Child's Nervous System (2020) 36:2789–2799
https://doi.org/10.1007/s00381-020-04560-3

ORIGINAL ARTICLE

Spontaneous rupture of middle fossa arachnoid cysts: surgical series from a single center pediatric hospital and literature review

Alberto Balestrino [1,2] · Gianluca Piatelli [2] · Alessandro Consales [2] · Armando Cama [2] · Andrea Rossi [3] · Mattia Pacetti [2] · Pietro Fiaschi [1,2] · Marco Pavanello [2]

Received: 13 January 2020 / Accepted: 27 February 2020 / Published online: 23 March 2020

Abstract

Purpose Arachnoid cysts may present with symptoms deriving from cyst rupture, usually causing intracystic hemorrhage and subdural hematoma or hygroma. Rupture is usually caused by minor trauma, spontaneous rupture is an exceptional event, and 57 cases have been described in literature. We here present and discuss the largest series of spontaneously ruptured middle fossa arachnoid cysts in order to investigate clinical presentation and best treatment available.

Methods We report a retrospective series of 17 pediatric patients surgically treated for middle fossa arachnoid cyst with signs of cyst rupture without a history of trauma in the previous 90 days. We describe clinical presentation, treatment, and outcome at follow-up discussing our results with a literature review including all reported cases of spontaneous rupture of middle fossa arachnoid cysts.

Results In our experience patients most frequently presented with subdural hygroma, in literature, a chronic hematoma was most frequently reported. Headache is the most reported symptom at presentation. Neurological deficits and consciousness alterations are rare. Surgical treatment may resolve brain compression only or reduce rupture recurrence risk. Conservative treatment has also been proposed. Different treatments are reported and discussed focusing on indications, contraindications, risks, and expected benefits.

Conclusion We propose, when safely possible, microsurgical cyst fenestration in skull base cisterns as the treatment of choice for these patients as long as it addresses both immediate decompression and risk of rupture recurrence. We report good outcomes and low incidence of complications from our series with a mean postoperative follow-up of 30 months.

Keywords Subdural hematoma · Subdural hygroma · Fenestration · Intracystic hemorrhage

Introduction

Arachnoid cysts (ACs) are cerebrospinal fluid collections surrounded by an arachnoid sheet believed to be of congenital origin. They are uncommon cystic lesions that represent 1% of all intracranial masses [1]. Nearly half of pediatric intracranial arachnoid cysts are located in the sylvian fissure/middle cranial fossa [2], and 60–80% of arachnoid cysts are discovered before the age of 15 years, mainly in the male population [3–5].

Although ACs usually remain stable in volume, they may enlarge with different reported mechanisms [6] causing symptoms related to direct compression of specific structures or intracranial hypertension.

They may also present with symptoms deriving from cyst rupture, usually causing intracystic hemorrhage and/or subdural hematoma or hygroma [7, 8]. Rupture is usually caused by minor head trauma, but spontaneous rupture may also happen [9].

Spontaneous rupture of middle fossa arachnoid cyst (MFAC) is a rare event; only 57 cases have been reported in literature (Table 1).

✉ Alberto Balestrino
alberto.balestrino@gmail.com

1 Division of Neurosurgery, Department of Neuroscience (DINOGMI), Ospedale Policlinico San Martino-IST, University of Genoa, Largo Rosanna Benzi 10, 16132 Genoa, Italy

2 Department of Neurosurgery, IRCCS Istituto Giannina Gaslini Children's Hospital, Genoa, Italy

3 Department of Neuroradiology, IRCCS Istituto Giannina Gaslini Children's Hospital, Genoa, Italy

Table 1 Literature review. Patients are grouped according to treatment received

Author and year		Sex, age	Side of the cyst	Exclusively intracystic bleeding	Subdural collection's content	Subdural collection's side	Headache	Nausea and vomiting	Neurological symptoms
Conservative treatment ($n = 9$)									
Inoue et al. [10]	1987	M, 7	Right	no	Chronic	Homolateral	No	No	Unspecified ocular palsy
Rakier et al. [11]	1995	F, 8	Right	No	CSF	Homolateral	Yes	No	No
Choong et al. [12]	1998	F, 9	Left	No	CSF	Bilateral	Yes	Yes	No
Henriques et al. [13]	2007	M, 10	Left	No	Homolateral csf, contralateral acute	Bilateral	Yes	No	6th nerve deficit
Katsaros et al. [14]	2008	F, 35	Right	Yes	No	No	Yes	No	No
Lohani et al. [15]	2013	M, 11	Right	No	Subacute	Homolateral and spinal	Yes	No	No
Marques et al. [16]	2014	M, 29	Left	No	CSF	Bilateral	Yes	Yes	No
Bora et al. [17]	2015	M, 2	Left	No	Chronic	Homolateral	No	Yes	Macrocrania
Adin et al. [7]	2017	M, 21	left	No	Subacute	Homolateral	Yes	No	No
Hematoma surgery ($n = 15$)									
Page et al. [18]	1987	F, 12	Left	No	Subacute	Homolateral	Yes	Yes	No
Rogers et al. [19]	1990	F, 6	Right	No	Chronic	Homolateral	Yes	No	No
Oka et al. [20]	1994	M, 24	Left	No	Chronic	Homolateral	Yes	No	No
Parsch et al. [8]	1997	NR, 27	Right	No	Chronic	Homolateral	Yes	No	Discrete left motor deficit
Ibarra et al. [21]	2000	M, 11	Left	No	Chronic	Homolateral	Yes	Yes	6th nerve deficit
Mori et al. [22]	2002	F, 11	Right	No	Chronic	Homolateral	Yes	Yes	No
Chan et al. [23]	2008	M, 29	Left	No	Chronic	Homolateral	Yes	No	Dizziness
Patel et al. [24]	2013	M, 9	Bilateral	No	Chronic	Unilateral right	Yes	Yes	No
Kang et al. [25]	2015	M, 13	Right	No	Chronic	Homolateral	Yes	Yes	No
Yuksel et al. [26]	2016	M, 17	Left	No	Chronic	Homolateral	Yes	No	6th nerve deficit
Wu et al. [27]	2018	F, 8	Left	No	Chronic	Homolateral	NR	NR	NR
		M, 19	Left	No	Chronic	Homolateral	NR	NR	NR
		M, 17	Left	No	Chronic	Homolateral	NR	NR	NR
		M, 41	Left	No	Chronic	Homolateral	NR	NR	NR
Kaszuba et al. [28]	2018	M, 47	Left	No	Subacute	Homolateral	Yes	Yes	Ataxia, dizziness
Shunting procedures ($n = 6$)									
Rogers et al. [19]	1990	M, 6	Right	No	Chronic	Homolateral	Yes	No	No
Albuquerque et al. [1]	1997	M, 10	Left	No	CSF	Homolateral	Yes	Yes	No
Poirrier et al. [29]	2004	M, 15	Right	No	CSF	Homolateral	Yes	Yes	Blurred vision
Cakir et al. [30]	2004	M, 9	Right	No	CSF	Homolateral	Yes	Yes	No
Gil-Gouveia et al. [31]	2010	F, 16	Left	No	Chronic	Homolateral	Yes	Yes	Horizontal diplopia
Khilji et al. [32]	2016	M, 9	Left	No	Chronic	Bilateral	Yes	Yes	No
Subdural cyst fenestration ($n = 14$)									
Cullis et al. [33]	1983	M, 11	Left	No	CSF	Homolateral	Yes	Yes	No
Page et al. [18]	1987	M, 17	Right	No	Chronic	Homolateral	Yes	Yes	No
Eustace et al. [34]	1992	F, 11	Left	No	Chronic	Homolateral	Yes	No	No
Iaconetta et al. [35]	2006	M, 13	Right	No	Chronic	Homolateral	Yes	Yes	No
Ziaka et al. [36]	2008	M, 38	Bilateral	No	Chronic	Unilateral left	Yes	No	Hyperreflexia on the right side and imbalance
Hong et al. [37]	2008	F, 11	Left	No	Chronic	Homolateral	Yes	Yes	No
Patel et al. [38]	2009	M, 22	Left	No	Subacute	Homolateral	Yes	Yes	No
Gündüz et al. [39]	2010	M, 57	Left	Yes	No	No	Yes	No	Aphasia
		F, 19	Left	No	Subacute	Homolateral	Yes	No	No
Shrestha et al. [9]	2014	M, 21	Left	No	Chronic	Homolateral	Yes	No	No
		M, 15	Left	No	Chronic	Homolateral	Yes	No	No

