J Neurosurg 110:1250–1255, 2009

Relationship between supratentorial arachnoid cyst and chronic subdural hematoma: neuroradiological evidence and surgical treatment

Clinical article

Maurizio Domenicucci, M.D.,[1] Natale Russo, M.D.,[1] Elisabetta Giugni, M.D.,[2] and Alberto Pierallini, M.D.[2]

[1]Departments of Neurological Sciences and Neurosurgery, University of Rome "Sapienza;" and [2]Department of Neuroradiology, IRCCS, San Raffaele, Rome, Italy

Object. Arachnoid cysts are relatively common congenital intracranial mass lesions that arise during the development of the meninges. They can be complicated by the formation of an ipsilateral chronic subdural hematoma (CSDH) after minor cranial trauma. Treatment of these coexisting conditions remains controversial. In this study the authors describe the anatomical, clinical, and neuroradiological features and outcome in a series of patients whose CSDH associated with arachnoid cysts were managed surgically by draining the hematoma alone and leaving the cyst intact. The authors based this surgical management on histological and neuroradiological observations concerning these associated medical conditions.

Methods. A series of 8 patients with CSDHs associated with arachnoid cysts underwent surgery to drain the hematoma though a bur hole. The arachnoid cyst was left intact. Postoperative follow-up included CT scanning and T1- and T2-weighted MR imaging.

Results. Clinical, anatomical, and radiological observations suggest that because separate membranes cover arachnoid cysts and the related hematoma, arachnoid cysts remain unaffected by the subdural bleeding. In the present study, these observations received support from the neuroimaging appearances, suggesting that arachnoid cysts related to hematoma contained only blood breakdown products from the hematoma that had filtered through the reciprocal dividing membranes.

Conclusions. Arachnoid cysts associated with SDH are anatomically separate conditions whose neurological symptoms respond to surgical drainage of the CSDH alone. *(DOI: 10.3171/2008.4.17509)*

Key Words • imaging • intracranial arachnoid cyst • minor head trauma • subdural hematoma

Arachnoid cysts are congenital intraarachnoid malformations of the meninges[18,24,25] and account for about 1% of all intracranial tumors.[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.[1,8,31] Patients with an AC are at increased risk of a CDSH, the most frequent hemorrhagic event associated with an AC developing.[6,22] In a study of 658 patients with CSDHs, Parsch et al.[22] reported a 2.43% incidence of ACs. How an AC favors the development of a CSDH remains unclear, although various pathogenic mechanisms have been proposed.[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.

Abbreviations used in this paper: AC = arachnoid cyst; CDSH = chronic subdural hematoma; CSF = cerebrospinal fluid.

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

TABLE 1: Summary of data in 8 patients with ACs in association with CSDH*

Case No.	Age (yrs), Sex	Location			AC: Preop Imaging Apearance			
		AC	CSDH	Minor Head Injury	CT	T1 MRI	T2 MRI	FLAIR
1	46, M	lt front	lt parietooccipital	MVA	CSF-like	CSF-like	CSF-like	CSF-like
2	7, M	lt temp	lt frontotemporal	soccer related	CSF-like†	–	–	–
3	21, M	lt temp	lt frontotemporal	accident	CSF-like	hyperintense	CSF-like	hyperintense
4	64, M	lt temp	lt hemispheric	MVA	CSF-like	parenchymal	CSF-like	hyperintense
		rt temp			CSF-like	CSF-like	CSF-like	CSF-like
5	41, M	rt temp	rt tempoparietal	bicycle accident	CSF-like	hyperintense	CSF-like	-
6	69, F	rt front	rt hemispheric	fall	CSF-like	parenchymal	CSF-like	hyperintense
7	31, F	rt emp	rt frontotemporal	MVA	CSF-like†	hyperintense	CSF-like	hyperintense
8	28, M	rt temp	rt frontotemporal	aggression	CSF-like	parenchymal	CSF-like	–

* MVA = motor vehicle accident.
† Scalloping inner table.

in the emergency department and 7 patients underwent MR imaging. One patient could not undergo cerebral MR imaging because, immediately after the CT scanning, he underwent an emergency operation for mild right hemiparesis.

