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Sinus pericranii: diagnostic and therapeutic considerations in 15 patients

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Abstract
Introduction Sinus pericranii (SP) is a rare, usually asymptomatic condition characterized by a large communication between the intra- and the extracranial venous drainage pathways in which blood may circulate bidirectionally through dilated veins of the skull. We describe our diagnostic and therapeutic experience with SP, with a special focus on the vascular analysis of digital subtraction angiography (DSA).
Methods DSA images of 15 patients were evaluated with regard to the delay in opacification of the scalp vessels, the absence or distortion of the superficial cortical veins in the vicinity of the SP, the drainage patterns of the superior sagittal sinus, and the degree of maturation of the venous outlets of the brain. SP were classified either as "dominant", if the main stream of contrast flow used the SP to drain the brain bypassing usual venous outlets, or as "accessory", if only a small part of the venous outflow occurred through the extradiploic vessels.
Results All patients presented with a nonpulsatile, soft-tissue mass. The lesion was on the midline in 14 of 15 patients, frontal in 12 patients, and parietal in 2 patients. In 13 patients, associated intracranial venous anomalies were present, eight of which were developmental venous anomalies. Seven patients had a dominant SP, and eight an accessory SP.
Conclusion SP can be considered the cutaneous sign of an underlying venous anomaly. If treatment is contemplated, analysis of the drainage pattern of the SP has to be performed. Treatment should be avoided in dominant SP or if its accessory role constitutes the only collateral pathway of an underlying venous anomaly.

Keywords Sinus pericranii · Developmental venous anomaly · Venous malformation · Emissary vein

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Introduction

Since its first description in 1850 [1], many different definitions of the so-called sinus pericranii (SP) have been proposed. Stromeyer, who coined the term in 1850 described SP as a "blood bag on the skull, in connection with the veins of the diploe and through these with the sinuses of the brain" [1]. This definition refers to SP as a simple outpouching of an intracranial sinus through a defect in the skull and has been adopted by some authors in the recent literature describing SP as a venous pouch that receives blood from and drains into the intracranial sinuses [2–8]. In distinction to this "cul-de-sac" definition, other

authors have defined SP as a communication between the intra- and the extracranial venous systems (i.e. comparable to an emissary vein) [9–11]. This definition was first introduced in 1936 when Fevre and Modec distinguished three different types of SP: (1) the closed system, which in fact is the cul-de-sac type described above, (2) the transcranial collateral which involves a communication between the intra- and extracranial venous systems, and (3) the anomaly draining intracranially [12]. We propose the following definition of SP: an emissary vein (in terms of its transosseous disposition and associated diploic drainage) with an increased subgaleal drainage (instead of an interperiostodural, i.e. sinus, drainage). In this definition, SP is regarded as a venous anomaly in which the communication between the intra- and the extracranial venous systems is not constituted by small anastomotic diploic (i.e. emissary) veins but by a network of thin-walled veins that form a varix on the external table of the skull. This varix is continuous with the pericranial veins of the scalp [13, 14].

Most SPs become clinically apparent as nonpulsatile soft-tissue masses that are located in the frontal region along or close to the midline and connect pericranial veins with the superior sagittal sinus (SSS) through a bony defect [15]. Off-midline locations have been described but are extremely rare [4, 16, 17].

We describe here our diagnostic and therapeutic experience in 15 patients with SP, with the following aims:

1. To propose a pretreatment assessment questionnaire to select patients for treatment of SP.
2. To establish the role of digital subtraction angiography (DSA) in the preoperative (either surgical or endovascular) assessment.
3. To propose a treatment strategy for the safe management of the anomaly, based on a new appraisal of SP as a cutaneous stigma (constituting a variant and not a disease) of an underlying intracranial vascular anomaly, which may range from simple varicose ectasia of the intradiploic veins (i.e. isolated SP), through developmental venous anomalies (DVA), to complex venous malformations.

Materials and methods

Patients were selected after a retrospective search through the databank of our hospital from 1989 to 2005 employing the search term “sinus pericranii”. The search revealed 15 patients (8 females and 7 males) whose age at presentation to our service ranged from birth to 19 years. Their clinical files and their imaging data were reviewed.