Table 1 (continued)

		F, 16	Left	No	Chronic	Homolateral	Yes	No	Dizziness
Hall et al. [40]	2017	M, 34	Right	No	Subacute	Homolateral	Yes	Yes	Mild left motor deficit
Aydogmus et al. [41]	2017	M, 15	Left	No	Subacute	Homolateral	Yes	Yes	No
Basal cyst fenestration ($n = 7$)									
Ergun et al. [42]	1997	M, 14	Left	No	CSF	Homolateral	Yes	Yes	Right motor deficit
Çayli [43]	2000	F, 12	Left	No	CSF	Homolateral	Yes	Yes	No
Galarza et al. [44]	2002	M, 17	NR	No	Chronic	NR	Yes	No	No
Slaviero et al. [45]	2008	M, 5	Left	No	CSF	Homolateral	Yes	No	No
Liu et al. [46]	2014	F, 5	Right	No	CSF	Homolateral	Yes	Yes	No
Shrestha et al. [9]	2014	F, 5	Right	No	Subacute	Homolateral	Yes	Yes	No
Adin et al. [7]	2018	M, 36	Right	No	Subacute	Homolateral	Yes	Yes	Decreased visual acuity
Cyst resection ($n = 4$)									
Chandra et al. [47]	2015	M, 12	Left	No	Chronic	Homolateral	Yes	Yes	No
Wu et al. [27]	2018	M, 1	Bilateral	No	Chronic	Bilateral	NR	NR	NR
		M, 12	Left	No	Chronic	Homolateral	NR	NR	NR
		M, 10	Right	No	Chronic	Homolateral	NR	NR	NR
Treatment not reported ($n = 2$)									
Sener [48]	1997	M, 12	Left	No	CSF	Homolateral	Yes	No	No
		M, 16	Left	No	Chronic	Homolateral	Yes	No	No

Author and year	Seizure	Other findings	Treatment	Follow-up (months)	Subdural collection at follow-up	Cyst volume at follow-up	Symptoms at follow-up
Conservative treatment ($n = 9$)							
Inoue et al. [10]	No		Observation	36	Regressed	Disappeared	Recovery
Rakier et al. [11]	No		Observation	NR	NR	NR	Recovery
Choong et al. [12]	No	Papilloedema	Acetazolamide	12	Regressed	Stable	Resolution of papilloedema
Henriques et al. [13]	No	Papilloedema	Observation	8	Regressed	Stable	Recovery
Katsaros et al. [14]	No		Observation	18	Regressed (intracystic)	Stable	Recovery
Lohani et al. [15]	No	Radicular leg pain	Steroid therapy	3	Regressed	Reduced	Recovery
Marques et al. [16]	No	Papilloedema	Initially conservative (acetazolamide), subsequent basal cyst fenestration at 2 months	0	Regressed	Reduced	NR
Bora et al. [17]	No		Observation	8	Reduced	Stable	Asymptomatic
Adin et al. [7]	No		Observation	6	NR	NR	Unchanged
Hematoma surgery ($n = 15$)							
Page et al. [18]	No	Papilloedema	Unspecified hematoma surgery	0	NR	NR	Recovery
Rogers et al. [19]	No	Papilloedema	Craniotomy for hematoma	NR	NR	NR	NR
Oka et al. [20]	No		Burr holes for hematoma	24	Regressed	Reduced	Recovery
Parsch et al. [8]	No		Craniotomy for hematoma	150	NR	NR	Minimal left motor deficit, rare headaches
Ibarra et al. [21]	No		Unspecified hematoma surgery	0	Regressed	Stable	Improvement of symptoms
Mori et al. [22]	No		Burr holes for hematoma	NR	NR	NR	Recovery
Chan et al. [23]	No		Burr holes for hematoma	NR	NR	NR	NR
Patel et al. [24]	No		Unspecified hematoma surgery	12	NR	Stable	Recovery
Kang et al. [25]	No		Burr holes for hematoma and the second burr holes for hematoma recurrence. Endovascular coiling of MMA	60	Regressed	NR	Recovery
Yuksel et al. [26]	No	Papilloedema	Craniotomy for hematoma	1	Regressed	Stable	Recovery
Wu et al. [27]	NR	NR	Burr holes for hematoma	NR	NR	NR	Recovery
	NR	NR	Burr holes for hematoma	NR	NR	NR	Recovery

