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Spontaneous Hemorrhage During Cranial Computed Tomography

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Computed tomographic (CT) scanning is very accurate in the recognition of intracerebral hemorrhage. However, when radiographic contrast material has been administered, there may be confusion between an unsuspected hematoma and an enhancing brain lesion. We recently encountered two cases of spontaneous hemorrhage occurring during the enhancement phase of the CT examination immediately after unenhanced scans showed no evidence of hemorrhage. The sudden appearance of increased attenuation presented the diagnostic problem of distinguishing between hemorrhage and an occult enhancing lesion.

Case Reports

Case 1

L. M., a 10-year-old American Indian girl, underwent bone marrow transplantation for aplastic anemia. Her posttransplantation course was complicated by increased clotting times attributed to disseminated intravascular coagulation and to graft-versus-host liver disease.

Seven days after transplantation, a seizure prompted CT scanning of the head; it was normal. Recurrent seizures and coma 23 days after transplantation led to repeat CT. Initial views, without contrast enhancement, showed ventricular enlargement when compared with the previous study. After contrast enhancement with 50 ml of meglumine diatrizoate (Hypaque 60%, Sterling Labs.), there was enhancement of vascular structures but no other change (fig. 1A). A question of enhancement along the tentorial edge (later shown to be normal) prompted repeat views, which demonstrated a collection of high density material near the left foramen of Monro (fig. 1B). The rapidly changing appearance of the ventricular density suggested hemorrhage, and an additional repeat view was performed with the patient's head in a left lateral decubitus position (fig. 1C). This view demonstrated the changed interface between the cerebrospinal fluid and the extravasated blood.

Although there was no immediate change in the patient's condition after CT, her subsequent neurologic status deteriorated progressively until her death 72 hr later. Autopsy revealed massive intraventricular hemorrhage.

Case 2

C. D., a 41-year-old man, had documented hemorrhage from an anterior cerebral artery aneurysm 1 month before follow-up CT. An intracranial shunt had been placed 7 days earlier to relieve acute hydrocephalus after the hemorrhage. He was stable and CT scanning was ordered to assess shunt function.

Initial views without contrast enhancement showed ventricular enlargement and a ventriculoperitoneal shunt tip (fig. 2A) [1], with no evidence of hemorrhage or mass effect. Enhanced views after infusion of 300 ml of meglumine iothalamate (Conray 30%, Mallinckrodt, Inc.) demonstrated an extensive, high density, left supracallosal mass (fig. 2B). The patient complained of severe headache immediately after the scan, and the diagnosis of acute intracerebral hemorrhage was made.

To exclude the possibility that the postcontrast density might represent extensive enhancement of a lesion poorly visualized on the initial unenhanced views (e.g., a recent infarct in the anterior cerebral artery distribution), scanning was repeated 24 hr later. This scan without enhancement again showed the high density lesion, confirming the diagnosis of hemorrhage (fig. 2C).

The patient's condition rapidly deteriorated, and he died 48 hr after the initial scan. Autopsy showed a large frontocallosal hematoma. Because of massive tissue destruction, it was not possible to distinguish between rebleeding from the aneurysm and hemorrhage into an area of infarction.

Discussion

In case 1, the hemorrhage actually occurred during the scanning procedure. The outpouring of iodinated blood on the CT scan was analogous to the extravasation of contrast material from an aneurysm during angiography. In case 2, the hemorrhage began between the unenhanced and enhanced scans. If the hemorrhage in either of these cases had been present on the unenhanced scans, there would

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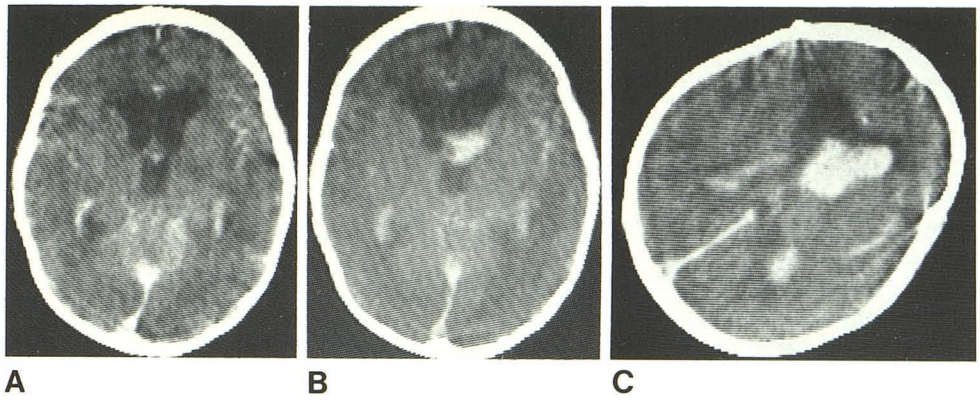

Fig. 1.—Case 1. **A**, Enhanced scan. Vascular structures and ventricular dilatation. **B**, 10 min later. High density near foramen of Monro of left ventricle. **C**, 10 min after **B**, left decubitus position. Fluid interface between cerebrospinal fluid and blood.

