eight BHAG were found, by order of frequency, in the occipital squama (OS), transverse sinus (TS), lateral lacuna of the superior sagittal sinus (LLSSS), and straight sinus (SS), with cerebellar tissue being the most frequently involved in BHAG (94.5 % of OS, 55 % of TS, 100 % SS BHAG). Multiple BHAG were found in 58 % of the patients (up to five per patient). Parenchymal signal and structural changes (SSCG) were observed in 46 % of BHAG (100 % were cerebellar). Three patients had pseudotumor cerebri (PTCS); one patient had only MRI signs of PTCS. Twenty-one percent of patients had intracranial conditions susceptible of increasing cerebrospinal fluid (CSF) pressure other than PTCS.

Conclusions BHAG occurred in the OS, TS, LLSSS, and the SS. SSCG of the herniated cerebellum were frequent and possibly result from tethering/strangulation in the AG. No

symptoms could be clearly attributed to BHAG, though in three cases of PTCS, TS BHAG could have contributed to sustaining the raised CSF pressure. Various factors are probably involved in the development of BHAG including normal pia-arachnoid bridges between the brain surface and the AG, hydrodynamic constrains on the brain and AG, and, in some cases, increased intracranial pressure.

Keywords Arachnoid granulations · Brain herniation · Parenchymal abnormalities · Acquired meningo-encephalocele

Abbreviations

AG	Arachnoid granulation
BH	Brain herniation
BHAG	Brain herniation into an AG
ITG	Inferior temporal gyrus
ISL	Inferior semilunar lobule
L	Left
LLSSS	Lateral lacunae of the SSS
LOTG	Lateral occipito-temporal gyrus
NC-CT	Non-contrast CT
OS	Occipital squama
PC-CT	Post-contrast CT
PTCS	Pseudotumor cerebri syndrome
R	Right
SiS	Sigmoid sinus
SS	Straight sinus
SSCG	Signal and structural changes
SSL	Superior semilunar lobule
SSS	Superior sagittal sinus
TS	Transverse sinus

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Introduction

Arachnoid granulations (AG), also known as Pacchionian granulations or Pacchionian bodies, develop from the arachnoid membrane [1, 2] and invaginate into the dura mater as they grow. Though Antonio Pacchioni is generally credited for describing AG for the first time in 1705, hence the eponymous designation, AG had already been reported by various investigators including Samuel Collins (1685), Alexis Littre (1684), and Thomas Willis (1664) and described and illustrated as far back as 1543 by Andreas Vesalius [1–3]. We owe Pacchioni a detailed description of AG and an attempt to explain their function. AG arise from arachnoid villi [2] that may be found on the visceral surface of the arachnoid and are filled with cerebrospinal fluid (CSF). CSF pulsations likely represent the principal mechanism of growth of arachnoid villi into AG. AG appear with age [2, 4], being virtually absent in early childhood and becoming a constant finding in adult life [1, 5]. As they grow, they invaginate into the dura forming a CSF-filled cavity and may erode into the calvarium [2, 3, 5]. Digitations of pia mater covering the brain surface extend into the AG, thus anchoring the brain to the dura matter, forming pia-arachnoid bridges [2, 3, 6, 7].

AG are found within dural venous sinuses or meningeal and diploic veins [2, 5, 8, 9], frequently located at points of cortical venous convergence into a dural sinus [1, 10]. AG may project into the lumen of the dural venous sinus and must be distinguished from thrombosis on imaging studies. Their most frequent location is within the lacunae laterales of the superior sagittal sinus (LLSSS) [2, 5]. They may also be found in the lumen of the superior sagittal sinus (SSS), the transverse sinuses (TS), the great vein of Galen/straight sinus junction, the straight sinus (SS), paracavernous sinuses, the middle meningeal veins in the middle cranial fossa, or in the tentorial sinuses [2, 5, 11]. AG in the TS most frequently arise from the infratentorial surface though they can also arise from the cerebral surface [5].

AG may also develop into the calvarium at a distance from a dural venous sinus and seemingly with no relation to venous structures, eroding the inner table, the diploe, and even the outer table. Although they may involve any part of the calvarium, they are frequently identified in the frontal bone and occipital squama, where they appear as well-marginated lytic lesions on plain films and CT and follow CSF signal on MRI [12–16]. The bony defect harboring these arachnoid granulations (arachnoid pits) may exceed 10 mm in diameter. AG in pneumatized regions of the base of the skull may provide a pathway for spontaneous CSF leaks and for meningocele/encephalomeningocele [17, 18].

The purpose of the present imaging study is to report cases of brain herniations (BH) into AG located in the dural venous sinuses and/or the calvarium that were encountered during routine diagnostic imaging investigations. BH into dural venous sinuses or the calvarial bony defects have rarely been reported

in the literature. To the best of our knowledge, this is the largest series of BH into AG (BHAG). Physiopathological and clinical considerations and a review of the literature are provided.

Materials

This is a retrospective study based on MRIs performed at the Hospital of Sion. A keyword search using “brain herniation”, “meningo-encephalocele”, and “arachnoid granulation” of our RIS PACS system (Radoffice, WDS technologies SA, Geneva, Switzerland) was performed. All patients gave informed consent for MRI±CT being performed during routine medical investigations. Informed consent of the patients for the study was waived by our IRB due to the retrospective nature of the study.

MRI were performed on a 1.5-T scan (Siemens Medical Systems, Erlangen, Germany). All MRI studies included non-contrast 3DT1 MPRAGE or post-gadolinium (0.1 mmol/kg) 3DT1 MPRAGE (Gd-3DT1) acquisitions, T2 Tirm dark fluid, TSE-T2 sequences, and susceptibility weighted imaging (SWI). In some cases, 3DT2 space or CISS acquisitions were also available. When CT studies (64 MDCT; GE, Fairfield, USA) were also available, they were included in the analysis. All cases of BHAG into a dural venous sinus or the calvarium were included. Patient medical history was analyzed. History and imaging signs of increased intracranial pressure or pseudotumor cerebri were sought for. The origin of the BH and the location of the BHAG were recorded. Signal or structural changes of the herniated brain were noted. In MRI studies with available Gd-3DT1 images, enhancement of the dura lining the AG in cases of OS BHAG was recorded. In the cases of BHAG into the calvarium where CT was available, erosions of the outer table were recorded.

Consensual analysis of the imaging and data was performed by a senior radiology resident and a senior board certified neuroradiologist (SM and DSM, respectively). All imaging sequences for each patient were analyzed at the same time.

A French and English literature review was performed with PubMed and Google. Cited references in pertinent published articles were also scrutinized in search for publications that could have been missed by the internet search.

Results

The keyword search of our RIS PACS system revealed 38 patients harboring a total of 68 BHAG between February 2013 and January 2016 (no cases found before February 2013). All patients were older than 18 years of age. Average age of patients was 63 ± 16.3 years and 66 % of patients were females.

BH were found by order of frequency in the OS, TS, LLSSS, and SS, all corresponding to typical locations of AG.

BH were surrounded by a CSF rim or plainly located within a uni- or multiloculated CSF-filled defect in the dural venous sinus, the frontal/parietal bone, or OS (Figs. 1, 2, 3, 4, 5, and 6). Furthermore, when located in the dural venous sinuses, a zone of venous convergence was found at the point of the BH or in its vicinity. Though no statistical analyses in terms of frequency were performed, OS BHAG often contained inferior cerebellar or vermician veins and bore a close anatomical relation with OS diploic veins.