Table 1 (continued)

	NR	NR	Burr holes for hematoma	NR	NR	NR	Recovery
	NR	NR	Burr holes for hematoma	NR	NR	NR	Recovery
Kaszuba et al. [28]	No		Craniotomy for hematoma	NR	NR	NR	Occasional headaches
Shunting procedures ($n = 6$)							
Rogers et al. [19]	No	Papilloedema, unilateral ocular proptosis	Craniotomy and cysto-peritoneal shunt	NR	NR	NR	NR
Albuquerque et al. [1]	No		Subdural-peritoneal shunt	NR	Regressed	Reduced	Recovery
Poirrier et al. [29]	No		Burr holes for subdural hematoma drainage and the second surgery for subdural-peritoneal shunt	1	Regressed	Reduced	Recovery
Cakir et al. [30]	No		Burr holes for subdural hematoma and the second surgery for subdural-peritoneal shunt	0	Reduced	Reduced	Recovery
Gil-Gouveia et al. [31]	No		Subdural-peritoneal shunt	12	Regressed	Stable	Recovery
Khilji et al. [32]	No	Papilloedema	Bilateral subdural-peritoneal shunt	0	NR	NR	NR
Subdural cyst fenestration ($n = 14$)							
Cullis et al. [33]	No		Craniotomy and subdural cyst fenestration	NR	NR	NR	NR
Page et al. [18]	No		Craniotomy and subdural cyst fenestration	0	NR	NR	Recovery
Eustace et al. [34]	No		Craniotomy and subdural cyst fenestration	0	NR	NR	Recovery
Iaconetta et al. [35]	No	Papilloedema	Craniotomy and subdural cyst fenestration	0	Regressed	Stable	Recovery
Ziaka et al. [36]	No		Craniotomy and subdural cyst fenestration	6	NR	NR	Recovery
Hong et al. [37]	No		Craniotomy and subdural cyst fenestration	0	Regressed	NR	Recovery
Patel et al. [38]	No		Craniotomy and subdural cyst fenestration	0	Reduced	Reduced	Recovery
Gündüz et al. [39]	No		Craniotomy and subdural cyst fenestration	0	NR	NR	Recovery
	No		Craniotomy and subdural cyst fenestration	0	NR	NR	Recovery
Shrestha et al. [9]	No	Homolateral cheek spasm	Craniotomy and subdural cyst fenestration	0	Regressed	Disappeared	Recovery
	No		Craniotomy and subdural cyst fenestration	0	Reduced	Stable	Recovery
	No		Craniotomy and subdural cyst fenestration	0	Reduced	Stable	NR
Hall et al. [40]	No		Craniotomy and subdural cyst fenestration	0	Reduced	NR	Recovery
Aydogmus et al. [41]	No		Large burr hole drainage and subdural fenestration of the cyst	NR	Regressed	Stable	Recovery
Basal cyst fenestration ($n = 7$)							
Ergun et al. [42]	No		Craniotomy and basal cyst fenestration	0	Reduced	Reduced	Improvement of motor deficit
Çayli [43]	No	Papilloedema	Craniotomy and basal cyst fenestration	NR	Reduced	Reduced	Recovery
Galarza et al. [44]	No	Papilloedema	Craniotomy and basal cyst fenestration	NR	NR	Reduced	NR
Slaviero et al. [45]	No		Craniotomy and basal cyst fenestration	8	Regressed	Reduced	Recovery
Liu et al. [46]	No	Irritability	Craniotomy and basal cyst fenestration	16	Regressed	NR	Recovery
Shrestha et al. [9]	No		Craniotomy and basal cyst fenestration	0	Reduced	Reduced	NR
Adin et al. [7]	No		Craniotomy and basal cyst fenestration	12	NR	NR	Recovery
Cyst resection ($n = 4$)							
Chandra et al. [47]	No		Craniotomy and AC resection	0	NR	NR	Recovery
Wu et al. [27]	NR	NR	Craniotomy and AC resection	NR	NR	NR	Recovery
	NR	NR	Craniotomy and AC resection	NR	NR	NR	Recovery
	NR	NR	Craniotomy and AC resection	NR	NR	NR	Recovery
Treatment not reported ($n = 2$)							
Sener [48]	No		NR	NR	NR	NR	NR
	No		NR	NR	NR	NR	NR

Whenever to treat them, timing of treatment and procedure of choice are still a matter of debate. Different surgical treatments have been proposed for cysts that are enlarging in volume or which become symptomatic. These procedures mainly consist in cerebrospinal fluid (CSF) diversion to peritoneum or to the physiological CSF pathways. Direct excision of the cyst, cyst fenestration, and shunting operation has been reported [27, 49, 50]. Given the rarity of the pathology, only case reports and very small case series have been reported; thus, clinical presentations of these patients have been poorly studied, and there is still uncertainty regarding the indications, feasibility, efficacy, and safety of the different surgical procedures reported. Our main purposes are to describe clinical presentation of these patients and to help physicians to critically decide which treatment will be best for these patients with more awareness of feasibility and efficacy of the different surgical procedures. To do so, we here present our series of 17 patients surgically treated for spontaneously ruptured MFAC that, to the best of our knowledge, is the largest case series in literature. We have then discussed and compared our results with an extensive literature review in order to clarify the current state of the art of treatment for this rare pathology. We have reported and discussed clinical presentation, indications, contraindications, risks, and benefits of the different treatments that have been proposed.

Materials and methods

We have retrospectively analyzed all patients operated for MFAC from 2008 to 2018 at the Neurosurgery Unit of the Istituto Giannina Gaslini Children's Hospital, Genoa, Italy. Inclusion criteria were presence on admission CT scan of MFAC with radiological signs of rupture defined as the presence of subdural hygroma or hematoma, intracystic hemorrhage, or intraparenchymal hematoma. Exclusion criteria were history of trauma in 3 months before presentation, aggregation, or coagulation dysfunctions either iatrogenic or pathological. We have reviewed demographic parameters, clinical data, radiological exams, pathology records, and follow-up documentation. Clinical presentation, surgical treatments, complication, and outcome at follow-up are reported. Volume of AC was described using Galassi's classification of MFAC [51].

