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Differentiation Between Meningeal Fibrosis and Chronic Subdural Hematoma After Ventricular Shunting: Value of Enhanced CT and MR Scans

Sylvie Destian[1]
Linda A. Heier[1]
Robert D. Zimmerman[1]
Susan Morgello[2]
Michael D. F. Deck[1]

Unenhanced CT in four patients with long-standing ventricular shunts demonstrated bilateral low-density extraaxial collections that were indistinguishable from chronic subdural hematomas. After administration of contrast material, however, there was marked enhancement of the collections as well as prominent paratentorial and parafalcial enhancement. MR imaging, performed in three patients, demonstrated the extent and paradural location of the collections better than CT did, but, as with unenhanced CT, the collections could not be distinguished from chronic subdural hematomas. On follow-up CT and MR, there was no change in the size, enhancement, or intensity of the collections. Histologic examination of biopsies from two patients demonstrated fibrosis of the meninges characterized by granulation tissue and collagen deposition.

Meningeal fibrosis is a rare postshunt phenomenon that may mimic chronic subdural hematoma on unenhanced CT and MR. Recognition of this entity is important, particularly if therapeutic intervention is being considered. Therefore, an enhanced CT or enhanced MR scan should be obtained in chronically shunted patients to differentiate between a drainable chronic subdural hematoma and meningeal fibrosis.

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The development of subdural hematomas after ventricular shunting is a well-known and well-reported complication [1–10]. Although there have been scattered reports, predominantly within the neurosurgical literature, describing thickening of subdural membranes in association with chronic subdural hematomas in both shunted and nonshunted patients [11–16], to our knowledge the radiologic features of meningeal fibrosis, as a distinct sequela of chronic ventricular shunting, have not been reported. Therefore, we present the CT and MR features of four patients who developed this rare and poorly understood postshunt phenomenon.

Materials and Methods

We retrospectively studied four patients, three women and one man 20–77 years old, in whom chronic subdural collections were discovered more than 2 years after ventricular shunt placement (Table 1). The subjects, collected over several years, included two patients who were shunted at other hospitals and presented to us only when they developed symptoms. Before shunting, the initial diagnoses included a colloid cyst, normal-pressure hydrocephalus, aqueductal stenosis, and Chiari II malformation with a meningomyelocele repaired at birth. The duration of the shunt before diagnosis of the subdural collections ranged from 2 to 14 years, and none of the patients were symptomatic during this interval. When they did become symptomatic, however, two patients presented with headache, one with lethargy, and one with diplopia. Repeated CSF examinations showed no evidence of meningitis. Two of the patients were biopsied as a result of the radiologic findings and persistent symptoms.

All of the patients were initially examined by CT with and without IV contrast material. Contiguous 10-mm-thick axial sections were obtained. Two of the patients were examined on a GE 8800 scanner, one on a GE 9800 scanner, and one on EMI 1005 and 5005 scanners. A follow-up CT examination was performed in all of the patients from 1 to 15 months after

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[1] Department of Radiology, Division of Neuroradiology, New York Hospital–Cornell Medical Center, 525 E. 68th St., New York, NY 10021. Address reprint requests to L. A. Heier.

[2] Department of Pathology (Neuropathology), New York Hospital–Cornell Medical Center, New York, NY 10021.

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TABLE 1: Clinical Summary of Patients with Meningeal Fibrosis

Case No.	Age	Gender	Preshunt Diagnosis	Duration of Shunt (years)	Symptoms	Pathology
1	77	F	Normal-pressure hydrocephalus	2–3	Headache	+
2	20	M	Chronic aqueduct stenosis	7–8	Diplopia	+
3	23	F	Chiari II meningomyelocele	13–14	Headache	–
4	45	F	Colloid cyst	2–3	Lethargy	–