Sixty-three percent of the BHAG were in the OS (43/68), 29.5 % in the TS (20/68), 6 % in the LLSSS (4/68), and 1.5 % in the SS (1/68). Seventy-eight percent of all BHAG arose from the cerebellum. Ninety-five percent of OS BHAG (41/43) arose from the cerebellum and 5 % from the occipital lobe (2/43). In the latter, the OS AG was located supratentorially above the torcular herophili. Eighty-eight percent of OS BHAG arose from the inferior semilunar lobule (ISL) (Fig. 3), 4.5 % from both the ISL and the superior semilunar lobule (SSL) (2/43) (Fig. 5), 4.5 % from the occipital lobe, and 2 % from the SSL (1/35). Herniated brain into TS AG arose from the inferior temporal gyrus (ITG) in 40 % of the cases (9/20) from the SSL in 55 % (11/20) (Fig. 2), and from the ITG and lateral occipito-temporal gyrus (LOTG) in 5 % of the cases (1/20) (Fig. 1). One hundred percent of SS BHAG arose from the SSL (1/1) (Fig. 4). One hundred percent of LLSSS BHAG arose from the superior frontal gyrus (Fig. 6), all eroding into the parietal bone except for one which eroded the frontal bone, immediately in front of the coronal suture.

Multiple BHAG were noted in 58 % of the patients (22/38). Eighty-two percent of the patients with multiple BHAG had two BHAG, 9 % had three BHAG, 4.5 % had four, and 4.5 % had five.

Signal and structural changes (SSCG) of the BH within the AG or the stem of the BH (Figs. 2 and 5) were noted in 46 % of the BHAG (31/68). These changes included focal brain atrophy (widening of sulci or folia) and/or hyper-T2 and T2-tirm signal consistent with gliosis (no restricted diffusion, no enhancement, no blood residues on SWI). SSCG were found in 53 % of OS BHAG (23/43), 35 % of TS BHAG (7/20), 100 % of SS BHAG (1/1), and 0 % of LLSSS BHAG. One hundred percent of SSCG involved herniated cerebellar tissue.

Enhancement of the dura mater lining the OS AG was observed in 19 of the 28 OS BHAG (68 %) where Gd-3DT1 sequences were available.

Non-contrast or gadolinium-enhanced volumetric T1 images and volumetric T2 images were respectively obtained in 100 % and 48 % of the studies. All BHAG were detectable on 3DT1 (with or without gadolinium enhancement) and 3DT2. Gd-3DT1 images were not superior to non-enhanced 3DT1 images for the detection of BHAG (100 % were visible on non-enhanced 3DT1 images), but they could better delineate the structures surrounding the BH (CSF rim, boundaries

of the dural venous sinus lumen), demonstrate enhancement of the dura lining OS AG, and show pial/diploic veins in the vicinity of the BHAG.

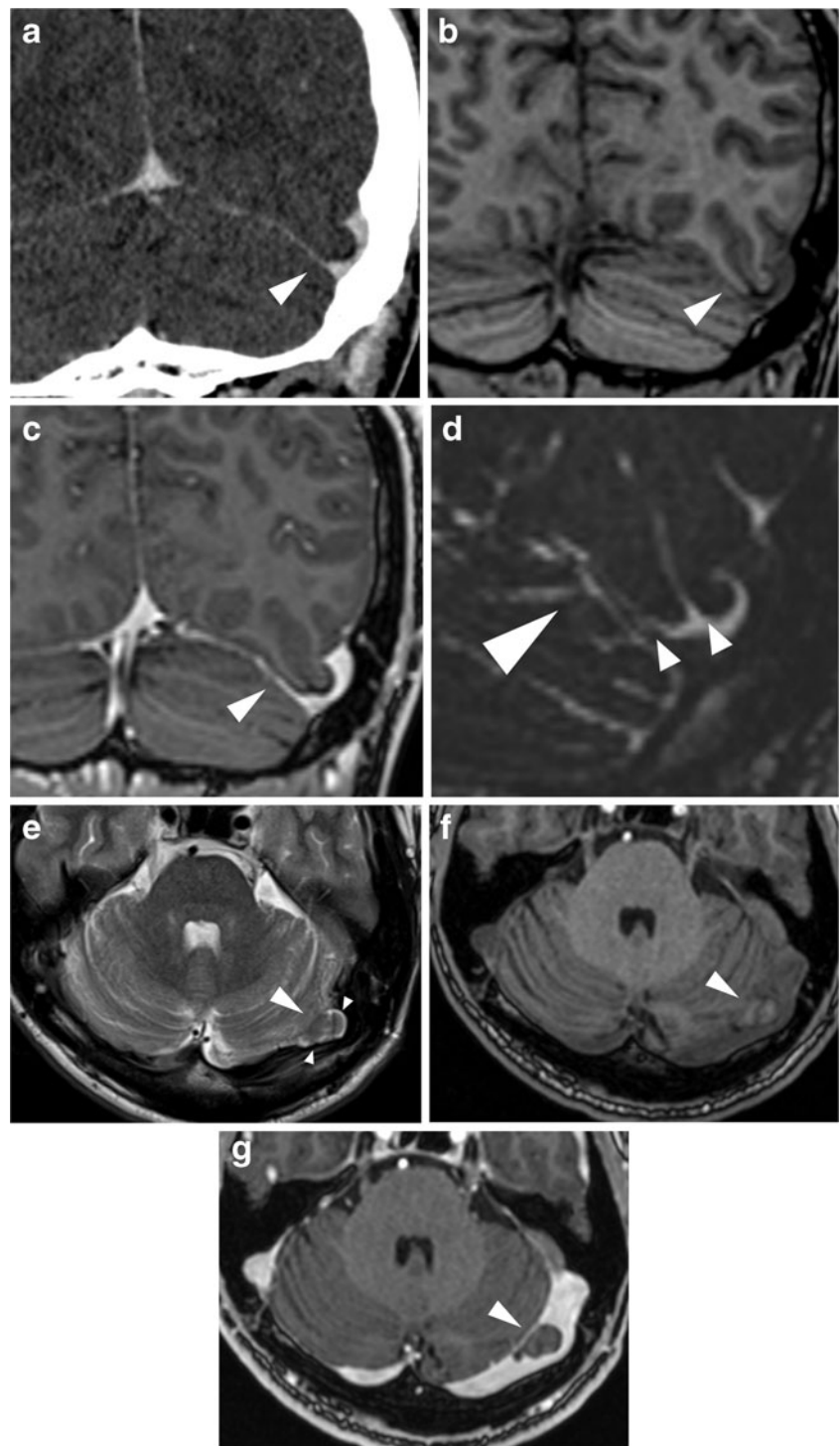
Eighty-four percent of the patients had a non-contrast CT study of which 63 % included contrast-enhanced CT (either CTA, CTV, or delayed venous phase CT). BHAG were identifiable retrospectively in 87 % of CT studies and suspected on CT prior to MRI in 58 % (initial imaging workup). Ninety percent of TS BHAG were identifiable retrospectively and 45 % were suspected on the initial CT (all were post-contrast CT) against 84 and 65 %, respectively, for OS BHAG. One hundred percent LLSSS BHAG were suspected on the initial study in the patient with LLSSS BHAG that had pre-MRI CT. The SS BHAG was not suspected on initial non-contrast and post-contrast CT but was identifiable retrospectively.

Erosions measuring up to 4 mm of the outer table of the calvarium were found in 13 % of BHAG (9/68), seven of which corresponded to OS BHAG and two to LLSSS BHAG (Fig. 6). No brain tissue was seen to herniate beyond the outer table. In one case of LLSSS BHAG (Fig. 6) and one case of OS BHAG, CSF from the BHAG was seen to extrude beyond the outer table defect on MRI (patients 34 and 37).

