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MR Characteristics of Subdural Hematomas and Hygromas at 1.5 T

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MR images of 24 patients with 33 subdural collections were retrospectively reviewed to determine the spectrum of MR findings associated with such lesions. The lesions were dated by history, when available. Hematomas were grouped as follows: acute, four; early subacute, four; late subacute, four; chronic, 13. Six collections were classified as rehemorrhage; and two patients had CSF hygromas. Subdural hematomas evolved in a pattern similar to intracerebral hemorrhage with the exception of chronic subdural hematomas, in which isointensity or hypointensity relative to gray matter was observed on short TR/TE images compared with the persistent very high signal intensity noted in chronic parenchymal hematomas. Hemosiderin was rarely seen in chronic hematomas. These findings are most likely the result of the absence of a blood-brain barrier, which allowed clearance and dilution of blood products. Subdural hematomas with repeat hemorrhage demonstrated multiple phases of bleeding with layering phenomenon and more frequent hemosiderin deposition. It is possible that the clearance of blood products, as observed in chronic subdural hematomas, is impaired or poorly functional when rehemorrhage occurs. The persistence of high signal from methemoglobin in a hematoma that is expected to be in the chronic phase also suggests repeated hemorrhage. Acute CSF subdural hygromas had signal intensities identical to CSF without MR evidence of blood products. At surgery, clear fluid under pressure was found.

MR imaging, with its unique ability to delineate the various phases of hemorrhage, is well suited to the evaluation of subdural hemorrhage.

Although the appearance and evolution of hemorrhage in the brain on high-field MR images is well established [1], subdural hemorrhage is less well characterized. Physiologically and anatomically, the subdural space differs from the brain parenchyma. It is a potential space-lacking tissue as well as a blood-brain barrier. Such differences may alter the MR appearance and evolution of subdural hemorrhage when compared with bleeding into the cerebral parenchyma. The known propensity for repeat hemorrhage in subdural hematomas may further complicate the MR signal intensity patterns. Our study is an attempt to categorize and understand the MR appearance of subdural hemorrhage.

Materials and Methods

The MR images of 24 patients (ages 1 month to 70 years) with subdural hematomas and hygromas were reviewed retrospectively. Imaging was performed on a 1.5-T GE imager using spin-echo (SE) sequences with 600/20 (TR/TE) and 2500–3000/30–80/2 and a matrix size of 128 × 256. Serial imaging was performed in four patients. Hemorrhages were classified by the patients' clinical history (i.e., time interval between insult and MR scan) as acute (<1 week old), early subacute (>1 week and <2 weeks old), late subacute (>2 weeks old and <1 month old), or chronic (>1 month old). These categories, which follow those developed by Gomori et al. [1, 2] for parenchymal hematomas, can be summarized as follows: Acute hematomas are characterized by hypointensity on long TR images. Early subacute hematomas have peripheral hyperintensity on short TR images and hypointensity centrally on long TR/TE sequences. The late subacute subdural hematoma is hyperintense on both long and

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short TR images. Chronic parenchymal hematomas are hyperintense on long and short TR images and are surrounded by a hemosiderin rim. In subdural hematomas the presence of hemosiderin is indicated by susceptibility changes (hypointensity on long TR/TE images) in thickened membranes or clumps of material.

Results

Thirty-three subdural collections were present in 24 patients and were grouped as follows: acute, four; early subacute, four; late subacute, four; chronic, 13; and rehemor-

