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SHORT ILLUSTRATED REVIEW

Cavernous angiomas of the lateral ventricles

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Abstract
Background Cavernous angiomas are vascular malformations which rarely involve the cavities of the lateral ventricles. Knowledge of the specific clinical and neuroradiological features displayed by these lesions is limited by the scarcity of patients included in the reported series.
Objective and methods The aim of this study was to compile and analyse the epidemiological, clinical, neuroradiological and surgical characteristics of these lesions as provided by the well-described examples reported in the scientific literature. A total of 49 were gathered, including three patients operated on recently in our Department.
Findings and conclusions Cavernomas developing within the ventricular cavities attain a larger size than parenchymal counterpart lesions, causing symptoms and signs derived mainly from the mass effect. The characteristic parenchymal hypointense rim is less frequently identified on T2-weighted echo-gradient MRI sequences. Total surgical excision is the treatment of choice for these lesions, yet the surgical routes employed may still be associated with a high rate of neurological complications.

Keywords Cavernoma · Cavernous angioma · Intraventricular tumour · Lateral ventricle

Introduction

Cavernous haemangiomas are vascular hamartomas which may be diagnosed in every region of the central nervous system. The first description of an intraventricular cavernous angioma was made by Finkelnburg in 1905 [8]. This topography only represents 2.5% to 14% of all intracranial cavernous angiomas [34, 37, 40]. The lateral ventricles are the most frequent site, followed by the third and fourth ventricles [30]. In this study, we have performed a systematic review of the 46 well-described cases of lateral ventricle cavernomas (LVC) reported in the literature to date and included three new patients treated in our Department. An analysis of the clinical and radiological features shared by these lesions was performed and the results of surgical excision were investigated in order to evaluate the role of such intervention.

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Literature review

Using the MEDLINE database, a systematic review of multi-language literature was carried out. An initial search was performed by using the English keywords “ventricular cavernoma” and “ventricular cavernous angioma”. A total of forty five articles were obtained. Once the selected papers were gathered, we reduced the analysis to the papers reporting LVC, which also provided an explicit description of the clinical, radiological and/ or therapeutic data. The lists of references from these articles were also reviewed. Overall, 46 examples of LVC, selected from 30 case-report articles [2, 4, 5, 7, 9, 12, 13, 15, 18–26, 28–30, 32, 36, 38, 39] and intracranial cavernoma series [1, 3, 33, 34, 37, 41], together with the three patients operated on in our department, were included in the present analysis. In addition, we reviewed the recent literature, using the same database, selecting those studies presenting essential information about the main aspects of the neuroradiological differential diagnosis and surgical treatment of lateral ventricle masses that were relevant.

Analysis

Table 1 summarises the clinical information recorded from the cases of LVC included in this study. The age distribution ranged from three days to seventy four years old (mean 28.43±20.59 years) with no difference in the sex distribution. Symptoms of high intracranial pressure were the most common (48.9%), followed by seizures (19.1%) and haemorrhage (17%). Symptomatic hydrocephalus was present in only three patients. The preferential location (n=47) was the right lateral ventricle (55.3%). A more precise analysis of the topographical distribution revealed the trigone and the frontal horn (40.5%) to be the ventricular segments most frequently affected (n=37). Diagnostic computed tomography (CT) and/ or magnetic resonance imaging (MRI) studies (n=41) usually showed a well-defined mass with a heterogeneous density and/or intensity. The size of lesions (n=36) ranged from 0.4 to 10 cm (mean 3.6±2 cm). Angiography (n=25) was either normal (40%) or displayed signs of an avascular mass (32%). A tumoural blush was observed in four lesions whereas an anomalous venous phase was found in three. The surgical approaches (n=34) included the posterior parietal or temporal trans-ventricular route for 21 lesions located at the trigone or temporal horn. In the case of frontal lesions, the frontal trans-ventricular (n=6) and interhemispheric trans-callosal (n=5) were the routes preferred. The degree of excision was reported as total in 35 patients and as partial in three others. Three patients were not operated on and had a fatal outcome. Overall, four fatal episodes were registered. Neurological morbidity was recorded in 41%, including permanent hemianopia and/or hemiparesis in twelve patients, dysphasia in two and an additional patient with post-operative epileptic seizures. Finally, one patient suffered a permanent coma status.

