



Review Article

The spectrum of venous anomalies associated with atretic parietal cephaloceles: A literature review

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ABSTRACT

Background: Parietal atretic cephalocele (PAC) is a small, subscalp lesion with underlying extracranial meningeal, neural, and glial tissues. In this paper, we analyze the related literature on the continuum of PAC-associated venous anomalies and report an exemplary case.

Methods: The PubMed Medline database was searched using the following search algorithm: (Atretic encephalocele) OR (Rudimentary meningocele,) OR (Atypical meningocele) OR (Meningocele manqué) OR (Meningeal heterotopia). Only papers detailing the venous anomalies associated with PACs have been included.

Results: A total of 30 papers in our search documented PAC-associated venous abnormalities. The overall number of cases reported was 68 (including our exemplary case). The most frequently identified associated venous anomaly was the presence of a “fenestrated superior sagittal sinus” recorded in 48.5% of cases ($n = 33$), followed closely by “persistent falcine sinus” in 47% ($n = 32$) and vertical embryonic positioning of the straight sinus (SS) in 44% ($n = 30$). The complete absence of a SS was reported in 39.7% ($n = 27$) and various anomalies of the Galenic system were reported in 26.8% of cases ($n = 12$).

Conclusion: Although benign in nature, PACs are often a marker for the presence of complex and variable cerebral venous malformations, requiring extensive preoperative imaging workup for both the superficial and deep venous systems to obtain an accurate understanding of the anatomy of the venous system and guide surgical planning.

Keywords: Parietal, Atretic cephalocele, Venous anomalies

INTRODUCTION

A “cephalocele” is a congenital herniation of intracranial contents through defects in the dura and cranium.^[30] There are four classes of cephaloceles, namely, meningoceles, meningoencephaloceles (encephaloceles), meningoencephalocystoceles (with parts of the ventricles), and atretic cephaloceles (ACs).^[6]

ACs differ from true cephaloceles by the presence of dural remnants, fibrous tissue, as well as dysplastic neuronal tissue and are thought to represent involuted true cephaloceles (encephaloceles and meningoceles).^[22] ACs are rare lesions, accounting for 1% of all

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cerebrospinal congenital anomalies and 37.5–50% of all cranial cephaloceles.^[37] ACs are further subdivided into “parietal” and “occipital” forms, in relation to their location adjacent to the vertex or the external occipital protuberance, respectively.^[2]

The concept of atretic parietal cephaloceles (APCs) as a separate entity was first investigated by Yokota *et al.*^[37] who defined them as small, skin-covered, noncystic, nodular, or flat vertex midline lesions. Parietal atretic cephaloceles (PACs) are distinct in their frequent associating midline cerebral and venous malformations that govern their presentation, management, and prognosis.^[37] The description of these venous malformations becomes blurred when it comes to occipital cephaloceles, considering the higher degree of anatomical venous variations possible in their vicinity.

A wide range of APC-associated venous anomalies has been reported using the magnetic resonance venography (MRV) techniques. In this review, we intend to examine the available literature on the venous anomalies that may coexist with APCs. Our second objective is to report a new venous anomaly associated with an adult PAC presented in our exemplary case.

MATERIALS AND METHODS

The PubMed Medline database was searched using the following search algorithm: (((atretic cephalocele [Title/Abstract]) OR (Rudimentary meningocele, [Title/Abstract])) OR (Atypical meningocele [Title/Abstract])) OR (meningocele manqué [Title/Abstract])) OR (meningeal heterotopia [Title/Abstract]). No study type or time restrictions were applied. The initial search yielded a total of 85 articles. The abstract screening was used to identify articles reporting on APC. This was followed by a detailed review to include only papers describing the associated venous anomalies if present. Besides, the reference section of these papers was screened for relevant citations. Paper identification was done by two independent reviewers. Any discrepancy was resolved by discussion or the opinion of a third reviewer. Information extraction was based on the following parameters: study characteristics (author, year), patient age, concurrent venous anomalies, management approach (conservative vs. surgical), and patient outcomes [Table 1].