DSA images of the SP were evaluated with regard to the delay in opacification of the scalp vessels, the absence or distortion of the superficial cortical veins in the vicinity of the SP, the drainage patterns of the SSS, and the degree of maturation of the venous outlets of the brain. SP were classified either as (1) dominant, if the main stream of contrast material flow used the SP to drain the brain, bypassing usual venous outlets, or as (2) accessory, if only a small part of the venous outflow occurred through the extradiploic vessels (Fig. 1).

Therapeutic choices were made based on the clinical and angiographic features of the SP, and the wishes of the patient. A clinical–neuroradiological questionnaire was developed that is presented in the Appendix. Only the most illustrative cases are described here; the others are briefly summarized in Table 1.

Results

Clinical features

All 15 patients presented at birth with a nonpulsatile, soft-tissue mass without a thrill. There were no patients with multiple SP. The lesion was located in the midline in 14 of 15 patients, and was frontal in 12 patients and parietal in 2 patients; in only one patient was the lesion in a parietal off-midline location. All SP were located cranial to the occipital bone and were in close spatial relationship to the midline sutures (from the metopic suture to the interparietal suture). Associated diseases were meningocele, oesophageal atresia, and a cerebrofacial arteriovenous metameric syndrome (CAMS-III) in one patient each; two patients presented with a facial haemangioma. In 13 patients, associated intracranial venous anomalies were present; these included solitary DVA (present in 8 patients), vein of Galen hypoplasia (in 2 patients), vein of Galen aneurysmal malformation (VGAM), dural sinus malformation (DSM) and an intraosseous AVM in one patient each.

Imaging features

In three patients ultrasonography was performed. Here a venous vessel crossing the hyperechoic cranial vault indicated the diagnosis of SP. Plain radiographs in two patients demonstrated a bony defect that was located in both patients on the midline (Figs. 1 and 2). On CT (nine patients) the SP was identified by the bony defect (present in all investigated patients), the midline location (all but one patient) and the vessel crossing this channel (Fig. 3).

MRI was performed in all 15 patients and revealed the venous varices typically as a flow void on T2-weighted images crossing the skull, and an associated DVA could be well depicted on contrast-enhanced studies.

On angiography (DSA), the venous system and the exact role played by the SP in the venous drainage could be

