A rachnoid cysts are congenital intraarachnoid malformations of the meninges\textsuperscript{18,24,25} and account for about 1\% of all intracranial tumors.\textsuperscript{20} These benign lesions often remain asymptomatic or can become progressively clinically manifest as the cyst exerts a mass effect on the surrounding cerebral parenchyma, especially in childhood or young adults.\textsuperscript{1,8,31} Patients with an AC are at increased risk of a CDSH, the most frequent hemorrhagic event associated with an AC developing.\textsuperscript{6,22} In a study of 658 patients with CSDHs, Parsch et al.\textsuperscript{22} reported a 2.43\% incidence of ACs. How an AC favors the development of a CDSH remains unclear, although various pathogenic mechanisms have been proposed.\textsuperscript{16,18} We designed this study to investigate the clinical, neurological, and radiological outcome of patients with CSDHs related to an AC and in whom treatment involved draining the CSDH while leaving the AC intact. We sought to relate the rationale for the surgical management we propose to the causative mechanisms underlying these associated conditions.

\textbf{Methods.} Eight patients with ipsilateral CSDHs related to an AC underwent surgery some time between 2000 and 2005 in the Department of Neurosurgery at the “Sapienza” University. Before coming to our attention, none of the patients had complained of neurological disorders or had received a diagnosis of AC. All patients had reported mild head trauma (Table 1) from 20 to 35 days before hospital admission (mean time 28 days). There were 6 males and 2 females age 7–69 years (mean age 38.3 years). Of the 8 patients, 6 were younger than 50 years old. All 8 patients underwent cerebral CT scanning.
Arachnoid cyst and subdural hematoma

Results

All CSDHs were ipsilateral to the AC, and in 1 patient (Case 4 [Table 1]) neuroimaging also detected a contralateral temporal AC. Of the 8 ACs, 6 involved the temporal fossa and 2 the frontal convexity. Computed tomography and MR imaging features of the CSDH were typical of a chronic subdural blood collection. In all cases CT scans showed that the AC was isointense to CSF. On T2-weighted MR images, all ACs appeared isointense to CSF, whereas on T1-weighted images, except those in Case 4, they appeared isointense to the brain or hyperintense to CSF. In the 5 patients in whom FLAIR sequences were acquired (excepted in Case 1 [Table 1]), the AC appeared isointense to CSF. On T2-weighted MR images, all ACs appeared isointense to CSF, whereas on T1-weighted images, except those in Case 4, they appeared isointense to the brain or hyperintense to CSF. In the 5 patients in whom FLAIR sequences were acquired (excepted in Case 1 [Table 1]), the AC appeared hyperintense to CSF. In all patients, surgery consisted of bur hole irrigation and drainage of the CSDH. The bur hole served to avoid rupturing the thin septal layer separating the hematoma from the cyst. None of the procedures led to cyst wall rupture or the need for fenestration. All patients underwent early postoperative cerebral CT scanning, and late MR imaging during follow-up. As they did on preoperative scans, the ACs appeared hyperintense to CSF on postoperative CT scans. Follow-up MR images of the ACs obtained within 2 weeks of surgery showed signal intensities similar to those on preoperative images with minimal or marked reexpansion of the cysts. On 4-month T1-weighted and FLAIR MR images, the signals progressively normalized and from 6 months after surgery onwards became of CSF type. In all cases, throughout follow-up period the T2-weighted signal from the AC content remained isointense to CSF, as it had been preoperatively. All patients resumed their daily activities within 15 days after surgery.

Illustrative Cases

Case 3

Examination. This 21-year-old man (Table 1) presented with a 20-day history of headache after a head injury caused by being run over by a motor vehicle. Brain CT scanning demonstrated a left frontotemporal CSDH in contact with an underlying temporal AC that appeared isodense to CSF. Preoperatively the CSDH exhibited a mixed, markedly and moderately high-intensity area on T1-weighted (Fig. 1A) and T2-weighted MR images, and the AC appeared as a markedly hyperintense area on T1- and T2-weighted MR images.

Operation and Postoperative Course. The patient underwent emergency surgery to evacuate the CSDH through a bur hole while the AC was left intact. During the postoperative course the patient’s neurological conditions normalized and his headache disappeared. One month after surgery, the left temporal CSDH had diminished in size and the middle fossa AC appeared hyperintense on T2-weighted FLAIR images and was of mixed signal intensity (iso- and hypointense) on T1-weighted images (Fig. 1B). At 6 months MR imaging showed that the CSDH had disappeared, and on all pulse sequences the signal in the left middle fossa AC had become isointense with CSF (Fig. 1C). At 6-month clinical follow-up the patient was completely free of neurological symptoms.

