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Intraparenchymal Blood-Fluid Levels: New CT Sign of Arteriovenous Malformation Rupture

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The computed tomographic (CT) findings of arteriovenous malformations of the brain have been well described [1–5]. Recently, Daniels et al. [6] reported three cases of arteriovenous malformations with cystic cavities on CT due to hematoma liquefaction, an unusual finding. We report three cases of ruptured arteriovenous malformations associated with blood-fluid levels within cystic cavities.

Case Reports

Case 1

A 64-year-old woman, in previous good health except for a questionable history of mild hypertension, complained of frontal headache with nausea and dizziness on the evening before admission. She was found unresponsive the next morning. Physical examination revealed a left hemiparesis and a blood pressure of 150/64 mm Hg.

CT revealed a large frontal intracerebral hematoma with minimal shift. Layering of blood within a cystic cavity below the hematoma was noted (Fig. 1). There was no enhancement after administering a 100 ml bolus injection of meglumine diatrizoate (Renografin 60).

Because of rapid deterioration, the patient was taken immediately

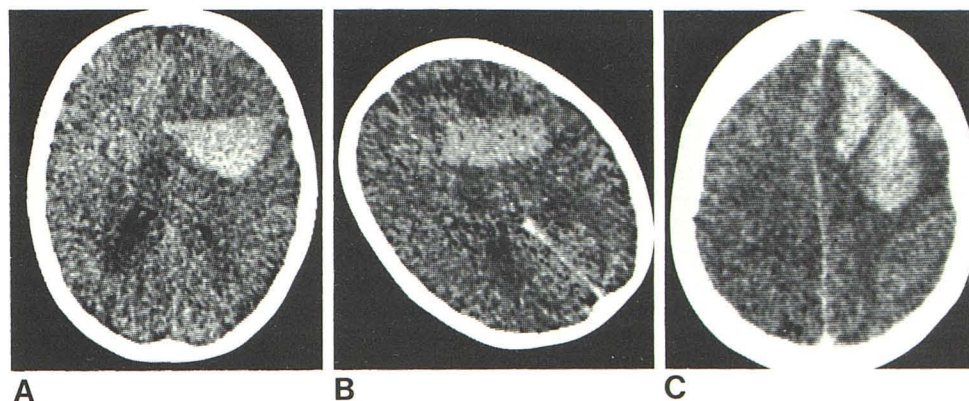
to surgery with the clinical diagnosis of either an arteriovenous malformation or a tumor. A right frontal craniotomy revealed a liquified hematoma, which was removed by suction. On the surface of the frontal lobe, there was a firm nodule containing a tangle of thrombosed small vessels. Pathology of the nodule confirmed an arteriovenous malformation. In addition, hemosiderin deposits within surrounding macrophages indicated a previous bleed.

Case 2

A 50-year-old woman with 2 years of recurring severe left frontal headaches was seen with an excruciating headache and vomiting followed by dysphasia. She was aphasic with a right facial weakness and neck stiffness. CT revealed a left frontal hematoma (fig. 2B) above a cystic cavity containing a blood-fluid level (fig. 2A). No enhancement was noted after contrast administration. Cerebral angiography failed to demonstrate a vascular malformation or tumor vascularity. A mass effect was present.

At surgery, an abnormal tangle of vessels was encountered and removed. A fresh hematoma was evacuated from a cystic cavity. Pathology confirmed the diagnosis of arteriovenous malformation. Hemosiderin-laden macrophages were present in the walls of the cyst.

Fig. 1.—Case 1. A, Noncontrast scan. Blood-fluid level in cystic space and mass effect in right frontal lobe. B, Layering effect preserved with head tilted. C, Higher section. Large hematoma abuts blood-fluid level.



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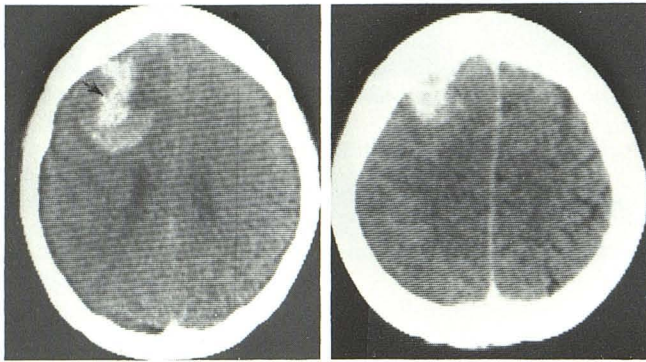


Fig. 2.—Case 2. Noncontrast scans. Blood-fluid level in left frontal lobe with extension of blood into cystic cavity (arrow) from overlying hematoma.

