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Neuroradiologic Diagnosis of Subdural Empyema and CT Limitations

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Five cases of subdural empyema are described. Two of the cases eluded a definitive computed tomography (CT) diagnosis despite classical clinical background. Extracerebral collection with definitive border enhancement was seen in the other three cases. Mass effect, present in all five cases, was related to the extracerebral collection in three cases and diffuse cerebral edema and/or infarction in two. Angiography in four cases initially demonstrated an extracerebral collection in three and inflammatory angiospasm in two. Repeat angiography demonstrated an extracerebral collection in the fourth case. In the proper clinical setting subdural empyema should be considered even in the absence of an extracerebral collection when mass effect or an infarction pattern is seen on CT. Angiography may be diagnostic in such cases. Hopefully, newer techniques will further the diagnostic efficacy of CT in this disease.

Computed tomography (CT) is essential for diagnosis and management of cerebral infections. Although extensive literature [1-8] is available for CT diagnosis of intracranial abscesses, subdural empyema specifically has not received adequate attention in recent reports. We describe our experience with five cases of subdural empyema, two of which had nonspecific CT findings, and the use of angiography in the appropriate clinical setting.

Case Reports

Case 1

A 21-year-old woman was seen with marked deterioration in sensorium, fever, and nuchal rigidity 3 weeks after a gunshot wound to the left orbit that had resulted in a dural laceration. Cerebrospinal fluid examination revealed leukocytosis with polymorphonuclear predominance. CT revealed a low density subdural collection with border enhancement on the left and midline shift to the right (fig. 1A). Angiography immediately after CT demonstrated a frontoparietal extracerebral collection without changes in the arterial caliber or occlusions of vessels (fig. 1B). At surgery a subdural empyema was drained.

Case 2

A 7-month-old girl had an upper respiratory tract infection that was treated with antibiotics 3 weeks before admission. Fever, vomiting, and lethargy developed 7 days before admission, and 5 days thereafter became obtunded with a stiff neck. At this time, provisional diagnosis of meningitis was made. On admission to Hermann Hospital in Houston, Tex. she was opisthotonic with deviation of head and eyes to the left and had rapid shallow breathing. Examination demonstrated sustained clonus on the right side and a possible mild right hemiparesis. Lumbar puncture revealed turbid, yellowish cerebrospinal fluid with a white blood cell count of 4,300/mm³ with polymorphonuclear predominance, protein of 10.16

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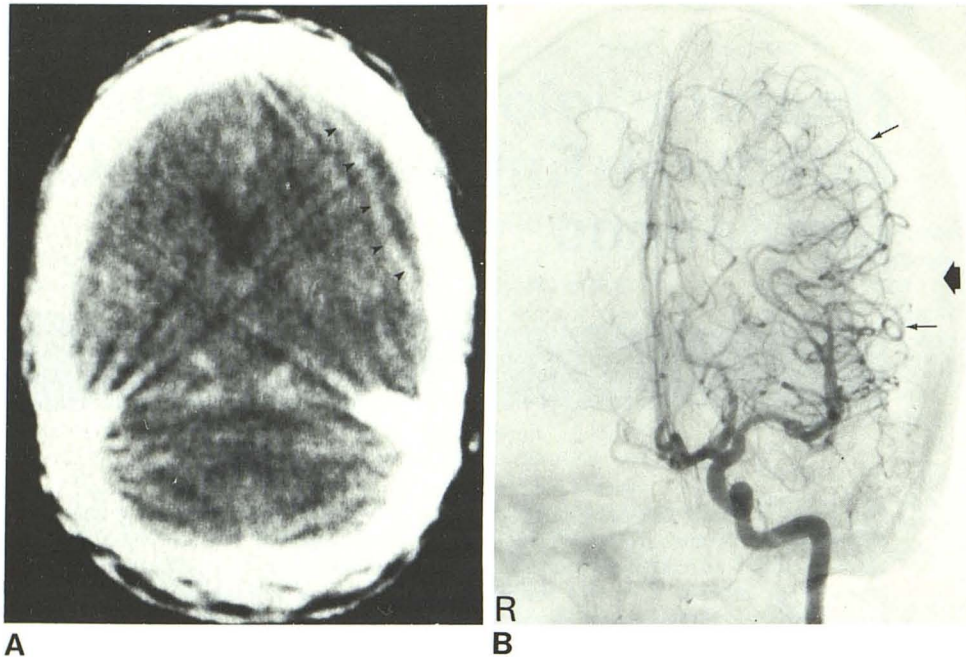


