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CASE REPORT Open Access

An arachnoid cyst rupture complicated with subdural hygroma in a middle-aged woman: a case report and review of the literature

Fakhreddin Saboonihaa[1*] and Ghasem Baghershahi[1]

Abstract

Background Arachnoid cysts (ACs) account for about 1% of all intracranial mass lesions. The Sylvian fissure is the most common site of ACs which are usually asymptomatic. Traumatic rupture of arachnoid cysts complicated with subdural hygroma is a relatively rare entity in adults especially in females. There is no consensus on their management and each case could add to previous experiences leading to more uniform therapeutic measurements. This case encompasses a combination of watchful strategy, limited surgery and ultimately successful cyst fenestration which highlights a good outcome without complications by adopting a stepwise procedure.

Case presentation Here, the case of a 51-year-old Persian woman being presented with a 2-month history of a localized headache which had begun 4 days after a minor head trauma. Early brain computed tomography (CT) obtained in another center at 4th day of post-injury which misinterpreted as an old infarct. Repeated CT in our center revealed a large AC of Sylvian fissure complicated with subdural hygroma. Initially a conservative management pursued but due to worsening of the symptoms, a burr-hole craniotomy performed for hygroma evacuation that subsequently followed by cyst fenestration about 1-month later.

Conclusion Previous awareness about radiographic characteristics of ACs as well as employing appropriate imaging modality are necessary for correct and timely diagnosis of symptomatic patients after minor head trauma because incidentally found ACs might be the underpinning causes of symptoms. Also, following a multistage therapeutic process along with the involvement of patients in decision-making are of paramount importance.

Keywords Arachnoid, Cyst, Sylvian, Hygroma, Rupture, Case report, Multiomic

Introduction

Arachnoid cysts (ACs) are benign collections of cerebrospinal fluid (CSF) in the arachnoid membranes which occur most often in the middle cranial fossa followed by the posterior fossa, convexity and suprasellar region [1, 2]. ACs are seen in about 4% of population and account for 1% of all intracranial masses [1, 3]. Most ACs are congenital (primary) in origin but a minority are secondary to head trauma, neoplasms, leptomeningitis and surgery [1] Ventriculomegaly or hydrocephalus is seen in many patients with interhemispheric ACs and is closely related to brain ventricular abnormalities rather than the mass effect only but is rarely encountered in the middle fossa ACs [28]. Corpus callosum agenesis is common among the intracranial abnormalities associated with ACs [29]. The majority of ACs are clinically asymptomatic but rarely, traumatic or spontaneous rupture of ACs might

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occur with cysts larger than 5 cm are more susceptible [4, 5]. Moreover, middle cranial fossa cysts are more likely to undergo traumatic subdural hematoma or hygroma and the risk of their rupture is twofold higher than the other ACs [2, 7]. Tearing of these cysts may present with headache, seizure and rarely hemiparesis [3]. CT imaging is often sufficient for diagnosis of ACs which show a Hounsfield attenuation values range from 10 to 20 (hypodense) [27].

This case describes a middle-aged woman with a 2-month history of progressive headache after a minor head trauma with a Galassi type II Sylvian fissure AC complicated with hygroma. Being in her midlife, female gender, family history of subependymoma in her nephew, initial misdiagnosis as an old infarct despite the lack of apparent motor deficits, obvious thinning of the ipsilateral temporal bone in the areas without direct contact with the cyst, no hemorrhagic transformation despite of long duration of hygroma as well as radiologically apparent outer membrane of the cyst demarcating subarachnoid and subdural spaces with a wide opening to the subdural space all are featured characteristics of this case.