RESULTS

Our literature review yielded a total of 85 articles reporting on APCs.^[1,4,6,8,-17,20,24-30,32,33,35-37,39] Only those papers detailing the venous anomalies associated with APCs were included, for an overall of 30 articles [Table 1]. The total number of reported cases was 68 (including the exemplary case). The age range of the patients was 1 day–38 years, of whom 88%

($n = 62/68$) belonged to the pediatric age group, and only 6 cases (12%) of adult PAC were reported. Two cases were diagnosed by second-trimester ultrasound.

The most commonly described associated venous anomaly was the presence of a “fenestrated superior sagittal sinus” (SSS) reported in 48.5% of the cases ($n = 33$), followed closely by “persistent falcine sinus” (PFS) in 47% ($n = 32$) and vertical embryonic positioning of the straight sinus (VEP SS) in 44% ($n = 30$). The complete absence of the sagittal sinus (SS) was reported in 39.7% ($n = 27$) and various abnormalities of the Galenic system were described in 26.8% of cases ($n = 12$). Our exemplary case was the first to report an enlarged vein of Trolard.

Other reported venous malformations were partially absent SS and duplicated SSS, collectively reported in 4.4% of the cases ($n = 3$).

Of all cases, only 38% underwent surgical excision ($n = 26$), while the remaining 62% ($n = 42$) were managed conservatively. Outcomes were reported for 35.3% ($n = 24$) patients. Of those, the majority (83%, $n = 20/24$) had good outcomes, with “mild” neurological deficit, recurrence, and death occurring in 4.2%, 8.3%, and 4.2% ($n = 1/24, 2/24, \text{ and } 1/24$), respectively.

Exemplary case

A 25-year-old male presented to the outpatient clinic having with a “scalp bulge.” On examination, the patient had a painless, small-sized midline mass situated in the parietal area. On palpation, the lesion was round, hard in texture, and fixed to surrounding structures, nontender, with intact overlying skin. The lesion was first noticed when the patient was 4 years old and has remained unchanged.

Brain computed tomography (CT) and magnetic resonance imaging (MRI) scans were ordered revealing a calvarium bifidum occupied by a heterogeneous cystic lesion with dysplastic brain parenchyma located in the frontal convexity

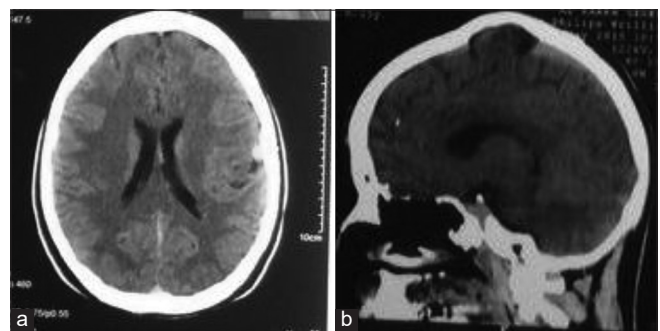


Figure 1: (a) Brain CT scan showing an enlarged left parietal cortical vessel, (b) sagittal brain CT scan showing a bulging at the vertex with protrusion of brain parenchyma inside and a cut in the SSS. CT.

Table 1: The spectrum of APC-associated venous anomalies.