Four patients had imaging signs of pseudotumor cerebri syndrome (PTCS). Imaging signs of PTCS were considered in the presence of enlarged sella, “empty or partially empty sella”, dilated optic nerve sheaths, tortuous optic nerves, and dilated CSF spaces of Meckel’s cave or around the oculomotor nerves and/or abducens nerves [19]. Three patients (patients 1, 8, and 38) had clinical symptoms of PTCS that were managed conservatively with acetazolamide treatment. Patients 1 and 8 had TS BHAG, arising from the ITG bilaterally for patient 1 and from the SSL bilaterally for patient 8. BHAG were responsible for severe bilateral TS stenosis in patient 8. In patient 1, there was severe stenosis of the right TS caused by the BHAG and AGs and a hypoplastic left TS with severe stenosis from AG and extrinsic compression from increased CSF pressure (left TS BHAG did not cause significant stenosis). As the patients evolved favorably under conservative medical treatment, no TS stenting was contemplated and no TS pressure measurements were performed. The third patient was recently diagnosed with PTCS (patient 38) secondary to venous outflow obstruction from a torcular herophili meningioma invading the origin of the right TS that was severely stenosed, and from a left TS severe stenosis secondary to AG containing herniated SSL. The patient is waiting for follow-up consult. One patient had imaging signs of PTCS (patient 7) without symptoms of PTCS and could have corresponded to a case of “burnt out” PTCS. In this patient, BHAG were located in the right TS and in the right OS.

Twenty-one percent of patients (8/38) had or had had intracranial conditions potentially causing chronically raised intracranial pressure (Table 1).

Fig. 1 Thirty-three-year-old male with subarachnoid hemorrhage of unknown origin (patient 15). Herniation of the ITG and LOTG (*large arrowhead*) is seen into the left TS. This BHAG was suspected on initial PC-CT. ITG looks normal. It is surrounded by CSF that contained an AG (*small arrowheads (d, e)*). Coronal MPR reformat: CTA (**a**), 3DT1 MPRAGE (**b**), Gd-3DT1 MPRAGE (**c**), 3DT2 space (**d**); axial: TSE-T2 (**e**), 3DT1 MPRAGE (**f**), Gd-3DT1 MPRAGE (**g**)



Discussion

Sixty-eight BHAG were found in 38 patients in this study based on MRI findings. Sixty-three percent occurred in the OS, 29.5 % in the TS, 6 % in the LLSSS, and 1.5 % in the SS. Average age was 63 years, suggesting that BHAG occur more frequently with advancing age. Two thirds of patients were females.

That BH occurred into AG is supported by several morphological features in the present study: BH were found in typical locations for AG (OS, TS, LLSSS, and SS); they were surrounded by a CSF rim or plainly located within a uni- or multiloculated CSF-filled defect in the dural venous sinus or calvarium; when located in the dural venous sinuses, a zone of venous convergence was generally found at the point of the BH

Fig. 2 Eighty-five-year-old woman with right frontal glioblastoma multiforme (patient 25). Bilateral SSL (*arrows*) herniation into AG in the TS (*large arrowheads (b–d)*). SSCG of the herniated SSL surrounded by gliosis (*small arrowheads (a)*). The herniated tissue appears tethered. Enhanced TS (*small double arrows (d, e)*) is visible around the BHAG. Coronal T2 TIRM dark fluid (*a*); axial TSE-T2 (*b, c*); reformat Gd-3DT1 MPRAGE (*d—axial oblique, e—coronal oblique*)

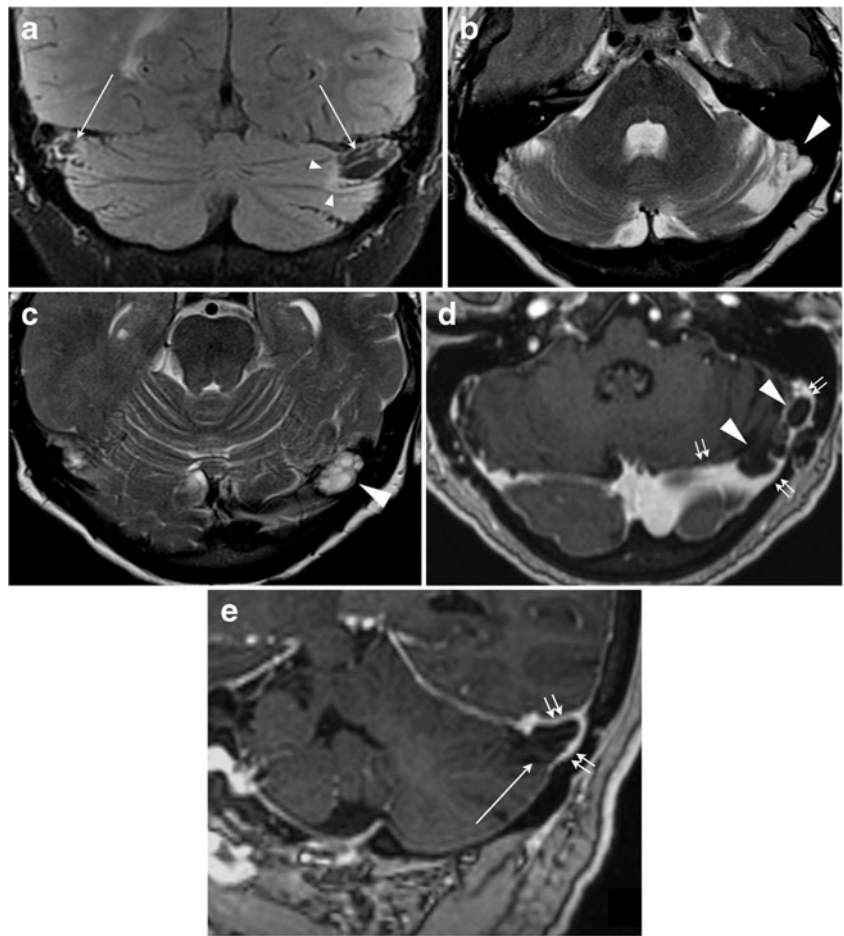


Fig. 3 Thirty-four-year-old woman with suspected meningitis and multiple BHAG (patient 24). There is a small herniation of normal looking ISL (*arrow*) into an AG of the OS (*arrowhead*) on the left side. Typical CSF filled and multilobulated appearance of the AG OS. There is substantial contrast enhancement of the wall of the AG OS (*arrowhead (d)*), which is much more conspicuous than normal looking dura mater (*small double arrow*). This enhancement could result from either tethering of the dura or inflammation. TSE-T2 (*a*), 3DT2 space (*b*), 3DT1 MPRAGE (*c*), and Gd-3DT1 MPRAGE (*d*)