A literature review was performed in order to find all reported cases of spontaneous rupture of arachnoid cysts of the middle cranial fossa. The latter was performed using an online database search (Medline/Pubmed) using combinations of the terms "arachnoid cyst", "subdural", "haemorrhage", "hemorrhage", "haematoma", "hematoma", "hygroma", "bleeding" and "rupture". We have excluded all cases with a history of trauma in 3 months before presentation. We have also excluded from our literature review all patients over 70 years of age, the ones with reported dementia, with blood coagulation or platelet aggregation anomalies as in these cases, other possible causes for subdural bleeding were coexisting.

Results

From 2008 to 2018, 17 patients were surgically treated in our institution for arachnoid cysts of middle cranial fossa with radiological signs of rupture without history of trauma in 3 months before presentation (Table 2). No blood coagulation or platelet aggregation disorders were found. No genetic syndromes were reported in our series.

Mean age at presentation was 8.3 years (range 3 months–14 years).

In two cases, bilateral cysts were found; thus, 17 patients presented 19 temporal arachnoid cysts. Fifty-three percent of them were classified as Galassi's type II, 41% were type III, and 6% were type I.

All patients presented with concomitant chronic subdural hematoma and/or subdural hygroma. We did not find patients with radiological signs of intracystic bleeding without subdural collection nor with a different CT density between cystic and subdural fluid.

Two patients (11.7%) presented bilateral cysts, while bilateral subdural blood collections were present in 47% of patients. There were globally 25 subdural blood collections; 76% of these were hygromas, 12% were chronic hematomas, and 12% were subacute hematomas. No acute hematomas are reported in our series.

The most frequently reported symptom at presentation was headache, reported by 82% of patients. Headache was graded with the visual analogue scale (VAS), defined as mild with a pain rated from 1 to 5 and severe if the pain was rated from 6 to 10. In 93% of cases, it was reported being severe and only in 7% was reported as mild. These headaches were continuous in 36% of cases and intermittent in 64%. A fundus oculi examination was available in 15 out of 17 patients. Papilloedema was reported in only 33% of these patients. Nausea and vomiting were reported in 35.3% of patients. Consciousness alterations were reported in 2 patients (12%); both of these patients had papilloedema. No seizures were reported. There was only one completely asymptomatic patient in which the cyst and the associated subdural collection were discovered as an incidental finding.

Microsurgical fenestration of the cyst into the skull base cisterns was the procedure of choice in 76% of patients; in 2 cases, basal fenestration was judged too hazardous due to the finding of thick basal arachnoid, and only subdural fenestration was performed. We usually enter the cyst and check if the skull base arachnoid is clear enough to permit safe fenestration. If it is safely possible, we always aim to perform a basal fenestration; otherwise if it is judged too hazardous, we

Table 2 Case series. Clinical and radiological presentation, treatment, and outcomes at follow-up

Patient	Sex, age	Galassi type cyst	Side of the cyst	Subdural collection's side	Subdural collection's content	Headache (I = intermittent, C = continuous) (M = mild, S = severe)	Nausea and vomiting	Fundus oculi	Other findings
1	F, 11	2	Left	Left	Csf	I, S	No	Papilloedema (improved after surgery)	Exophtalmus, mild left facial deficit
2	M, 6	3	Bilateral	Left	Chronic hematoma	I, S	Yes	Normal	
3	M, 10	2	Right	Right	Chronic hematoma	I, M	No	Papilloedema more on the right side (improved after surgery)	Right exophtalmus
4	M, 5	2	Left	Left	Subacute hematoma	C, S	Yes	Papilloedema (improved after surgery)	
5	M, 9	2	Left	Bilateral	Csf	C,S	Yes	Normal	
6	M, 14	3	Left	Bilateral	Csf	I, S	No	Normal	
7	M, 6	3	Right	Bilateral	Csf	I, S	Yes	Normal	Irritability
8	F, 1	2	Left	Bilateral right > left	Csf	No	No	Normal	Macrocrania
9	M, 13	2	Left	Left	Subacute hematoma	I, S	Yes	Normal	
10	F, 10	3	Right	Bilateral	Csf	C, S	No	Normal	
11	M, 13	3	Left	Left	Csf	I, S	Yes	Normal	
12	M, 3 months	1	Bilateral	Bilateral	Left csf, right subacute	No	No	Normal	Macrocrania
13	F, 8	2	Left	Left	Csf	I, S	No	Not available	
14	M, 10	2	Left	Bilateral	Csf	No	No	Not available	
15	M, 7	3	Left	Left	Csf	C, S	No	Papilloedema (resolved after surgery)	
16	M, 3	3	Right	Bilateral	Csf	C, S	Yes	Papilloedema (improved after surgery)	
17	M, 7	2	Left	Left	Chronic hematoma	I, S	No	Normal	Mild left palpebral ptosis

Patient	Surgical treatment	Complications	Rescue surgery	Follow-up time (months)	Cyst's volume at follow-up	Galassi classification at follow-up	Symptoms at follow-up
1	Basal fenestration			27	Reduction	1	Regressed
2	(1) Bilateral cysto-peritoneal shunt, (2) endoscopic right basal fenestration, (3) left basal fenestration not possible			54	Bilateral reduction	2	Regressed
3	Subdural fenestration (basal technically not possible)	Homolateral epidural hematoma	Hematoma evacuation	48	Slight reduction	2	Regressed
4	Emergent burr holes for hematoma drainage and subsequent basal fenestration (cyst was not detected at the first CT)			48	Stable	2	Regressed
5	Basal fenestration			40	Reduced	1	Regressed
6	Basal fenestration			44	Slightly reduced	3	Regressed
7	Basal fenestration			39	Slightly increased	3	Regressed
8	Basal fenestration			21	Slightly reduced	2	Normalized skull
9	Basal fenestration			29	Stable		Persisting headache and vomiting (1/month)
10	External cyst derivation and subsequent subdural fenestration			36	Slightly reduced	3	Regressed
11	Basal fenestration	CSF fistula	Revision	20	Reduced	1	Regressed
12	Burr holes for hematoma			15	Disappeared	0	Regressed
13	Basal fenestration			6	Reduced	2	Regressed
14	Basal fenestration			16	Slightly reduced	2	No symptoms
15	Basal fenestration			16	Stable	3	Regressed
16	Basal fenestration			3	Slightly reduced	3	Regressed
17	Basal fenestration			56	Reduced	1	Regressed

fenestrate the cyst only into the subdural space. We think that this procedure, when safely feasible, is the most effective in relieving symptoms and preventing re-rupture of the cyst.