Fig. 1.—Case 1. **A**, Contrast-enhanced scan. Low density extracerebral collection with border enhancement on left (*arrowheads*) and midline shift of frontal horns to right. **B**, Midarterial phase of left internal carotid angiogram. Extracerebral crescentic collection (*arrows*) without any definite caliber changes of cerebral vasculature.

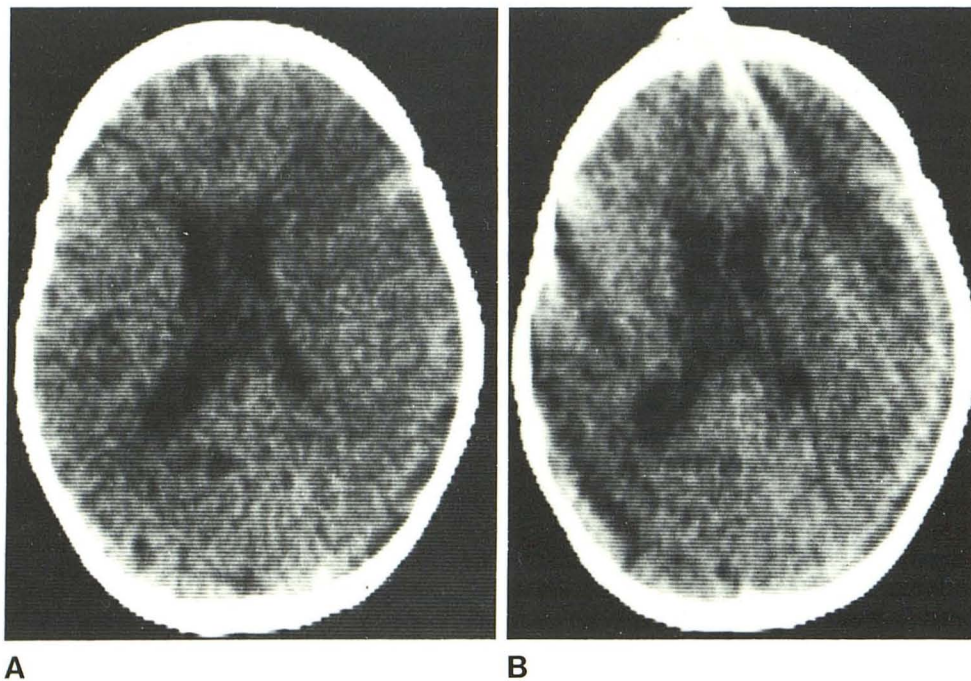


Fig. 2.—Case 2. **A**, Low density extracerebral collection on left. **B**, border enhancement after contrast injection.

g/dl, and glucose of 4 g/dl. The patient was put on a combination of antibiotics.

CT 1 day after admission showed a subdural collection of fluid with medial rim enhancement (fig. 2). Mass effect in the form of diminution of sulci on the periphery of the left hemisphere was seen. A diagnosis of subdural empyema was suggested. After CT a left parietal craniotomy revealed yellow mucoid pus along with a yellow membrane covering the brain surface. *H. influenzae* type B was cultured from the pus. Follow-up CT 5 days after admission showed a low density zone in the left frontal area suggestive of edema or infarction. Angiography 12 days after admission revealed a residual

left extracerebral collection. At 20 days after admission, the patient's neurologic status was gradually improving.