Authors	Year	Number of cases	Age range	Associated venous anomalies	Management	Outcome
Inoue <i>et al.</i> ^[13]	1938	3	6 m–7 y	• Long vein of Galen (3)	Surgical excision	Good
Patterson <i>et al.</i> ^[28]	1998	8	1 d–3 y	• VEP SS (3) • VEP SS (6) • Fenestration of SSS (4)	Surgical excision	6 Normal 1 with mild motor delay 1 died at 3 y
Martínez-Lage <i>et al.</i> ^[20]	1997	1	38 y	VEP SS	Conservative	N/A
McLone and De Leon ^[24]	1998	1	7 m	Abnormal Galenic system	N/A	N/A
Brunelle <i>et al.</i> ^[4]	2000	27	N/A	• Persistent falcine sinus (PFS) (27) • Absent straight sinus (23) • Fenestrated SSS (23) • Absent vein of Galen (4)	N/A	N/A
Morioka <i>et al.</i> ^[25]	2009	3	1–18 y	VEP SS	Surgical excision (2) Conservative (1)	N/A
Yoshida <i>et al.</i> ^[38]	2006	1	13 y	VEP SS	Surgical excision	N/A
Kim <i>et al.</i> ^[15]	2006	1	11 y	• PFS • Abnormal Galenic system • Absent straight sinus	Surgical excision	Good
Wong <i>et al.</i> ^[35]	2010	1	6 y	VEP SS	Surgical excision	Good
van Laak <i>et al.</i> ^[34]	2010	1	4 m	VEP SS	Conservative	N/A
Kumar <i>et al.</i> ^[16]	2010	1	4 y	VEP SS	N/A	N/A
Şengöz <i>et al.</i> ^[30]	2011	1	23 y	• PFS • Abnormal Galenic system • Abnormal straight sinus	Conservative	N/A
Hsu <i>et al.</i> ^[12]	2011	1	16 y	• PFS • Partial absence of SS	N/A	N/A
Yilmaz <i>et al.</i> ^[36]	2011	1	36 y	VEP SS	Surgical excision	Recurrence
Muralidharan <i>et al.</i> ^[27]	2013	1	22 m	VEP SS	Conservative	N/A
Leykamm <i>et al.</i> ^[17]	2013	1	1 d	• VEP SS • Fenestrated SSS	Surgical excision	Good
Siverino <i>et al.</i> ^[31]	2015	1	3 y	• VEP SS • Fenestrated SSS • Abnormal Galenic system	Surgical excision	Good
Bick <i>et al.</i> ^[3]	2015	1	6 m	VEP SS	Surgical excision	N/A
Anand <i>et al.</i> ^[11]	2015	1	4 w	VEP SS	Surgical excision	Good
Ertuğrul <i>et al.</i> ^[8]	2015	1	25 y	VEP SS	Surgical excision	Recurrence
Kumar <i>et al.</i> ^[14]	2016	1	1 y	• VEP SS • Fenestrated SSS	N/A	N/A
Gulati <i>et al.</i> ^[11]	2016	1	6 m	• VEP SS • Fenestrated SSS	Surgical excision	Good
Santos <i>et al.</i> ^[29]	2016	1	22 w (intrauterine)	• VEP SS	Surgical excision	Good
Demir <i>et al.</i> ^[6]	2016	1	47 y	• Fenestrated SSS	Conservative	N/A
Murakami <i>et al.</i> ^[26]	2017	1	1 m	• VEP SS • SS and SSS deviated to right	Surgical excision	Good
Gagliardo <i>et al.</i> ^[10]	2018	1	Newborn	• VEP SS • Duplicated SSS	Conservative	Good
Siverino <i>et al.</i> ^[31]	2015	1	23 w (prenatal)	VEP SS	Surgical excision	Good
Tsitouridis <i>et al.</i> ^[32]	2005	2	2 m 8 m	• PFS • Absent SS	N/A	N/A
Favoreel <i>et al.</i> ^[9]	2015	1	1 y	Abnormal Galenic system	N/A	N/A
Present case	2020	1	25 y	• Fenestrated SSS • Enlarged vein of Trolard	Conservative	Good

VEP SS: Vertical embryonic positioning of the straight sinus, SS: Straight sinus, SSS: Superior sagittal sinus, PFS: Persistent falcine sinus, APC: Atretic parietal cephalocele