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 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 invariably appeared isodense 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

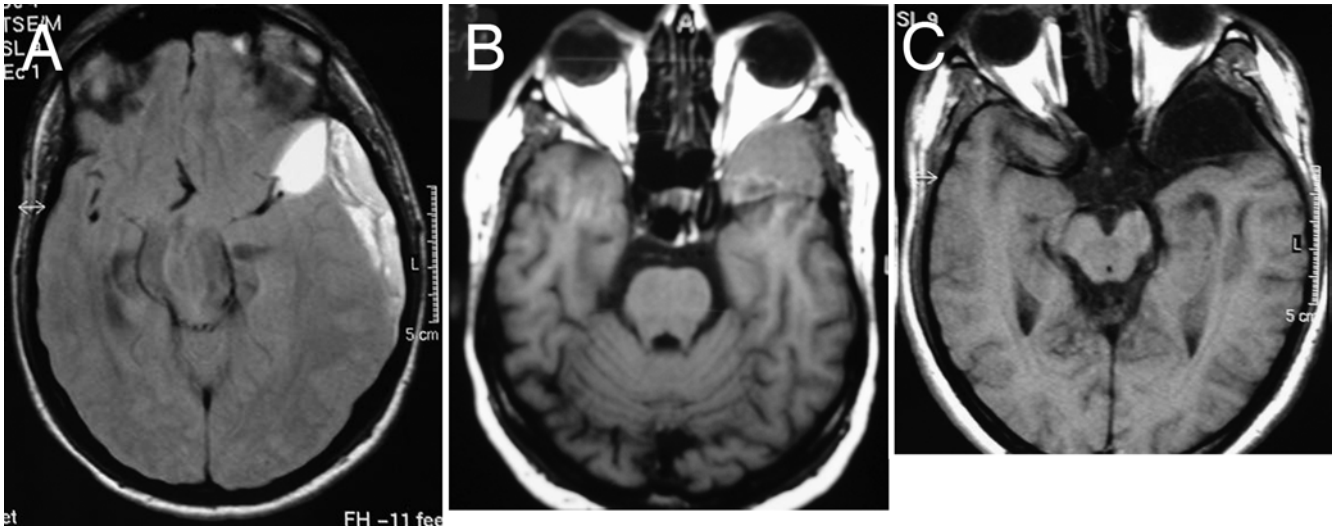

FIG. 1. Axial T1-weighted MR imaging studies. A: Preoperative image demonstrating the hematoma in the subdural space, which appears as a mixed, markedly and moderately high-intensity area; the AC appears markedly hyperintense. B: Postoperative image obtained 1 month after surgery showing the middle fossa AC appearing nonhomogeneously isointense to brain parenchyma. C: Postoperative image obtained 6 months after surgery showing that the signal intensity of the AC in the left middle fossa has changed and is now isointense with CSF.

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.[2,16,29] In our cases, as in reported cases,[3,4,21,30] 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,[24,27] owing to changes in mesenchymal condensation or CSF flow into the pia-arachnoid space.[18] Ultrastructural studies that involved transmission electron microscopy have specified that the cyst lining consists of single or multiple layers of cells[24,28] and that ACs are wholly situated within the arachnoid membrane.[24]

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.[5,9] In an electronic micro-