TABLE 1: Summary of 24 Patients with Subdural Hematomas*

Case No.	Age	Interval	Comment
Acute SDH (<1 week old)			
1.	55 yr	1 day	MVA
2.	8 yr	3 days	Hemophilia
	(patient also had a late subacute SDH)		
3.	2 mo	5 days	Skull fracture
4.	33 yr	5 days	MVA
Early subacute SDH (>1 week and <2 weeks old)			
5.	5 yr	9 days	Preop debris
6.	1 mo	13 days	MVA
7.	55 yr	14 days	MVA
8.	59 yr	14 days	Shunt out
Late subacute SDH (>2 weeks and <1 month old)			
9.	6 mo	10 days	Abuse
10.	4 mo	Unknown*	Abuse
11.	5 yr	23 days	MVA
Chronic SDH (>1 month old)			
12.	2 yr	30 days	Postop
13.	70 yr	>30 days, est	On Coumadin
14.	9 wk	>30 days, est	Abuse
15.	4 mo	>30 days, est	Abuse
16.	4 mo	>30 days, est	Abuse
17.	5 mo	>30 days, est	Abuse
18.	15 mo	>30 days, est	Abuse
Subdural hygroma			
19.	7 wk	3 days	Head trauma
20.	4 yr	15 days	MVA
Rehemorrhage			
21.	4 yr	8 mo	(Follow-up scan 4 months later). New bleeding in known chronic collections.
22.	8 yr	9 mo	(Follow-up scan 42 days later). New bleeding in known chronic collections.
23.	31 yr	7 mo	Since initial trauma, 2-week history of headache, presented with papilledema.
24.	41 yr	Unknown	Autopsy revealed chronic SDH with acute rehemorrhage.

Note.—est = estimate (in some cases the exact date of injury was unknown but a reasonable estimate could be made); unknown* = interval from injury to examination was unknown, classified with SDH of similar signal intensities; MVA = motor vehicle accident.

* Hematomas were bilateral in nine patients, for a total of 33.

rhage, six. There were two hygromas. The results are presented in Table 1. Subdural hematomas evolved in a pattern similar to parenchymal hematomas in the acute and subacute stages. Acute subdural hematomas were characterized by

the presence of hypointensity on long TR/TE images (Fig. 1). In the early subacute stage we noted a rim of high signal intensity, presumably due to free methemoglobin, on all pulse sequences surrounding a center of low signal intensity most

Fig. 1.—Case 2: hemophiliac with acute right suboccipital SDH and incidentally discovered left subacute SDH.

A, CT scan shows hyperdense acute right occipital SDH.

B, MR image, 2500/80, shows low signal intensity of deoxyhemoglobin in acute right SDH and high signal from methemoglobin in subacute left SDH. Note absence of hemosiderin in left subacute SDH.

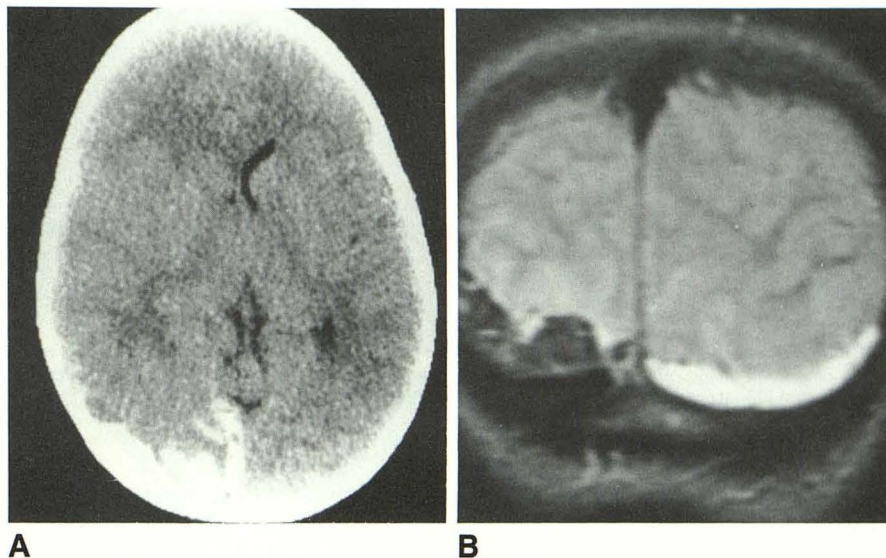


Fig. 2.—Case 7: early subacute SDH 14 days after motor vehicle accident.

A, Sagittal MR image, 600/20, shows peripheral high signal intensity of extensive frontotemporal SDH.