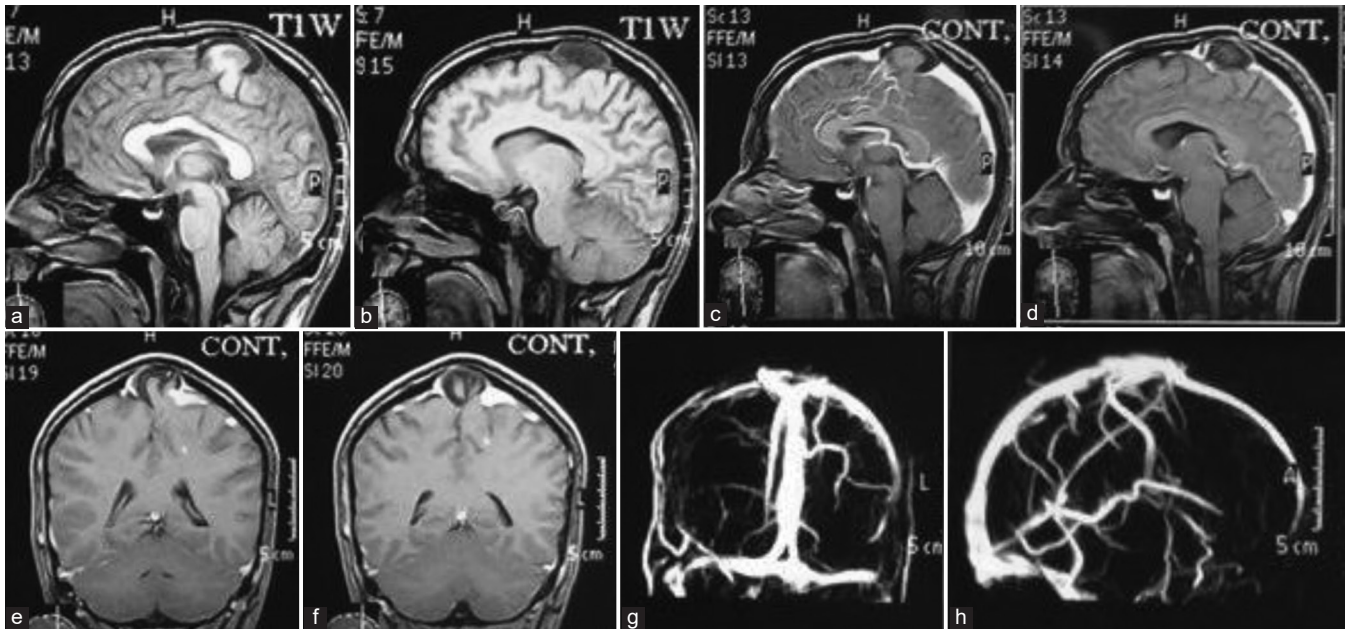


Figure 2: Brain MRI: (a and b) T1-weighted images MRI (a) revealed a midline parietal lesion with brain parenchyma protrudes inside, (b) isolated cyst like lesion. (c and d) T1 with contrast showed cut in the superior sagittal sinus SSS with enlarged draining veins drains to the left side of SSS, and a midline parietal mass. (e and f) Coronal section of T1-weighted MRI with contrast show fenestration of SSS with brain parenchyma inside the mass. (g and h) MRV reveals abnormalities and fenestration of SSS by enlarged left cortical vein arising from convexity ending in SSS with initial suspicion of DAVF. MRI: Magnetic resonance imaging, SSS: Superior sagittal sinus, MRV: Magnetic resonance venography, DAVF: Dural arteriovenous fistula.

[Figures 1 and 2]. The MRV revealed an anomalous vein, along with a focal indentation of the SSS on the left side, suggesting a vascular malformation. Next, a CT angiogram (CTA) was done, revealing a large oval filling defect in the middle third of the SSS (about 44×22 mm) with some opacified intrinsic filling vessels. The CTA also showed a grossly dilated left cortical parietal vein extending from the Sylvian fissure to open in the SSS at the site of the filling defect [Figure 3]. At this point, type 1 dural arteriovenous fistula (DAVF) and a PAC with associated venous anomalies were the two main differentials.

A digital subtraction angiography was performed which excluded DAVF and the diagnosis of an enlarged vein of Trolard associated with fenestration of the SSS in the setting of a small atretic parietal encephalocele was made [Figure 4]. The patient was managed conservatively. His follow-up MRI scans at 6 months and 2 years showed no new findings.

DISCUSSION

Nomenclature and classification of ACs

Alternative terms that have been used to describe ACs include meningocele manqué, abortive, rudimentary, occult, sequestered, atypical meningocele, and atypical heterotopia.^[6] Although these terms have been used