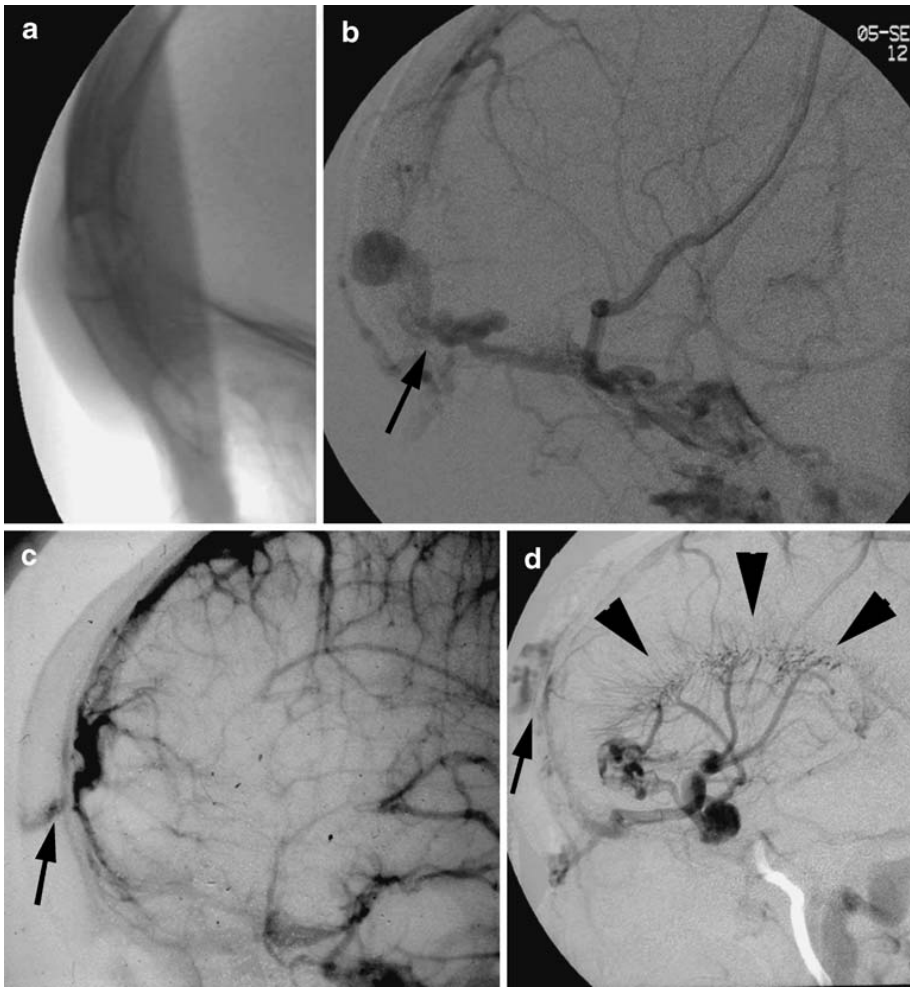

Fig. 1 Different types of dominant and accessory SP. **a** Bony defect demonstrated on a plain radiograph. **b** The main stream of contrast material flow uses the SP (*arrow*) to drain the brain bypassing the usual venous outlets (dominant SP, no therapy). **c** Only a small part of the venous outflow occurs through the extradiploic vessels, and all other sinuses are patent and mature (accessory SP). **d** Although the normal sinuses are patent and drainage is directed via those channels, the large DVA (*arrowheads*) uses the SP as the sole drainage pathway. Although the SP is accessory in terms of normal brain drainage, it is important for the drainage of the DVA and closure is, therefore, contraindicated

visualized. The SP was visualized as a prominent vascular channel that connected the intracranial with the extracranial venous circulation in all patients. Based on the angiographic criteria mentioned above, we identified seven patients (about 46%) with a dominant angiographic pattern, i.e. in these seven patients, the SP played the major role in drainage of the brain. In the remaining eight patients, the role played by the SP was accessory (see Fig. 1 for examples).

Therapeutic decisions

Treatment of the SP was considered in six patients, four of them by a surgical approach alone. In one patient a combined approach was performed in order to treat the complex venous drainage (through the SP) of an associated facial haemangioma. In one patient endovascular therapy had to be performed to treat bleeding complications after a surgical approach. No treatment of the SP was considered appropriate in nine patients, but five of these were treated with interventional neuroradiological procedures for associated diseases including embolization of a DSM (Fig. 4), an AVM, a VGAM, a haemangioma and a venous malformation.

In the untreated patients, there was no tendency for the SP to grow, and spontaneous resolution of the communication between the SP and the SSS occurred in two patients.

In order to assess the clinical impact of the anomaly on the patients a clinical–neuroradiological questionnaire was developed based on our experience in the process of therapeutic decision making and as a result of the analysis of the patients presented here. This questionnaire is reproduced in the Appendix.

Discussion

SP is a rare, usually asymptomatic condition that is characterized according to our definition by an abnormal