Case report

A 51-year-old woman presented to our center with a 2-month history of progressive localized headache which had been developed 4 days after a minor head trauma to the left temporal area. Initial brain CT scan which performed in another center revealed a large hypodensity at the left middle fossa that attributed mistakenly by treating neurologist to an old middle cerebral artery infarction despite the absence of significant motor deficits on index visit (Fig. 1). The patient was treated with ibuprofen 400 mg twice daily, without any response. Seven weeks later, the patient visited in our center because of the worsening of the headache and emerging new symptoms including morning nausea and vomiting. There was no history of previous neurological illness. She did not use illicit drugs or alcohol. The family history included rheumatoid arthritis (RA) in her smaller sister. On examination, the patient was conscious and oriented to time, location and person. Pupils were normal size and reactive and other cranial nerves were intact. An upward left Babinski reflex test was seen. Based on the history and examinations, raised intracranial pressure was suspected and a repeat brain CT scan showed reduced size of the initial lesion which linked to a large left frontotemporoparietal subdural collection isodense to CSF without significant midline shift consistent with an AC complicated with subdural hygroma (Fig. 2). Because of the patient's preference, a conservative management pursued. Three weeks later, headache intensified with superimposed diplopia. Magnetic resonance imaging (MRI) revealed a large AC whose outer membrane ruptured into the subdural space leading to a large left subdural effusion and severe midline shift (Fig. 3). The patient underwent a

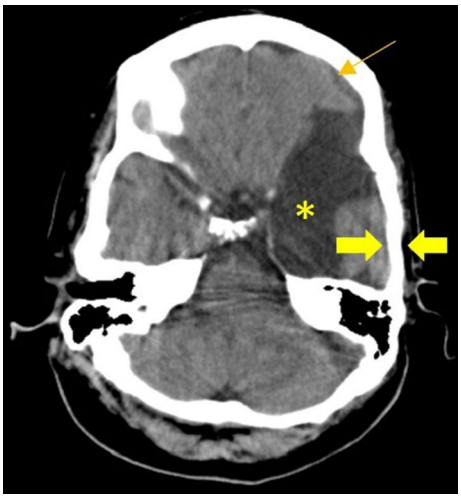

Fig. 1 Initial unenhanced CT scan of the head which obtained because of the headache onset at the 4th day after minor head trauma to the left temporal bone. Axial view shows a rectangular-shaped lesion of cerebrospinal fluid density (Hounsfield unit, + 8) filling the middle cranial fossa just at the anatomic site of Sylvian fissure consistent with Galassi type II arachnoid cyst (asterisk).A small subdural collection at the left frontal region connecting to the cyst is also evident (orange arrow). Moreover, thinning of the ipsilateral temporal bone was seen (yellow-filled arrows)

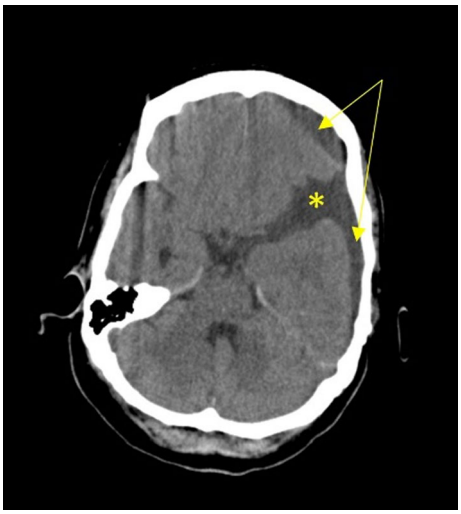

Fig. 2 Non-contrast brain CT scan performed in our center because of the headache exacerbation 7 weeks after trauma. The arachnoid cyst reduced in size (asterisk) extending to a left frontotemporoparietal subdural collection isodense to cerebrospinal fluid suggestive of rupturing of the cyst into the subdural space complicated with hygroma (yellow arrows)

