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Spinal subarachnoid clot detected by CT.

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Astrocytoma with Calvarial Erosion

A 26-year-old woman was seen with a solid, low-grade astrocytoma with resultant erosion of the calvaria. Although calvarial erosion from gliomas has been reported [1–4], it has not been demonstrated with computed tomography (CT). Our patient had an 8 month history of tinnitis, visual field defects, headaches, and one episode of seizure activity. Skull radiographs and CT demonstrated a poorly marginated, 5 cm bony erosion of the left lateral surface of the frontal bone (fig. 1). A mass immediately adjacent to the area of erosion was also evident with CT. The mass produced cerebral edema that resulted in effacement and contralateral shift of the lateral ventricles. The erosion was from pressure atrophy of the inner table and thinning of the diploë.

Calvarial erosion caused by a superficially located glioma is rare [5]. Additional intracranial masses that may produce calvarial erosion include meningioma [1, 3, 5], intracranial cysts [1], dural metastases [3], chronic juvenile subdural hematomas [2, 3], and anomalous development of the cisterna magna [2, 3]. The CT appearance of the frontal lobe mass in our case was not characteristic of either meningioma or intracranial cyst [6]. Metastatic neoplasm was a consideration but was unlikely because of the patient's age. The CT findings in our case are most suggestive of calvarial erosion from glioma.

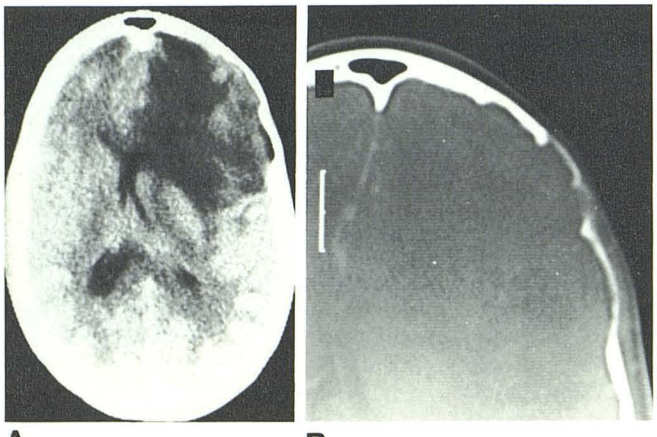

Fig. 1.—**A**, Large left frontal mass with edema and contralateral shift. **B**, Associated calvarial erosion was better identified on bone window setting.

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Spinal Subarachnoid Clot Detected by CT

We studied a case of subarachnoid hematoma after traumatic lumbar puncture [1–4] in a patient with pneumococcal meningitis. This 20-year-old woman had a history of Still disease and had recently developed visual hallucinations. Neurologic examination was normal. The patient was on aspirin, 13 tablets a day, and prednisone, 25 mg four times a day. Lumbar puncture, arthrocentesis, and blood culture were all positive for pneumococcus. After the last lumbar puncture, the patient developed urinary retention as well as fecal incontinence. Because of these new symptoms, CT of the spine was performed and showed areas of increased density in the dural sac at the level of the cauda equina (fig. 1A). In view of the patient's history of multiple traumatic spinal taps, a diagnosis of spinal subarachnoid hematoma was made. Metrizamide myelography and subsequent CT showed filling defects in the dural sac (fig. 1B) without evidence of subdural or epidural hematoma. During lumbar puncture for myelography,