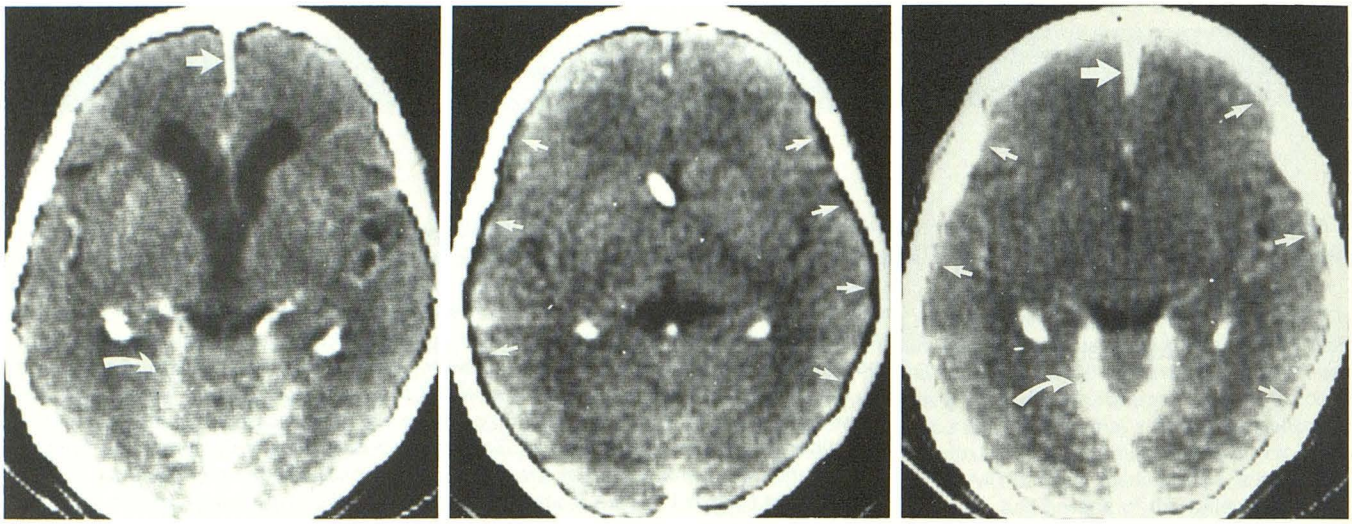


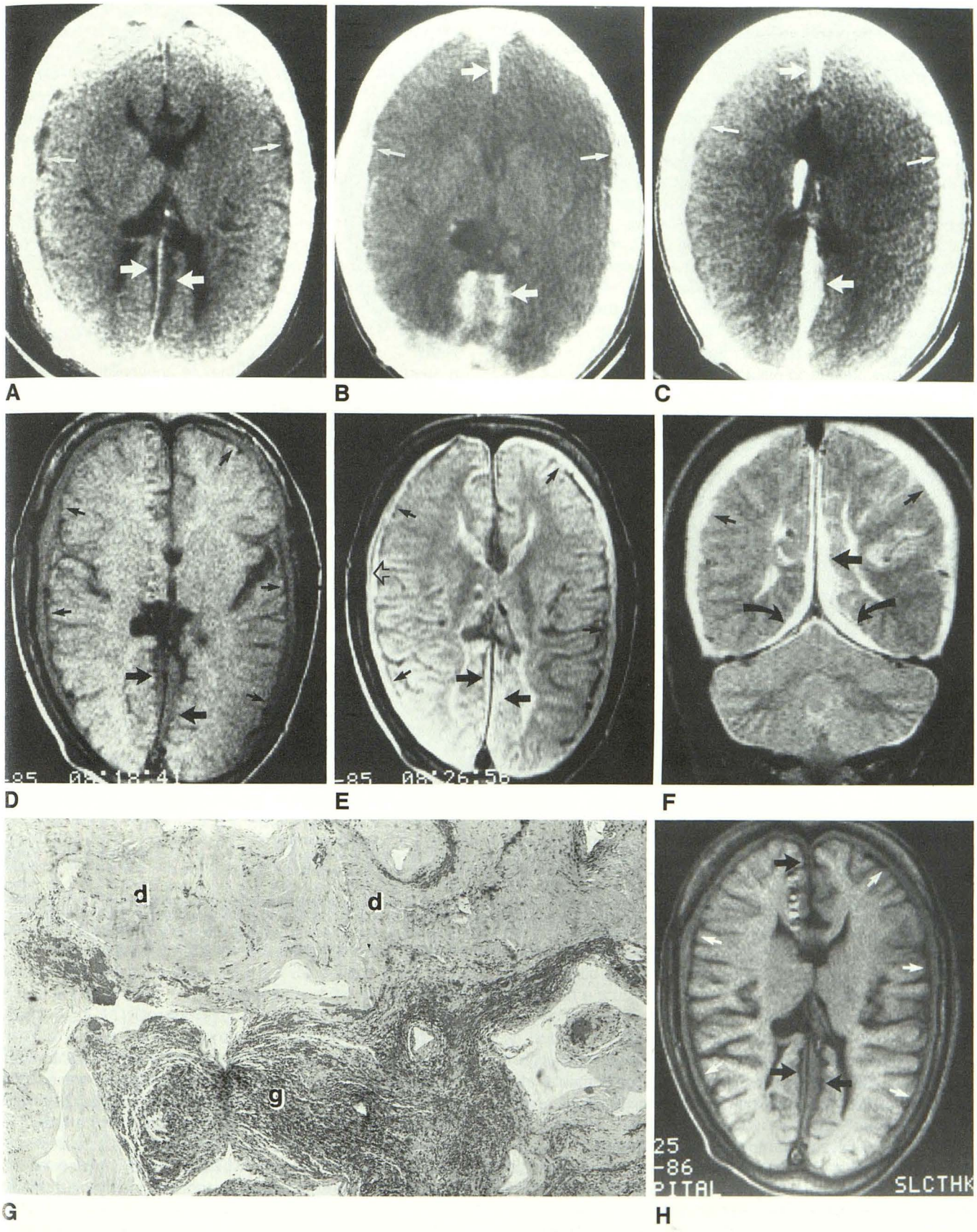
Fig. 1.—Case 1: 77-year-old woman shunted because of normal-pressure hydrocephalus.
***A*, Preshunt contrast-enhanced CT scan shows hydrocephalus and normal enhancement of falx (*straight arrow*) and tentorial incisura (*curved arrow*).**
***B*, 2 years 7 months after shunt placement. Unenhanced CT scan shows ventricular shunt, small ventricles, and small bilateral low-density convexity collections (*arrows*).**
***C*, Postshunt contrast-enhanced CT scan shows marked enhancement of convexity collections (*small straight arrows*), as well as prominent falcial (*large straight arrow*) and tentorial (*curved arrow*) enhancement.**