Table 1 Patient demographics and clinical data

Patient	Gender	Concomitant disease	Clinical features	Intracranial venous anomalies	Neuroradiological examinations	Role of SP in venous drainage	Type of treatment	Outcome
1	F	No	Mild cardiac failure, macrocrania, cosmetic problem	Dural sinus malformation	US, CT, MRI and angiography	Dominant (dominant with regard to subjacent venous malformation)	None; gluing of the dural sinus malformation	Worsening
2	M	Mild macrocrania	Hemicrania, increasing frontal mass. Cosmetic problem	DVA, intrasinusal foreign body, septal venous anomaly, atrial vein substituting an internal cerebral vein	Radiography, MRI/ MRA, angiography	Dominant	None (follow-up of the foreign body (is it removable?)	Lost to follow-up
3	F	Blue stain in left hemiface	Frontal mass	Lobar DVA, dysplastic SSS, absence of internal cerebral veins, anomalous drainage of cavernous sinus	Radiography, US, CT, angiography (Fig. 1)	Accessory (dominant with regard to subjacent DVA)	None	Spontaneous resolution of the SP
4	M	No	Frontoparietal mass; cosmetic problem	Partially malformative SSS	CT, MRI, MRA; DSA after attempted surgery (Fig. 4)	Dominant	Surgical followed by endovascular (glue) and direct puncture	Recovery of blood loss; decreasing volume of haematoma and occlusion of SP
5	F	Posterior meningocele	Frontal mass	Vein of Galen aplasia, sinus falcinus	MRI, angiography	Dominant	Surgical	Lost to follow-up
6	M	No	Median frontal mass	Deep DVA, vein of Galen and straight sinus aplasia, sinus falcinus	MRI, MRA, CT, US, angiography	Accessory (dominant with regard to subjacent DVA)	Surgical	Spontaneous resolution of the communication with SSS; safe surgical removal
7	F	No	Median frontal mass	Deep DVA with absence of the basal vein of Rosenthal	MRI, angiography	Dominant	Surgical (bypass)	Good
8	F	No	Parietal off-midline soft lesion, slightly left-sided	Venous anatomical variations	CT, MRI, MRA, angiography (Fig. 2)	Accessory	Surgical	Good
9	M	No	Red-bluish spot in frontal midline	Frontal lobe DVA, osseous drainage of the frontal lobe, venous malformation at the tip of the nose	MRI, MRA, angiography	Accessory, but to be reassessed after treatment of venous malformation	None; presurgical embolization of the venous malformation	Good

10	M	Oesophageal atresia	Median parietal mass	None	CT, MRI, MRA, angiography (Fig. 3)	Accessory	None; asymptomatic parietal lesion. Follow-up	Stable
11	M	Mild macrocrania	Small median frontal mass; mild neurocognitive delay	Vein of Galen aneurysmal malformation, sinus falcinus	CT, MRI, MRA, angiography	Dominant	None; angiography was performed to treat a vein of Galen aneurysmal malformation	Good
12	F	CAMS III	Signs and symptoms due to osseous AVM (haemorrhage)	Multiple intracerebral AVM, mandibular AVM, DVA	MRI, MRA, angiography	Accessory	None; multiple endovascular session to treat osseous AVM	Stable
13	F	Left-sided intraorbital haemangioma	Exophthalmia and painful cutaneous lesion	DVA	MRI, MRA, angiography	Dominant	None	Stable
14	F	Facial midline venous malformation	Median frontal mass; cosmetic problem	None	MRA, angiography after surgical approach	Accessory (accessory with regard to haemangioma)	Surgical and endovascular (coils); embolization of the capillary lesion in order to perform a surgical approach	Stable; significant cosmetic problems
15	M	No	Left hemiplegia, seizure, headache, frontal soft mass	Right lobar proliferative angiopathy	CT, MRI, MRA, fMRI, angiography	Dominant	None	Stable, worsening angiopathy