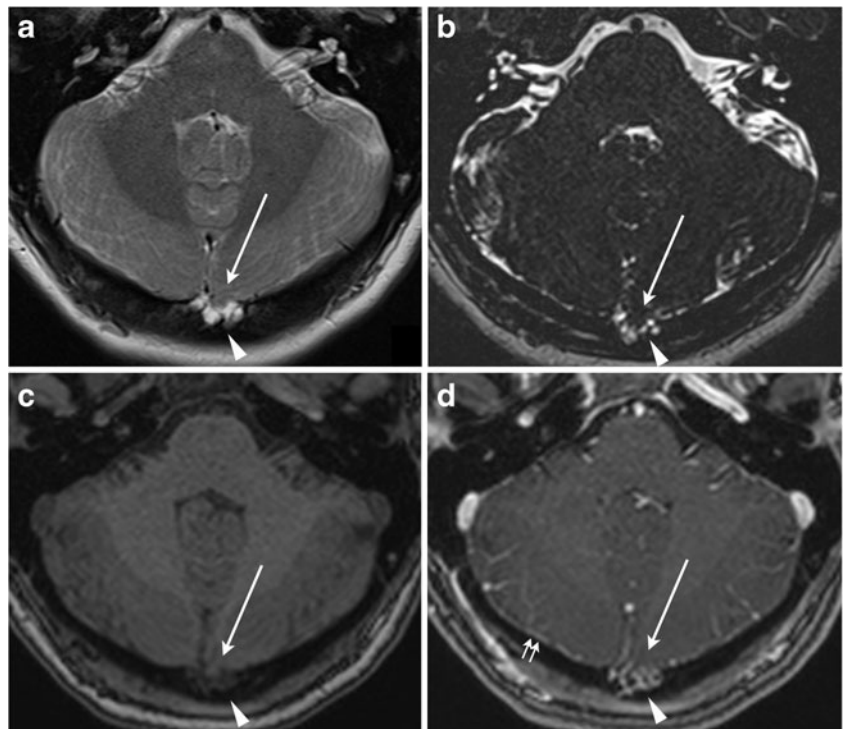
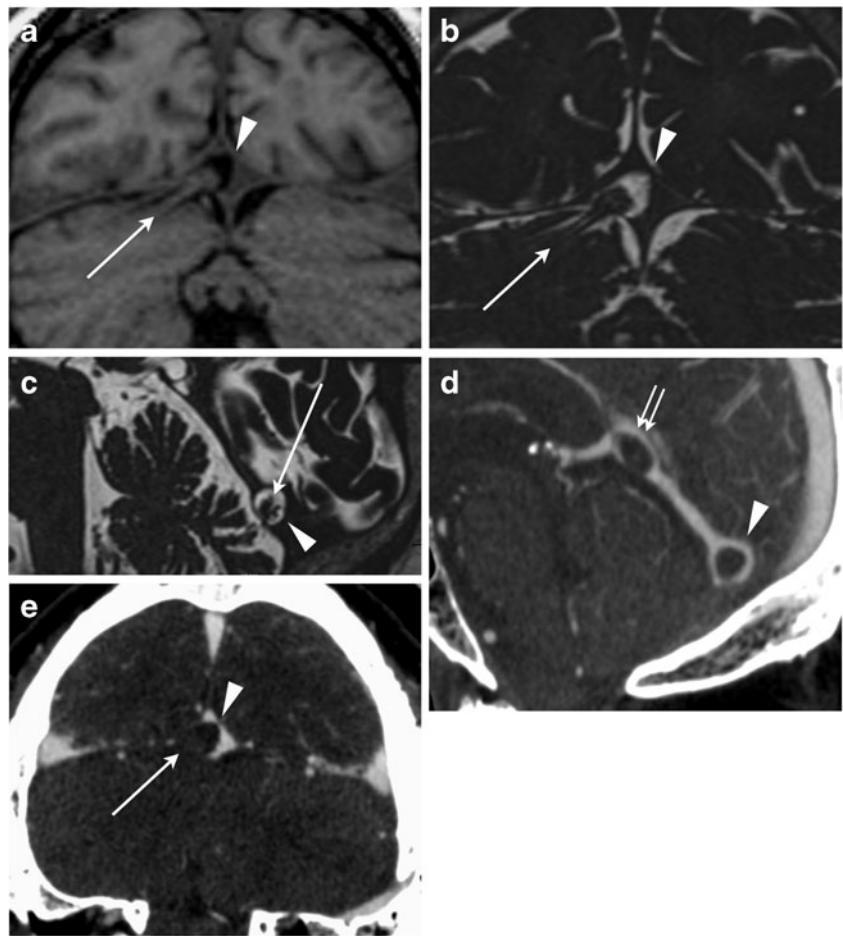


Fig. 4 Seventy-four-year-old male with transient global amnesia (patient 22). Herniation of the SSL (*arrow*) into an AG of the distal SS (*arrowhead*). The herniated SSL looks tethered and slight SSCG are observed. This BHAG was mistaken for a large AG of the SS on contrast-enhanced CT (**d, e**), though the herniated SSL was visible (**e**) and retrospectively identified as a BHAG after the MRI study. A second large AG (*double arrow* (**d**)) was also visible on CT at the great vein of Galen and SS junction but contained no brain tissue. Coronal oblique reformat 3DT1 MPRAGE (**a**) and 3DT2 space (**b**); sagittal reformat 3DT2 space (**c**), sagittal and coronal oblique reformat CTA (**d, e**)

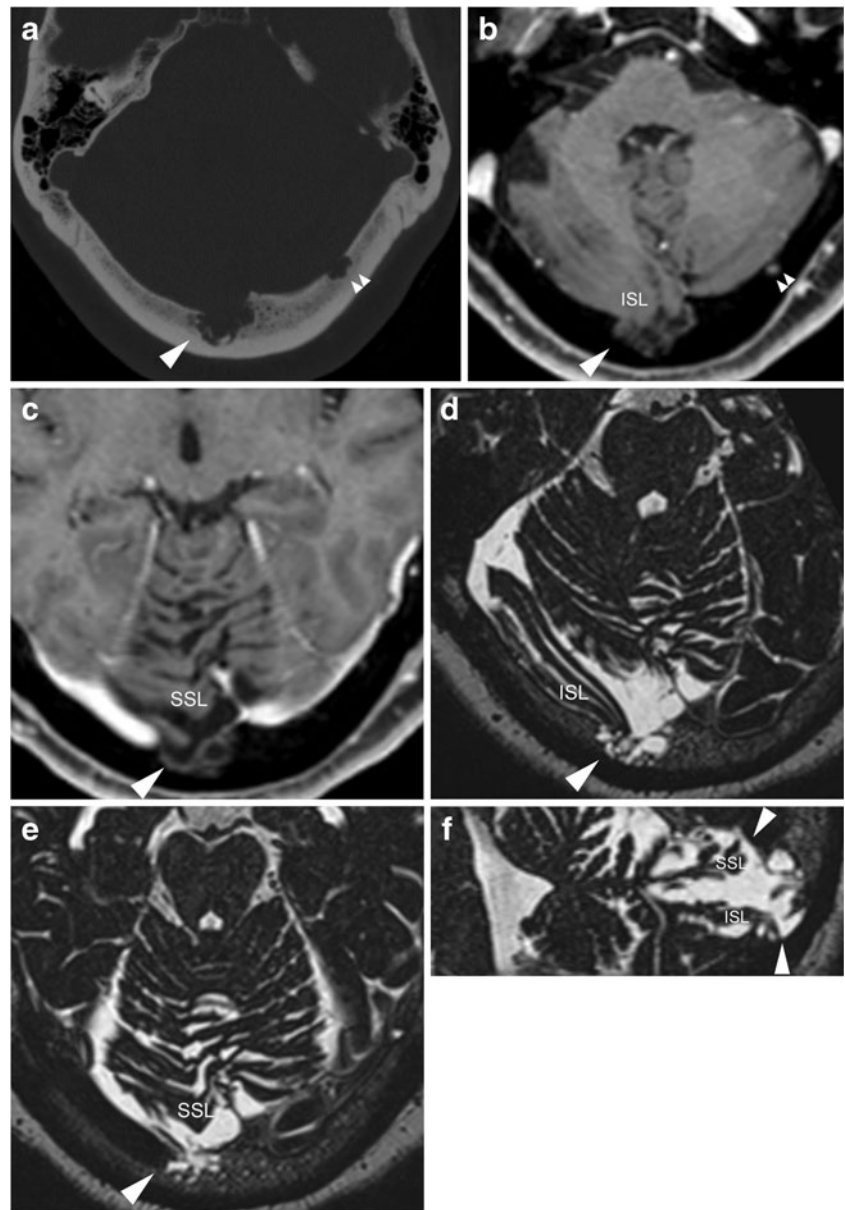


or in its vicinity; and when located in the OS, the osseous defect harboring the BH often contained inferior cerebellar or vermian veins and/or bore a close anatomical relation with OS diploic veins. There is also evidence in the literature to support that the BH reported in our series occur in AG. In a paper on venous pseudotumor cerebri [20], it is mentioned that “small hernias of brain tissue are frequently seen in the base of arachnoid granulations at craniotomy” (M Besser; personal communication). No follow-up publication to this “personal communication” could be found to provide further details. More importantly, Wolbach [7] presented nine autopsy cases of “hernias of the cerebrum and cerebellum” due to increased intracranial pressure, six of which had BH into dural venous sinuses or into the occipital squama (Table 2). In five of these cases, the author specified that BH entered arachnoid villi and he provided microscopic correlation of one case where distorted cerebellar tissue herniated into large arachnoid villi in the right lateral sinus. Beneke, quoted by Wolbach [7], described an autopsy case of intracranial hypertension secondary to a choroid plexus papilloma in which he found numerous BH into arachnoid villi along the superior sagittal sinus. These findings were confirmed histologically as quoted by Wolbach: “The hernias consisted of brain tissue in various stages of