We have performed shunting procedures in 2 patients in order to immediately decompress the cyst and fenestrate it in a second surgical procedure. In one case, bilateral Galassi type III cysts were first shunted into the peritoneum; then, in a second surgical time, the right cyst was endoscopically fenestrated into basal cisterns and in a third operation, the same procedure was attempted on the left side but not performed because of excessively high risks due to thick basal arachnoid. On the right side, the shunt was ligated and left in place; on the other side, it was kept functioning. The second patient was treated first with external cyst derivation and later with subdural fenestration because basal fenestration was not possible.

Only one patient of 3 months of age was treated with hematoma drainage via burr holes without approaching the cyst. We thought that in a small child, this was the safest procedure, avoiding risks of a more invasive surgery. At follow-up, the cysts disappeared and no further intervention was needed.

Interestingly, one patient presented with a spontaneous subacute hematoma, isodense to brain parenchyma, and was treated with burr holes in an urgent surgery setting; a postoperative CT scan revealed the presence of a Galassi type II temporal cyst that was then treated in a second surgical time with microsurgical fenestration into basal cisterns. We reported two surgical complications: one epidural hematoma that needed surgical evacuation and one CSF fistula that needed a revision surgery.

The average postoperative follow-up was 30.5 months. All patients experienced complete regression of symptoms at follow-up except for one patient that still complained of headaches and vomiting approximately once a month. Even if cyst volume reduction was not the purpose of treatment, reduction resulting in a lower postoperative Galassi classification type was reported in 37% of the cysts. Thirty-two percent had a slight visible reduction without lowering of the Galassi score, 16% were found to be stable at follow-up, and 2 bilateral cysts (10%) disappeared. Only 1 (5%) Galassi type 3 cyst showed slight increase in volume. The latter was anyway clinically asymptomatic at follow-up. No cases of hydrocephalus were reported at follow-up.

Discussion

Presentation

Arachnoid cysts of the middle cranial fossa account for 34% of all intracranial arachnoid cysts in adults [52] and 46% of all intracranial arachnoid cysts in children [2]. Although the majority of AC remain stable during life, natural history can quickly change by rupture of the cyst, usually defined by development of intracystic hemorrhage or subdural hematomas or hygromas. Minor head trauma is a known risk factor for AC rupture. Even if not usual, spontaneous rupture can also happen.

We have found 57 cases of spontaneous MFAC rupture reported in the literature (Table 1); 72.7% were males and 27.3% were females. Mean age was 16.7 years (range 1–47 years), higher than the mean age of our patients of 8.3 years (range 3 months–14 years) (Table 3).

Arachnoid cyst volume is considered a risk factor for rupture, especially those cysts larger than 5 cm in maximal diameter which have a higher risk of rupture [53]. This latter finding seems not to be confirmed by our series. We have divided patients according to Galassi's classification (Table 2) of MFAC and compared our results with those of a large series of unruptured symptomatic MFAC [54]. In our series, 6% were type 1, 53% were type 2, and 41% were type 3, while in the reference article, they were 15% type 1, 54% type 2, and 31% type 3. We may hypothesize that type 1 MFAC is less prone to spontaneous rupture, while type 2 cysts have a slightly higher risk. Unfortunately, these data were not comparable with the data from our literature review because we found that AC volumes were rarely reported.

The most frequent MFAC radiological sign of rupture is a subdural collection; although rare, a purely intracystic bleeding is possible. While no patients from our series presented with purely intracystic hemorrhage, we found two such patients (3.5%) reported in the literature [14, 39].

In our series, the subdural collection was most frequently a hygroma while in literature (where patient's mean age was higher than in our series), the most frequent finding was a chronic hematoma. A subacute subdural hematoma as MFAC rupture presentation is possible; conversely acute spontaneous subdural hematoma seems to be exceptional as we have found only one reported case [13].

Ruptured MFAC is more frequently found on the left side with a 2:1 ratio.

Interestingly, all patients with bilateral subdural collection had the same collection content on both sides except one patient [13] in whom there was a hygroma homolateral to the cyst and a contralateral subacute hematoma. This might be explained by a rebleeding inside the contralateral collection.

Clinical findings at presentation were unfortunately available in only 47 out of 57 patients from our literature review.

In literature, 95.9% of patients reported headache as presenting symptom; in our series, only 82% had headache at presentation. It is of note that 2 of the 3 patients that did not report headache in our series were under 1 year of age, so that the still opened skull sutures allowed enlargement of the skull compensating the raised intracranial pressure. This is confirmed by the finding of macrocrania reported in both of these patients. In agreement with this explanation, 1 of the 2 patients from the literature that did not report headache was 2 years old

Table 3 Result comparison between our series and literature

		Gaslini Children's Hospital	Literature review
	Patients	17	57
Presentation	Age at presentation in years, mean (range)	8.3 (3 months–14 years)	16.6 (1–47)
	Sex	76.5% males, 23.5% females	73.2% males, 26.8% females
	Side of the cyst	64.7% left, 23.5% right, 11.8% bilateral	62.5% left, 32.1% right, 5.3% bilateral
	Exclusively intracystic bleeding	0	3.5%
	Content of subdural collection	76% hygroma, 12% chronic hematoma, 12% subacute hematoma	25% hygroma, 56.6% chronic hematoma, 16.7% subacute hematoma, 1.7% acute hematoma
	Headache	82.3%	96%
	Nausea and vomit	41.2%	54%
	Papilloedema	33.3%	11 reported cases
	Asymptomatic	5.9%	0
	Impaired consciousness	11.8%	6%
Treatment and outcome	Treatment	5.5% hematoma drainage only, 5.5% shunting procedures, 11.2% subdural cyst fenestration, 77.8% basal cyst fenestration	16.4% conservative, 27.3% hematoma drainage only, 10.9% shunting procedures, 25.4% subdural cyst fenestration, 12.7% basal cyst fenestration, 7.3% AC resection
	Mean follow-up time	29.9 months	11 months
	Symptoms at follow-up	94.1% asymptomatic, 5.9% partial recovery	89.1% asymptomatic, 8.7% partial recovery, 2.2% no improvement

[17] and had macrocrania at presentation that was likely caused by the raised intracranial pressure. At presentation, he complained of nausea and vomiting. Given the fact that he had a chronic subdural hematoma, we suppose that AC rupture happened at a time when sutures were still opened leading to an increase of skull circumference; conversely by the time of presentation, cranial sutures were closed so that we may hypothesize that rebleeding or expansion of the subdural hematoma led to clinical symptom development. He was probably too young to be able to report headache, and nausea and vomiting were the only visible symptoms of intracranial hypertension.