Illustrative clinical examples

Patient 1

This 60 year old woman presented with acute onset headache, vomiting and recent memory impairment. The MRI showed a lesion located within the left frontal horn. It seemed to arise from the anterior thalamus (Fig. 1a) and displayed no remarkable angiographic findings. The initial diagnostic suspicion was subependymoma. The mass was totally removed through a left frontal trans-ventricular approach. The macroscopic appearance was that of an organised haematoma. Microscopically, it proved to be a cavernous angioma showing discrete areas of recent haemorrhage. In the post-operative period, the patient suffered from a transient nominal dysphasia and right upper limb monoparesis, with total recovery of both symptoms after rehabilitation. The patient remains asymptomatic after a four year follow-up period.

Patient 2

A 70 year old man who complained of disorientation, memory loss, urinary incontinence and gait apraxia during the preceding two months was admitted to our department. A large ventricular mass which filled the left frontal horn and caused obstructive hydrocephalus was detected (Fig. 1b). Angiography demonstrated displacement of the normal vasculature. The provisional diagnosis was that of neurocytoma. A right frontal ventriculo-peritoneal shunt was initially inserted with improvement of the symptoms. A stereotactic biopsy was then performed, without a definite diagnosis, which caused an intra-lesional haemorrhage without clinical repercussion. Finally, the lesion was macroscopically removed through a left frontal trans-ventricular approach. The microscopic study showed a cavernous angioma with signs of haemorrhage at various stages. Post-operative MRI disclosed a small remnant of the lesion adjacent to the anterior left thalamus. The patient did well without neurological deficits and no regrowth of the lesion has been detected three years after the surgical procedure.

Patient 3

This 66 year old man presented with recurrent episodes of absence seizures during the last 2 years. The cerebral MRI showed a mass located within the left ventricular atrium (Figs. 1c and d) which was angiographically silent. The neuroradiological diagnosis was that of cavernoma. It was completely removed through a left posterior parietal trans-ventricular approach. A grossly calcified mass adherent to the choroid plexus was identified at the surgical procedure. The histopathological analysis confirmed the diagnosis of cavernous angioma. Post-operatively, the patient suffered a generalised seizure which was treated with phenytoin. He recovered successfully without neurological symptoms. No cavernoma rests were identified on the post-operative MRI studies after one year follow-up.

Discussion

Clinical features

Growth of cavernous angiomas depends mainly on recurrent episodes of intralesional haemorrhage (usually self-limited and of a moderate volume) in addition to the proliferation of the endothelial cells which are lining the