B, MR image, 2500/80, shows peripheral hyperintensity of methemoglobin surrounding a center of low signal intensity deoxy- or intracellular methemoglobin (arrows).

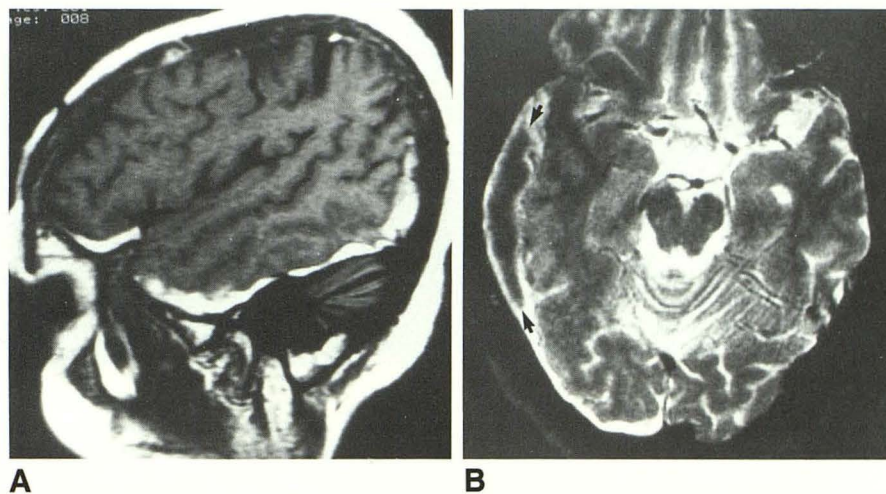
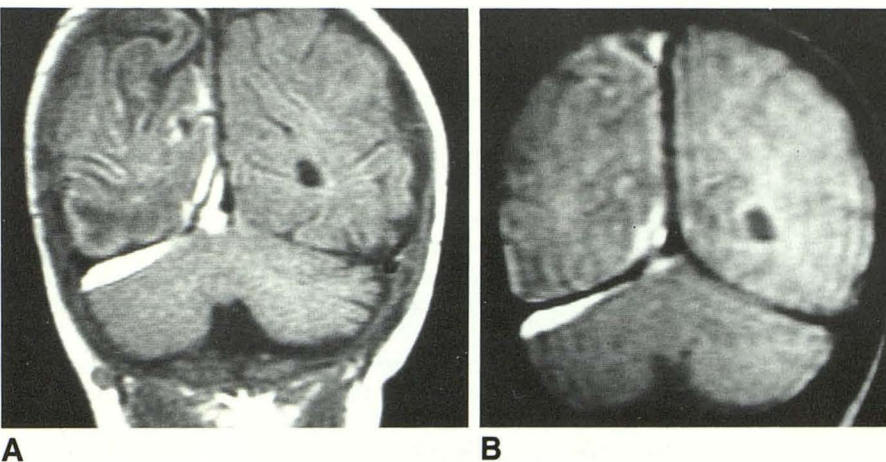


Fig. 3.—Case 10: late subacute SDH in a case of child abuse. Exact interval from injury to examination is uncertain.

A, Coronal MR image, 600/20, shows high signal intensity in small right tentorial SDH.

B, Coronal MR image, 3000/30, also shows high signal intensity in right tentorial SDH.