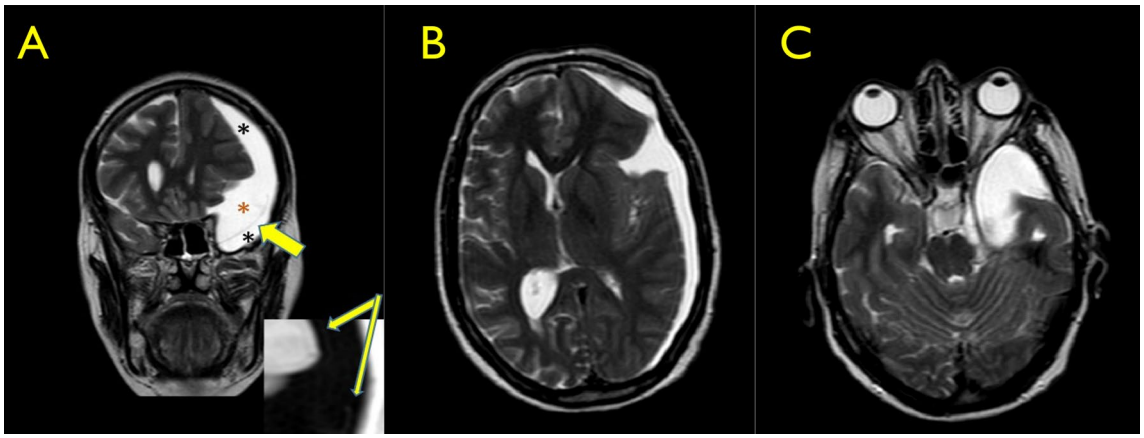

Fig. 3 MRI of the brain requested 10 weeks post-injury because of the headache aggravation superimposed by diplopia. **A** Coronal T2-weighted MRI revealed a large arachnoid cyst in the left middle cranial fossa containing fluid of CSF intensity (brown asterisk) extending to subdural space(black asterisks).The membrane separating arachnoid cyst and the subdural hygroma is clearly obvious (yellow-filled arrows in the main image and inset). Note: For better delineation of the upper part of the cyst membrane, the inset has been artificially inverse-colored. **B** Axial T2-weighted image revealed a large left frontotemporoparietal subdural hygroma leading to severe midline shift and nearly complete effacement of left lateral ventricle **C** Axial T2-weighted image showing the rectangular arachnoid cyst at the middle fossa corresponding to Galassi type II arachnoid cyst

burr-hole craniotomy for evacuation of the hygroma. The symptoms decreased but not completely resolved. Post-operation CT scan revealed markedly reduced volume of subdural hygroma but the cyst remained unchanged probably due to communication with underlying subarachnoid space. However, during surgery a slit-valve or tunnel-shaped canal was not seen grossly (Fig. 4). Hence, a repeat surgery was scheduled for 1-month later in which the cyst adjoined to the underlying arachnoid space with multiple fenestrations with complete resolution of the symptoms. The brain CT scan obtained immediately after surgery did not show a significant reduction in the cyst size. However, long-term imaging follow-up was planned for better evaluation of the changes in cyst morphology.

Discussion

ACs are leptomeningeal-lined CSF-filled sacs characterized by hyperplastic arachnoid cells, increased collagen and the lack of normal spider-like trabeculations. ACs mechanism of formation is not fully understood but the leading theory supposes that they classically result

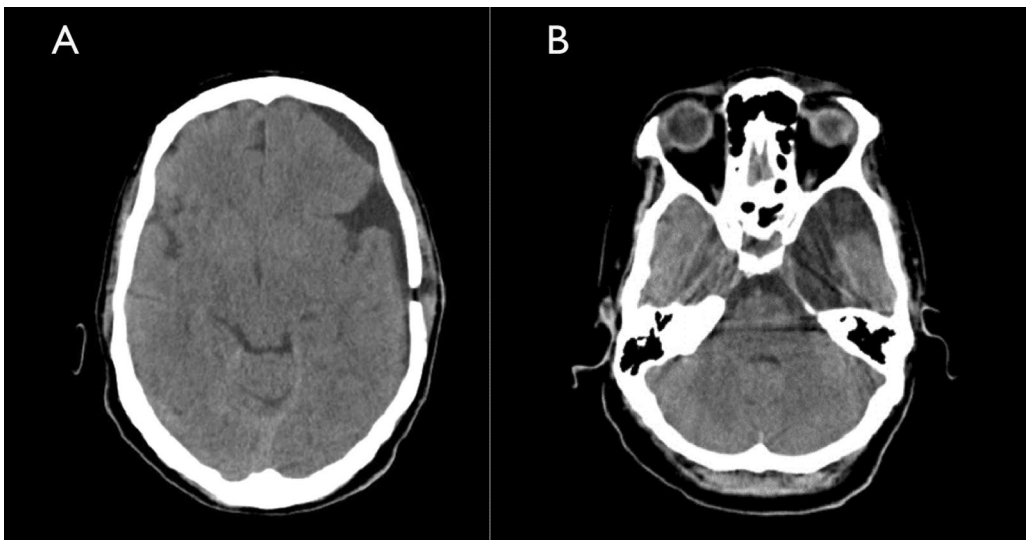

Fig. 4 Unenhanced computed tomography of the brain obtained immediately postoperation. **A** Axial view shows that the size of the left subdural hygroma markedly decreased. The site of burr-hole craniotomy could be seen. **B** axial view demonstrating that the size of cyst did not significantly reduced compared to the preoperative MRI.