Table 1 Lateral ventricle cavernomas reported in the literature

Patient	Reference	Sex/age	Presentation	Size (cm)	Location (side/horn)	Approach	Extent of resection	Outcome
1	Merrit [22]	F/16y	H	4.5	L/T	PTTV	T	Coma
2	Arnstein et al. [2]	M/3d	H	Large	R/NA	NO	NO	Death
3	McGuire et al. [21]	M/3m	HCP	2	BV/F	NA	NA	NA
4	Schneider and Liss [33]	F/33y	M, S	8	R/NA	PTTV	T	Hemianopia
5		F/33y	M, S	10	R/NA	PTTV	T	Hemianopia
6	Jain [13]	M/15y	M	5	R/F	FTV	T	Asympt
7	McConnel and Leonard [20]	F/31y	H	0.5	R/O	NO	NO	Death
8	Towfighi et al. [39]	F/74	H	0.4	BV/F	NA	T	NA
9	Coin et al. [5]	F/36y	S	2	R/T	NA	T	Hemianopia
10	Numaguchi et al. [28]	M/43y	M	Large	R/T	NA	T	Hemianopia, hemiplegia
11	Pau and Orunesu [29]	NA/56y	H	NA	L/T	NO	NO	Death
12	Namba et al. [25]	F/45y	H	3	R/B	NA	P	Improved
13	Iwasa et al. [12]	F/8d	HCP	3	L/T	PTTV, S	T	Asympt
14	Chadduck et al. [4]	F/21y	S	3	R/T	PTTV	T	Hemianopia
15		F/29y	M	5	R/NA	PTTV	T	Asympt
16		F/4m	S	4	R/T	PTTV	T	Improved
17	Simard et al. [34]	M/22y	M	NA	R/NA	NA	NA	NA
18		F/13y	M	NA	R/NA	NA	NA	NA
19	Yamasaki et al. [41]	M/73y	M	3	R/T	PTTV	T	Hemianopia, hemiparesis
20	Suzuki [36]	M/40y	M	3	NA/NA	NA	T	Improved
21	Sabatier et al. [32]	M/9m	H	2	L/F	NO	NO	Cerebellar syndrome
22	Tatagiba et al. [37]	M/33y	H	3	L/F	FTV	T	Asympt
23		M/35y	S, M	4	R/T	OTV	T	Death
24		F/24y	M	3	L/T	PTTV	T	Asympt
25	Miyagi et al. [24]	F/3y	M	2	L/T	TSTV	T	Hemiparesis
26	Lynch et al. [19]	F/39y	S	NA	R/F	NA	P	Asympt
27		M/5y	S	NA	R/F	NA	T	Asympt
28		F/10y	M	NA	R/NA	NA	T	Asympt
29	Kaim et al. [15]	M/64y	M	1.5	R/F	IHTC	T	NA
30	Reyns et al. [30]	F/16y	M	4	L/F	SB, FTV, D	T	Improved
31		M/36y	S	4	BV/F	IHTC, D	T	Hemiparesis, dysphasia
32	Fagundes-Pereyra et al. [7]	F/15y	M	4.1	L/F	IHTC	T	Asympt
33	Attar et al. [3]	M/30y	M	NA	L/NA	PTTV	NA	Asympt
34		M/30y	M	NA	L/NA	PTTV	NA	Asympt
35		M/18y	M	NA	L/NA	PTTV	NA	Asympt
36		M/30y	M	NA	L/NA	FTV	NA	Asympt
37	Nieto et al. [26]	F/11y	S	5	L/T	PTTV	T	Hemianopia
38	Tatsui et al. [38]	F/17y	S	1.5	R/T	PTTV	T	Asympt
39		M/52y	M	1.5	R/Te	PTTV	T	Asympt
40	Michaelson et al. [23]	F/22y	M	7.5	R/Te	PTTV	T	Asympt
41	Kumar et al. [18]	M/8y	M	5	R/T	PTTV	T	Asympt
42		F/19y	S, M	5	L/T	PTTV	T	Hemiparesis, dysphasia, hemianopia
43		M/20y	M	5	R/T	PTTV	T	Asympt
44	Alves de Sousa [1]	NA/NA	NA	6	R/F	IHTC	T	Asympt
45		NA/NA	NA	NA	NA/F	IHTC	T	Asympt
46	Darder et al. [9]	M/25y	M	2	R/T	PTTV	T	Asympt
47	Present cases	F/60y	M	2	L/F	FTV	T	Hemiparesis, memory impairment
48		M/70y	HCP	3.5	L/F	S, SB, FTV	P	Asympt
49		M/66y	S	2.7	L/T	PTTV	T	Seizure

NA (all categories): information not available; sex—M: male, F: female; age—d: days, m: months, y: years; onset—A: acute, I: insidious; presentation—H: haemorrhage, M: mass effect, S: seizures, HCP: hydrocephalus; side—R: right, L: left, BV: bi-ventricular; horn—F: frontal, B: body, T: trigone, Te: temporal, A: atrium, O: occipital; approach—NO: not operated, FTV: frontal trans-ventricular, PTTV: parietal–temporal trans-ventricular, IHTC: interhemispheric trans-callosal, OTV: occipital trans-ventricular, TSTV: trans-sylvian trans-ventricular, S: ventriculo-peritoneal shunt, D: external ventricular drain, SB: stereotactic biopsy; extent of resection—NO: not operated, T: total, P: partial; outcome—Asympt: asymptomatic