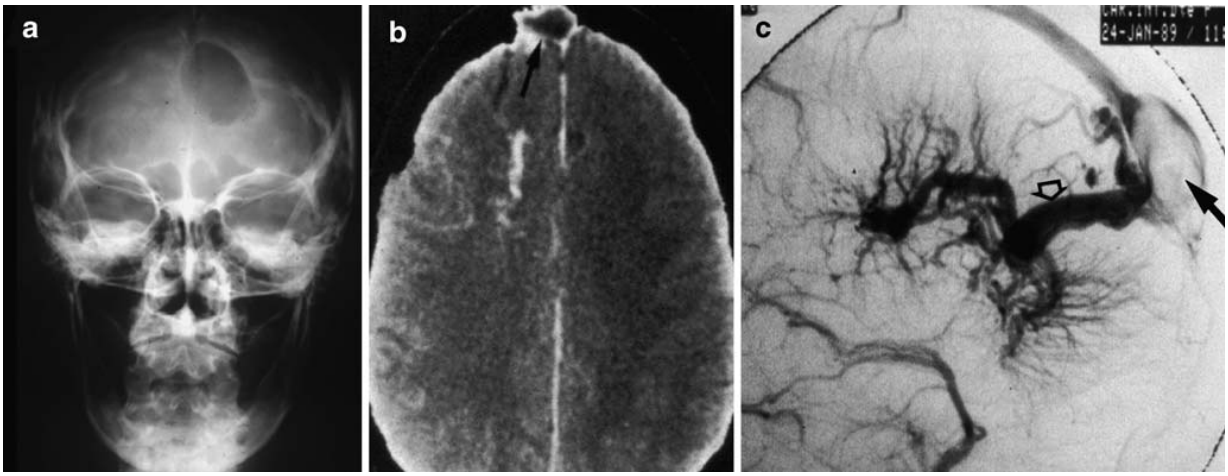

Fig. 2 SP on plain skull radiograph (**a**), contrast-enhanced CT scan (**b**) and DSA (**c**). The radiograph (**a**) demonstrates the bony defect on the midline, the CT scan (**b**) demonstrates a DVA in close spatial relationship to the SP (*black arrow*), and the angiogram (**c**) demonstrates drainage of the DVA (*open arrow*) only into the SP (*black arrow*). Ablation of the SP would lead to venous congestion since it represents the only possible drainage pathway for the large DVA

communication between the intra- and the extracranial venous drainage pathways in which blood may circulate bidirectionally through dilated vessels of the scalp. According to its transosseous disposition and diploic associated drainage, SP is similar to an emissary vein. In contrast to a simple emissary vein that has a small anastomotic channel without relevance in draining the underlying brain and no associated venous ectasia, the SP is an alternative pathway of drainage that is constituted by a network of thin-walled veins that form a varix on the external table of the skull. The aetiology of SP is unknown; the development of SP after head trauma favours an acquired pathophysiology [15]. However, its frequent association with intracranial DVA or other anomalies [18–23] has led authors to support a congenital cause (transient venous hypertension in the late embryonic period influencing venous development has been suggested as a early triggering event) [19]. The SSS is formed from an interperiostodural plexus that has both bony and pial afferents and that, during embryonic development, regresses and comes together to form a few prominent venous channels [24]. This regression is mediated by both bony and pial signalling. We speculate that the frequent association of SP with a DVA points to an anomalous signal reaching the epidural space to produce the necessary plexus confluence during embryonic development which in turn may lead to an overtriggering of diploic veins that converge to become an SP. Although hypothetical, this would take into account the association of SP with various venous anomalies, including DVAs and the absence or malformation of normal sinuses and veins.

Affected patients typically present with a soft mass which is variable in size (increasing during Valsalva manoeuvre) and fixed to the subjacent scalp [14]. In most patients it occurs along the midline in the frontal region (53% in our series), while off-midline locations are exceptional (one in our series) [16, 25]. Multiplicity, although reported [20], was not encountered in our series. All SPs discussed here were cranial to the occipital bone along the midline sutures extending from the posterior fontanelle to the forehead. An SP located caudal to the posterior fontanelle, although reported, seems to be the exception [26].

Clinical symptoms of SP are typically mild (headache, vertigo and nausea) and the main complaint is usually cosmetic. The age at diagnosis may vary from birth (in patients with wide SP) up to the third decade of life. Although a male predominance has been previously described, our series did not confirm this trend (seven males and eight females). SP can be associated with craniostenosis [26–28] where it probably represents an alternative drainage to the narrowed outlets at the cranial base. The prognosis is almost always good, and the natural history commonly shows no further evolution in size after puberty and a low risk of spontaneous or traumatic bleeding; spontaneous involution or partial thrombosis has also been described [3]. Treatment is often unnecessary, although surgical ligature or endovascular embolization may be carried out for cosmetic reasons [29].

The clinical features and objective examination, including manual and dynamic examination of the lesion (Valsalva manoeuvre), are strongly suggestive of the diagnosis which is easily confirmed by ultrasonography and colour-Doppler scans. During neuroradiological work-up conventional radiography now plays only a minor role; however, focused CT (bone window with reduced mAS that covers only the bony defect is sufficient) is required to demonstrate the bony defect. Contrast-enhanced magnetic resonance imaging (MRI) and magnetic resonance angiog-