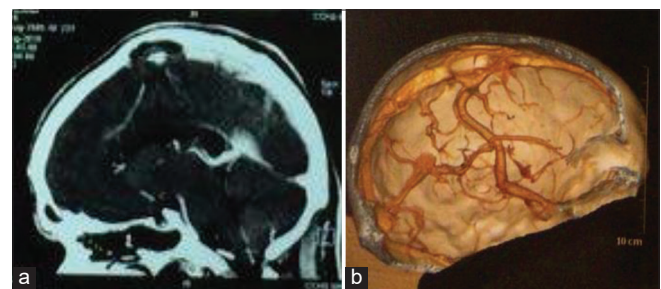


Figure 3: Cerebral CT angiography (a) sagittal section: midline mass with surrounding vascularity and cut in the SSS. (b) 3D-reconstructed image showed an enlarged vein arising from Sylvian fissure, crossing over the parietal cortex, and end in SSS. CT: Computed tomography, SSS: Superior sagittal sinus.

interchangeably, differences between them do exist, as identified by Lopez *et al.*^[18]

Martinez-Lage *et al.* classified ACs into two types. Type 1, which is limited to the stalk of the lesion, contains arachnoid tissue as well as tangles of anomalous blood vessels and is covered by hairy skin. Type 2 extends to the dome of the lesion and consists of meningeal tissue intermingled with dermal and fibrous tissue, as well as clusters of anomalous blood vessels, extending as a net, and ectopic neural or glial element.^[20-22] For the majority of cases, no pathological data were available and this parameter was hence excluded from the final analysis.

Evolving terminology for atretic parietal cephalocele (APC)-associated venous anomalies

The cooccurrence of APCs with venous malformations was first described by McLaurin *et al.* in 1964 who described anomalies such as fenestrated SSS and VEP SS.^[23]

Typical angiographic features of VEP SS include elongated internal cerebral veins that join a small single vein the cistern (vein of Galen), which coalesces with a shortened vertically positioned SS in the falx to join the SSS.^[26] Before the advent of CISS and 3T-T2R MRI sequences, the detailed course of these anomalies was less clearly delineated. For example,

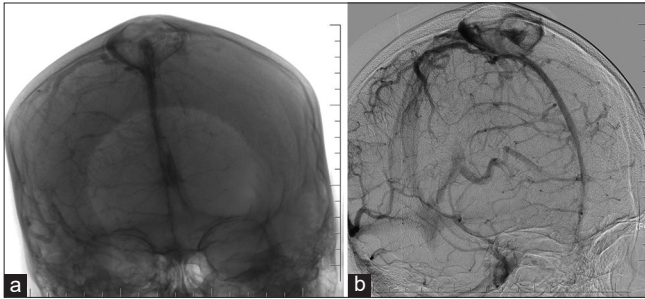


Figure 4: (a and b) Digital (a) and native (b) images of the cerebral angiogram show fenestration of the superior sagittal sinus due to interposition of the encephalocele also causing a bony defect in the convexity with enlarged vein of Trolard on the left side.

the elongated ICVs were referred to as an “elongated vein of Galen.” In other studies, conventional MRI images failed to show the SS, and thus, “absent SS with an anomalous Galenic system” was the reported anomaly.

Etiopathogenetic and embryonic correlations of APCs and their associated venous anomalies

Both the etiology and embryological basis of APC and their associated venous anomalies are currently a source of controversy. Both genetic and environmental factors are thought to play a role in the pathogenesis of AC, including Vitamin A, teratogens, X-ray, folic acid antagonists, trypan blue, triamcinolone, and malnutrition.^[20-22] Familial cases of ACs have also been rarely reported, although an exact genetic marker has not been identified.^[5,19,39]

As to their embryonic origin of the APCs, several theories have been proposed, including failure of neural tube closure, normal closure followed by an abnormal reopening of the neural tube, partial regression of an intrauterine meningoencephalocele, a sequel of a primary mesenchymal injury, the persistence of neural crest remnants, or a nuchal bleb.^[3,5,7,21,33] [Figure 5]. The presence of both skin and neural defects suggests a shared embryonic origin for the malformations since both tissues are derived from the ectoderm.