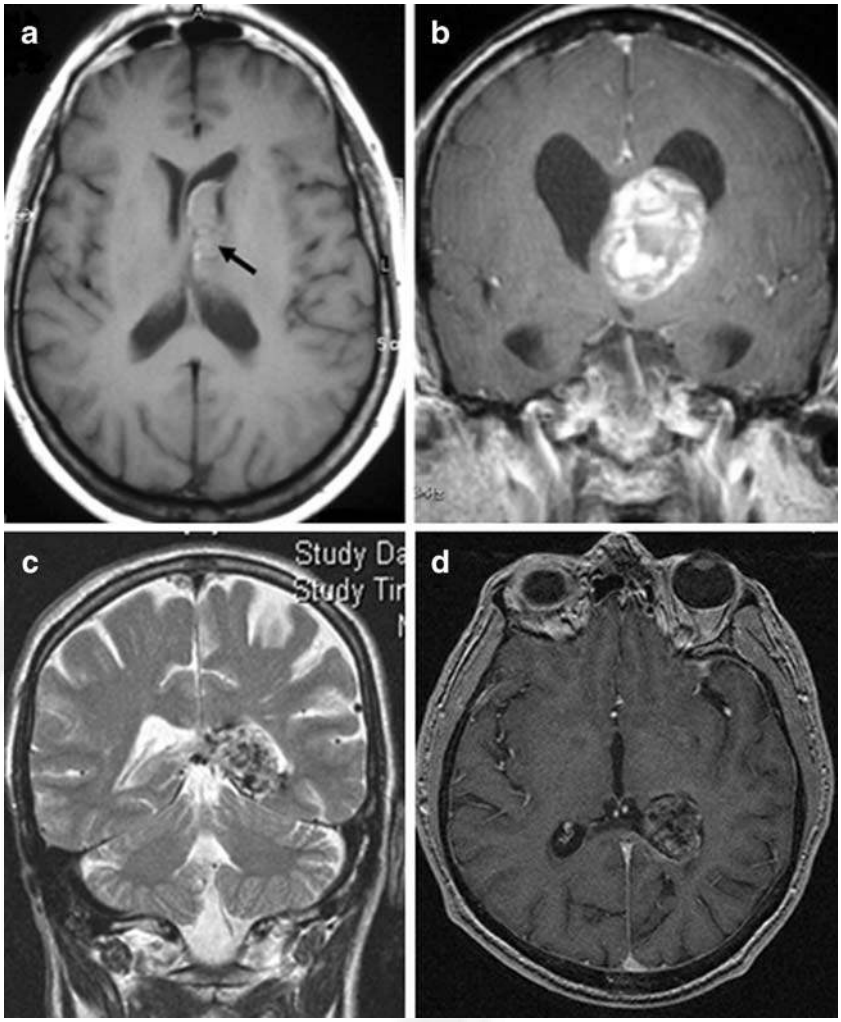

Fig. 1 **a** Patient 1. Pre-operative axial MRI study showing a solid, T1-weighted isointense intra-ventricular mass of 2×1 cm with a thalamic extension (*arrow*) and peripheral hyperintense signal corresponding to recent bleeding areas. **b** Patient 2. This coronal T1-weighted MRI displays a left ventricular mass of 3.5×2.5 cm that obstructs both foramina of Monro, causing bi-ventricular hydrocephalus. The lesion presents hypo- and hyperintense signal areas and intense heterogeneous Gadolinium enhancement. (**c** and **d**) Patient 3. Pre-operative coronal T2-weighted (**c**) and axial T1-weighted gadolinium-enhanced (**d**) MRI studies showing a 2.7×2.5 cm heterogeneous mass occupying the left atrium

caverns [10, 17, 27]. Cavernomas developing within a ventricular cavity, without the restrictions imposed by the brain parenchyma, attain a larger size (mean of 3.6 cm) than intra-parenchymal counterparts. As a result, patients harbouring LVCs usually present symptoms derived from the mass effect of the lesion. This fact can be related to the larger size attained by LVCs at the time of diagnosis. Less frequently, the clinical onset is caused by an acute haemorrhage or seizures [30, 35, 40].

Diagnosis

LVCs typically appear as well-delineated heterogeneous masses, without enhancement after contrast administration in both CT and MRI. These findings are consistent with areas of calcification and blood at various stages of degradation within the cavernoma [30, 38]. The characteristic hyposignal rim on the T2 echo-gradient weighted MRI observed in parenchymal cavernous angiomas is lacking in most LVCs due to the absence of bleeding into the surrounding brain tissue and was found only in five lesions, including our third patient [15, 23, 26, 30].

Cavernomas have been classically considered as angiographically occult or cryptic vascular malformations. Nevertheless, a tumoural blush [4, 25, 35, 37] or a feeding artery can be identified on the cerebral angiogram [4]. Numaguchi et al. have described the presence of tiny strands of the contrast medium in the avascular mass in the capillary and venous phase, without large draining veins or early venous filling being observed [28]. The coexistence of LVC with a medullary venous malformation that participates in the compensatory venous drainage of the lesion has also been reported [12, 24]. These associated venous malformations are not always detected in the pre-operative angiogram. The development of post-operative brain oedema and/or venous infarction, after occluding this compensatory venous drainage during the removal of the cavernoma, has been reported [24].