Case 3

An 11-year-old boy was admitted with a diagnosis of meningitis. He had complained of headaches 4 days before admission. During the next 24 hr he also developed malaise, fever, and neck pain. At this time the patient was treated with oral antibiotics, but 1 day before admission he developed staggering gait, nuchal rigidity, muscle weakness, and spiking temperatures and he had a gener-

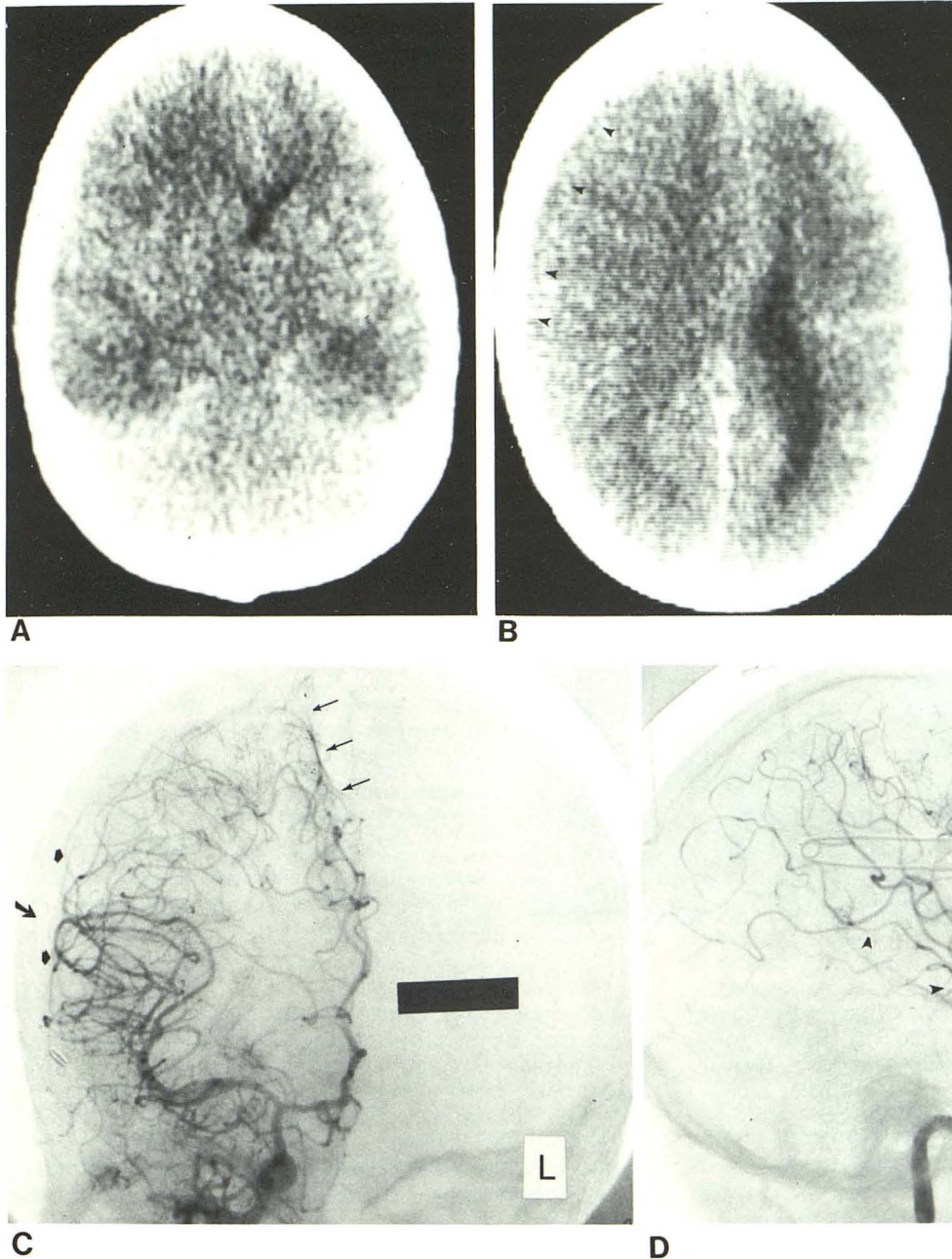


Fig. 3.—Case 3. A and B, Contrast-enhanced scans. Right hemispheric mass effect secondary to diffuse cerebral edema with midline shift to left and obliteration of right lateral ventricle. Small extracerebral low density crescentic zone suggested (B, arrowheads). C, Anteroposterior view. Arterial phase of right internal carotid injection. Parafalcine (thin arrows) and right hemispheric (short arrows) extracerebral collection. Inner table of skull (longer thick arrow). D, Caliber changes in cerebral arteries (arrowheads). Prominent anterior falcine arteries (arrows).

alized seizure. On admission, the cerebrospinal fluid revealed a white blood cell count of $5,200/\text{mm}^3$, glucose of 6.9 g/dl, and protein of 91 g/dl. Gram stain was negative.