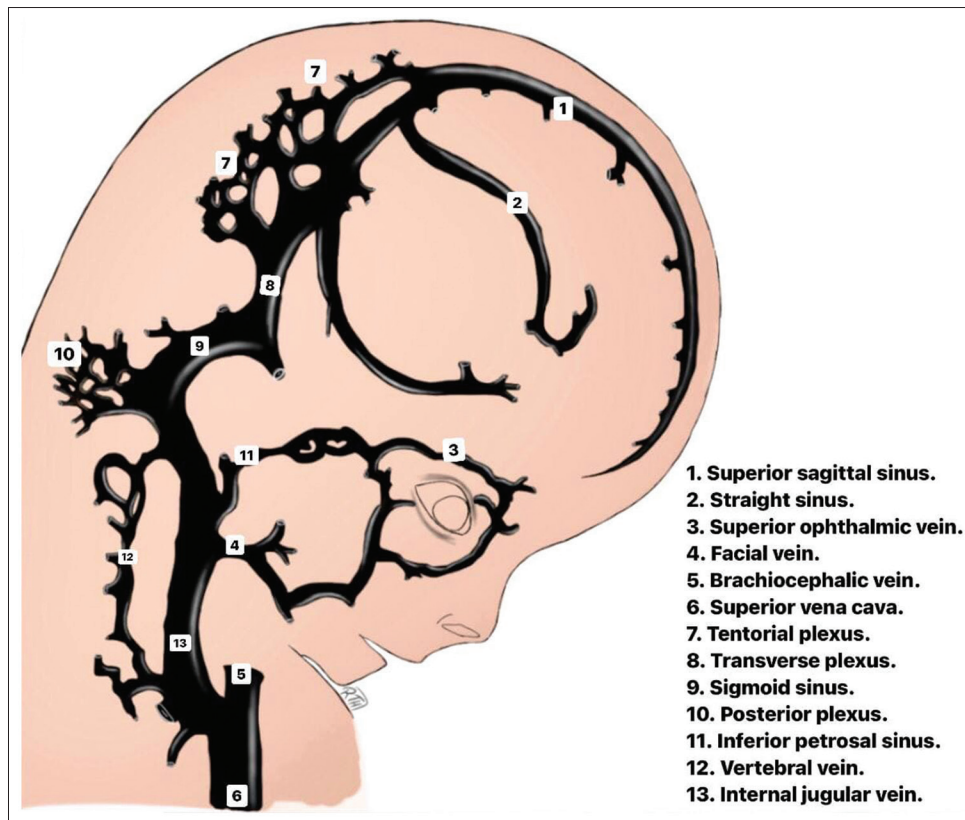


Figure 5: The embryologic pattern of the intracranial venous system.

Fenestration of the SSS was the most commonly reported APC-associated venous anomaly. The SSS forms during the 35–50 gestational days from the fusion of the marginal sinuses. The presence of the AC and its associated fibrous tracts is thought to interrupt this process, resulting in splitting or “fenestration” of the SSS.^[31]

Surgical perspective

APCs typically present as a skin-covered midline scalp mass.^[35] Clinically, most patients with atretic encephaloceles are asymptomatic and usually discovered incidentally. Surgery is advised for esthetically unpleasant lesions, those with, or at high risk of, ulceration or rupture due to their prominent location, masses causing headache due to stretching of the dura, and lesions requiring histological diagnosis.^[35]

Preoperative imaging of both the superficial and deep venous systems is mandatory in cases of APCs, given the high incidence of associated venous anomalies. Interestingly, while these venous anomalies rarely necessitate formal intradural exploration in pediatric practice, intradural extension was required in our case, raising the question of whether these anomalies progress over time and, thus, the importance of surgery timing. The surgery typically involves excising of the mass with cranioplasty, without intervening with the venous anomalies, as they participate in normal venous drainage. In the absence of other intracranial anomalies, the prognosis of APCs is generally good.^[31]

Study limitations and future research directions

One limitation of this review is that the majority of the studies were single case reports or small series, with limited data on patient outcomes. For a robust conclusion on the prognostic significance of specific venous anomalies, larger multicenter, outcome-oriented studies are required. Furthermore, more angiographic studies that could better delineate the anatomy of the associated venous anomalies and address the inconsistencies in the terminology used to describe them are the way forward toward a better understanding of APCs.

CONCLUSION

Although benign in nature, PACs are also a marker for the presence of a spectrum of cerebral venous malformations of which the neurosurgeons and neuroradiologists should be vigilant. When PAC is suspected, a comprehensive preoperative imaging workup for both the superficial and deep venous systems is, therefore, required to obtain an accurate understanding of the course of the venous system and use these data to inform surgical planning.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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