Although we do not know from our literature review how many patients underwent a fundus oculi examination, papilloedema was reported in 11 patients (19.6%) [12, 13, 16, 18, 19, 26, 32, 35, 43, 44]. In our series, 33% of the patients who underwent a fundus oculi examination had papilloedema. Even if we do not have direct data regarding intracranial pressure neither from our experience nor from literature, we may suppose that headache in these patients is caused by meningeal irritation by blood instead of being a symptom of intracranial hypertension, because of the presence of papilloedema in only a minority of cases.

We did not find any correlation between papilloedema and the presence of nausea and vomiting neither in literature nor in our results. Thus, we hypothesize that in a substantial number of patients, these are signs of meningeal irritation as well.

Neurological deficits at presentation are usually found in a minority of patients with ruptured MFAC; in the literature, we found that the most frequently reported ones were hemiparesis, present in 8.2% of cases and ocular palsy in 10.2%. In our series, neurological deficits were even less frequently reported, possibly because of the younger age of our patients.

Treatment

Consensus regarding the procedure of choice in ruptured MFAC is lacking. Different surgical treatments were proposed that may address the cyst itself or only the subdural collection. Subdural fluid may be drained via single or multiple burr holes or craniotomy. Arachnoid cyst itself is treated with direct resection of the cyst, with shunting procedures into the peritoneum or into subcutaneously implanted reservoirs or with fenestration of cyst walls either into the subdural space or into the skull base cisterns.

Conservative treatment with follow up alone or with medical therapy with acetazolamide or steroids has also been proposed. Spontaneous resolution of MFAC has been reported for unruptured cysts since 1985 [55]. We have found 9 papers reporting conservative treatment for ruptured MFAC [7, 10–17]. Good clinical outcomes are reported but long-term follow-up is lacking in most of these reports. One of these cases needed surgical fenestration due to enlargement of subdural hygroma with midline shift after 2 months of observation along with acetazolamide therapy [16].

Surgery may address only the subdural collection which is usually thought to be the cause of brain compression leading to symptoms. Burr holes or craniotomy may be used to evacuate the hematoma. This treatment is a less invasive option with shorter surgical time, but it does not resolve the cyst itself that may remain excluded from CSF circulation and therefore carries the risk rupture recurrence. We have generally found good outcomes reported except for few cases that reported persistent symptoms at follow-up even if improved (Table 1).

Cysts may be decompressed by means of shunting procedures that may aim to create a shunt directly between cyst and peritoneum or between subdural space and peritoneum. These procedures allow immediate decompression of the subdural collection preventing at the same time future complications deriving from re-rupture or cyst expansion. Disadvantages are risks of shunt failure and lifelong shunt dependence.

Treatment may directly address the cyst by means of fenestration of the capsule decompressing the cyst in order to reduce mass effect and risk of rupture. Fenestration may be performed either into the subdural space or into the skull base cisterns. In these procedures, the subdural collection is encountered on the surgical route and therefore drained.

All the patients from our series experienced complete symptom relief at follow-up, except for the single one that still complained of rare headaches (Table 2).

Microsurgical fenestration of the cyst has been reported in literature (Table 1) even if technical limitations due to patient anatomical differences have been scantly described. Regarding surgical fenestration of ruptured MFAC, we have found good outcomes reported in the literature (Table 1). These data however lack of a consistent follow-up period (mean 0.5 months for subdural fenestration procedures and 7.2 months for basal fenestration procedures). All cases for whom follow-up data were available showed full recovery without procedure-related complications or rupture recurrence. Our series, considering only microsurgical basal cyst fenestration procedures, with a mean follow-up of 29.9 months, confirms the effectiveness and safety of these techniques adding an adequate postoperative follow-up time to the already reported cases. Even if the current best level of evidence regarding the treatment of choice for these patients is still low, based on our results and our literature review, we think that microsurgical fenestration of the cyst in the skull base cisterns, when safely feasible, is an efficient and safe procedure that addresses both the acute compression and the risk of rupture recurrence. This procedure also avoids the risks related to implantation of permanent devices that is needed in shunting procedures.

Due to the retrospective nature of our study and the rarity of the pathology, our recommendation is still based on weak evidences and future prospective, and more targeted studies are needed. Current evidences due to the limited reported follow-up do not seem to clearly show that conservative treatment is a good option for these patients; thus, at today, we tend to discourage this approach. Further studies with better evidences and longer follow-up times are mandatory to evaluate this option.

Conclusions

Spontaneous rupture of MFAC is a rare but possible event. Development of a subdural collection is the most frequent radiological sign of rupture. Headache and nausea are the most frequent symptoms at presentation; focal neurological deficits are relatively rare but possible.

From our experience and from the presented literature review, we think that spontaneously ruptured MFAC will benefit from operative rather than conservative treatment. Of all the surgical procedures reported, we suggest that microsurgical fenestration of the cyst into the skull base cisterns, when safely feasible, is the most effective and safe procedure addressing both the acute compression and the risk of rupture recurrence.

Limitations

Patients from our series have been treated according to surgeon's preference based on the single cases; the absence of standardization of treatment may limit the comparability of our results.

Even if to our knowledge this is the largest reported series of patients treated for spontaneous rupture of MFAC, due to the rarity of the pathology, numbers are still relatively small.

Given the retrospective nature of our study, we think that this topic needs future prospective randomized trials investigating the best treatment for these patients.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

References

1. Albuquerque FC, Giannotta SL (1997) Arachnoid cyst rupture producing subdural hygroma and intracranial hypertension: case reports. Neurosurgery 41:951–956. https://doi.org/10.1097/00006123-199710000-00036
2. Al-Holou WN, Yew AY, Boomsaad ZE et al (2010) Prevalence and natural history of arachnoid cysts in children. J Neurosurg Pediatr 5:578–585. https://doi.org/10.3171/2010.2.PEDS09464
3. Galassi E, Tognetti F, Frank F et al (1985) Infratentorial arachnoid cysts. J Neurosurg 63:210–217. https://doi.org/10.3171/jns.1985.63.2.0210