CT scan 1 day after admission was interpreted as showing right cerebral edema with displacement of the midline structures to the left (figs. 3A and 3B). In retrospect, the contrast-enhanced scan showed an equivocal low density crescentic zone with slight border enhancement paralleling the inner table of the skull. The patient deteriorated neurologically 2 days after admission, developing a right third, a right sixth, and a left seventh nerve palsy. A left hemiparesis was also found. Clinical diagnosis of encephalitis was made and the patient was taken to surgery for a brain biopsy. A large amount of subdural pus was encountered on a right temporo-parietal craniotomy. Unidentified Gram-negative anaerobic rods were grown on culture. After operation, angiography was performed

on two separate occasions to localize other sites of subdural empyema. Extracerebral collections were observed on both cerebral convexities and with the interhemispheric fissure. Attenuation in the caliber of peripheral vessels as well as vessels at the base of the brain was noted. Prominent anterior falcine arteries were observed (figs. 3C and 3D). Pansinusitis was diagnosed on subsequent sinus and skull radiographs. After a protracted course, the patient was discharged. When last seen 17 months after his initial admission, he was neurologically normal, seizure-free, and doing well in school.

Case 4

A 31-year-old man was seen at another hospital with a 2 week history of severe headache, elevated temperature, and numbness and weakness of the right leg. Over an 8 hr period this progressed