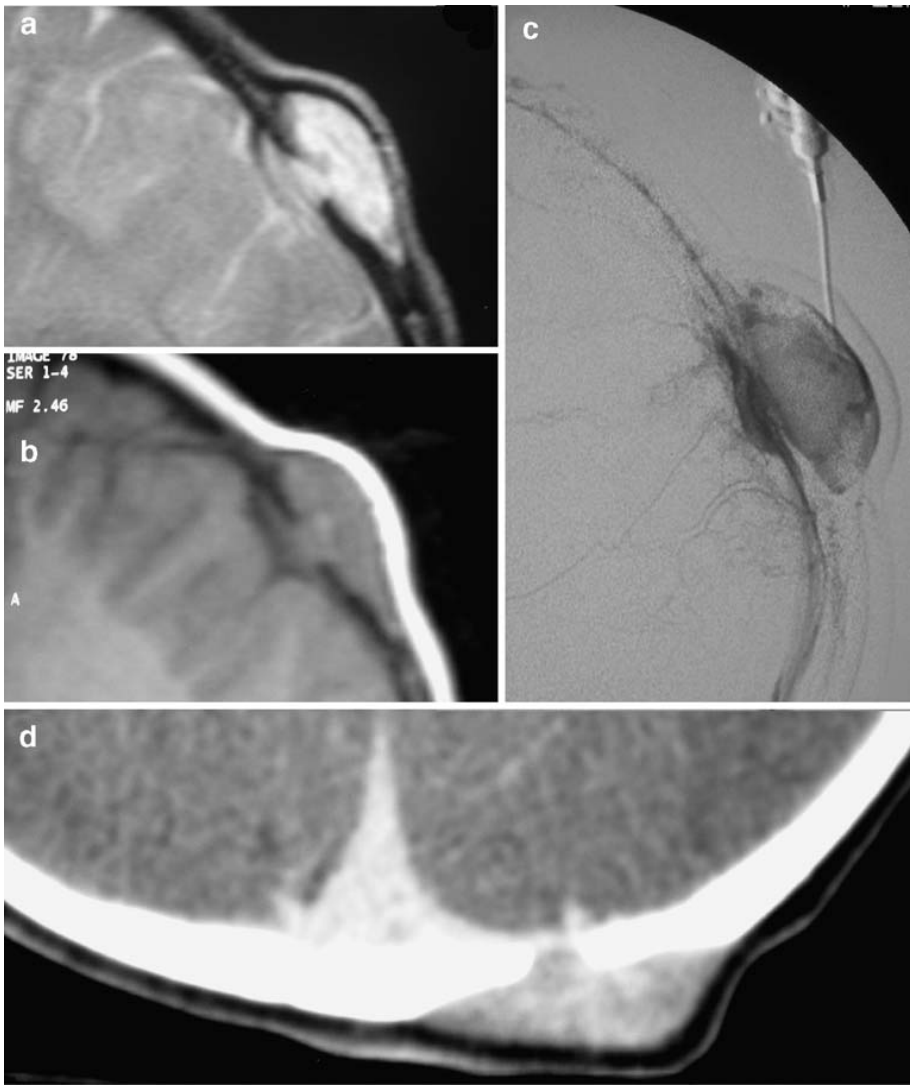

Fig. 3 Off-midline location of a SP on MRI (**a, b**), CT (**d**) and DSA (**c**). After direct puncture of the SP (**c**) the communication with the SSS can be appreciated; percutaneous therapy had therefore to be strictly avoided. The well-marginated bony defect can be appreciated on contrast-enhanced CT (**d**)

raphy (MRA) are the methods of choice as they demonstrate the presence of the SP and its drainage into the dural venous sinuses, usually the SSS. MR evaluation should also include assessment of the superficial cortical veins adjacent to the SP, associated venous anomalies, and the maturation of the jugular bulbs as well as the other venous outlets of the brain. Using these noninvasive imaging techniques, the diagnosis of SP can be easily established [29]. DSA is therefore usually not necessary, but once therapy is contemplated, it should be performed to assess the intracranial venous dynamics and the role of the SP in venous drainage.