disorganization, some containing vessels of considerable size, which were carried along from their original positions... The entrance of the hernias into the dura takes place through endothelial lined fissures, which Beneke states normally contain processes of arachnoid tissue. These arachnoid processes, he goes on to say, are the smallest “Anlagen” of the Pacchionian granulations”.

Descriptions of BHAG in the radiological literature are scant (summarized in Table 2). Kollar et al. [21] reported a case of TS dura arteriovenous fistula (DAVF) containing a round filling defect in the fistulous portion of the TS on digital subtraction angiography (DSA). The DAVF was embolized and subsequently resected surgically. Herniated brain tissue was found within the resected TS that corresponded to the filling defect observed on DSA. Other than its ectopic location, the herniated brain was histologically normal. Liang et al. [22] mention that two out of 433 filling defects in the dural venous sinuses on MRI were due to invaginated brain into the dural venous sinus. Their figure 5 illustrates an occipital lobe BH into the right TS, though no other information concerning these two cases was provided. Recently, five individual case reports and a series of five cases [23–28] reported a total of ten patients harboring 13 BH. A study including 20 patients and

Fig. 5 Seventy-year-old female with squamous cell carcinoma of the base of the tongue, cerebral metastasis, and meningeal carcinosis (patient 14). There is a giant AG of the OS (AGOS—*arrowhead*) on the midline and a small one on the left (*double arrowhead*). The giant AGOS contains herniated, tethered ISL and SSL with mild SSCG from the right cerebellum. Note how the right cerebellar hemisphere is distorted from the attraction into the AG. In comparison with Fig. 4, there is faint contrast enhancement of the walls of the AGOS (b, c) identical to that of normal dura. Non-contrast CT (a); reformat Gd-3DT1 MPRAGE (b, c); reformat 3DT2 (d–f)

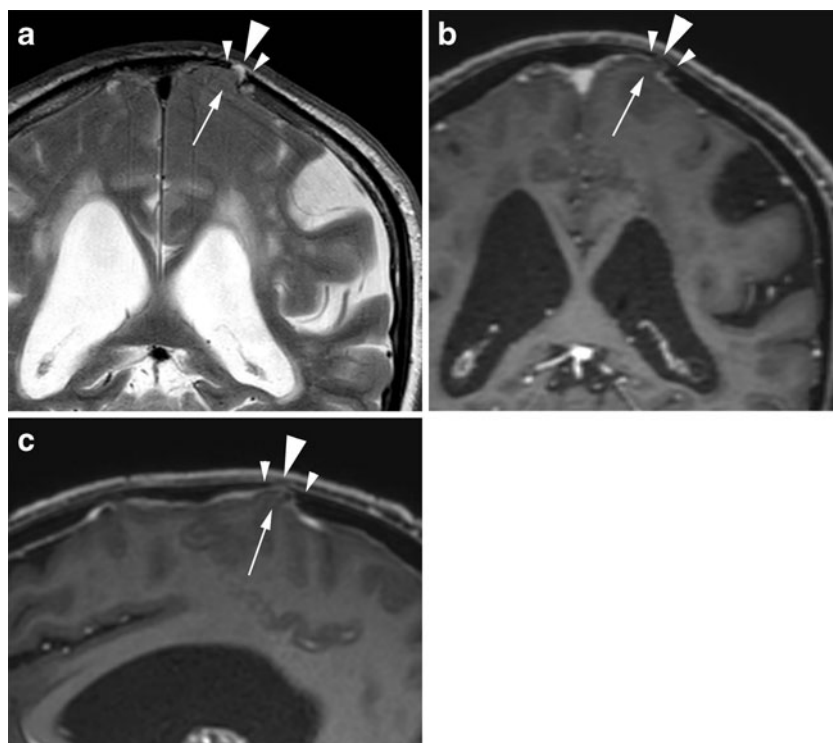


21 BH [29] was published while the present manuscript was being finalized. In this study, 62 % of BH occurred in the TS, 19 % in the torcular, 14 % into the OS, and 5 % into the sigmoid sinus. Four cases involved pediatric patients of 8, 9, and 11 (two patients) years of age [24–26, 29], the rest of BHAG being encountered in adult patients as in the present series. Some authors have suggested that these BH occurred into AG [24, 27]; others do not take a clear position [23, 25] or suggest that BH could also invaginate into dural venous sinuses at points of dural weakness or where cortical veins enter the venous sinus without clearly ruling out AG [26, 29].

Several authors have referred to BHAG into dural venous sinuses as encephaloceles [23, 27, 28]. Battal et al. [26, 29] argue, however, that BH into dural venous sinuses and calvarium do not represent encephaloceles as “encephaloceles are

composed of meninges and brain extruding outside the skull”. In our study, no brain tissue was seen to herniate outside the outer table of the calvarium. Erosions of the outer table were, however, observed in 13 % of BHAG, and in two of those cases, CSF from the BHAG extruded outside the outer table on MRI (Fig. 6). Beneke, cited by Wolbach [7], described an autopsy case of “papilloma of ependymal origin arising from the choroid plexus. There were symptoms [of increased intracranial pressure] for about 1 year before death... There was marked internal hydrocephalus. The [brain] hernias were particularly numerous in the regions of the arachnoid villi along the superior longitudinal sinus. A number had completely perforated the bone”. An analogy can be drawn between BHAG of the skull vault and dural venous sinuses and temporal and sphenoidal acquired meningo-encephaloceles where AG

Fig. 6 Seventy-eight-year-old male with left sigmoid sinus DAVF and normal pressure hydrocephalus harboring two LLSSS BHAG from the SFG (patient 32). The more posteriorly located of the two BHAG (*large arrowhead*) is seen to cross the entire thickness of the calvarium, eroding through the outer table (*small arrowheads*). The herniated SFG (*arrow*) reaches the diploic spaces, while the CSF component from the AG surrounding the BH extrudes slightly beyond the outer surface of the outer table. **a** Coronal TSE-T2, **b** coronal reformat Gd-3DT1 MPRAGE, **c** sagittal oblique reformat Gd-3DT1 MPRAGE



located in the cranial fossae seem to be implicated in the development of the meningo-encephalocele [18]. Finally, the definition of “cele” (from the greek word “Kele”) is that of “a swelling or hernia in a specified part” (Oxford, British and World English Dictionary, Online). BHAG, therefore, correspond to a form of acquired meningo-encephalocele.