the initial CT study. Three of the four patients were also examined by MR within 10 days of the initial CT examination. Two of the patients were examined on a Technicare 0.6-T superconductive magnet and one patient was examined on a GE 1.5-T superconductive magnet. Short and long TR axial spin-echo pulse sequences were performed in each case. In addition, a long TR coronal sequence was performed in one case and a short TR sagittal sequence was performed in another. Short TR images were obtained by using 500–600/20–32 (TR/TE) and long TR images were obtained by using 1500–2150/32–96. Scans obtained on the 0.6-T scanner were obtained with two excitations, a 196 × 256 matrix for the short TR sequences, a 128 × 256 matrix for the long TR sequences, and a slice thickness of 7.5 mm with a 2.5-mm gap. Scans obtained on the 1.5-T magnet were obtained with one excitation, a 256 × 256 matrix, and a slice thickness of 5 mm with a 2-mm gap. A follow-up MR examination was performed in one patient 17 months after the initial MR examination.

Results

Unenhanced CT scans in all patients demonstrated a ventricular shunt, small ventricles, and bilateral low-density extraaxial collections indistinguishable from chronic subdural hematomas, without mass effect or calcification (Figs. 1B, 2A, and 3A). After administration of contrast material, there

Fig. 2 (facing page).—Case 2: 20-year-old man 7 years 5 months after shunt placement for aqueductal stenosis.
***A*, Postshunt unenhanced CT scan shows small ventricles, small bilateral low-density convexity (*thin arrows*), and interhemispheric (*thick arrows*) collections.**
***B* and *C*, Contrast-enhanced CT scans at slightly lower (*B*) and higher (*C*) levels show ventricular shunt and enhancement of convexity (*thin arrows*) and interhemispheric (*thick arrows*) collections.**
***D–F*, Postshunt 0.6-T MR scans 3 days later show bilateral convexity (*small solid straight arrows*), interhemispheric (*large solid straight arrows*), and paratentorial (*curved arrows*) collections that are hypointense relative to brain and hyperintense relative to CSF on short TR/short TE, 500/32 (*D*), sequences and hyperintense relative to brain and CSF on long TR/intermediate TE, 2150/64 (*E*), and long TR/long TE, 1500/90 (*F*), sequences. Hypointense falx and tentorium are easily distinguished from adjacent collections. Note linear hypointensity within right convexity collection (*open arrow*).**
***G*, Low-power photomicrograph of biopsy specimen obtained a few days after MR scans shows dura (d) and subdural collection of granulation (g) tissue with prominent vascularity and collagen bundles. (H and E, original magnification ×25)**
***H*, Follow-up 0.6-T short TR/short TE scan, 500/32, 17 months later shows no major change in size or intensity of convexity (*white arrows*) or interhemispheric (*black arrows*) collections.**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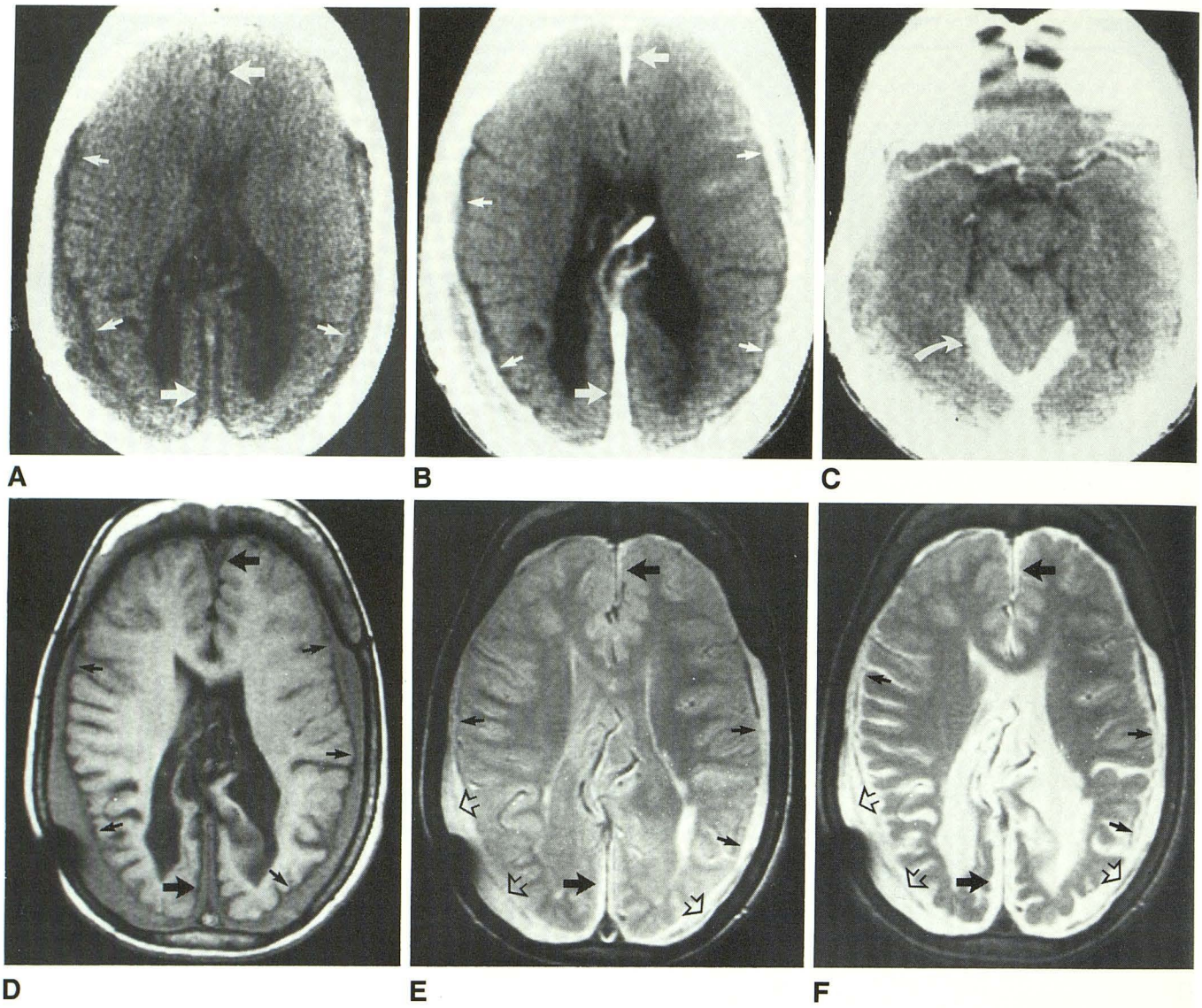

Fig. 3.—Case 3: 23-year-old woman shunted because of Chiari II malformation with repaired meningomyelocele.