Despite the numerous descriptions of SP in the literature, the pattern of flow towards the anomaly has not been extensively reviewed. On the basis of the results of angiography (evaluating the delay of opacification of the vessels, the absence or distortion of superficial cortical veins in the vicinity of the SP, the drainage patterns of the SSS, and the assessment of the degree of maturation of the venous outlets of the brain), two basic SP patterns were identified: (1) "dominant", if the main stream of contrast material flow uses the SP to drain the brain bypassing the usual venous outlets, and (2) "accessory", if only a small part of the venous outflow occurs through the extradiploic vessels. Since the treatment of the anomaly is generally conservative, the clinical–neuroradiological questionnaire that we propose in the Appendix might help in identifying those SPs that should be surgically or endovascularly removed because of clinical or psychological problems, and to choose the treatment best suited to a specific patient based both on this simple differentiation of dominant and accessory drainage and other related issues (such as

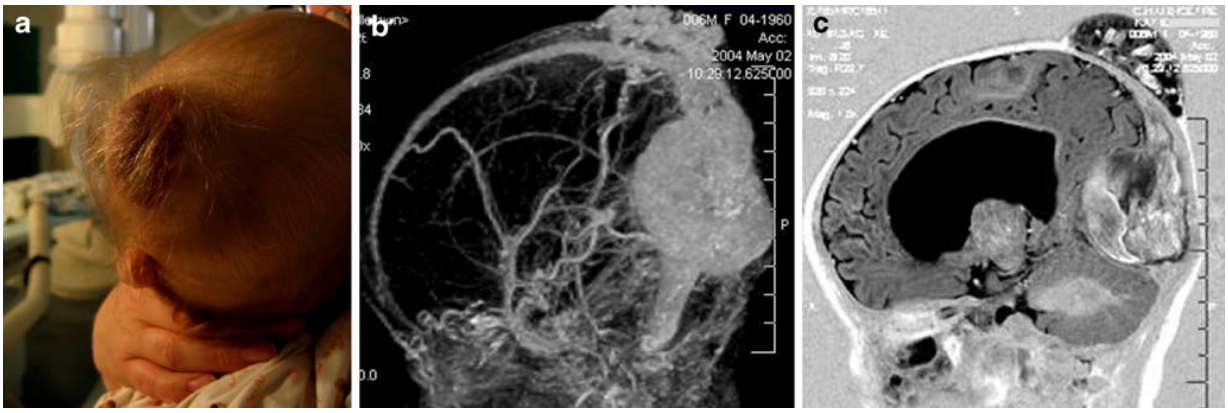

Fig. 4 Antenatal ultrasonography of this female showed increasing heart ventricular size and a wide peripheral intracranial lesion lying posteriorly between the cerebral hemispheres and splaying the leaves of the falx, initially interpreted as a huge haematoma. Postnatal MRI and ultrasonography displayed an occipital DSM associated with a parietal SP (**a–c**). DSA was carried out and confirmed the diagnosis of DSM of the torcular and of the right transverse sinus, detected the presence of intralesional thrombus formation, and also showed the complete maturation of the normal venous outlets with regression of the median occipital sinus. The importance of the SP as a collateral drainage pathway in the presence of complete thrombosis of the DSM was identified, and closure of the SP was therefore avoided

cosmetic considerations). This questionnaire, determines first from a clinical point of view whether or not further diagnostic steps should to be taken after the clinical diagnosis is established. The salient issues are the problems experienced by the patient (psychological problems due to cosmetic issues) and the clinical presentation. If the psychological problems are severe and/or if the presentation of the SP is unusual (e.g. off-midline location) further diagnostic steps need to be taken. The following imaging modalities may be used: CT in the bone window to demonstrate the margins of the bony communication, or MRI and angiography during which the role of the SP in the venous drainage of the brain should be carefully evaluated. In patients in whom the diagnostic process demonstrates no contraindication to treating the lesion, therapy can finally be considered and should be tailored to the angiomorphology of the lesion.

Seven of 15 patients showed a dominant angiographic pattern that represented a contraindication to both surgical and endovascular treatment in view of the potentially life-threatening complications, including bleeding (with the surgical approach) and venous congestion and haemorrhage (with both endovascular and surgical strategies). In the remaining cases the cerebral venous drainage pattern was "accessory", but in four patients the SP constituted the only drainage pathway of an underlying DVA, a dural sinus malformation or a haemangioma. As a consequence, in these patients any aggressive treatment (occlusion) would have led to venous congestion or bleeding, and was avoided. In addition to the differentiation of the drainage role played by the SP, our questionnaire adds additional points to be kept in mind by the treating physician that might be of importance in the decision-making process. In particular, a clinical and psychological assessment that points towards associated diseases, additional clinical symptoms or problems might be helpful in identifying those patients who might require additional diagnostic procedures, while the imaging assessment can aid in the identification of risks and benefits associated with treatment (see the Appendix).