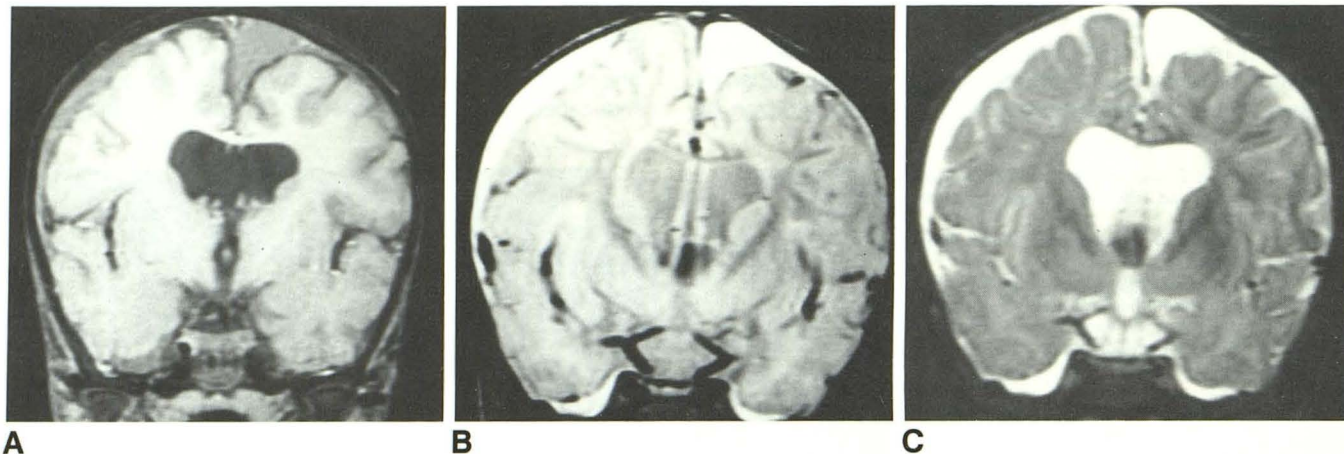


Fig. 4.—Case 14: chronic SDH in a case of child abuse. Injury interval estimated at >30 days.

A, Coronal MR image, 600/20, shows extracerebral collection with signal intensity isointense relative to gray matter in vertex and convexities.

B, Coronal MR image, 3000/30, shows hyperintensity of bilateral chronic SDH while ventricular CSF remains hypointense.

C, Coronal MR image, 3000/80, shows hyperintensity of SDH.

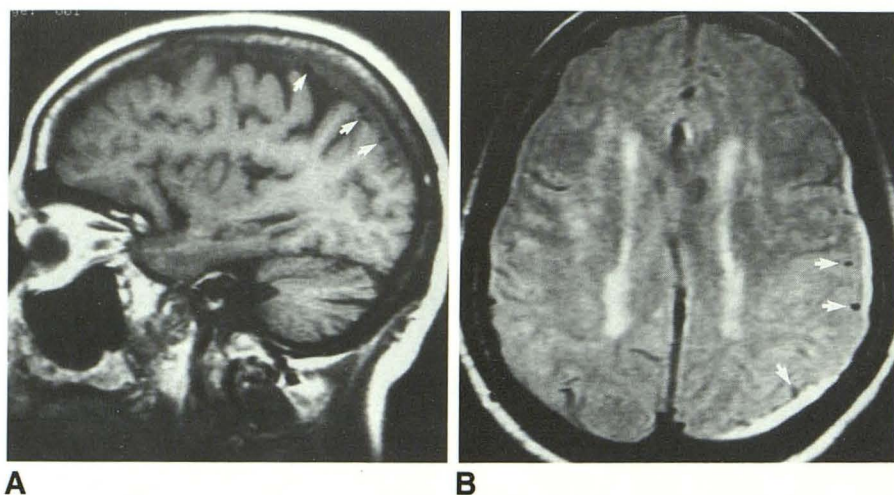


Fig. 5.—Case 13: elderly patient on Coumadin.

A, Sagittal MR image, 600/20, of a small chronic SDH in an elderly patient with large sulci showing inward displacement of veins (arrows) by SDH, a useful anatomic criterion for distinguishing subdural collections from simply enlarged CSF spaces.

B, Axial MR image, 3000/80, shows hyperintense chronic SDH and inwardly displaced veins (arrows).

marked on long TR/TE images (Fig. 2). In the late subacute hematoma, the entire volume of the collection had high signal intensity on all pulse sequences (Fig. 3). No hemosiderin was seen in acute or subacute collections.