***A–C*, 13 years 9 months after shunt placement, unenhanced CT scan (*A*) shows low-density convexity (*small arrows*) and interhemispheric (*large arrows*) collections that enhance markedly after administration of IV contrast material (*B*); there is also marked paratentorial (*curved arrow*) enhancement (*C*).**

***D–F*, Postshunt 1.5-T MR scans 10 days later show normal thickness and appearance of falx and bilateral convexity (*small solid arrows*) and parafalcial (*large solid arrows*) collections, which are hypointense relative to brain and hyperintense relative to CSF on short TR/short TE, 600/20 (*D*), sequences; hyperintense relative to brain and CSF on long TR/intermediate TE, 2000/40 (*E*), sequences; and hyperintense relative to brain and isointense relative to CSF on long TR/long TE, 2000/80 (*F*), sequences. Note linear hypointensities (*open arrows*) within convexity collections on long TR images.**

was marked enhancement of the convexity collections as well as prominent paratentorial and parafalcial enhancement (Figs. 1C, 2B, 2C, 3B, and 3C), compared with preshunt scans when available (Fig. 1A).

On MR, the collections appeared hypointense relative to gray matter and mildly hyperintense relative to CSF on short TR/short TE sequences (Figs. 2D, 2H, and 3D) and hyperintense relative to gray matter and CSF on long TR/intermediate TE sequences (Figs. 2E and 3E). On the long TR/long TE sequences, the collections appeared hyperintense relative to gray matter in all patients, mildly hyperintense relative to CSF in two patients, and isointense relative to CSF in one patient (Figs. 2F and 3F). In two of the patients, linear hypointensities were seen within the subdural collections on the long TR sequences (Figs. 3E and 3F). The extent of the collections was better demonstrated on MR than on CT. Moreover, on MR, the appearance of the falx and tentorium was normal, visible as thin dark lines adjacent to the collections (Figs. 2D–2F, 2H, and 3D–3F), while on CT the enhanced dura merged with, and was indistinguishable from, the enhancing collec-

tions. Thus, MR demonstrated that the collections were truly parafalcial and paratentorial, lying adjacent to, rather than within, the dura.

Follow-up CT examinations demonstrated no change in the size or enhancement of the collections. A follow-up MR examination (Fig. 2H) demonstrated no appreciable change in the size or intensity of the collections.

Histologic examinations of biopsy specimens in two patients revealed granulation tissue and marked fibrosis within the subdural space. Figure 2G is a photomicrograph of a histologic section obtained from the patient with aqueductal stenosis (Fig. 2). It demonstrates the vascular granulation tissue intermixed with collagen bundles in the subdural space.

Discussion

Many complications of ventricular shunting have been reported in the literature [1–10], including shunt malfunction, ventriculitis, and subdural hematoma. One complication that has received little attention is meningeal fibrosis in the subdural space.

Although the unenhanced CT appearances of the subdural collections in our series were indistinguishable from chronic subdural hematomas, it is clear from the enhancement after contrast administration that the collections contained little or no fluid. The parafalcial enhancement seen in Figure 1C is particularly striking when compared with the preshunt scan (Fig. 1A) in the same patient.

MR demonstrated the extent of the collections better than CT did, but again, as with unenhanced CT, the collections on MR were indistinguishable from chronic subdural hematomas. Although the signal intensities of the collections on MR are more typical of chronic hemorrhage than of fibrosis, which is typically hypointense, it is possible that fibrosis in the subdural space has a higher water content than fibrosis elsewhere in the body, or that the signal intensity reflects recurrent hemorrhage and/or organizing hemorrhage intermixed with fibrosis. The linear hypointensities visible within the convexity collections (Figs. 2E, 3E, and 3F) may represent thickened fibrous membranes or acute hemorrhage. The normal appearance of the falx and tentorium as thin dark lines on the MR scans clearly indicates that the collections are parafalcial and paratentorial, rather than dural.