4. Higashi S, Yamashita J, Yamamoto Y, Izumi K (1992) Hemifacial spasm associated with a cerebellopontine angle arachnoid cyst in a young adult. Surg Neurol 37:289–292. https://doi.org/10.1016/0090-3019(92)90155-g
5. Hoffman HJ, Hendrick EB, Humphreys RP, Armstrong EA (1982) Investigation and management of suprasellar arachnoid cysts. J Neurosurg 57:597–602. https://doi.org/10.3171/jns.1982.57.5.0597
6. Gosalakkal JA (2002) Intracranial arachnoid cysts in children: a review of pathogenesis, clinical features, and management. Pediatr Neurol 26:93–98. https://doi.org/10.1016/S0887-8994(01)00329-0
7. Adin ME, Yıldız MS, Deniz MA, Behzadi AH, Mata-Mbemba D (2018) Arachnoid cysts with spontaneous intracystic hemorrhage and associated subdural hematoma: report of management and follow-up of 2 cases. Radiol Case Reports 13:516–521. https://doi.org/10.1016/j.radcr.2017.12.006
8. Parsch CS, Krauß J, Hofmann E et al (1997) Arachnoid cysts associated with subdural hematomas and hygromas: analysis of 16 cases, long-term follow-up, and review of the literature. Neurosurgery 40:483–490. https://doi.org/10.1097/00006123-199703000-00010
9. You C, Shrestha R (2014) Spontaneous chronic subdural hematoma associated with arachnoid cyst in children and young adults. Asian J Neurosurg 9:168–172. https://doi.org/10.4103/1793-5482.142739
10. Inoue T, Matsushima T, Tashima S et al (1987) Spontaneous disappearance of a middle fossa arachnoid cyst associated with subdural hematoma. Surg Neurol 28:447–450. https://doi.org/10.1016/0090-3019(87)90228-X
11. Rakier A, Feinsod M (1995) Gradual resolution of an arachnoid cyst after spontaneous rupture into the subdural space: case report. J Neurosurg 83:1085–1086. https://doi.org/10.3171/jns.1995.83.6.1085
12. Choong CT, Lee SH (1998) Subdural hygroma in association with middle fossa arachnoid cyst: acetazolamide therapy. Brain Dev 20: 319–322. https://doi.org/10.1016/S0387-7604(98)00039-4
13. Henriques JG, Pianetti Filho G, KSW H et al (2007) Spontaneous acute subdural hematoma contralateral to an arachnoid cyst. Arq Neuropsiquiatr 65:1034–1036. https://doi.org/10.1590/S0004-282X2007000600025
14. Katsaros VK, Flaskas T, Stavrinou LC et al (2008) Intracranial hemorrhagic arachnoid cyst: the role of diffusion-weighted imaging. Neuroradiol J 21:543–546. https://doi.org/10.1177/197140090802100412
15. Lohani S, Robertson RL, Proctor MR (2013) Ruptured temporal lobe arachnoid cyst presenting with severe back pain. J Neurosurg Pediatr 12:281–283. https://doi.org/10.3171/2013.6.PEDS13122
16. Marques IB, Vieira Barbosa J (2014) Arachnoid cyst spontaneous rupture. Acta Medica Port 27:137–141. https://doi.org/10.20344/amp.2017
17. Bora A, Yokuş A, Batur A, Bulut MD, Yavuz A, Gülşen İ, Özgökçe M, Arslan M (2015) Spontaneous rupture of the middle fossa arachnoid cyst into the subdural space: case report. Polish J Radiol 80: 324–327. https://doi.org/10.12659/PJR.893928
18. Page A, Paxton RM, Mohan D (1987) A reappraisal of the relationship between arachnoid cysts of the middle fossa and chronic subdural. J Neurol Neurosurg Psychiatry 50:1001–1007. https://doi.org/10.1136/jnnp.50.8.1001
19. Rogers MA, Klug GL, Siu KH (1990) Middle fossa arachnoid cysts in association with subdural haematomas. A review and recommendations for management. Br J Neurosurg 4:497–502. https://doi.org/10.3109/02688699008993799
20. Oka Y, Kumon Y, Ohta S et al (1994) Chronic subdural hematoma associated with middle fossa arachnoid cysts-three case reports. Neurol Med Chir (Tokyo) 34:95–99
21. Ibarra R, Kesava PP (2000) Role of MR imaging in the diagnosis of complicated arachnoid cyst. Pediatr Radiol 30:329–331. https://doi.org/10.1007/s002470050751
22. Mori K, Yamamoto T, Horinaka N, Maeda M (2002) Arachnoid cyst is a risk factor for chronic subdural hematoma in juveniles: twelve cases of chronic subdural hematoma associated with arachnoid cyst. J Neurotrauma 19:1017–1027. https://doi.org/10.1089/089771502760341938
23. Chan JY, Huang CT, Liu YK et al (2008) Chronic subdural hematoma associated with arachnoid cyst in young adults: a case report. Kaohsiung J Med Sci 24:41–44. https://doi.org/10.1016/S1607-551X(08)70072-X
24. Patel RA, Levy ML, Crawford JR (2013) Spontaneous subdural haemorrhage in a child with bilateral middle cranial fossa arachnoid cysts. BMJ Case Rep:1–2. https://doi.org/10.1136/bcr-2013-200802
25. Kang J, Whang K, Hong S-K, Pyen JS, Cho SM, Kim JY, Kim SH, Oh JW (2015) Middle meningeal artery embolization in recurrent chronic subdural hematoma combined with arachnoid cyst. Korean J Neurotrauma 11:187–190. https://doi.org/10.13004/kjnt.2015.11.2.187
26. Yüksel MO, Gürbüz MS, Senol M, Karaarslan N (2016) Spontaneous subdural haematoma developing secondary to arachnoid cyst rupture. J Clin Diagnostic Res 10:PD05–PD06. https://doi.org/10.7860/JCDR/2016/21056.8708
27. Wu X, Li G, Zhao J, Zhu X, Zhang Y, Hou K (2018) Arachnoid cyst–associated chronic subdural hematoma: report of 14 cases and a systematic literature review. World Neurosurg 109:e118–e130. https://doi.org/10.1016/j.wneu.2017.09.115
28. Kaszuba M, Tan L, Moftakhar R, Kasliwal M (2018) Nontraumatic subdural hematoma and intracystic hemorrhage associated with a middle fossa arachnoid cyst. Asian J Neurosurg 13:116–118. https://doi.org/10.4103/1793-5482.181124
29. Poirrier ALML, Ngosso-Tetanye I, Mouchamps M, Misson JP (2004) Spontaneous arachnoid cyst rupture in a previously asymptomatic child: a case report. Eur J Paediatr Neurol 8:247–251. https://doi.org/10.1016/j.ejpn.2004.04.005
30. Cakir E, Kayhankuzeyli SOC et al (2004) Arachnoid cyst rupture with subdural hygroma: case report and literature review. Neurocirugia (Astur) 15:72–75
31. Gil-Gouveia R, Miguens J, Coiteiro D (2010) Rupture of middle fossa arachnoid cyst simulating new-onset migraine with aura: clinical notes. Headache 50:314–319. https://doi.org/10.1111/j.1526-4610.2009.01573.x
32. Khilji MF, Jeswani NL, Hamid RS, Al Azri F (2016) Spontaneous arachnoid cyst rupture with subdural hygroma in a child. Case Rep Emerg Med 2016:1–3. https://doi.org/10.1155/2016/6964713
33. Cullis PA, Gilroy J (1983) Arachnoid cyst with rupture into the subdural space. J Neurol Neurosurg Psychiatry 46:454–456. https://doi.org/10.1136/jnnp.46.5.454
34. Eustace S, Toland J, Stack J (1992) CT and MRI of arachnoid cyst with complicating intracystic and subdural haemorrhage. J Comput Assist Tomogr 16:995–997. https://doi.org/10.1097/00004728-199211000-00034
35. Iaconetta G, Esposito M, Maiuri F, Cappabianca P (2006) Arachnoid cyst with intracystic haemorrhage and subdural haematoma: case report and literature review. Neurol Sci 26:451–455. https://doi.org/10.1007/s10072-006-0532-z
36. Ziaka DS, Kouyialis AT, Boviatsis EJ, Sakas DE (2008) Asymptomatic massive subdural hematoma in a patient with bitemporal agenesis and bilateral temporal arachnoid cysts. South Med J 101:324–326. https://doi.org/10.1097/SMJ.0b013e3181646a5c
37. Hong J-C, Kim M-S, Chang C-H, Kim S-H (2008) Arachnoid cyst with spontaneous intracystic hemorrhage and chronic subdural