Subdural hematomas differed significantly from parenchymal hematomas in the chronic phase (Fig. 4). The signal intensity ranged from slightly hypointense to isointense relative to gray matter on short TR/TE images, in contrast to the persistent high signal intensity seen in chronic parenchymal hematomas on both long TR/short TE and long TR/TE images [1]. In contrast to brain hemorrhage, hemosiderin was detected in only one of 13 chronic subdural hematomas. The presence of hemosiderin was noted to occur in the presence of either thickened membranes or was associated with clumps of material, which demonstrated susceptibility changes on long TR images. An anatomic observation in MR images of subdural hematomas that has proved useful in angiography is the inward displacement of superficial veins against the

brain by the subdural collection. Although this sign is not needed to diagnose the presence of subdural hematomas on MR, it can be useful in differentiating chronic subdural collections from enlarged CSF spaces resulting from atrophy (Fig. 5).

Chronic subdural hematomas with rehemorrhage demonstrated several unique characteristics, including layering phenomenon and more consistent hemosiderin deposition. The evolution from acute to subacute rehemorrhage and from the subacute to the chronic phase was demonstrated on serial scans in one patient (Fig. 6). Persistence of high signal intensity, typical of the subacute stage, beyond its expected time interval was present in two collections, which were repeatedly rehemorrhaging, as documented either clinically or by CT (Fig. 7). This patient also showed membranous deposition of hemosiderin (Fig. 7B).

In one patient, a surgically proved bilateral acute subdural CSF hygroma was present, which contained no blood prod-