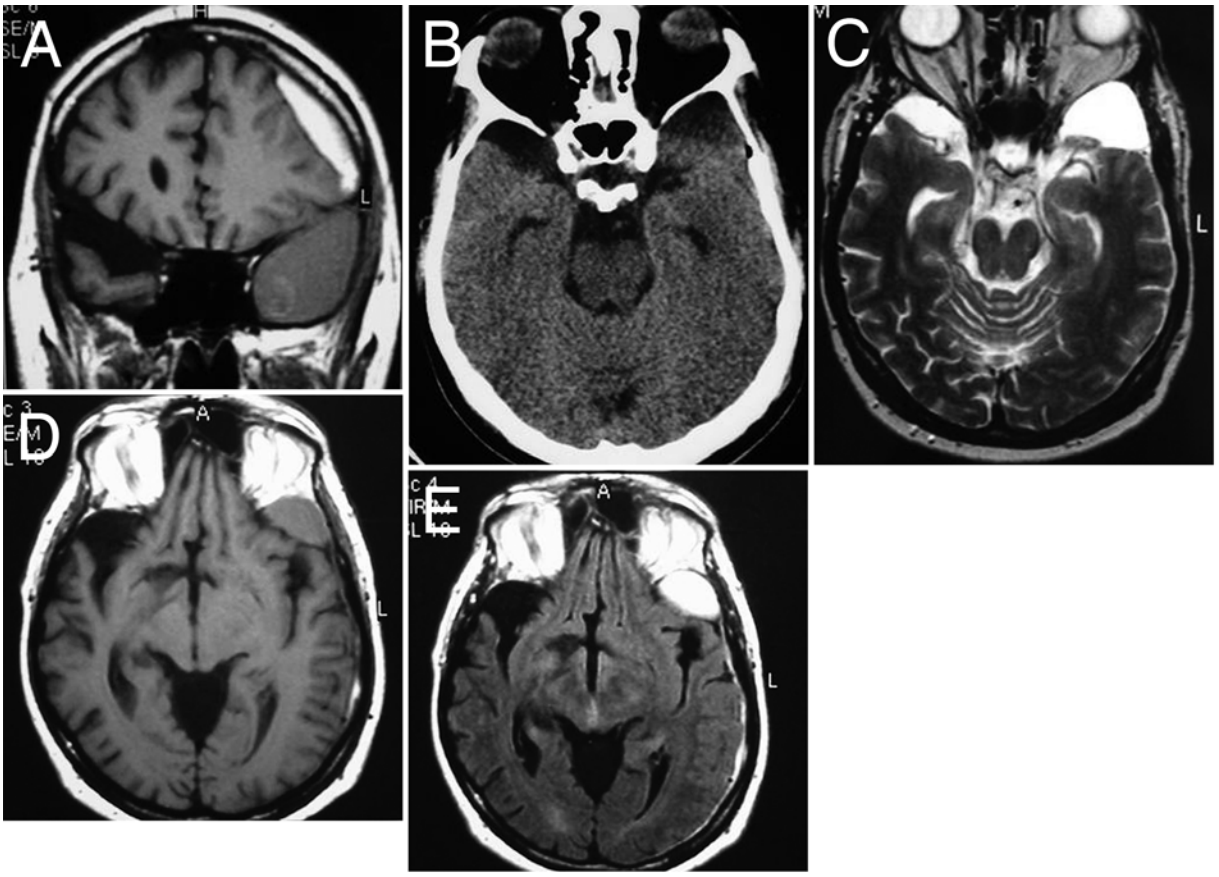

FIG. 2. Imaging studies. Preoperative coronal T1-weighted MR image showing the hematoma in the left frontotemporal and parietal subdural space appearing as a markedly high-intensity area and the middle fossa AC in the middle fossa appearing isointense to brain parenchyma. The contralateral middle fossa AC appears isointense with CSF. B: Postoperative CT scan obtained 2 days after surgery documenting the disappearance of the SDH, whereas both ACs appear isointense with CSF. C: Postoperative T2-weighted MR image acquired 20 days after surgery showing that both ACs in the middle fossa appear isointense to CSF. D: Postoperative T1-weighted image demonstrating that the left middle fossa AC appears isointense to the brain parenchyma, whereas the contralateral cyst is still isointense with CSF. E: On this T2-weighted FLAIR image the lesion appears hyperintense.

scopic study, Haines et al.[9] 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.[5] From anatomical findings and studies involving transmission electron microscopy,[24,27,28] 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,[17] appearing hyperdense, isodense, or hypodense,[19] 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.[12] On T1-weighted and FLAIR images the cystic, hematic, 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 AC 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.[11] 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,[7] and often appeared nonhomogenous. 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[16,22] that nevertheless provided no MR imaging information on bleeding within the AC. Other investigators have evacuated the CSDH and inserted a cystoperitoneal shunt.[1,15,21,26] Some have proposed using craniectomy or craniotomy with evacuation of the CSDH and fenestration of the AC.[21,22] There are only a few published cases in which authors described a CSDH associated with an AC that were treated conservatively.[22] 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.[16,23,30] In all our cases, as in previously reported series,[4,23] 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,[10,13,14] 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 intraarachnoid. 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

1. Arai H, Sato K, Wachi A, Okuda O, Takeda N: Arachnoid cysts of the middle cranial fossa: experience with 77 patients who were treated with cystoperitoneal shunting. **Neurosurgery 39:**1108–1112, 1996
2. Asghar M, Adhiyaman V, Greenway MW, Bhowmick BK, Bates A: Chronic subdural haematoma in the elderly–a North Wales experience. **J R Soc Med 95:**290–292, 2002
3. Cappelen J, Unsgaard G: Arachnoid cysts of the middle cranial fossa and traumatic complications. **Childs Nerv Syst 2:**225–227, 1986
4. Demetriades AK, McEvoy AW, Kitchen ND: Subdural haematoma associated with an arachnoid cyst after repetitive minor heading injury in ball games. **Br J Sports Med 38:**E8, 2004
5. Domenicucci M, Strzelecki JW, Delfini R: Acute posttraumatic subdural hematoma: "intradural" computed tomographic appearance as a favourable prognostic factor. **Neurosurgery 42:**51–55, 1998
6. Foelholm R, Waltimo O: Epidemiology of chronic subdural haematoma. **Acta Neurochir (Wien) 32:**247–250, 1975
7. Fujisawa H, Nomura S, Kajiwara K, Kato S, Fuji M, Suzuki M: Various magnetic resonance imaging patterns of chronic subdural hematomas: indicators of the pathogenesis? **Neurol Med Chir (Tokyo) 46:**333–338, 2006
8. Go KG, Houthoff HJ, Blaauweh EH, Havinga P, Hartsuiker J: Arachnoid cysts of the sylvian fissure – evidence of fluid secretion. **J Neurosurg 60:**803–813, 1984
9. Haines DE, Harkey HL, Al-Mefty O: The "subdural" space: A new look at an outdated concept. **Neurosurgery 32:**111–120, 1993
10. Huang D, Abe T, Kojima K, Tanaka N, Watauabe M, Ohkura A, et al: Intracystic hemorrhage of the middle fossa arachnoid cyst and subdural hematoma caused by ruptured mid-