Though rare, BHAG may be encountered during routine brain imaging. An incidence of 0.32 % has recently been reported [29]. They must be distinguished from venous sinus thrombosis, dural-based tumors, or other normal filling defects of dural venous sinuses, such as septa, AG, fat, etc. [22, 30, 31]. MRI readily demonstrated BHAG, particularly if high-resolution volumetric T1 and T2 sequences are obtained, in keeping with findings from Battal et al. [29]. Indeed, conventional brain sequences have proved to be inadequate for the detection of small BH due to thick slice thickness [29]. In our study, CT was also effective in demonstrating BHAG, which were retrospectively identified in 87 % of CT studies and suspected on the initial CT prior to MRI in 58 % of the cases. Though OS BHAG are readily detectable on non-contrast CT (NC-CT), post-contrast CT (PC-CT) was generally necessary to identify TS BHAG. PC-CT shows a filling defect in the dural venous sinus that can draw attention to an otherwise unsuspected BH due to the herniated brain appearing isodense to the dural venous sinus on NC-CT.

All OS and SS BHAG arose from the cerebellar hemispheres except for two OS BHAG in one patient that were located supratentorially, arising from the occipital lobe. Interestingly, 55 % of TS BHAG arose infratentorially from

the SSL, while 45 % arose from the inferior surface of the temporal lobe. Herniation of the cerebellum into the undersurface of the TS is possible because AG principally develop on the cerebellar surface of the TS as reported by Le Gros Clark [5]. BHAG locations differed from previous reports, with the most frequent BHAG location being the OS followed by the TS, as opposed to the TS being the most frequent location in the radiological literature [21–27, 29]. Cerebellar herniation into TS AG was previously reported in the radiological literature by Battal et al. [29] in one case and by Chan et al. [24], though close inspection of their figures 1–3 suggests that this could have in fact been a case of ISL herniation into an OS AG. Wolbach [7] reported cerebellar herniations into AG of the lateral sinuses (transverse and sigmoid sinuses) in four out of the six autopsy cases described above. BHAG into LL SSS have been described by Wolbach [7] (three cases) and by Beneke, cited by Wolbach [7] (one case), but no reference was found in the radiological literature. Unlike other authors, we did not encounter BH into the sigmoid sinus or the torcular herophili [29], or occipital lobe BH into the TS [22, 29].

Multiple BHAG were a frequent finding in the present study, being identified in 58 % of the patients with a maximum of five BHAG in one case (patient 36). This is a higher rate than reported by Battal et al. [29] in the largest series up to day with 1 patient out of 20 (5 %) harboring multiple BHAGs. A case of three BHAG was also reported by Battal et al. [26] in their series of five patients. Wolbach [7], however, reports multiple BHAG in 100 % of his patients, with multiple BHAG being found both in the same location and in different locations at the same time.

Table 1 Patient demographics, clinical findings, BHAG characteristics, and PTCS

Patient number	Age	Sex	Clinical findings	Location of BH	BHAG/origin	SSCG	Dural enhancement of AG (OS only)	PTCS	PTCS signs on imaging, but no clinical signs of PTCS	Conditions possibly increasing intracranial pressure chronically (not including PTCS)
1	35	F	PTCS	L TS / L ITG R TS / R ITG		No No		Yes No	No No	No No
2	75	F	Depression / L1 fracture with post-traumatic epidural DAVF	L OS / L ISL		Yes	No	No	No	No
3	58	F	Vertigo	L OS / L ISL R OS / R ISL		Yes Yes	NC	No	No	No
4	19	M	Syncope	R TS / R ITG		No	NC	No	No	No
5	76	F	Left hemispheric border zone infarcts, severe left ICA stenosis	R OS / R ISL		No	NC	No	No	No
6	63	M	Frontal intraparenchymal sarcoid lesion	R OS / R ISL R OS / R ISL		Yes Yes	No No	No	No	Yes
7	80	F	Confused state, pulmonary embolism	R TS / R ITG R OS / R ISL and R SSL		No Yes	NC	No	Yes	No
8	76	F	PTCS, vertigo	R TS / R SSL L TS / L SSL		No No		Yes No	No	No
9	44	M	Head trauma, diffuse axonal injury	L OS / L ISL L OS / L ISL		No Yes	NC	No	No	No
10	50	F	VA dissection	L OS / LISL		No	Yes	No	No	No
11	74	M	Left MCA stroke, amyloid angiopathy	L OS / LISL		No	Yes	No	No	No
12	70	F	Multiple cardioembolic infarcts	L OS / LISL		No	No	No	No	No
13	56	M	Cranio-cerebral trauma	L OS / LISL		Yes	Yes	No	No	No
14	70	F	Squamous cell carcinoma base of tongue, cerebral metastasis and meningeal carcinomatosis	R OS / R ISL and R SSL		Yes	No	No	No	Yes
15	33	M	Spontaneous SAH of unknown origin	L TS / L LOTG and LITG		No		No	No	No
16	71	M	Frontal meningioma	L OS / L ISL R OS / R ISL		Yes No	Yes Yes	No No	No No	Yes Yes
17	44	F	Multiple sclerosis	L OS / L ISL R OS / R ISL		No No	No No	No	No	No
18	63	M	Parkinson's disease	L TS / L SSL L OS / L ISL		Yes Yes	NC	No	No	No
19	70	M	Right frontal anaplastic ependymoma	R OS / R ISL		Yes	Yes	No	No	Yes
20	57	M	Headache, high blood pressure	L OS / L ISL L OS / L ISL		Yes Yes	Yes Yes	No	No	No
21	73	F	T-Cell lymphoma (extracranial)	L TS / L ITG		No	Yes	No	No	No
22	74	M	Transient global amnesia	L OS / L ISL R OS / R ISL		No No	Yes Yes	No	No	No
23	34	F	Suspected transient ischemic attack	SS / R SSL L OS / L ISL		Yes Yes	NC	No No	No No	No No

Table 1 (continued)

Patient number	Age	Sex	Clinical findings	Location BHAG/origin of BH	SSCG	Dural enhancement of AG (OS only)	PTCS	PTCS signs on imaging, but no clinical signs of PTCS	Conditions possibly increasing intracranial pressure chronically (not including PTCS)
24	34	F	Suspected viral meningitis	L TS / L ITG R TS / R ITG L OS / L ISL R OS / R ISL	No No No No	Yes Yes	No No	No	No
25	85	F	Right frontal GBM	L TS / L SSL R TS / R SSL	Yes Yes		No No	No	Yes
26	65	F	Cognitive decline	L OS / L ISL R OS / R ISL	Yes Yes	NC	No No	No	No
27	50	F	Giant right clinoidal meningioma	L TS / L SSL	Yes		No	No	Yes
28	76	F	Right ACA stroke	L TS / L SSL L OS / L ISL	No No	NC	No No	No	No
29	58	F	Headache	L TS / L ITG L OS / L ISL	No Yes	Yes Yes	No No	No	No
30	70	F	Vertigo	R OS / R ISL	Yes		No	No	No
31	85	F	Left MCA stroke	L OS / L ISL R OS / R ISL	Yes Yes	Yes Yes	No No	No	No
32	78	M	Left sigmoid sinus DAVF and normal pressure hydrocephalus	R OS / R ISL L OS / L ISL L LLSSS / SFG L LLSSS / SFG	No No No No	NC No No No	No No No	No	Yes
33	75	M	Left MCA TIA	R OS / R OL R OS / R OL	No No	NC	No	No	No
34	77	F	Right cerebellar hypertensive hemorrhage	L LLSSS / SFG R LLSSS / SFG	No No	No No	No	No	No
35	75	F	Cognitive decline, parietal meningioma (8 mm)	L OS / L ISL R OS / R ISL	Yes Yes	No Yes	No No	No	No
36	55	F	Large left vestibular schwannoma with posterior fossa mass effect, hydrocephalus	R TS / R SSL L OS / L ISL L OS / L ISL L OS / L ISL R OS / R ISL	No No No No No	Yes Yes Yes Yes No	No No No No	No	Yes
37	67	F	Right central retinal artery occlusion	R OS / R ISL L TS / L SSL R TS / R SSL	No Yes Yes		No No	No	No
38	68	F	PTCS and torcular 3 cm meningioma invading R TS	L TS / L SSL	Yes		Yes	Yes	No