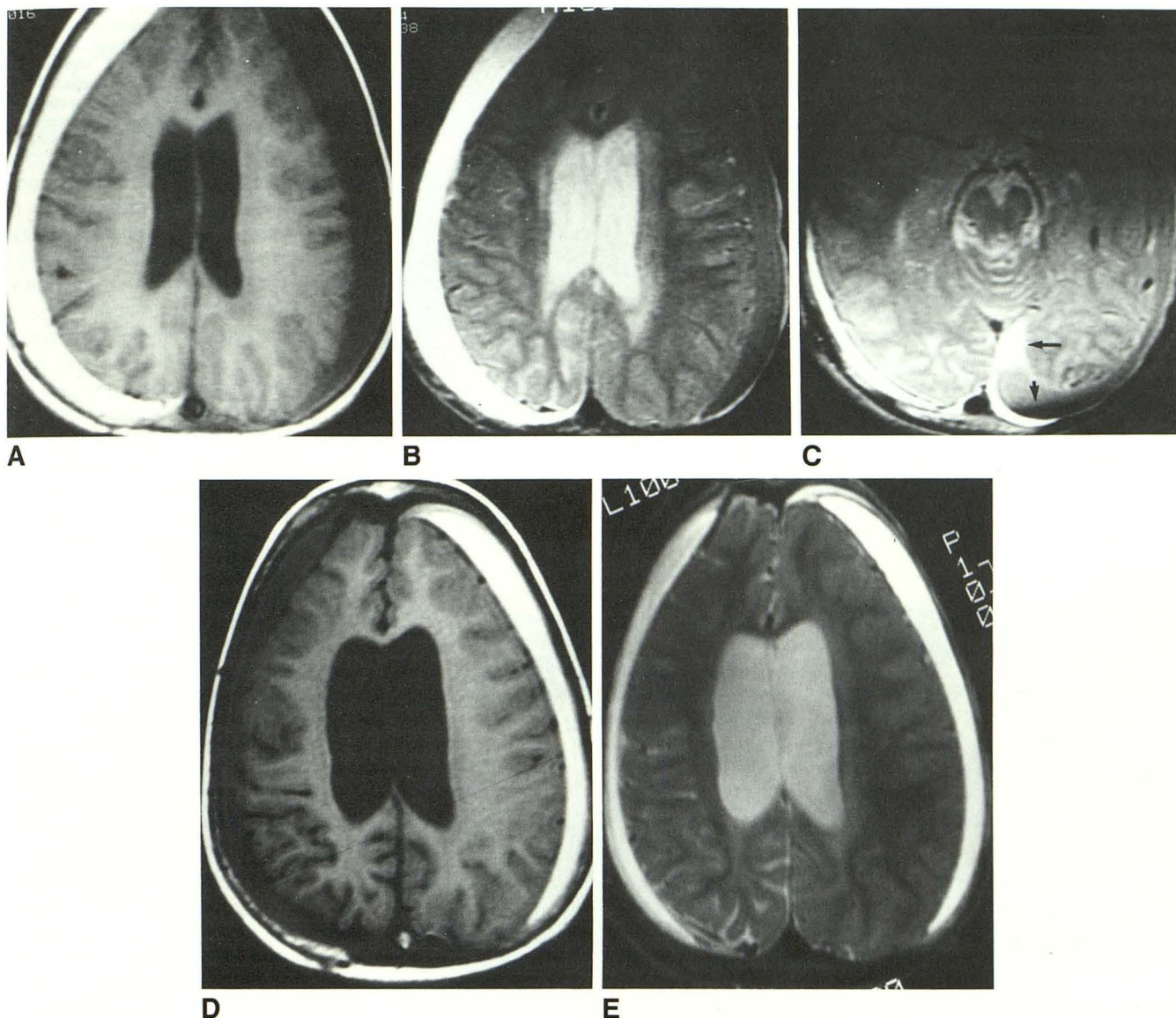


Fig. 6.—Case 21: routine follow-up scan in patient with known chronic SDH of 8 months duration after shunting for a tectal glioma shows acute rehemorrhage on the left and a right-sided subacute rehemorrhage. Although clinically asymptomatic, this bleeding into preexisting chronic subdural collections was new compared with prior CT scans.

A, MR image, 600/25, shows a high-intensity, right-sided subacute SDH as well as a low-intensity acute rehemorrhage of left SDH. On this scan alone it cannot be determined whether the low signal intensity on the left is due to a CSF collection or deoxyhemoglobin.

B, MR image, 3000/80, shows diffuse low-intensity susceptibility effects in the collection on the left, confirming the presence of deoxyhemoglobin in an acute rehemorrhage. The right-sided subacute rehemorrhage remains high in signal intensity.

C, MR image, 3000/80, shows layering effect and loculations. There is a dependent layer of low-intensity deoxyhemoglobin (*short arrow*) and a plasma supernatant of high signal intensity (*long arrow*). There is also an area of loculated high signal fluid posteromedially.

D–E, Repeat scans 4 months after initial scan show the evolution from subacute to chronic SDH on the right and from acute to subacute on the left. The persistence of the high signal from methemoglobin on the left over 4 months is most likely due to repeated hemorrhage.

D, MR image, 600/25, shows evolution of right-sided SDH from high intensity subacute to low-intensity chronic SDH. The left-sided collection has evolved from acute to subacute.

E, MR image, 3000/80, shows high signal intensity in both collections.

ucts. These collections demonstrated signal intensities that were identical to CSF (Fig. 8). In this case, surgery revealed clear CSF under pressure in the subdural space.

Discussion

The MR characteristics of acute and subacute subdural hematomas are similar to those of intraparenchymal hemor-

rhage [1, 2], whereas chronic subdural hematomas differ from parenchymal hematomas in several ways. As opposed to the typical parenchymal hematoma, which is markedly hyperintense on short TR/TE images, a chronic subdural hematoma will usually be slightly hypointense to isointense relative to gray matter on short TR/TE images. This loss of the T1 shortening effect appears to result from a decrease in the concentration of free methemoglobin by either dilution, ab-

