A Form of Dysplasia or a Fortuitous Association? A Cerebral Aneurysm Inside an Arachnoid Cyst: Case Report

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OBJECTIVE: Although arachnoid cysts and intracranial aneurysms are very common lesions, their association in the same patient is rare. We present a case of a middle cerebral artery aneurysm ruptured into an arachnoid cyst. We found only six cases with intracystic hemorrhage reported in the literature. The presence of an arachnoid cyst can mislead clinical presentation. The patient presented a paradoxically small temporal fossa and thickening of the temporal and sphenoid bone. The authors suggest that this uncommon association (arachnoid cyst, atypical cranial vault, and "mirror-like" cerebral aneurysm) could represent a form of dysplasia.

CLINICAL PRESENTATION: A 46-year-old patient presented with a 3-week history of slight headaches, which had worsened in the last 3 days before presentation. Computed tomographic scans showed a cystic lesion located in the middle cranial fossa and sylvian fissure with suspected aneurysm dilation inside. Magnetic resonance imaging scans showed an intracystic hemorrhage but not subarachnoid hemorrhage. Paradoxically, changes in the cranial vault around the cyst were noted. Digital subtraction angiography showed bilateral "mirror" middle cerebral artery aneurysms.

INTERVENTION: A large right pterional craniotomy was performed with full microsurgical removal of the arachnoid cyst walls and aneurysm clipping. The aneurysm was in the medial wall of the arachnoid cyst with its dome inside the cyst. The contralateral aneurysm was clipped 2 weeks later. The follow-up period was uneventful, and the patient returned to normal life.

CONCLUSION: Rupture of a cerebral aneurysm into an arachnoid cyst is rare. Clinical presentation may be unusual because the cyst can prevent subarachnoid hemorrhage. A middle fossa cranial arachnoid cyst in the presence of temporal bone depression, small middle fossa, and thickness of squamous temporal bone and the lesser wing of sphenoid is rare and suggests that congenital factors may play an important role in their development. The exceptional association between "mirror" aneurysms and arachnoid cyst with bone changes suggests a possible congenital form of dysplasia.

KEYWORDS: Arachnoid cyst, Cerebral aneurysm, Cranial vault, Dysplasia, Intracystic hemorrhage

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Intracranial aneurysms and arachnoid cysts are common pathologies whose etiologies have not been completely resolved. Rupture of an aneurysm causes classical sudden and intense headache, motivating the patient to seek medical attention. Bleeding of a cerebral aneurysm inside an arachnoid cyst has rarely been described. In a search of the literature, we found only six previously published cases (2, 5, 9, 10, 12, 31). We present another case of a bilateral middle cerebral artery (MCA) aneurysm that ruptured into a sylvian fissure arachnoid cyst. The clinical presentation in this case was unusual, leading to a delay in medical attention. The patient also presented with a history of temporal region cranial vault change since an early age. Apparent paradoxical vault changes in patients with arachnoid cysts have been described previously (19). To our knowledge,

no relationship between these findings has yet been discussed in the literature.