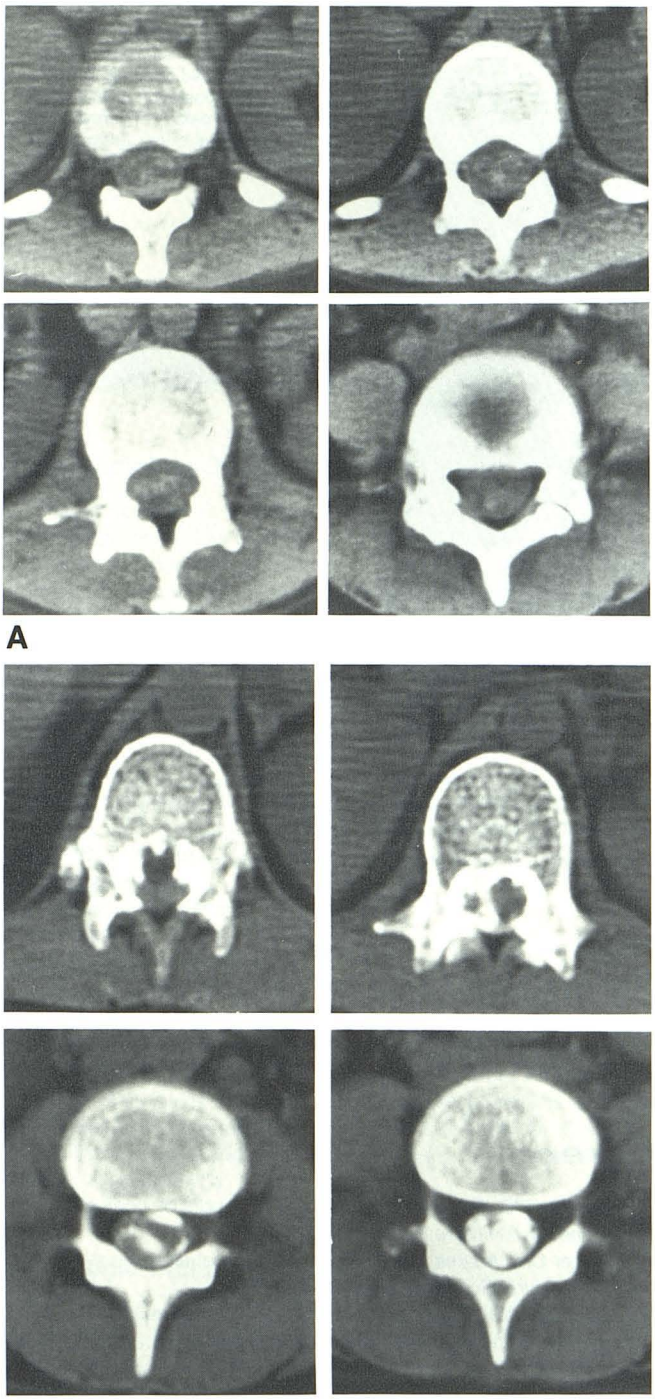

Fig. 1.—**A**, CT of lower thoracic and upper lumbar spine. Patchy areas of increased density in dural sac represent blood clots. **B**, CT metrizamide myelogram. Multiple filling defects and linear lucencies represent blood clots.

bleeding at the site of the puncture was prolonged, and laboratory tests showed thrombocytopenia. Repeat CT of the spine 8 days later failed to demonstrate previously noted increased densities of the hematoma. We suggest that the subarachnoid hematoma was caused by repeated spinal tap, abnormal coagulation, and possible engorged spinal vessels from a reactive process secondary to meningitis. C1–C2 puncture for myelography was avoided in this patient to prevent possible hematoma in the high cervical region and its grave consequences. Spinal CT is an essential study in cases with rapid progression of neurologic symptoms after traumatic spinal tap to rule out subarachnoid hematoma.

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Transient Global Amnesia: Complication of Arterial DSA

Transient global amnesia (TGA) is a clinical syndrome described by Bender [1] and Fisher and Adams [2] that consists of a single episode of the inability to assimilate new information for a finite period of time while retaining remote memory. This phenomenon has been described with selective cerebral [3–6] and coronary [7] angiography. A review of the literature revealed no report of TGA in nonselective angiography. Our report describes TGA in a nonselective digital subtraction angiographic (DSA) study of the aortic arch and cerebral circulation.

A 59-year-old man was referred for "dizziness." A 4 French pigtail catheter (Mallinckrodt) was placed in the ascending aorta via a right brachial artery approach, and the patient received four injections of 20 ml/sec for 2 sec (40 ml/injection) of Conray 43 (Mallinckrodt). The patient received our standard injection series of 160 ml of contrast material. This large volume of contrast material insures excellent demonstration of the carotid bifurcations. At 202 mg I/ml, 160 ml of Conray 43 gives the patient only 2.3 g more iodine than 100 ml of Hypaque 50 (Winthrop-Breon).

After the catheter was removed from the brachial artery and while hemostasis was being secured, the patient became confused and mildly agitated, repeating the questions, "Where am I?" and "What am I here for?" Physical examination revealed a normal pulse and blood pressure. Neurologic examination revealed no abnormalities except for the failure to remember three objects at 3 min. The patient correctly remembered his name, address, and where he worked. The patient recovered fully within 3 hr and had no residual deficit 2 months later. DSA showed a 25% concentric stenosis caused by atherosclerotic plaque in the proximal right internal carotid artery (fig. 1A) and a diminutive vertebrobasilar system (fig. 1B) without atherosclerotic stenosis.

TGA is a syndrome defined by Mathew and Meyer [6] as the sudden onset of episodes of loss of memory for recent events and inability to recall recently learned information, associated with retrograde amnesia. During the amnesic event the patient is alert and retains most of his personal identity. However, the patient is confused and upset about the memory loss. Repetitive questioning after having been given the answer is common.