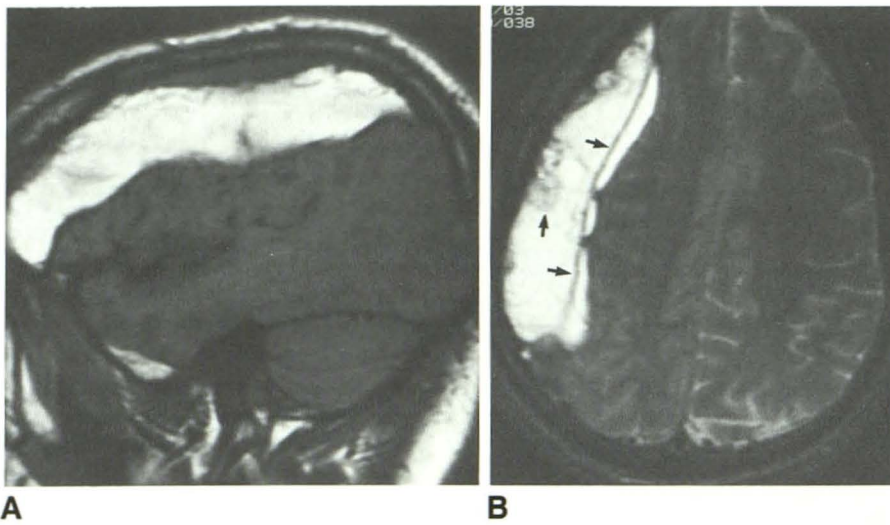


Fig. 7.—Case 23: patient with head trauma 7 months previously presented with a 2-week history of increasing headache and papilledema.

A, Sagittal MR image, 600/20, shows high signal intensity subacute rehemorrhage.

B, Axial MR image, 3000/80, demonstrates high signal intensity collection with thickened membranes showing hypointensities characteristic of hemosiderin (arrows).

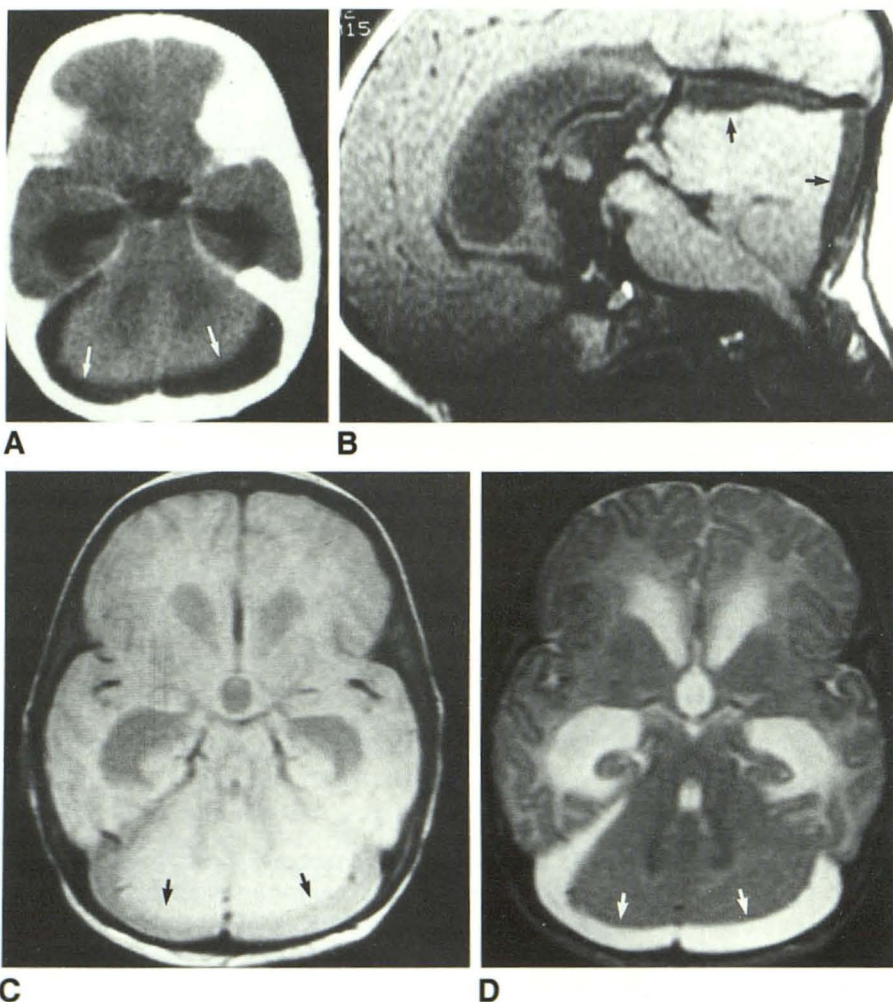


Fig. 8.—7-week-old girl was accidentally dropped on her head and presented to the emergency room 3 days later obtunded and with cerebellar signs.