Case Report

A 46-year-old woman was admitted to our neurosurgical service in March 2002 with a 3-week history of sporadic headaches without vomiting that resolved with analgesia. She did not seek medical attention because she considered her symptoms unimportant. Three days before admission, she presented a new sudden headache associated with vomiting. There was no history of trauma or nervous system infection. Morbid antecedents were arterial hypertension, smoking, and a chronic polyradiculopathy. Her neurological examination was normal, without meningial signs. Since infancy, she had noted a cranial deformity with depression (atrophy) in the right temporal bone (*Fig. 1*). Computed tomographic (CT) scans showed a right cystic lesion located in the right middle fossa and sylvian fissure with intracystic hemorrhage. CT scans showed an increased thickness in the squamous temporal bone and lesser sphenoidal wing, with slight depression on convexity and small middle fossa. Contrast CT scans showed saccular dilation of the MCA suggesting an aneurysm (*Fig. 2, A–C*). Magnetic resonance imaging scans showed a cystic lesion in the right middle fossa and sylvian fissure with a slightly increased signal on T1-weighted and proton density images, isointense in fluid-attenuated inversion recovery images, and high in T2-weighted images, suggesting an intracystic hemorrhage. There were also signs of hemosiderin in the cyst boundaries and a void signal in the right MCA (*Fig. 2, D–F*). These clinical and image findings suggested a ruptured aneurysm into cystic lesion, which led us to perform digital subtraction angiography. A superiorly projecting ruptured right MCA bifurcation saccular aneurysm was identified along with a smaller unruptured left MCA bifurcation aneurysm in the mirror position (*Fig. 3*). There was no vasospasm. The patient underwent a large right pterional craniotomy for cyst evacuation and aneurysm clipping (*Fig. 4*). During surgery, the duramater was bulging and in tension. After opening the dura, the arachnoid cyst appeared to be bulging in the sylvian fissure. The cyst appeared blood-tinged and its boundaries were impregnated with hemosiderin. The wall was opened and fully removed. The cyst contained hemorrhagic, xantochromic fluid under pressure. The pathologist confirmed arachnoid cyst. The base of aneurysm was firmly adhered to the medial wall of the cyst, which was dissected, and the aneurysm was clipped. Two weeks later, the left aneurysm was clipped. The patient was discharged after another 5 days without deficits and returned to her normal activites.

DISCUSSION

An association between a ruptured aneurysm and an arachnoid cyst is rare. Only six have been previously reported with intracystic hemorrhage (ICyH) (*Table 1*) (2, 5, 9, 10, 12, 31). Jinkins et al. (12) were the first to describe a ruptured MCA aneurysm with subarachnoid hemorrhage (SAH) discharging within convexity arachnoid cyst. Hirose et al. (9) described a carotid artery bifurcation aneurysm into a media fossa arachnoid cyst without SAH. Barker et al. (2) described the rupture of an internal carotid-posterior communicating artery aneurysm with SAH and ICyH. Huang et al. (10) described an ICyH and subdural hematoma caused by MCA aneurysm rupture. Zanini et al. (31) described an ICyH with SAH caused by a carotid artery bifurcation aneurysm and discussed the etiopathogenetic mechanisms. Berhouma et al. (5) recently reported bilateral "mirror" MCA aneurysms with ICyH, SDH, and SAH. There are other reported cases of associated aneurysms and arachnoid cysts; however, all are without ICyH (3, 15, 24). Recent descriptions of parenchymal cysts around cerebral aneurysms (4, 22) and spontaneous intracystic hemorrhages without aneurysms (11) are not related to our subject.

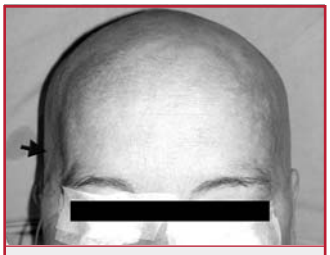

FIGURE 1. *Cranial vault deformity in the temporal region* (black arrow).

Clinical Presentation

This is the second case in our department in which an aneurysm ruptured into an arachnoid cyst. As in the previous case (31), this patient presented with slight to moderate headache, without vomiting or compromising consciousness. The symptoms did not motivate the patient to seek medical help because the headaches were slight and transient. Only when symptoms worsened, days after the first onset, did the patient seek medical attention at the hospital. Other unusual manifestations from ruptured aneurysms are the absence of meningeal signs (5, 9) and transient motor deficits (5, 31). The presence of the arachnoid cyst probably prevented the spread of blood into the arachnoid space. Nevertheless, some blood can spread to the subarachnoid (2, 12, 31) or subdural (5, 10) space causing typical SAH symptoms.