F female, M male, ACA anterior cerebral artery, AG arachnoid granulation, BH brain herniation, BHAG brain herniation into an AG, DAVF dural arteriovenous fistula, ICA internal carotid artery, ITG inferior temporal gyrus, ISL inferior semilunar lobule, LL .SSS lateral lacunae of the superior sagittal sinus, L left, LOTG lateral occipito-temporal gyrus, MCA middle cerebral artery, NC no contrast-enhanced MRI available, OL occipital lobe, OS occipital squama, PC-CT post-contrast CT, PTCS pseudotumor cerebri syndrome, R right, SFG superior frontal gyrus, SS straight sinus, SSSG signal and structural changes, SSL superior semilunar lobule, TIA transient ischemic attack, TS transverse sinus, VA vertebral artery

Table 2 Summary of BHAG found in the literature

Case	Authors year (ref.)	Case number	Age	Sex	Location of BH	Hemiated parenchyma	Presentation/clinical findings
Radiological literature							
1	Battal et al. 2015 [29]	1	21	M	Left TS	Left ITG	Treated left frontal AVM
2		2	24	F	Right TS	Right ITG	Headache, left hemifacial spasm
3		3	34	M	Left TS	Left ITG	Right cerebellar-brainstem chronic infarction
4		4	34	F	Torcular	Right cerebellar hemisphere	Left parietal diffuse astrocytoma
5		5	39	F	Torcular	Left cerebellar hemisphere	Vascular headache
6		6	52	M	Right TS	Right ITG	Vertigo, hypertension, right SNHL
7		7	11	M	Left TS	Left ITG	Headache
8		8	40	M	Torcular	Left cerebellar hemisphere	Headache, vertigo
9		9	20	M	Left TS	LITG	Vascular headache
10		10	71	F	Right TS	Right occipital lobe	Right parietal meningioma
11		11	67	M	Right TS	Right occipital lobe	Right temporal anaplastic meningioma
12		12	31	F	Left TS	Left cerebellar hemisphere	Headache
13		13	35	F	Torcular	Left cerebellar hemisphere	Multiple sclerosis
14		13	35	F	Left TS	Left occipital lobe	Multiple sclerosis
15		14	25	M	Occipital bone	Left cerebellar hemisphere	Right frontal focal cortical dysplasia, epilepsy
17		15	21	M	Left TS	Left ITG	Headache
18		16	51	F	Right TS	Right ITG	Headache
19		17	50	F	Occipital bone	Right cerebellar hemisphere	Neck pain
20		18	64	F	Occipital bone	Right cerebellar hemisphere	Left temporal meningioma
21		19	21	M	Right TS-SIS border	Right ITG and DVA	Syncope
22		20	38	F	Left TS	Left ITG	Headache, vertigo, left temporal arachnoid cyst
23	Asadi et al. 2015 [28]	1	51	F	Left TS	Left ITG	Syncope and confusion preceded by 3-week history of twitching of left face and arm
24	Kocyıgit et al. 2015 [25]	1	9	F	Right TS	Right ITG	Mild head trauma
25	Battal et al. 2014 [26]	1	11	F	Right TS	Right ITG	Growth retardation due to growth hormone deficiency
26		2	63	F	Occipital bone	Right cerebellar hemisphere	Hearing loss
27		3	19	M	Right TS	Right ITG	Syncope
28		4	56	M	Right TS	Right ITG	Headache and CSF leak/ethmoidal bone encephalocele
29		4	56	M	Left TS	Left ITG	Headache and CSF leak/ethmoidal bone encephalocele
30		4	56	M	Left SS	Left cerebellar hemisphere	Headache and CSF leak/ethmoidal bone encephalocele
33		5	68	M	Left TS	Left ITG	Chronic headache
34	Karatag et al. 2013 [23]	1	31	F	Left SIS	Left ITG	Headache/cerebellar ectopia
35	Coban et al. 2013 [27]	1	31	F	Left TS	Left ITG	5-day history of dizziness and imbalance
36	Chan et al. 2011 [24]	1	8	F	Right TS	Right cerebellar hemisphere	Severe traumatic brain injury
37	Kollar et al. 1998 [21]	1	56	F	Left TS	Left ITG	Left transverse DAVF with pulsatile tinnitus

Table 2 (continued)

Case	Anatomo-pathological literature Authors year (ref)	Case number	Age	Sex	Location of BH R lateral sinus ^c	Hemiated parenchyma Cerebellum	Duration of increased intracranial pressure Acute stroke for 13 days prior to demise	Findings at autopsy
38	Wolbach 1908 [7]	2	39	F	R lateral sinus ^c	Cerebellum	Signs of ICP for several years and bilateral hemianopsia for 2 years	Massive hemorrhage into right basal ganglia. <i>Histology:</i> large arachnoid villi containing at their bases, small masses of distorted cerebellar tissue
39		5	39	M	L lateral sinus ^c	Cerebellum	Signs of ICP for 5 years	Adenocarcinoma of pituitary body
40		6	37	M	L and R LSSS R lateral sinus ^c	Cerebrum Supratentorial ^a	Signs of ICP for 6 months	Base of the brain, the left side, small round cell sarcoma
41		7	50	M	L and R LSSS L lateral sinus ^c	Multiple from cerebrum Multiple from cerebellum	Signs of ICP for 6 months	Metastatic carcinoma; left side of cerebellum and superior frontal gyrus
42		8	52	M	Occipital sinus ^b R LSSS	Multiple from cerebrum Right cerebrum	Signs of ICP for 6 months	Glioma of right temporal lobe
43		9	39	F	L and R lateral sinus ^c SSS	Left and right cerebellum Multiple from cerebrum	Signs of ICP for several years	Glioma, infiltrating and involving the median portions of both frontal lobes
44	Beneke, 1898 (cited by Wolbach 1908 [7])	1	?	?	SSS or LSSS	Multiple from cerebrum	Signs of ICP for about 1 year	Choroid plexus papilloma with hydrocephalus. The hernias consisted of brain tissue in various stages of disorganization, some containing vessels of considerable size, which were carried along from their original positions

AVM arteriovenous malformation, *CSF* cerebrospinal fluid, *DAVF* dural arteriovenous fistula, *DVA* developmental venous anomaly, *ICP* increased intracranial pressure, *ITG* inferior temporal gyrus, *LLSSS* lateral lacunae SSS, *SIS* sigmoid sinus, *SSS* superior sagittal sinus, *TS* transverse sinus

^aNot specified

^bPresumably a persistent occipital sinus

^cWolbach uses the term lateral sinus to name conjointly the transverse and sigmoid sinuses

In all but one patient, patients died with signs of or from longstanding increased intracranial pressure that could account for the higher number of BHAG encountered by Wolbach.

Physiopathological considerations

The mechanism by which the brain may herniate into an AG likely responds to a combination of anatomical and hydrodynamic (mechanical) factors. As mentioned earlier, AGs originate in the arachnoid membrane as arachnoid villousities. As AG grow due to the continuous effect of CSF pulsations, they invaginate through points of dural weakness, dura mater defects, and/or points of venous convergence, drawing the adjacent pia mater from the surface of the brain into the AG [2, 7, 32]. A pia-arachnoid bridge is thus formed, constituting a potential scaffold through which the brain can be pulled into the AG. The same pulsatile forces that drive the growth and progressive invagination of AG into the dura and calvarium may act on the brain resulting in BHAG. In the case of TS BHAG arising from the temporal lobe BHAG, the temporal lobe lies in intimate contact with the dural venous sinus, likely favoring the herniation. This is not the case in TS BHAG arising from the cerebellum or the SS BHAG (Figs. 2 and 3). Pre-existing pia-arachnoid bridges between the AG and the brain could explain how the cerebellum may herniate cranially into the TS and SS AG, seemingly against gravity and CSF pulsations. In the same line of thought, Wolbach [7] states, “The more compact layers of the pia and arachnoid extend upwards into the villus, around the periphery of its base. This arrangement practically makes a break in the pia arachnoid covering of the brain and probably is of mechanical importance in the formation of hernias”.