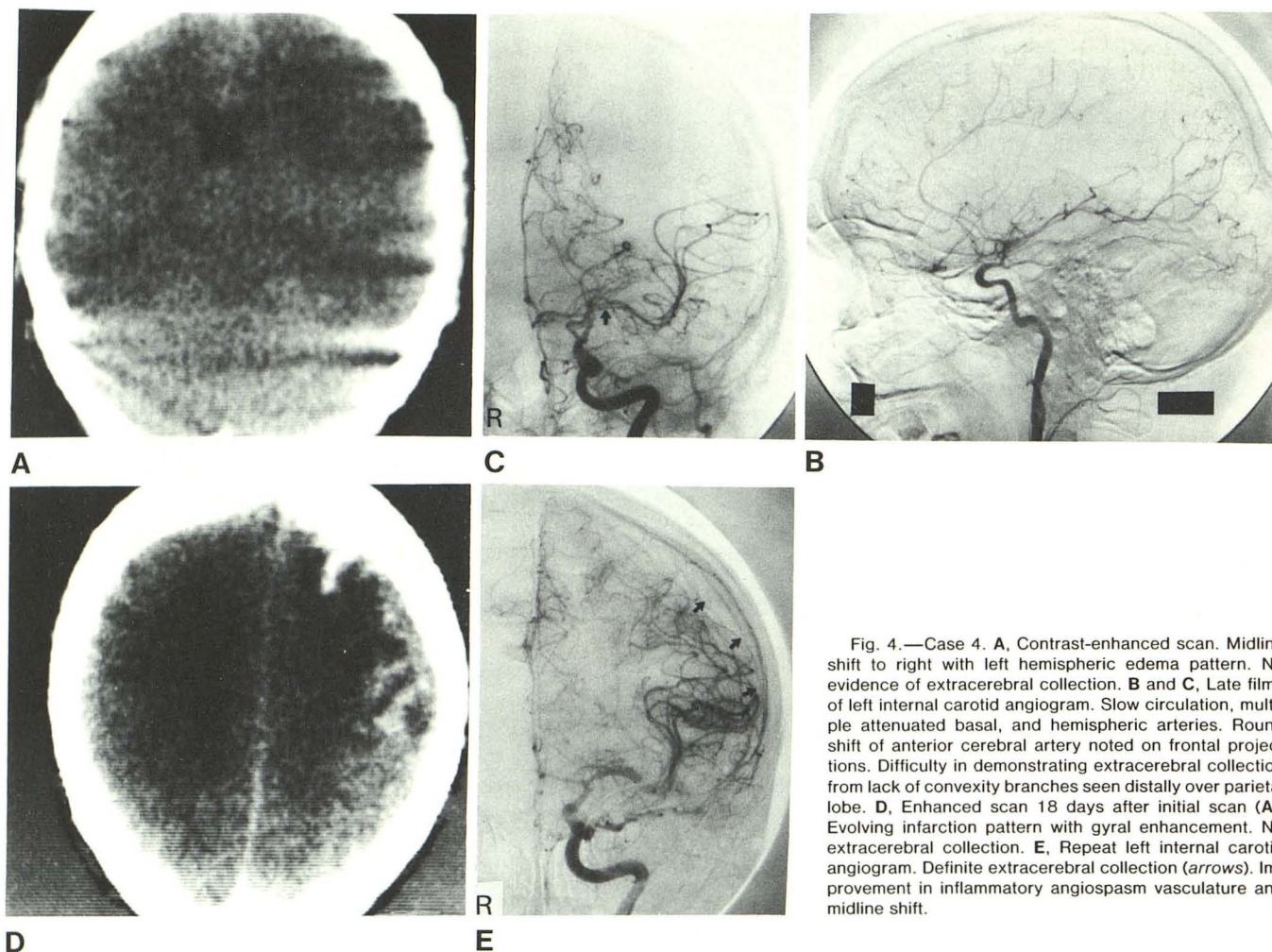


Fig. 4.—Case 4. **A**, Contrast-enhanced scan. Midline shift to right with left hemispheric edema pattern. No evidence of extracerebral collection. **B** and **C**, Late films of left internal carotid angiogram. Slow circulation, multiple attenuated basal, and hemispheric arteries. Round shift of anterior cerebral artery noted on frontal projections. Difficulty in demonstrating extracerebral collection from lack of convexity branches seen distally over parietal lobe. **D**, Enhanced scan 18 days after initial scan (**A**). Evolving infarction pattern with gyral enhancement. No extracerebral collection. **E**, Repeat left internal carotid angiogram. Definite extracerebral collection (arrows). Improvement in inflammatory angiospasm vasculature and midline shift.

to a right hemiplegia. After a focal seizure, the patient was transferred to Hermann Hospital. At this time he was found to have nuchal rigidity, slurred speech, and depressed mentation. Lumbar puncture revealed an opening pressure of 230 mm water, protein of 16 g/dl, and white blood cell count of 1,000/mm³ with 35% lymphocytes. Treatment was continued with chloramphenicol, penicillin, and streptomycin, which was begun at the other hospital.

Admission CT showed a midline shift from left to right with a low density zone of edema in the left frontal lobe (fig. 4A). No extracerebral collection was noted on either the pre- or postcontrast scans. Sinus series showed clouding of the left frontal sinus with loss of mucoperiosteal line consistent with sinusitis. Angiography revealed a prolonged circulation time and several poorly opacifying middle cerebral artery branches. A round shift to the left anterior cerebral artery was also observed (figs. 4B and 4C). CT scans over the next 18 days demonstrated a gyral enhancement pattern with improvement in mass effect (fig. 4D). Repeat angiography 18 days after the initial study revealed an extracerebral collection and improvement in the diffuse arterial changes (fig. 4E). A subdural empyema was evacuated through a left frontoparietal craniotomy. Follow-up almost 1 year after initial admission revealed a residual right hemiparesis. A low density zone indicating infarction of the left frontal lobe was seen on CT.