Arachnoid Cyst Etiopathogenesis

Arachnoid cysts are cavities filled by cerebrospinal fluid-like fluid. They occur inside the arachnoid membranes and are, in fact, intra-arachnoid. They make up 1% of all intracranial mass lesions and are found in 0.1% of autopsies (21). The most frequent location, nearly half of all cases, is the middle fossa. They are considered the result of abnormal arachnoid layer development during the embryo stage of life. However their precise pathogenesis remains unclear. There are three popular theories: dysembryogenesis of the arachnoid membrane related to a primary disturbance of the primitive mesenchyma adjacent to the neural tube; lobe agenesia resulting in secondary expansion of the cerebrospinal fluid space; and acquired process resulting from infectious, inflammatory, traumatic, or hemorrhagic events in the fetus or early life (16, 21). The arachnoid cyst may

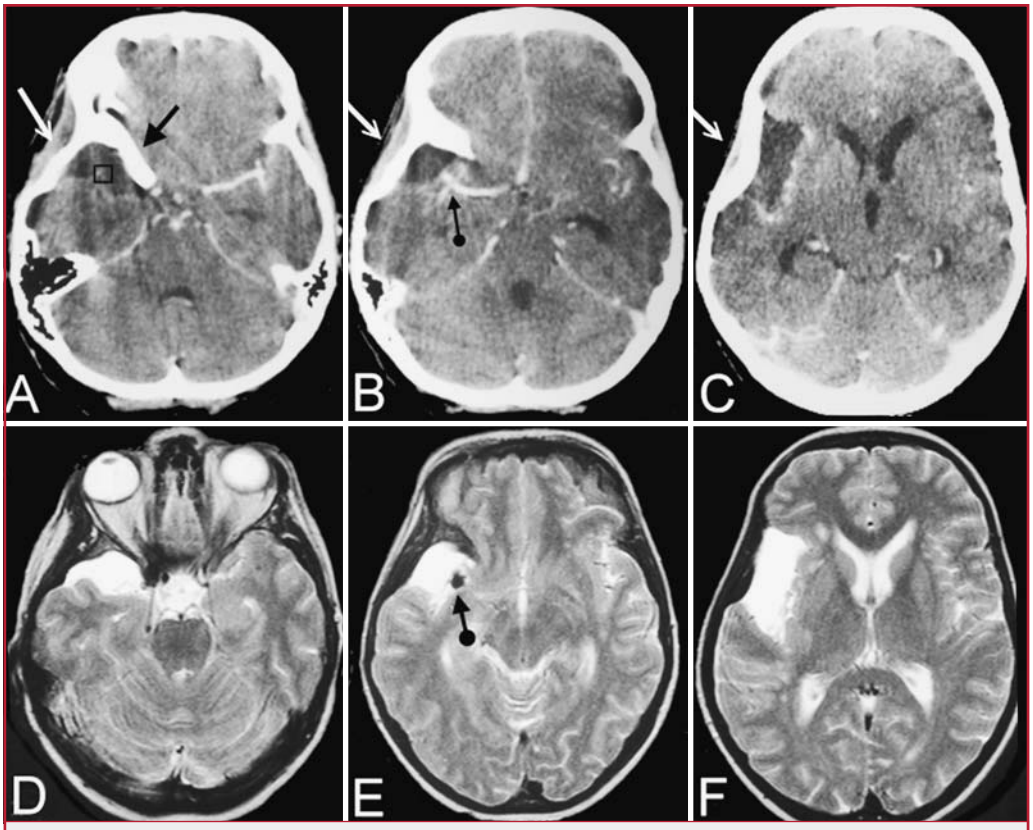

FIGURE 2. **A–C**, *contrasted CT scans showing a depression in the squamous temporal bone* (white arrow), *small middle fossa* (black square), *thickness of squamous temporal bone and lesser wing of the sphenoid* (black arrow), *and middle cerebral artery dilation suggesting aneurysm.* **D–F**, *axial T2-weighted magnetic resonance imaging scans showing an arachnoid cyst in the small middle cranial fossa and sylvian fissure. Note the presence of flow void at the medial wall of the cyst* (black arrow).

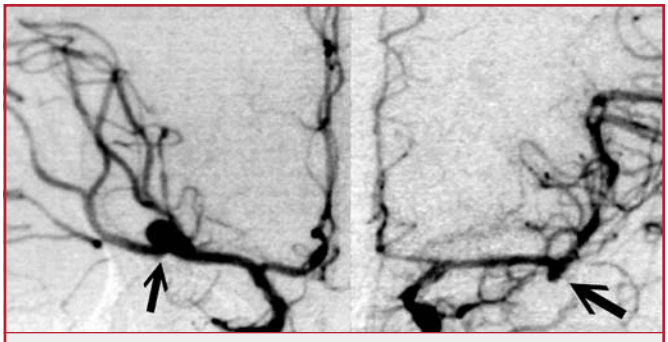

FIGURE 3. *Digital subtraction angiography showing a bilateral "mirror-like" aneurysm* (arrows) *at MCA bifurcation.*