Case 4

Examination. This 64-year-old man (Table 1) presented for investigation of a 2-week history of worsening headache after a motor vehicle accident that caused minor head trauma. A CT brain scan obtained in the emergency department revealed a left-sided CSDH. Contiguous with the CSDH was a left temporal AC. Another AC was visible in the right contralateral temporal area. On CT scans...
both cysts appeared isodense to CSF. Brain with T1-weighted MR (Fig. 2A) and FLAIR images demonstrated an area of homogeneous signal intensity hyperintense to CSF, confirming the left hemisphere blood collection in contiguity with the AC. Magnetic resonance imaging, like CT scanning, also revealed the right temporal AC contralateral to the CSDH, which was isointense to CSF on all sequences.

**Operation and Postoperative Course.** The patient underwent emergency surgery to evacuate the CSDH through a bur hole while the AC was left intact. During the postoperative course the patient’s neurological conditions gradually improved and ultimately returned to normal. A follow-up CT scan obtained on Day 2 (Fig. 2B) showed that the CSDH had disappeared and the ACs persisted, their content appearing isodense to CSF. At 20 days MR imaging (Fig. 2C–E) demonstrated analogous findings to preoperative images. On T2-weighted sequences both ACs exhibited CSF-like hyperintensity. The left middle fossa AC appeared isointense to brain parenchyma on T1-weighted and hyperintense on FLAIR images. Follow-up MR imaging performed at 8 months showed that the left-sided AC had normalized, and on all sequences its content exhibited a CSF-like signal identical to that of the contralateral AC. At 16 months the patient was completely free of neurological symptoms.

**Discussion**

The excellent surgical outcome in the 8 patients in our series suggests that patients with AC-associated CSDH can be successfully treated by undergoing CSDH drainage alone while the AC is left intact. All our patients’ neurological symptoms regressed within days of surgery, and at long-term follow-up (6 months–2 years) none of them had recurrent lesions. The patients’ postoperative clinical course resembled that of patients with the more usual CSDH uncomplicated by an AC. In none of our patients did pre- or postoperative MR imaging scans show AC features that were considered compatible with bleeding. Most of our patients in whom a CSDH was associated with an AC were young, and as in cases involving an isolated CSDH, their CSDH manifested typically within a few weeks after a minor head injury and their neurological symptoms were due to intracranial hypertension. In our cases, as in reported cases, the CSDH was ipsilateral to the AC and the AC caused no symptoms until the CSDH manifested neurologically. These data suggest that when a CSDH and AC are diagnosed in the same patient, the AC is an incidental finding and that the neurological symptoms arise exclusively from the mass effect as the CSDH develops.

**Histological Data and Pathogenesis**

Our decision to manage the AC and CSDH as 2 separate noncommunicating entities derived from current histological knowledge. How an AC form remains unclear. The lesion could arise from a minor developmental aberration of the subarachnoid space owing to changes in mesenchymal condensation or CSF flow into the pia-arachnoid space. Ultrastructural studies that involved transmission electron microscopy have specified that the cyst lining consists of single or multiple layers of cells and that ACs are wholly situated within the arachnoid membrane.

Current histological knowledge also leaves the pathogenesis of CSDH unclear. Researchers over the past decades investigating the anatomical and pathophysiological features of the meninges have prompted a new look at the concept of the subdural space, thus leading to a reappraisal of the approach to SDHs. In an electronic micro-
scopic study, Haines et al. described the dural border cells that extend from the dura and adhere tightly to the arachnoid membrane, delimiting a “potential space”—namely, the “subdural compartment.” This space remains virtual until it is created and opened by a traumatic event. In an earlier study of patients with posttraumatic acute SDH, investigators found that neuroimaging studies showed the hematoma in the “intradural” compartment. From anatomical findings and studies involving transmission electron microscopy, we conjecture that the membrane separating an AC from a CSDH forms when the membranous structures of the AC and CSDH overlap and adhere tightly to each other. The presence of a congenital arachnoid malformation such as an AC weakens the subdural compartment at the level of the junction of the dural border cells and favors subsequent bleeding, causing a minimal and asymptomatic acute SDH that ultimately evolves into a symptomatic CSDH.

Accordingly, because a membrane separates the 2 cavities and prevents communication, surgically managing both associated conditions by simply draining under local anesthesia the CSDH seems therapeutically appropriate and minimally invasive.