from developmental splitting of bilayer arachnoid membrane with subsequent intra-arachnoid space expansion due to a ball-valve mechanism [12, 14]. While most cases are considered idiopathic or congenital, Mendelian inheritance of ACs in some instances such as mucopolysaccharidoses and acrocallosal syndromes has been reported. Additionally, familial clustering, sidedness and gender predominance all argue in favor of an underlying genetic mechanism with about 20% of ACs are attributable to genetic variants [8, 12, 14, 15]. ACs are well associated with some disorders such as glutaric aciduria type 1 and Sjogren syndrome [29]. Also, an association between meningiomas and middle fossa ACs has been reported. By considering the specificity of the location of two disorders, local alterations such as localized release of bone growth factors, e.g., IGF-1,IGF-2 and PDGF which all are involved in the bone formation have been supposed further supporting the idea of an underlying neurodevelopmental process leading to both bone remodeling and cyst formation as in our case [29]. Sylvian arachnoid cysts (SACs) are the most common type of ACs in adult and pediatric patients with a marked tendency to occur in men and on the left side which are also the most susceptible types to be ruptured with a cumulative risk of up to 20% of subdural hematoma [2, 7]. In a recent study carried out by Dirik, et.al (2023), it has been shown that up to 25% of pediatric patients who were diagnosed with primary ACs by MRI, being suffered mental retardation suggesting a maldevelopmental process [13]. Middle cranial fossa cysts could result in thinning and scalloping of the overlying bone even in Galassi type I cysts regardless of their size as well as proptosis because of temporal bone remodeling [19, 20]. They are classified into three types according to Galassi classification. Galassi type 1 cyst is small semicircular cyst confined to the anterior part of temporal fossa showing free communication with the subarachnoid space; type 2 is medium-sized quadrangular cyst with slow connection to the subarachnoid space; and type 3 is large oval cyst with mass effect and midline shift usually without cisternal connection [3, 9, 19]. Middle fossa ACs are generally asymptomatic but rarely might undergo spontaneous enlargement or disappearance as well as traumatic rupture leading to subdural hematoma or hygroma most often occurring in Galassi type II and III cysts [3, 6]. The growth rates of ACs in adults are around 2–3% increase in size which are often lower than pediatric population [26].The most common symptoms of middle fossa ACs is headache [13]. Interestingly, all reported cases of cyst rupture leading to subdural hygroma have been reported in the context of mild head injury and the vast majority of complications have been occurred in the middle cranial fossa cysts. Probable explanations for these observations, may be disappearance of the cysts after severe trauma or death owing to trauma severity itself, malignant complications such as severe subdural or intracystic hemorrhage masking the previous hygroma as well as overlooking the cysts in the setting of multiple injuries. The mechanism of rupture is not known but the compression of SAC against the ipsilateral sphenoid wing or a direct injury to it via thinned temporal bone have been suggested. Hygroma is thought to originate from the disruption of the cyst wall with subsequent CSF leakage into the subdural space. Some authors suggested that the cyst rupture first results in the hygroma formation leading to disruption of the bridging veins and small bleedings which in turn, causes the chronic subdural hematoma. This would explain more prevalent subdural hematomas in adult patients compared to children because the child become symptomatic earlier due to their brain trophic changes [7]. There are much debates regarding the best treatment strategy for ACs in pediatric and adults. Treatment options of asymptomatic patients are controversial but for symptomatic cysts, the treatment modalities include expectant management, cyst shunting and microsurgical or endoscopic fenestration [2]. The surgical approach is determined by the location of the cyst as well as the presence or absence of involvement of the adjacent structures [16]. In terms of endoscopy, the best outcomes have been achieved with suprasellar cysts. But, regarding the middle fossa cysts, endoscopic approach remains controversial with some authors prefer microsurgery over endoscopy in this area [17]. Samuel et.al in their study found that endoscopic and microsurgical approaches result in comparable clinical and radiological outcomes. They showed that shunting was associated with a greater reduction in the cyst volume than other surgical techniques whereas endoscopic fenestration led to a shorter length of admission. In subgroup analysis they detected no significant difference in percentage cyst volume reduction between endoscopic and open surgery (30% vs. 26%). Moreover, the study revealed that the therapeutic benefits of cyst volume reduction depend more on the location of the cyst rather than the absolute volume reduction with the best outcomes in midline and posterior fossa cysts [18]. This finding may explain successful conservative management of otherwise asymptomatic ACs of middle fossa which complicated with subdural hygroma or hematoma. For example, Maher et.al reported a case series of eight pediatric patients presented with subdural hygroma leading to symptomatic presentation in otherwise asymptomatic ACs. Seven out of eight patient managed nonsurgically and the authors suggested that the decision to surgically treat symptomatic hygromas associated with previously asymptomatic arachnoid cysts should be made carefully and only after taking into account the