A correct pre-operative diagnosis of a cavernous angioma was obtained in only five out of the 14 reports

providing this information, including our third patient. This was probably as a consequence of the uncommon location and the specific radiological characteristics displayed by this subgroup of cavernomas [9, 23, 38, 41]. LVCs are frequently misdiagnosed as tumours [14, 16, 18, 26, 30, 37, 38] and this can lead to the use of invasive diagnostic procedures such as stereotactic biopsy [30], which can cause iatrogenic bleeding, as observed in our third patient. However, previous studies analysing the use of stereotactic biopsies in parenchymal cavernomas concluded that this procedure can be performed safely in these patients, despite the apparent risk of haemorrhage [30, 34].

Treatment

LVCs can usually be easily dissected from the ventricular walls [20, 26]. However, on occasion the lesion cannot be distinguished from an organised haematoma. A few show an intimate relationship with the choroid plexus [9], as was noted in our third patient. Some authors have proposed that LVCs could originate at the subependymal region [4], disrupting the ependymal layer during their growth and finally invading the ventricle. We found a subependymal component of the lesion in our first patient, adjacent to the left thalamus. Nevertheless, the origin of LVCs still remains unclear.

The surgical anatomy and approaches to the lateral ventricles have been extensively described elsewhere [6, 31]. The preferable routes for the resection of cavernomas located within the frontal horn are either the trans-cortical transventricular or the interhemispheric trans-callosal approaches [1]. Trans-temporal and superior parietal approaches have been used most frequently for the excision of trigonal and temporal lesions [18]. The trans-sylvian trans-ventricular approach is a good alternative for the resection of trigonal cavernomas with the benefit of a minimal disruption of the visual pathways [24, 42].

Due to the low frequency of hydrocephalus associated with LVCs (6.1%) and the large tumour volume at the time of diagnosis, a neuroendoscopic approach is seldom indicated. Stereotactic or frameless navigation devices could help during the ventricular approach [9]. Nevertheless, they become useless after opening the ventricular cavity, due to the significant degree of brain shift that follows the release of cerebrospinal fluid. For this reason, some authors recommend the use of ultrasound to guide the approach and control the degree of removal [18].

The management of hydrocephalus associated with LVC has not been well established. An early resection of the mass might solve the cerebrospinal fluid obstruction. In fact, the presence of ventricular dilatation may help during the surgical procedure. However, the insertion of a ventriculo-peritoneal shunt before removing the lesion represents a safe choice, as we performed in our second patient, because it allowed an early relief of the symptoms of high intracranial pressure whilst studying the mass. The avascular nature of cavernomas minimises the risk of shunt device obstruction caused by intra-operative bleeding during lesion removal.

Outcome

After the surgical procedure, 65% of the patients were asymptomatic or improved from their initial symptoms. The most frequent permanent post-operative deficit was a contralateral homonymous hemianopia, observed in eight patients (Table 1). This deficit was mostly (62.5%) a direct consequence of the trans-cortical approach to the ventricular system [4, 5, 26, 28, 33]. Four fatalities have been reported among the examples of LVC included in this review. Three patients who did not undergo surgery died after an acute haemorrhage [2, 20, 29], whereas one patient suffered an extensive sinus thrombosis after the surgical procedure [37].

Conclusion

Cavernomas must be included in the differential diagnosis of patients harbouring a lateral ventricle mass, although an accurate pre-operative diagnosis remains challenging due to their low frequency and their atypical clinical and neuroradiological characteristics. A radical removal of the lesion is recommended in most patients [30]. Modern microsurgical techniques guarantee a safe and complete resection of LVC but a high rate of post-operative neurological deficits is still associated with these procedures.

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Comment

Carrasco et al reported three rare cases of the lateral ventricle CM and had the systematic review of the whole reported similar cases in the literature. They analyzed the radiological findings, surgical strategies and the outcomes of these lesions. They concluded that radical surgery was still the treatment of choice, with low mortality but high neurological morbidity. This report offered the general outline of this rare subgroup of cavernoma.

But the definition of ventricle cavernoma is still not clear. Actually many lesions are the cavernoma arising from the thalamus or basal ganglia.

Intralesional hemorrhages and lesion expansion may involve the lateral wall of the ventricle and protrude into the ventricle cativity. In the case of thalamus cavernoma, it is not uncommon in the clinical experience.

Surgical treatment of the CM in that area should be carefully balanced between the outcome and the natural course. Accurate localization of the lesions with neuronavigation can offer individualized surgical approaches and avoid additional surgical trauma to the eloquent area. We agree that radical resection of the lesions is the treatment of choice. Partial resection of the lesion or biopsy is not recommended.

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