In agreement with our results, a review of the literature showed that most SPs occur in association with a large spectrum of venous malformative abnormalities (above all DVAs) [18–23], supporting the hypothesis of a common underlying venous anomalous or malformative condition. We found a wide spectrum of venous anomalies ranging from simple anatomical variations, to DVAs (as the most common anomaly), through hypoplasia, aplasia or malformations of the vein of Galen, to dural sinus malformations associated with severe cardiac failure. We did not find any other visible intracranial anomaly in only two patients (14%); this could be considered the simplest end of the wide spectrum. These considerations may support the understanding of SP as the extracranial counterpart of a DVA, and not only a mere expression of the hydrodynamic balance between intracerebral veins, cerebrospinal fluid spaces and the pericranial veins of the scalp.

Conclusion

Most SPs can be considered the cutaneous sign of an underlying venous anomaly, and should therefore be investigated with angiography before any surgical or endovascular treatment is contemplated. In order to assess the role played by the collateral venous outflow pathway, a

simple qualitative scale ("dominant" or "accessory") in regard to general or local venous outflow may be helpful. Dominant SPs should not be treated. Accessory SPs should also not be treated if the SP represents the only predictable collateral pathway in case of thrombosis of the underlying anomaly. Only accessory SPs associated with complete maturation of normal brain outlets (e.g. jugular bulbs) can be safely treated, either surgically or with embolization, according to the clinical features of the SP, the experience of the treating team and the wishes of the patient.

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

Appendix: Clinical and imaging questionnaire

A. General assessment

List of questions for clinical assessment; if an answer is "No" skip to Pretreatment assessment.

AI. Clinical assessment

- Has any other differential diagnosis been excluded (haemangioma, cephalocele, venous malformation, etc)?
- Is the SP located in a typical midline position?
- Are cosmetic problems the only complaint? If no, specify (physical, e.g. size, position or associated dermatological problems; clinical, e.g. visual impairment, headache, bleeding, pain)
- Can the clinical or physical complaints be treated conservatively?
- Is there any premature syndromic or non-syndromic fusion of the cranial sutures (craniosynostosis, achondroplasia, etc)?

AII. Psychological assessment

- If the cosmetic problem is the main complaint, is the problem tolerable?
- Can the patient or the patient's parents understand that treating a venous anomaly is not mandatory and could be associated with a life-threatening risk? Do they agree with the decision to abstain from treatment?
- Is the psychological, social and academic development of the patient normal?

B. Pretreatment assessment

If treatment (surgical or endovascular) is under consideration, CT (bone window), MRI (angiographic study, MRA) and DSA (studying the late venous phase) must be performed or reviewed; 'negative' answers point to abstention from treatment.

BI. CT

- Is the bony communication between the intra- and extracranial compartments subjacent to the SP single and well-marginated?
- Have phleboliths been searched for and then excluded (within the lesion)?

BII. MRI (MRA and phase-contrast sequence for flow-MRI)

- Is there a no-flow or slow-flow connection between the SP and the intracranial sinus system?
- Have any associated venous malformations near the SP been excluded?

BIII. DSA

- Is the role of the SP in the global venous drainage accessory (see main text)?
- If an associated subjacent venous malformation is present, is the role of the SP in the local venous drainage accessory?
- Are the normal cerebral venous outlets patent?
- Are the jugular bulbs mature?

C. Treatment

The choice of treatment depends on the physical and clinical features of the SP as well as on the experience of the treating team and the wishes of the patient. Endovascular embolization can be presurgical

CI. Surgical. In patients with an uncomplicated, small, thin-linked, soft SP

CII. Endovascular. In patients with a wide-linked, large, stretched-out SP

- Transvenous (gluing and/or coiling)
- Percutaneous (gluing)

CIII. Abstention from treatment

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