Signal and structural changes (SSCG) of the HB were frequently observed (46 % of BHAG). In all cases, SSCG involved cerebellar tissue, with no cases of supratentorial SSCG being found. Progressive thinning of the herniated cerebellum towards the AG and variable degrees of distortion of the cerebellum around the stem of the BH were observed (Figs. 2 and 5). We found no mention of SSCG of BHAG in the radiological literature. Distorted cerebellar tissue herniating into the lateral sinus (transverse and sigmoid sinuses) was, however, observed by Wolbach [7], and Beneke, quoted by Wolbach [7], found that “the hernias consisted of brain tissue in various stages of disorganization”. These autopsy findings could correspond to the SSCG encountered in cerebellar BHAG in our study.

Tethering of the herniated cerebellum by pia-arachnoid bridges or strangulation of the BH at the neck of AG or a combination of both could account for SSCG and distortion of the cerebellum. Tethering of the cerebellum is likely favored by it being suspended from the dura, rather than resting on it, as is the case for the undersurface of the temporal lobe. In addition, continuous downward forces pulling the cerebellum away from its dural attachments could further enhance tethering. Indeed, no SSCG were observed in TS BHAG

arising from the inferior surface of the temporal lobe, where, because the temporal lobe lies against the dural venous sinuses, tethering is unlikely. Finally, inflammatory processes leading to fibrous tissue within the AG contributing to strangulation and tethering cannot be discarded, in keeping with the progressive fibrosis observed in mature AG [2]. Enhancement of the dural lining of the AG was found in up to 68 % of the cases where volumetric Gd-enhanced T1 studies were available. Although in some cases the dural enhancement around BHAG was linear and as intense as in normal dura, in other cases, enhancement was very conspicuous (Fig. 4) suggesting inflammatory changes. Alternatively, this conspicuous dural enhancement could represent focal pachymeningitis secondary to the dura being pulled by the pia-arachnoid bridges and BH attaching it to the brain surface.

BHAG as a cause of neurological symptoms

A cause to effect relation between BHAG and neurological symptoms is difficult to ascertain in both the present study and previous reported cases, with the majority of presenting conditions being unrelated to BHAG (see Table 1).

In the three patients with PTCS of our series, venous outflow obstruction from stenosis caused by the BHAG could have contributed to raising CSF pressure. Two of these patients evolved favorably under conservative medical treatment; therefore, no TS stenting was contemplated and no TS pressure measurements were performed. The hemodynamic significance of the stenosis was, therefore, not quantified. The third case of PTCS was recently diagnosed and has not yet returned for follow-up. Whether the BHAG were the cause of the PTCS or the result of raised CSF pressure is impossible to determine in the absence of imaging studies prior to the development of PTCS. BHAG into the TS could, however, contribute to the PTCS by causing a venous outflow obstruction in both situations. BHAG into the TS from CSF hypertension could accentuate the stenosis and further raise the venous pressure, thus perpetuating and worsening a pre-existing PTCS. The question of whether BHAG into a dural venous sinus is a contraindication to stenting in cases of refractory PTCS with pressure gradient across the stenosis must be raised. Compression of viable HB by the stent could be of concern in cases where the neck of the AG is narrow.

In the published radiological literature, there is no specific mention of patients with PTCS and venous outflow obstruction caused by BHAG. Battal et al. [26] describe a patient with a medical history of spontaneous CSF leak presumably from a ethmoidal encephalocele, who carried two TS temporal lobe BHAG, one sigmoid sinus left cerebellar hemisphere BHAG and bilaterally hypoplastic transverse sinuses; though not specifically mentioned, PTCS would be a reasonable assumption in this case.

A relation with headaches is suggested by Battal et al. [29] as patients with headaches in their series of BHAG were higher than the general population, though this difference did not reach statistical significance. Coban et al. [27] describe a case of a 31-year-old patient with a 5-day history of dizziness, imbalance, and pressure sensation in the left ear and suggested that symptoms were caused by a left TS BHAG from the inferior temporal lobe. There is no mention of how the patient evolved clinically. A cause to effect relation is therefore difficult to establish for this case.

Finally, SSCG were frequently observed in the present study, but none involved frontal, temporal, or occipital lobe BH, all SSCG being exclusively found in the cerebellum. Supratentorial BH with SSCG could theoretically cause seizures. Indeed, temporal lobe encephaloceles have been shown to present with seizures as reported by Saavalainen et al. [33] in a series of 23 patients of which 12 underwent surgery. Histopathological examination of the resected herniated brain showed gliosis in all cases and mild cortical laminar disorganization in 42 %. These changes could, when imaged, correspond to SSCG observed on MRI in our study. One could posit that SSCG are necessary for BH to cause seizures, with the reserve that strangulated herniated brain in an AG could be sufficient to induce seizures and that BH without SSCG on imaging findings does not exclude minor gliotic changes and mild cortical laminar disorganization.

Clinical conditions leading to the formation of BHAG

In the present series, four patients had imaging findings of PTCS (three with clinical signs of PTCS, one without) corresponding to 10.5 % of the cases. A 10.5 % incidence of PTCS/“burnt out PTCS” in the present series is significantly higher (P value <0.0001) than the 0.9/100,000 annual incidence of PTCS in the general population [34]. Eight additional patients (21 %) had or had had intracranial conditions potentially causing chronically raised intracranial pressure (Table 1). Put together, 32 % patients (8/38) had or may have had increased intracranial pressure. In the series by Battal et al. [29], 20 % of the patients presented with intracranial mass lesions that could have led to increased intracranial pressure. In the autopsy cases reported by Wolbach and Beneke, all patients had died with signs of or from increased intracranial pressure from various conditions, and presented with multiple BHAG each. Overall, these findings suggest that increased intracranial pressure may bring about the formation of BHAG, though it is not a prerequisite. Indeed, given that the majority of the patients with BHAG do not have increased intracranial pressure, the chronic effect of CSF pulsations and pia-arachnoid bridges described above may generally suffice for BHAG to develop.

Limitations of the present study

The present study carries limitations inherent to all retrospective studies. Discrepancies exist between our study and previously reported cases, mainly concerning the location of BHAG, the origin of the BH, and the presence of SSCG that have not previously been reported. BHAG locations not reported in the study may have been missed given the setup of the study (keyword search).

Conclusion

BHAG represent a form of acquired meningo-encephaloceles. They occur, by order of frequency, in the OS, the TS, the LLSSS, and the SS, though other locations have been described in the literature. When located in a dural venous sinus, BHAG must be distinguished from thrombosis or other meningeal base processes such as meningioma. SSCG of the herniated cerebellum (but not of the temporal lobes) were frequent findings and possibly result from tethering and/or strangulation of the cerebellar tissue in the AG. No clinical symptoms could be clearly attributed to BHAG, though in our cases of venous PTCS, TS BHAG could have contributed to sustaining the raised CSF pressure. Various factors are likely involved in the development of BHAG, including normal pia-arachnoid bridges between the brain surface and the AG and hydrodynamic constrains on the brain and AG. Raised intracranial pressure may favor the development of BHAG. Scrutiny for imaging and clinical signs and conditions leading to raised intracranial/CSF pressure is, therefore, recommended when BHAG is discovered.

Compliance with ethical standards We declare that while all patients gave informed consent for imaging studies, due to the retrospective nature of this work, Institutional Review Board approval was not required.

Conflict of interest We declare that we have no conflict of interest.

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