Case 5

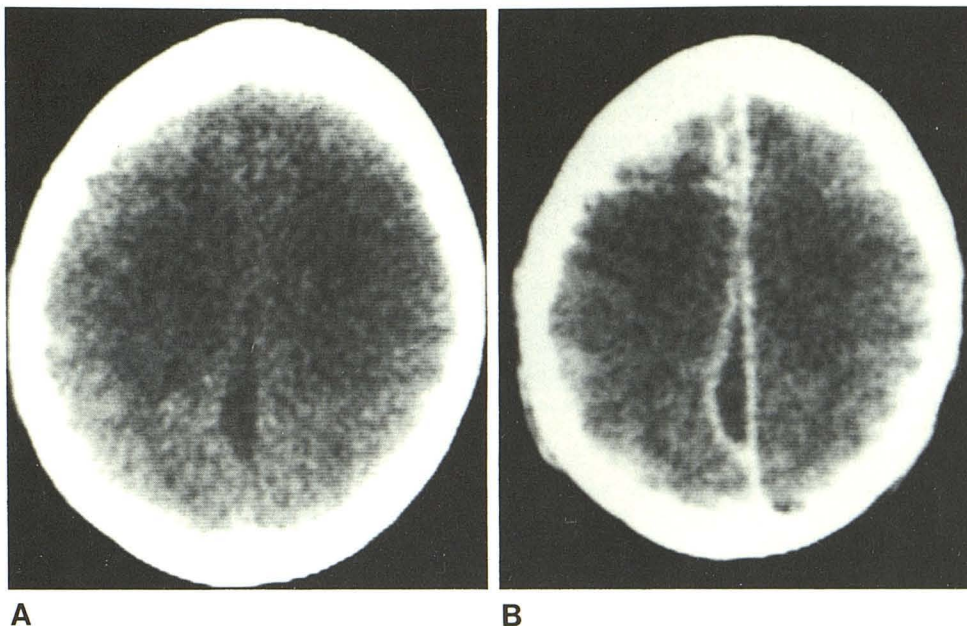
An 18-year-old man was transferred from another hospital with a 6 day history of headache, fever, chills, and photophobia. Lumbar

puncture at the other hospital showed a white blood cell count of 1,000/mm³ with polymorphonuclear predominance; glucose, 4.5 g/dl; and protein, 12.2 g/dl. Peripheral white blood cell count was 19,200/mm³. Subsequently, the patient became stuporous and developed right leg weakness and bilateral papilledema. Paranasal sinus radiographs showed frontal sinusitis. The patient was then transferred to St. Luke's Episcopal Hospital in Houston, Tex. where the frontal sinuses were drained. Despite antibiotic treatment the patient deteriorated more the next day. CT showed what was interpreted as a right frontal abscess and associated interhemispheric empyema (fig. 5). This was confirmed at surgery where a brain abscess and interhemispheric empyema were drained. After repeat drainage of the interhemispheric empyema, the patient had a satisfactory hospital course and was subsequently discharged.

Discussion

Subdural empyema, a neurosurgical emergency [9], should be considered when a patient has fever and focal neurologic deficits. It should also be suspected in a patient with meningitis who is refractory to medical therapy. Concomitant or previous history of paranasal sinusitis, osteomyelitis of the calvarium, otitis media, or postcraniotomy infection should alert the clinician to the possibility of subdural empyema [10]. Symptoms are related to an extracerebral collection of pus and increased intracranial pres-

Fig. 5.—Case 5. **A**, Low density interhemispheric collection. **B**, Peripheral enhancement after contrast injection. Subdural empyema proven at surgery.



sure. Venous thromboses and infarction may ensue and lead to further complications such as seizures, hemiparesis, hemianopsia, and aphasia [11]. The course is often catastrophic and mortality may range from 25% to 40% [12].