be a manifestation of an underlying extracellular matrix defect in polycystic kidney disease (PKD) (24). The frequent association between arachnoid cysts and other malformations, such as Type I neurofibromatosis, Marfan syndrome, PKD, and callosum agenesis, supports a congenital or genetically related pathogenesis (24, 26). It is frequently associated with the thinning and expansion (bulging) of the adjacent bones (temporal bone and the lesser sphenoidal wing), causing a larger than normal middle fossa. However, an apparently paradoxical vault change related to arachnoid cyst has been described by Redla et al. (19). They reported three cases of arachnoid cysts in which the middle cranial fossa was paradoxically small and had thickening of the squamous temporal bone and lesser sphenoidal wing. They reinforce the temporal lobe hypoplasia theory as the initial event followed by compensatory bone changes, before the appearance of the arachnoid cyst. In our patient, the arachnoid cyst was asymptomatic and became symptomatic only after ICyH caused by aneurysm rupture. The cranial vault alterations were probably congenital. In the presence of an arachnoid cyst, it presented a paradoxically small middle fossa, depression of the temporal region, and thickening of the squamous temporal bone and the lesser wing of the sphenoid.

Aneurysm Etiopathogenesis

Sacular aneurysms in the cerebral circulation are relatively common. The lifetime prevalence of these aneurysms ranges from 2 to 5.5% (29, 30). The precise development, growth, and rupture mechanisms of intracranial aneurysms are still to be discovered (7, 13, 30). The effects of hemodynamic stress causing degenerative vascular diseases and known risk factors such as smoking, contraceptives, and hypertension provide evidence favoring an acquired etiopathogenesis (28, 30). Several recent studies on aneurysm wall biology have highlighted the importance of inflammatory response in ruptured cerebral aneurysms (7, 8, 13, 29). A disruption of the arterial wall extracellular matrix, compromising the strength and elasticity of the intracranial arteries is a likely predisposing factor in aneurysm formation. Even so, genetic factors could also play an important role in their formation (17, 23, 30) in some cases. Family predisposition and an association with other inheritable disorders of connective diseases, such as PKD, Ehlers-Danlos disease Type IV, and Marfan's syndrome, and the extracellular matrix support this (6, 27, 30). A congenital decrease in extracellular matrix structural proteins has been