benign natural course of this condition [10]. Also, Parsch, et.al reported a good outcome with drainage of the subdural fluid or craniotomy in 13 cases and conservative treatment in two cases and concluded that performing cyst diversion or fenestration at the time of drainage of a hematoma or hygroma in previously asymptomatic arachnoid cysts is not necessary for all patients. [11]. Matthew, et.al reported uneventful conservative management of a previously healthy 14-month old male who was diagnosed with a large left AC complicated with a large subdural hygroma and significant midline shift [21].

Beside, we found only two case reports in the literature regarding the concurrent existence of RA and AC in the same patient. The first case was a 34-year-old woman with a long history of RA who had developed a severe headache 10 days prior to presentation and brain CT scan revealed a huge left middle fossa AC [22]. The second one, was a 40-year-old male who was diagnosed with Rhupus that in work-up for his headache, a large symmetric arachnoid cyst of the posterior fossa along with a hypoplastic right transverse sinus had been detected [23]. It is not definitely known whether simultaneous occurrence of RA and AC in these two rare cases is incidental thereby indicating the congenital origin of the ACs or is the result of leptomeningeal or brain tissue inflammation leading to secondary arachnoid cysts. However, by considering the location, sidedness and size of ACs, the hypoplastic transverse sinus in one patient, as well as the lack of clinical evidence of CNS involvement, it seems that the ACs in these two patients were more probably developmental in origin. Whether there is a common genetic susceptibility for RA and AC formation remains to be further elucidated. Our patient's smaller sister being suffering from RA might favor a familial clustering for two disorders.