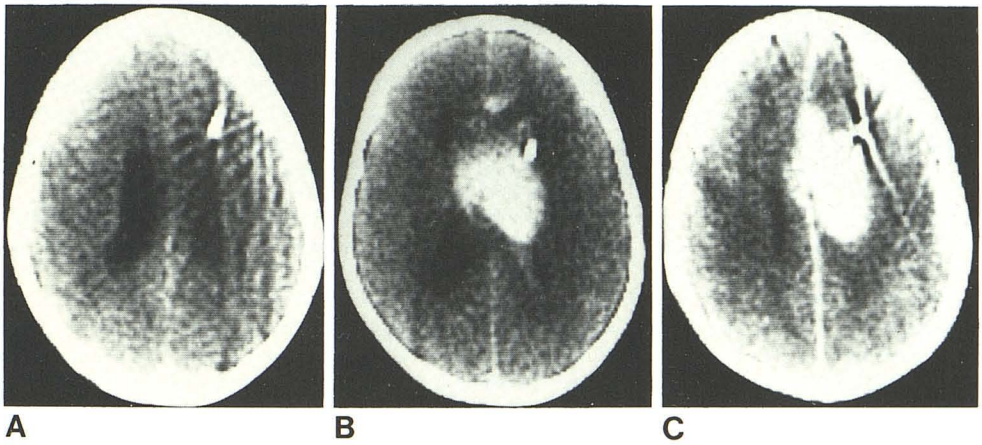

Fig. 2.—Case 2. **A**, Unenhanced scan with shunt tip. No evidence of hematoma. **B**, After enhancement. Large, left, parasagittal, frontoparietal, high density mass lesion extends into corpus callosum. **C**, Unenhanced scan 24 hr later confirms diagnosis of hemorrhage. (Reprinted from [1].)

have been little problem in diagnosis. However, the appearance of increased density after enhancement can be seen with the passage of contrast material across an abnormal blood brain barrier, as with infarction, infection, and tumor. In order to consider the correct diagnosis, we postulated that hemorrhage occurred during the short time in which contrast material was administered and/or scanning performed. In these cases, several factors led us to the correct diagnosis.

In case 1, the intraventricular location of the lesion and the shifting cerebrospinal fluid/blood interface suggested that iodinated blood was filling the lateral ventricle. In addition, although gradual enhancement of tumors and infarctions resulting in delayed visualization of these lesions has been reported, the delayed high density is neither as sudden nor as intense as seen here [2]. In case 2, the sudden appearance of mass effect where none had been present on the unenhanced scan and the persistence of the density 24 hr later, after clearance of the contrast material, excluded an occult enhancing lesion.

It is interesting to speculate on the contribution of injected radiographic contrast material to the production of these hemorrhagic events. While new bleeding from an aneurysm during cerebral angiography is a well known occurrence, there is disagreement whether pressure changes from a selective arterial injection may cause the rupture or whether the hemorrhage is coincidental [3]. Of course, no selective high pressure arterial injections were performed in our cases. The role of increased blood volume producing significant blood pressure alterations after an injection of a hypertonic solution is speculative. Assuming an instantaneous injection and immediate dilution of the hypertonic contrast agents by the movement of extravascular water, there was about a 9% increase in blood volume in case 1 and a 10% increase in case 2. Obviously, however, the relatively slow rate of intravenous injection and the delays in extravascular water movement, along with equilibration of the contrast material in other water compartments and its excretion by the kidneys, mean that the increase in blood volume and subsequent blood pressure alterations would be substantially less than these estimates. Of greater import might be the blood pressure alterations that occur during any stressful procedure, whether an arterial or a venous injection. Finally, however, contrast agents are known to transiently inhibit blood coagulation factors and platelet aggregation [4]. Both of our patients had ample reason for spontaneous intracranial hemorrhage (thrombocytopenia and disseminated intravascular coagulation in case 1, and a documented aneurysm and possibly infarcted tissue in case 2). A depressed ability to clot induced by the contrast material may have been additive in either of these cases. Of course, the hemorrhages we observed may well have been coincidental and unrelated to any of the factors discussed above.

A recent article [5] described the normal perfusion of iodinated contrast material into the ventricular system with-

out the presence of hemorrhage. However, this occurred during cerebral death and the role of this factor in the abnormal perfusion of the choroid plexus is speculated. Our two cases are not analogous.

The time course of CT demonstration of acute intracranial hematoma has not been determined. In describing in vitro experiments, Norman et al. [6] and New and Scott [7] agree that hemoconcentration is an important factor in the production of the high density lesion, which apparently occurs in the absence of clot retraction. However, the exact time sequence in vivo is still uncertain. Unfortunately, our cases of freshly extravasated blood do not offer insight into this question, since the extravasated blood contained a high concentration of iodine. In case 1, the density of the extravasated intraventricular blood was nearly identical to the density of the circulating blood in the straight sinus, so that extravascular hemoconcentration was unnecessary for the visualization of this "iodinated" hemorrhage. Duplicate CT scans with differing beam kilovoltage may have shown the relative contributions of hemoconcentration and iodine [8]. Of course, observation of ongoing hemorrhage on unenhanced scans will demonstrate the rapidity of hemoconcentration and its effect on CT visualizaiton of acute intracerebral hematoma.

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