Radiological Features

Preoperative cranial CT scans, obtained in all 8 patients, invariably demonstrated the AC and CSDH: the ACs always appeared similar to CSF in signal density. Conversely, CSDHs differed from the brain parenchyma and AC in density, appearing hyperdense, isodense, or hypodense, according to whether bleeding was long lasting or recent. In Cases 2 and 7, CT scans documented scalloping always involving the roof and middle skull base. In all patients the ACs also retained their CSF density on follow-up images, whereas in most cases after the CSDH was removed the AC reexpanded.

Preoperative MR imaging in 7 cases also identified and distinguished the AC, CSDH, and brain parenchyma. On T1-weighted and FLAIR images the cystic, hemorrhagic, and parenchymal components were clearly distinct and identifiable, whereas on T2-weighted images the in-
tensity of the AC content became indistinguishable from CSF. The T1-weighted images best distinguished between the cystic content and the content of the CSDH. On T1-weighted sequences the cyst generally appears homogeneous to brain parenchyma, whereas the CSDH is hyperintense, sometimes also appearing nonhomogeneous. On FLAIR sequences the AC is distinguishable from CSF in the subarachnoid spaces because it appears markedly hyperintense. In Case 4, the 2 coexisting ACs gave us a unique opportunity to compare an uncomplicated and complicated by a CSDH. In accordance with previous imaging findings, on all MR images of the AC uncomplicated by a CSDH, the AC and CSF signal intensity corresponded. Conversely, on T1-weighted MR images, the CSDH-associated AC appeared isodense to the parenchyma, whereas on FLAIR it was hyperintense, and on both sequences the signal was invariably homogeneous. In most of our cases the CSDH appeared hyperintense on all sequences, as others have reported for CSDH, and often appeared nonhomogeneous. Collectively, these findings on the relative MR imaging signal intensity features of ACs and CSDHs imply that the 2 entities differ in content and that MR imaging can easily distinguish one from the other. Whereas a CSDH always contains some blood, an AC displays no features frankly compatible with blood despite its contact with the CSDH. Magnetic resonance imaging signals from the content of complicated and uncomplicated AC differ, and these variations are visualized best on T1-weighted and FLAIR images.

Our new findings in this series support our earlier supposition that the subdural bleeding responsible for a CSDH leaves the associated AC only slightly changed in content. When an AC comes into contact with a CSDH, the blood breakdown products may filter through the dividing anatomical membrane and alter the CSF signal of an AC on some MR images, but they are insufficient to alter the density of their signal on CT scans. This explanation receives support also from early postoperative MR images that showed that the AC retained the signal characteristics seen when it remained in contact with the CSDH, but as follow-up lengthened it progressively regained CSF signal intensity in all sequences, a neuroimaging feature typical of an uncomplicated AC.

**Treatment**

The good results we obtained by performing bur hole irrigation of the CSDH and by leaving the cyst opening intact agree with the few previous reports that nevertheless provided no MR imaging information on bleeding within the AC. Other investigators have evacuated the CSDH and inserted a cystoperitoneal shunt. Some have proposed using craniectomy or craniotomy with evacuation of the CSDH and fenestration of the AC. There are only a few published cases in which authors described a CSDH associated with an AC that were treated conservatively. Most of these patients harboring these lesions had only mild neurological symptoms and the CSDH resolved spontaneously. In general, patients with an AC-associated CSDH are managed surgically, although the optimum surgical approach remains controversial. In all our cases, as in previously reported series, the ACs were asymptomatic and detected incidentally when the patient presented with a CSDH. In our patients, none of whose CT and MR images documented frank blood within the cyst, surgically evacuating the CSDH alone promoted a rapid and complete recovery of the neurological deficits, underscoring that the AC induced no mass effect. In rare reported cases, because CT and MR imaging documented the presence of fresh blood, the AC had to be opened.

**Conclusions**

Rather than being an incidental association, a CSDH may develop when an AC, which formed during dysembryonic development of the meninges, weakens the subdural compartment. Because a thin membrane separates the 2 entities, the bleeding responsible for a CSDH remains confined within the intradural compartment, whereas an AC remains exclusively intracranial. Blood degradation products that filter through the dividing membrane can alter the MR imaging signal but leave the CT CSF signal unchanged. These anatomical and imaging features explain the good outcome after surgical treatment to evacuate the CSDH leaving the membrane separating it from the AC intact.

**Disclaimer**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

**References**

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