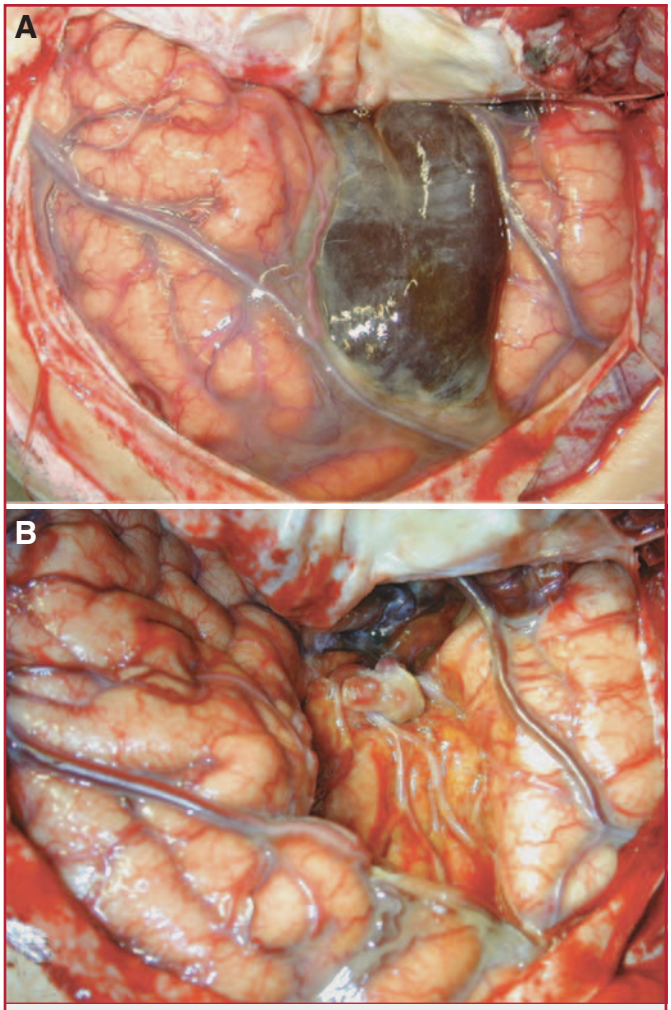

FIGURE 4. **A**, *intraoperative photograph showing the bulging sylvian fissure arachnoid cyst with intracystic hemorrhage.* **B**, *intraoperative photograph showing the cavity after removal of the full arachnoid cyst walls showing the aneurysm. Note the presence of hemosiderin in the cavity boundaries.*

seen in aneurysms (20). Collagen deficiency has also been proposed (14, 17, 20). Recently, Baccin et al. (1) reported a case of multiple mirror aneurysms associated with familial aneurysmal disease and, thus, suggested that an earlier defect in embryogenesis may be involved in multiple and mirror aneurysms (1). Multiple aneurysms are encountered in a variety of disorders such as PKD, fibromuscular dysplasia, Marfan syndrome, and Type I neurofibromatosis, which may result from defects in the mesodermal-neural crest or focal endothelial cell during embryonic stages.

Arachnoid Cyst and Aneurysm Association

There are few reports of an association between unruptured aneurysms and arachnoid cysts (3, 15, 24), and the prevalence of this association in the normal population is unknown. However, some embryological aspects of both pathologies and reports of unusual malformations might lead to speculation on a common pathophysiological pathway. There is controversy concerning the histogenesis of the arachnoid layer and the arterial layer involved in aneurysm formation. However, both apparently originate from the mesoderm, the primitive mesenchyme that surrounds the neural tube (16, 21), which is also involved in producing collagen for the extracellular matrix in the cerebral vessels. Schievink et al. (24) reported a case with generalized connective diseases and presented an association between cardiovascular disturbance (carotid-jugular fistulae, arterial dissection, arterial wall irregularities), skin (facial hemiatrophy), and multiple arachnoid cysts. In another report, Schievink et al. (25) described a child with progressive hemifacial atrophy (Parry-Romberg syndrome) and intracranial aneurysm disease. Another communication reports the same association (18). They suggest a malformation hypothesis in which both vascular and mesenchymal structures with common progenitors, derived from neural crest cells, are involved at the same time. The presence of the arachnoid cyst and an intracranial aneurysm in inheritable connective tissue disorders are higher than in the normal population. Analyzing 247 patients with PKD, Schievink et al. (26) detected 20 (8.1%) harboring arachnoid cysts and 27 (10.8%) with intracranial aneurysms. However, they did not find any correlation

TABLE 1. Reports of ruptured intracranial aneurysm and arachnoid cyst hemorrhage[a]

Series (ref. no.)	Age (yr)/sex	Aneurysm location	Cyst location	Presentation
Jinkins et al., 1987 (12)	40/M	MCA	Right convexity	IcyH, SAH
Hirose et al., 1995 (9)	45/M	BifCA	Left middle fossa	IcyH
Barker et al., 1998 (2)	29/F	PCoA	Left middle fossa	IcyH, SAH
Huang et al., 1999 (10)	61/F	MCA	Left middle fossa	IcyH, SDH
Zanini et al., 2000 (31)	35/M	BifCA	Left sylvian fissure	IcyH, SAH
Berhouma et al., 2006 (5)	37/F	Bilateral "mirror" MCA	Left middle fossa and sylvian fissure	IcyH, SAH, SDH
Current study	46/F	Bilateral "mirror" MCA	Right middle fossa and sylvian fissure	IcyH

[a] MCA, middle cerebral artery; IcyH, intracystic hemorrhage; SAH, subarachnoid hemorrhage; BifCA, bifurcation carotid artery; PCoA, posterior communicating artery; SDH, subdural hematoma.

between arachnoid cysts and intracranial aneurysms and, because of the relatively small number of aneurysm patients in the study, they could not draw a definitive conclusion. Perhaps the arachnoid cyst and intracranial aneurysm represent a distinct epiphenomenon of a single dysembryogenesis in some specific cases. Notwithstanding the fact that no concrete evidence of connective disease, PKD, or Parry-Romberg disease was found in our patient, the cranial vault changes and arachnoid cyst could represent a localized dysembryogenesis or a mild form of extracellular matrix disturbance.