Although subdural hematomas usually develop days to weeks after the procedure, it is not uncommon for chronic subdural hematomas to be detected months or even years later [3–5, 9–11]. Depending on the subjects and the methods used in different series, the reported prevalence of postshunt subdural hematomas has ranged from 3% to 28% [2–7, 9, 10]. The prevalence in adults, because of their fixed head size, is higher than that in children [2, 5, 9], and the prevalence in patients shunted for normal-pressure hydrocephalus is higher than that in patients shunted for obstructive hydrocephalus, most likely because of preexisting cortical atrophy [4, 9, 10]. Although many patients are asymptomatic, the most common presenting symptoms are headache and/or drowsiness and confusion [4–9]. Rapid decompression of the ventricular system with or without minor trauma, leading to disruption of bridging veins, is thought to represent the major etiologic factor [3–5, 9–11]. Although acute postshunt subdural hematomas are usually small and self-limited, some may persist or expand [1, 9], resulting in persistent symptoms requiring surgical evacuation.

Initially, a subdural clot consists of RBCs, fibrin, and platelets. As it organizes, there is an ingrowth of capillaries on the dural side of the clot, and fibroblasts on the dural surface proliferate. Together, the neocapillaries and fibroblasts form the outer membrane. At 1 month, the outer membrane is approximately equal in thickness to the dura [17], and it continues to thicken over time. Fibroblasts migrate from the dura to the arachnoid side of the clot, resulting in the formation of a thin inner membrane that remains relatively avascular compared with the outer membrane. Eventually, there is scant vascularization of the inner membrane as well. Because the basement membrane of the neocapillaries within the inner and outer membranes is incomplete or absent, expansion of the subdural clot as a result of spontaneous or posttraumatic recurrent bleeding is not uncommon, particularly in chronic hematomas [18, 19]. With recurrent bleeding, the continuing process of clot organization and membrane formation may result in a thick collagenous scar, similar in appearance to dura. In rare instances, calcification or ossification of the scar may occur [8, 11, 14, 17, 18].

The cause of meningeal fibrosis is unclear, but it probably represents a complication of one or more subdural hemorrhages resulting in a thick zone of fibrosis. Six reports of surgical cases have described the presence of thick subdural membranes in association with chronic subdural hematomas in shunted and nonshunted patients [12–16]. Furthermore, Barmier et al. [11] described bilateral enhancing chronic subdural hematomas on CT in a 10-year-old child who had been shunted 8 years before. Histology of the biopsy specimen showed organized hematoma and granulation tissue. Collins and Pucci [12] and Obenchain and Becker [13] reported progressive thickening of the membranes in patients with persistent subdural collections and regression of the membranes when the subdural fluid was removed, leading the authors to believe that membrane thickening is probably a reactive phenomenon. Although this may be true, they had difficulty correlating membrane thickness and success of internal drainage of the subdural collections, but noted a positive correlation between persistent symptoms and failure of internal drainage. Perhaps the fluid was loculated, or there was no fluid at all in those patients in whom drainage failed. In fact, in a study of chronically shunted patients, Faulhauer and Schmitz [15] refer to five chronic subdural hematomas as subdural callus because of their gross appearance at surgery, describing them as "unusual, thick, fibrous membranes containing pulpy and fatty material and little or no liquid." Emery [16] reported gross and microscopic thickening and increased vascularity of the meninges in children with chronic ventricular shunts, most of whom had a collagenous layer attached to the inner surface of the dura. Although it is possible that membrane formation initially may be a reaction to the persistent subdural fluid, the presence of subdural material in any form (e.g., fluid, soft tissue, or even a shunt catheter) may stimulate progressive membrane thickening.

Because our cases were collected retrospectively, and two patients were shunted at other institutions, we cannot say in what percentage of shunted patients meningeal fibrosis develops. Moreover, because these patient were not followed routinely while they were asymptomatic, it is difficult to determine when the collections developed or over what period of time the changes occurred.

Scattered reports of individual cases of meningeal fibrosis [11–16] have described the findings at surgery. The presence of the fibrosis was unsuspected preoperatively, and there is no mention of the prognosis or indications for therapy. It is too early to speculate on the prognosis of the individuals in our own series. When and in what cases therapy is indicated, as well as whether excision of the fibrous tissue or steroid therapy would alleviate the symptoms, have yet to be determined.

On the basis of our limited series, MR was better than CT in delineating the extent of the subdural collections. However, unenhanced CT and MR were unable to reliably distinguish between chronic subdural hematomas and meningeal fibrosis. The distinction between the two entities has practical therapeutic implications in those patients with persistent symptoms and persistent radiologic abnormalities. Particularly when surgical intervention is being considered, enhanced CT or enhanced MR should be performed to differentiate between a drainable chronic subdural hematoma and meningeal fibrosis in chronically shunted patients.

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