dle cerebral artery aneurysm. **AJNR Am J Neuroradiol 20:**1284–1286, 1999
11. Heier LA, Zimmerman RD, Amster JL, Gandy SE, Deck MD: Magnetic resonance imaging of arachnoid cysts. **Clin Imaging 13:**281–291, 1989
12. Ibarra R, Kesava PP: Role of MR imaging in the diagnosis of complicated arachnoid cyst. **Pediatr Radiol 30:**329–331, 2000
13. Ide C, De Coene B, Gilliard C, Pollo C, Hoebeke M, Godfraind C, et al: Hemorrhagic arachnoid cyst with third nerve paresis: CT and MR findings. **AJNR Am J Neuroradiol 18:**1407–1410, 1997
14. Ildan F, Cetinalp E, Bagdatoglu H, Boyar B, Uzuneyuoglu Z: Arachnoid cyst with traumatic intracystic hemorrhage unassociated with subdural hematoma. **Neurosurg Rev 17:**229–232, 1994
15. Kulali A, Von Wild K: Post-traumatic subdural hygroma as a complication of arachnoid cysts of the middle fossa. **Neurosurg Rev 12 (1 Suppl):**508–513, 1989
16. Mori K, Yamamoto T, Horinaka N, Maeda M: 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, 2002
17. Munk PL, Robertson WD, Durity FA: Middle fossa arachnoid cyst and subdural hematoma: CT study. **J Assist Tomogr 12:**1073–1075, 1988
18. Naidich TP, McLone DG, Radkowski MA: Intracranial arachnoid cysts. **Pediatr Neurosci 12:**112–122, 1986
19. Ochi M, Morikawa M, Ogino A, Nagaoki K, Hayashi K: Supratentorial arachnoid cyst and associated subdural hematoma: neuroradiologic studies. **Eur Radiol 6:**640–644, 1996
20. Osborn AG: **Diagnostic Neuroradiology.** St Louis: Mosby, 1994, pp 639–641
21. Page A, Paxton RM, Mohan D: A reappraisal of the relationship between arachnoid cysts of the middle fossa and chronic subdural haematoma. **J Neurol Neurosurg Psychiatry 50:**1001–1007, 1987
22. Parsch CS, Krauss J, Hofmann E, Meixensberger J, Roosen K: 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, 1997
23. Prabhu VC, Bailes JE: Chronic subdural hematoma complicating arachnoid cyst secondary to soccer-related head injury: case report. **Neurosurgery 50:**195–198, 2002
24. Rengachary SS, Watanabe I: Ultrastructure and pathogenesis of intracranial arachnoid cysts. **J Neuropathol Exp Neurol 40:**61–83, 1981
25. Robinson RG: The temporal lobe agenesis syndrome. **Brain 87:**87–106, 1964
26. Rogers MA, Klug GL, Siu KH: Middle fossa arachnoid cysts in association with subdural haematomas. A review and recommendations for management. **Br J Neurosurg 4:**497–502, 1990
27. Schachenmayr W, Friede RL: Fine structure of arachnoid cysts. **J Neuropathol Exp Neurol 38:**434–446, 1979
28. Starkman SP, Brown TC, Linell EA: Cerebral arachnoid cysts. **J Neuropathol Exp Neurol 17:**484–500, 1958
29. Ulmer S, Engellandt K, Stiller U, Nabavi A, Jansen O, Mehdorn MH: Chronic subdural hemorrhage into a giant arachnoidal cyst (Galassi classification type III). **J Comput Assist Tomogr 26:**647–653, 2002
30. van Burken MMG, Sarioglu AC, O'Donnell HD: Supratentorial arachnoid cyst with intracystic and subdural haematoma. **Neurochirurgia (Stuttg) 35:**199–203, 1992
31. Williams B, Guthkelch AN: Why do central arachnoid pouches expand? **J Neurol Neurosurg Psychiatry 37:**1085–1092, 1974

Manuscript submitted November 15, 2007.
Accepted April 8, 2008.
Please include this information when citing this paper: published online October 31, 2008; DOI: 10.3171/2008.4.17509.
Address correspondence to: Maurizio Domenicucci, M.D., Via A. Graf, 42 - 00137, Rome, Italy. email: smcapone@libero.it.