Computed tomography has become essential for diagnosis and management of cerebral infection [8]. However, subdural empyema has not received specific attention in recent literature. Zimmerman et al. [8] reported that subdural empyema may appear as an extracerebral low density collection with mass effect. Medial or border enhancement has not been described in subdural empyema on contrast-enhanced scans.

We found that subdural empyema may be seen with nonspecific CT findings. Three of our five cases revealed a definite low density extracerebral collection with medial or border enhancement (figs. 1A, 1B, 2A, 2B, 5A, and 5B). Case 3 revealed a subtle low density extracerebral collection without definite border enhancement (fig. 3). Case 4 was initially seen as diffuse edema of a cerebral hemisphere that evolved to an infarction pattern with peripheral gyral enhancement (figs. 4A and 4D). Our only constant CT finding was mass effect. The mass effect in cases 1 and 2 may be totally explained on the basis of an extracerebral collection. The explanation for inadequate detection of low density extracerebral collections in cases 3 and 4 may be that these collections were small in total volume and/or were isodense in relation to the surrounding cerebral tissue.

We believe that the focal neurologic deficit is due more to the response of the cerebral vasculature and brain to the inflammatory process and less to the mass effect of the extracerebral collection. Therefore, even small extracerebral collections of pus, relatively unresolved on current CT scans, may produce severe neurologic changes. The absence of medial or border enhancement in some cases may be related to a short time interval from onset of the infectious process, with its effect on cerebral vessels and brain, and the CT scan. It is presumed that the border enhancement

we observed in three cases is due to development of a granulation tissue wall over a period of 3 weeks. Analogously, this generalization is supported by the fact that the enhanced fibrous tissue wall of intracerebral abscess may not be observed until the third week of its natural history [7]. Conceivably, sequential CT scans might demonstrate the evolution of subdural empyema and appearance of border enhancement. A definite cerebral edema pattern, as visualized in cases 3 and 4, probably is responsible for the major part of the observed mass effect. This edema pattern is probably related to the cerebral ischemia secondary to an inflammatory angiospasm shown on subsequent angiograms. Concomitant venous thrombosis or cerebritis may also contribute to the development of cerebral edema.

Kim et al. [2] described these specific angiographic signs in subdural empyema: (1) an irregular border of the extracerebral collection; (2) a thickened vascular wall of the dura; and (3) a semilunar avascular zone in the lateral view. In addition, the following angiographic findings of meningeal inflammation may be present: (4) spasm of large arteries at the base of the brain with or without segmental arterial dilatation; (5) multiple peripheral arterial stenoses; and (6) enlargement of the meningeal arteries.

Angiography in cases 1–4 revealed a subdural collection in each case, although temporal relation of CT to angiography varied from case to case (see Case Reports). Cases 3 and 4 showed definite spasm of large arteries at the base of the brain (figs. 3C and 4B). Multiple peripheral arterial stenoses with reduced flow to the hemispheres were also observed in case 3. The presence of angiospasm and a prolonged circulation time in case 4 prevented the detection of an extracerebral collection on the first angiogram (figs. 4B and 4C). Dilatation of the anterior falcine artery was noted in case 1 (fig. 3D).

The neuroradiologic diagnosis of subdural empyema may be elusive if CT findings alone are considered. We believe that subdural empyema should be considered in the proper

clinical setting when mass effect, diffuse cerebral edema, or an infarction pattern is seen on CT without a definite extracerebral collection. In these cases arteriography should be promptly performed although, as case 4 illustrates, angiography may not always provide an answer. Moreover, difficulty may occur in demonstrating interhemispheric, base of brain, and posterior fossa extracerebral collections by angiography. The subdural empyema seen in figure 5 may only have been demonstrated by CT. On the other hand newer CT techniques including high dose contrast infusion, delayed scanning, and coronal imaging (direct or reconstructed) may further the diagnostic efficacy of CT for subdural empyema. It is hoped refinements of CT technique will lessen the need for angiography in this surgical emergency.

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