hematoma. J Korean Neurosurg Soc 43:54–56. https://doi.org/10.3340/jkns.2008.43.1.54
38. Patel AP, Oliverio PJ, Kurtom KH, Roberti F (2009) Spontaneous subdural hematoma and intracystic hemorrhage in an arachnoid cyst. Radiol Case Rep 4:298. https://doi.org/10.2484/rcr.v4i3.298
39. Gũndũz B, Yassa MIK, Ofluoğlu E et al (2010) Two cases of arachnoid cyst complicated by spontaneous intracystic hemorrhage. Neurol India 58:312–315. https://doi.org/10.4103/0028-3886.63795
40. Hall A, White MAJ, Myles L (2017) Spontaneous subdural haemorrhage from an arachnoid cyst: a case report and literature review. Br J Neurosurg 31:607–610. https://doi.org/10.1080/02688697.2016.1187255
41. Aydogmus E, Hicdonmez T (2017) Spontaneous intracystic haemorrhage of an arachnoid cyst associated with a subacute subdural hematoma: a case report and literature review. Turk Neurosurg:1–5. https://doi.org/10.5137/1019-5149.jtn.20885-17.2
42. Ergün R, Ökten AI, Beşkonakli E et al (1997) Unusual complication of arachnoid cyst: spontaneous rupture into the subdural space. Acta Neurochir 139:692–694. https://doi.org/10.1007/BF01412007
43. Çayli SR (2000) Arachnoid cyst with spontaneous rupture into the subdural space. Br J Neurosurg 14:568–570. https://doi.org/10.1080/02688690050206738
44. Galarza M, Pomata HB, Pueyrredón F et al (2002) Symptomatic supratentorial arachnoid cysts in children. Pediatr Neurol 27:180–185. https://doi.org/10.1016/S0887-8994(02)00414-9
45. Slaviero F, Frighetto L, Azambuja ND et al (2008) Middle cranial fossa arachnoid cysts complicated with subdural collections. Arq Neuropsiquiatr 66:913–915. https://doi.org/10.1590/S0004-282X2008000600033
46. Liu Z, Xu P, Li Q, Liu H, Chen N, Xu J (2014) Arachnoid cysts with subdural hematoma or intracystic hemorrhage in children. Pediatr Emerg Care 30:345–351. https://doi.org/10.1097/PEC.0000000000000128
47. Chandra VVR, Prasad BC, Subramanium CS, Kumar R (2015) Spontaneous intracystic hemorrhage complicating an intracranial arachnoid cyst. J Neurosci Rural Pract 6:629–630. https://doi.org/10.4103/0976-3147.169773
48. Sener RN Arachnoid cysts associated with post-traumatic and spontaneous rupture into the subdural space. Comput Med Imaging Graph 21:341–344
49. Levy ML (2005) Microsurgical Keyhole Approach for Middle Fossa. 56:1138–1145. https://doi.org/10.1227/01.NEU.0000155088.70249.45
50. Xu S, Wang Y, Luo Q et al (2016) Endoscopic fenestration of twenty-six patients with middle fossa arachnoid cyst. J Craniofac Surg 27:973–975. https://doi.org/10.1097/SCS.0000000000002624
51. Galassi E, Tognetti F, Gaist G et al (1982) Ct scan and metrizamide CT cisternography in arachnoid cysts of the middle cranial fossa: classification and pathophysiological aspects. Surg Neurol 17:363–369. https://doi.org/10.1016/0090-3019(82)90315-9
52. Al-Holou WN, Terman S, Kilburg C et al (2013) Prevalence and natural history of arachnoid cysts in adults. J Neurosurg 118:222–231
53. Cress M, Kestle JRW, Holubkov R, Riva-Cambrin J (2013) Risk factors for pediatric arachnoid cyst rupture/hemorrhage: a case-control study. Neurosurgery 72:716–722. https://doi.org/10.1227/NEU.0b013e318285b3a4
54. Zeng L, Feng L, Wang J, Li J, Wang Y, Chen J, Chen J, Lei T, Li L (2008) Comparative study on two surgical procedures for middle cranial fossa arachnoid cysts. J Huazhong Univ Sci Technol Med Sci 28:431–434. https://doi.org/10.1007/s11596-008-0412-2
55. Beltramello A, Mazza C (1985) Spontaneous disappearance of a large middle fossa arachnoid cyst. Surg Neurol 24:181–183. https://doi.org/10.1016/0090-3019(85)90182-X