There are some clinical pearls regarding the presented case. First, the rupture of the cyst into the subdural space after minor head trauma is justified by the thinning of the ipsilateral temporal bone thereby facilitating energy transfer to the inferior structures. Surprisingly, initial brain CT scan showed that the cyst primarily has not been juxtaposed with the thinned area of the temporal bone. This finding could not fully explain the bone remodeling through direct mass effect by cyst itself, rather raises the possibility of a shared neurodevelopmental abnormality in the same side. As a result, the thinned and remodeled temporal bone might be more compliant to the sudden raise in intracranial pressure thereby preventing more acute severe symptoms needing urgent measures. Second, the absence of hemorrhagic transformation in such a large cyst despite of the passage of about 3 months from the incident trauma, demonstrates that the chronicity of hygroma is not sufficient for hemorrhagic conversion in adults and that other additional factors such as the rate of expansion and venous compliance may play more important role. Moreover, wide opening of the outer membrane of the cyst into the subdural space might decrease the pressure gradient between subarachnoid and subdural space thereby preventing bleeding via reduced shear stress imposed on bridging veins. In addition, despite the lack of a significant reduction in the size of the cyst immediately after surgery, the patient's symptoms completely resolved after hygroma evacuation without any recurrence after 3-month follow-up. This finding is concordant with the Hall, et.al study that proposed volume reduction effects are more prominent in the midline and posterior fossa ACs suggesting a constraining effect imposed by dura matter on the brain tissue which quickly removes by draining even small volumes similar to the cardiac tamponade. This finding may clarify the good outcomes which have been reported by some authors using the expectant management of otherwise asymptomatic ACs complicated with subdural hygroma despite incomplete disappearance of the cysts. More importantly, after detailed investigation, it had been revealed that her nephew (the son of her smaller sister) has been suffering a chronic headache with a small well-demarcated right lateral intraventricular mass most consistent with subependymoma in the brain MRI. Interestingly in the 5th edition (2021) of the world health organization classification of central nervous system tumours, a sporadic case of subependymoma with germ-line mutation in the protein tyrosine phosphatase nonreceptor-type 11 gene (PTPN11) in a patient with Noonan syndrome (OMIM 163950) has been reported. Surprisingly, Kundishora, et.al (2023) in their multiomic analysis classified the gene PTPN11 as an AC high-confidence risk gene which had ≥ 2 damaging de novo variants (DNVs). [12, 24]. PTPN11 encodes the nonreceptor protein tyrosine phosphatase which is a signaling protein involved in the process of cell growth and differentiation. This gene has been associated with a variety of human cancers but to date only one case of subependymoma has been reported in association with its mutation [25]. In our opinion, considering the existence of AC and subependymoma in two close-related members of a family in the context of the recent discovery of a common genetic susceptibility locus for both disorders is the most innovative aspect of our case that might not be coincidental and merits further assessment.

Conclusions

ACs are most commonly discovered as incidental findings during neuroimaging for another reason. There is much discrepancy about the ideal management of either

symptomatic or asymptomatic cases. The arachnoid cysts have a heterogeneous clinical course which further complicates decision-making. Therapeutic options include watchful waiting strategy, microsurgical or endoscopic fenestration and shunting. However, the best approach largely depends on the symptoms that would be really attributable to the cysts, the size and anatomical location of them, the patient's preferences, the presence of complications related to the cyst rupture (intracystic hemorrhage, subdural hematoma or hygroma) and both the surgeon and institutional experiences. Traumatic rupture of ACs complicated with subdural hygroma is a relatively rare condition especially in adult females. Traditionally, these patients had been recommended surgery, however some recent reports oppose to this routine strategy. So, disagreement is more pronounced regarding the management of these subgroup due to lack of systemic and controlled studies and relative rarity of cases. Until conducting prospective long-term controlled trials and emerging well-organized protocols, the management of these patients should be performed on an individualized basis. A recent interesting study published by Nature Medicine (2023) might add new insights into the management of ACs. The authors of the study found that about 20% of cases of ACs are attributable to DNVs indicating genomic variants as an independent overlooked contributors to the ACs pathogenesis. This discovery has some important implications. First, seven out of the genes harbored DNVs, are essential in brain development via transcriptional or epigenomic regulatory role demonstrating that some ACs may represent an anatomic correlates of an underlying disorder in the brain development. Second, these findings may explain the unsuccessful outcomes after surgery of the middle fossa ACs aimed to treat seizure, psychiatric symptoms and language or motor delay which initially had been attributed to the mass effect of cyst itself whereas might be due to coexistence of a neurodevelopmental sequel . The authors concluded that the discovery of DNVs would increase the threshold for surgery in this population in the future [12]. So, the decision for surgical management of the middle fossa ACs should be made more cautiously.

Finally, we think that raising the suspicion for any possible connection between ACs and other genetic conditions such as benign brain tumors (e.g., subependymoma) and connective tissue disorders as well as addressing the unmet need for familial screening of ACs and their related anomalies, are the most important goals of this case report.

Abbreviations

ACs	Arachnoid cysts
SACs	Sylvian arachnoid cysts
CSF	Cerebrospinal fluid
CT	Computed tomography
PTPN11	Protein tyrosine phosphatase nonreceptor- type 11
DNVs	Damaging de novo variants
IGF-1,2	Insulin growth factor1, 2
PDGF	Platelet-derived growth factor
RA	Rheumatoid arthritis
MRI	Magnetic resonance imaging

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Competing interests
The authors declare that they have no competing interests.

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