Lack of supportive tissue surrounding the vessels, which occurs inside of the cyst, may contribute to the development, increase, and rupture of an aneurysm. In this and our previous case report, there were close relationships between both lesions, with the aneurysm inside the arachnoid cyst suggesting a basic underlying cause. However, as both pathologies are very common and no molecular data were provided, their association can only be fortuitous and many more cases studying molecular alterations of extracellular matrix would be needed to support this suggested underlying association. Reports of positive associations between, at first, unrelated lesions can induce erroneous conclusions. However, careful analysis of uncommon cases such as this one may contribute to an understanding of the still unresolved questions surrounding these common pathologies.

CONCLUSION

The rupture of a cerebral aneurysm into an arachnoid cyst has rarely been described in the literature. The clinical presentation may be unusual because the cyst can prevent subarachnoid hemorrhage. Atypical cranial vault changes can occur in the presence of an arachnoid cyst. In our opinion, this uncommon association, an arachnoid cyst, cranial vault deformity, and bilateral "mirror" cerebral aneurysm, strongly suggests a mild form of a dysplasia.

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COMMENTS

Arachnoid cysts or aneurysms are different lesions that are usually treated in neurosurgical practice. Both isolated lesions have a high incidence, but the presence of aneurysms and arachnoid cysts in different locations in the cranial vault within the same patient seems to be rare. Small arachnoid cysts are often found incidentally in association with different brain pathologies, such as tumors, arteriovenous malformations, and others around or at some distance from the former. Indeed, the suggested mild forms of connective diseases could also be a reasonable explanation for the isolated aneurysms or arachnoid cysts that are not associated with familial disorders or known connective disorders.

The angiogram shows a mirror-like middle cerebral artery bifurcation aneurysm with its usual configuration in both sides. Despite the bigger size of the intra-arachnoid cyst aneurysm, the direction of the bleeding was very similar to the usual place from which those aneurysms bleed, the sylvian fissure or temporal lobe, where the arachnoid cyst is located.

For all of these reasons, we agree with the authors that the common disorder can be found in both pathologies. However, we also think that the number of variables is stronger to support the idea of fortuitous finding until more cases can be studied and some evidence of dysplasia can be demonstrated.

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The authors present an interesting observation on the common occurrence of an arachnoid cyst and intracranial aneurysm formation. Six cases have been published in the literature so far. A mild form of dysplasia is suggested. The etiopathogenesis of aneurysms comprises not only the effects of hemodynamic stress, risk factors, e.g., smoking, contraceptives, hypertension, or inflammations, but also genetic factors. Some hereditary diseases are known to be associated with aneurysm formation (e.g., polycystic kidney disease, Ehlers Danlos syndrome, and Marfan syndrome). Gene coding for structural proteins of the extracellular matrix are also associated with aneurysm formation and are the focus of molecular investigations. If the authors' hypothesis that an arachnoid cyst and intracranial aneurysms are a mild form of dysplasia is true, molecular investigations should follow in order to elucidate a common molecular pathway.

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Cerebral aneurysms are more common than arachnoid cysts in most autopsy studies. On the other hand, some of the cysts, especially those that are small in size, may be missed during autopsies. The authors have described a very rare event of a middle cerebral artery bifurcation aneurysm that ruptured into an arachnoid cyst without more widespread subarachnoid hemorrhage. However, having these two lesions together seems to be more of a coincidence than a mutual pathobiology, as we should still see it happen more often in clinical practice. One must still remember that a patient with a sudden headache has an aneurysm rupture until proven otherwise and, with modern computed tomographic angiography, this can very easily and noninvasively be ruled out (1).

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