A, CT scan shows hypodense bilateral posterior fossa fluid collections (arrows) and acute hydrocephalus with temporal horn dilatation.

B, Sagittal MR image, 600/20, shows collections around cerebellum identical in signal intensity to CSF (arrows).

C and D, Axial MR images, 3000/30 (C) and 3000/80 (D), show that signal intensity of collections is identical to CSF (arrows).

sorption, and/or degradation. This is clearly shown on longitudinal imaging of the subdural hematoma shown in Figure 6, where hyperintensity on short TR/TE images converts to isointensity 4 months later.

The infrequent presence of hemosiderin, manifested by hypointensity on long TR/TE images, in chronic subdural hematomas as noted in this study is quite different from that in chronic intraparenchymal hematomas, in which it is a constant feature [3]. However, the marked hypointensity of hemosiderin was seen in thickened membranes and multiple clumps in three of four of the subdural collections that had rehemorrhaged. It is possible that, owing to the absence of a blood-brain barrier in the subdural space, hemosiderin is largely resorbed into the bloodstream. In repeat hemorrhages the clearance mechanism may be poorly functional, resulting in greater hemosiderin deposition.

Rehemorrhage is a frequent phenomenon with subdural hemorrhage and MR offers excellent visualization of this process. Because of the presence of a vascular membrane, subdural hematomas are prone to rehemorrhaging even without clinically evident trauma [4, 5]. Studies utilizing radio-nuclide-tagged red blood cells indicate that repeat hemorrhage in chronic subdural hematomas occurs at the average rate of 10.2% of the subdural hematoma's volume per day [5]. MR demonstrated repeat hemorrhages with layering and dilution of deoxyhemoglobin (MR changes of acute hemorrhage) in known chronic collections. Subacute hemorrhage was also seen in hematomas known to be chronic by clinical history.

Much of the previous literature emphasized the superior ability of MR to visualize subdural hematomas that were isodense on CT with little emphasis on the appearance or evolution of hemorrhage [6-8]. Hosoda et al. [9], using a much broader classification of chronic SDH, described a series of 20 chronic subdural hematomas that varied in appearance from hyper- to hypointensity on short TR images. However, if their individual cases are categorized by our time criteria, the hyperintense collections are subacute and the hypointense collections are chronic, as in our cases.

In addition to the observations made on the appearance and evolution of subdural hemorrhages, MR demonstrated nonhemorrhagic acute subdural hygromas in two cases. These collections displayed signal intensities that followed CSF without MR evidence of hemorrhage. In one of these

cases fluid analysis demonstrated nonhemorrhagic, clear CSF. Such collections are presumably due to tears in the arachnoid membrane [10].

In conclusion, acute and subacute subdural hematomas follow the signal intensity pattern at 1.5 T of intraparenchymal brain hemorrhage, but they differ in the chronic phase. As opposed to the typical chronic parenchymal hematoma, which is markedly hyperintense on short TR/TE images, the chronic subdural hematoma may be slightly hypointense to isointense relative to gray matter on short TR/TE images. We hypothesize that these signal intensity changes are the result of a decrease in the concentration of free methemoglobin by either dilution, absorption, and/or degradation. Hemosiderin, which is a constant feature of parenchymal hematomas, was seen only rarely in chronic subdural hematomas. The phenomenon of repeated hemorrhage was characterized by layering effects as well as more frequent hemosiderin deposition. MR, with its unique ability to image the various phases of hemorrhage, is well suited to the evaluation of subdural hemorrhage.

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