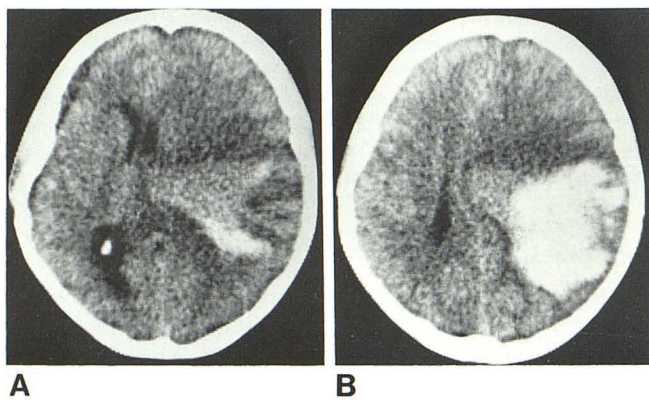


Fig. 3.—Case 3. **A**, Noncontrast scan. Blood-fluid level in left frontoparietal region with marked mass effect. **B**, Massive hematoma in parietal lobe adjacent to blood-fluid level.

Case 3

A 22-year-old man in previous good health collapsed suddenly at work. He was found mute with a right hemiparesis and left gaze preference. He had a varying blood pressure and an unstable respiratory pattern necessitating intubation. The consulting neurologist felt that he might have a left hemispheric mass lesion and recommended angiography, which revealed a mass effect in the left frontoparietal region. A small arteriovenous malformation of about 1 cm was seen in the peripheral frontal region fed by an ascending branch of the middle cerebral artery associated with arteriovenous shunting into the vein of Trolard.

After transfer to our hospital, CT demonstrated a large frontoparietal hematoma. Anterior to the hematoma, a cystic cavity containing a blood-fluid level was observed with marked displacement of the lateral ventricles (fig. 3). There was no evidence of enhancement peripheral to the hematoma. At surgery, the hematoma was evacuated by suction. Owing to inadequate exposure, a cystic cavity was not identified. Superficially, firm nodule resembling an arteriovenous malformation was found and removed. In addition, the brain in the region of the hematoma was gliotic. A biopsy was performed.

Histologically, a plexus of abnormal vessels was seen with irregular muscular walls characteristic of an arteriovenous malformation. There were adjacent "clefts" surrounded by reactive gliotic tissue and macrophages laden with hemosiderin granules, suggesting a previous bleed.

Discussion

Arteriovenous malformations have a varied CT appearance. On noncontrast scans, their attenuation values can range from hypo- to hyperdense, occasionally containing an admixture of both [1]. They may not enhance with contrast material. Hypodensity without enhancement has been attributed to encephalomalacia (from redirection of blood to the arteriovenous malformation) [1] or liquefaction of the hematoma [7]. Daniels et al. [6] considered their three cases of arteriovenous malformations with low densities to be due to hematoma liquefaction.

Patients harboring an arteriovenous malformation usually have either a hemorrhagic episode or seizure. However, some hemorrhages may be located in a neurologically silent area or the headaches may be too mild to warrant medical attention. The incidence of asymptomatic ruptures has been cited as 11% [8]. The CT appearance of an arteriovenous malformation rupture usually consists of an intraparenchymal hematoma that may extend intraventricularly or into the subarachnoid space. Contiguous enhancement in small malformations is difficult to detect because the enhancement can be mistaken easily for part of the hematoma.

Hemorrhage from rupture of an arteriovenous malformation producing a blood-fluid level has not been reported previously. A blood-fluid level, a familiar finding in subdural and intraventricular hemorrhage, implies the presence of a preexisting fluid-filled space in which red blood cell settling can occur. In intraparenchymal hemorrhage, because of the absence of a potential space, clot formation takes place without sedimentation. The finding of intraparenchymal blood-fluid levels in our cases suggests extravasation of blood into preexisting cystic cavities.

In addition, our cases had other common features. The lesions were located in the frontal lobe, a well known silent area of the brain. Also, all the lesions were seen when the rupture occurred beyond the confines of the cystic cavity, with sudden brain compression precipitating neurologic symptoms.

It is interesting that in all our patients, histologic evaluation revealed hemosiderin-laden macrophages, providing proof of previous subclinical hemorrhages. Since arteriovenous malformations are prone to recurrent ruptures, this concept of silent microhemorrhages is not new [5]. The presence of "clefts" surrounded by reactive gliosis suggests that this may have occurred extensively in case 3. It is quite feasible that repeated minor ruptures from the same focus may have resulted in cystic cavities; the patients were never seen until there was a major rupture from a new focus. In all three cases, a hematoma abutted the blood-fluid-containing cavity.

While a cystic cavity is a nonspecific sign, the finding of an intraparenchymal blood-fluid level within a cystic cavity is possibly specific for an arteriovenous malformation rupture. Therefore, angiography is recommended before surgery. The main differential diagnosis is an intratumoral hemorrhage. Whereas contrast CT may not detect a residual part of the lesion, cerebral angiography may define and differentiate an arteriovenous malformation from a tumor.

Addendum

Since submission of this manuscript, we have found two reports of intraparenchymal blood-fluid level; both concern intratumoral bleed (melanoma, two cases; pontine glioma, one case): Zimmerman RA, Balaniuk LT. Computed tomography of acute intratumoral hemorrhage. *Radiology* **1980**;135:355-359 and Dublin AB, Norman D. Fluid-fluid level in cystic cerebral metastatic melanoma. *J Comput Assist Tomogr* **1979**;3:650-652.

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