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CT Recognition of Subcortical Hematomas

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Subcortical hematomas develop in brain trauma and less commonly in hypertensive intracerebral hemorrhage. Six cases are reported that exhibit a spectrum of computed tomographic (CT) findings in this entity. Pathologic correlates in four cases are presented. It is theorized that subcortical hematomas form in trauma secondary to shearing stresses in the brain. Differential movement of gray and white matter may disrupt cortical medullary vessels. Rupture of degenerative vessels at this junction may account for hypertensive hematomas.

Neuropathologic literature over a period of 60 years has documented subcortical intracerebral bleeding secondary to trauma [1] and hypertension [2]. It has also been recognized in computed tomography (CT) of brain trauma [3]. Continued technical improvement in CT is promoting better resolution of gray and white matter. An understanding of the etiology and siting of these lesions would seem useful in neuro-CT interpretation. In this paper, we examine information about the gray-white matter junction in this reference. Selected case reports place the subject in a clinical context.

Case Reports

Case 1

An 18-year-old man sustained multiple injuries, including closed head trauma, a laceration of the liver, and right hip and rib fractures, in an automobile accident. Neurologic examination revealed stupor and left hemiparesis. A nonenhanced CT scan showed a subcortical hematoma (fig. 1) in the right frontal region, with surrounding edema. There was also a small adjacent epidural hematoma. A mildly depressed right frontal skull fracture was present.

Repeat scan 2 days after admission and treatment for his injuries again delineated the subcortical hematoma. He gradually improved and was discharged neurologically intact 6 weeks after admission.

Case 2

A 57-year-old man was found unconscious after a fall. CT (fig. 2A) showed a right frontal hematoma and a small, right subdural blood collection. A higher CT section (fig. 2B) brought out a 2 cm diameter hematoma with some surrounding edema at the junction of the right temporal and parietal lobes. The hematoma did not extend to the brain surface. The right frontal hematoma and a part of the frontal lobe that was necrotic were removed. The remaining right frontal lobe developed an abscess. Pulmonary complications occurred, and the patient also had liver decompensation.

At postmortem, a moderate-sized abscess was present in the right frontal lobe. A 2.5 cm diameter contusion was present in the superoposterior aspect of the right temporal lobe (fig. 2C). The hematoma had expanded the white matter of the inferior temporal gyrus and extended vertically to the white matter of the middle temporal gyrus. The gray matter

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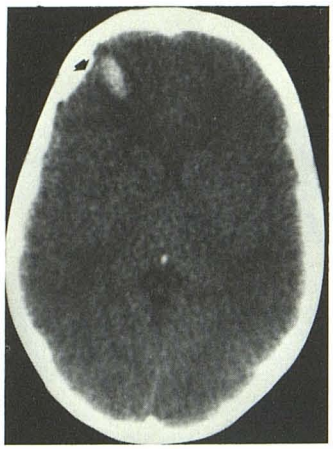
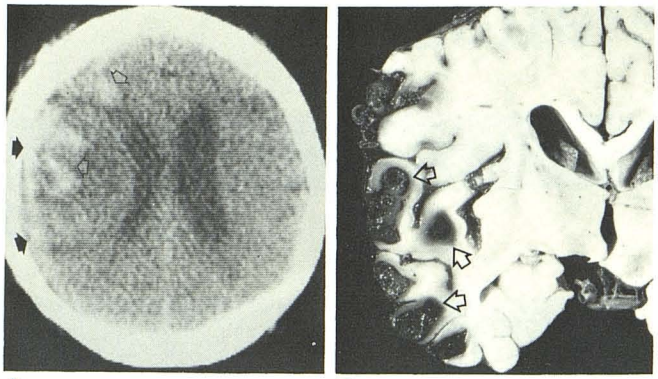
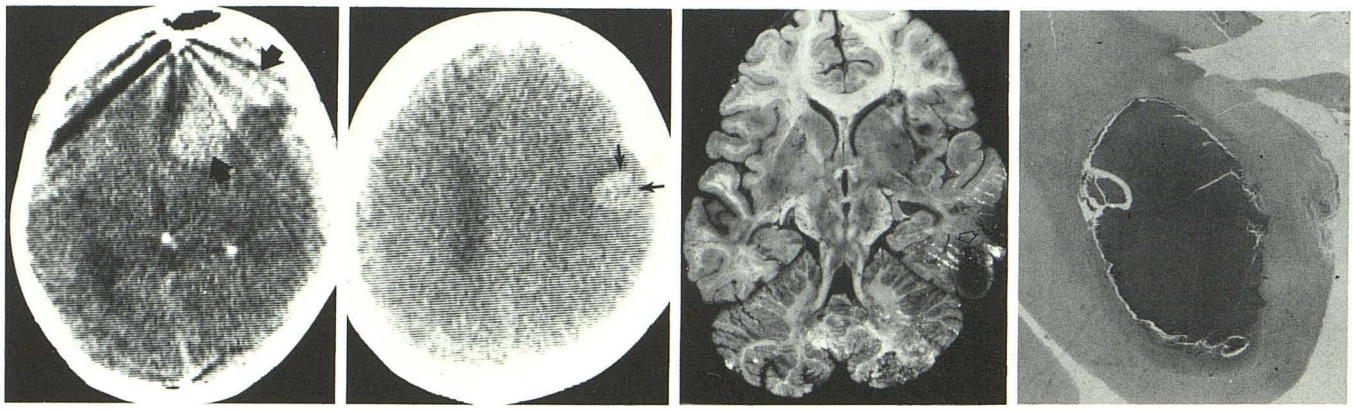


Fig. 1.—Case 1. Posttraumatic subcortical hematoma in right frontal region. Small adjacent epidural hematoma (*arrow*). Frontal fracture also present.



A B

Fig. 3.—Case 3. **A**, Focal areas of hemorrhage after trauma in right cortical and subcortical region (*open arrows*). Extracerebral collection of blood (*solid arrows*). **B**, Subcortical hemorrhage with extension to several brain surfaces (*arrows*).



A B C D

Fig. 2.—Case 2. **A**, Posttraumatic left frontal hematoma (*arrows*) **B**, Localized postparietotemporal hematoma (*arrows*) with adjacent edema. Lesion in subcortical position. Edema compresses left lateral ventricle. **C**, Autopsy. Part of hematoma in inferior temporal gyrus (*arrow*). **D**, Microscopic section. Containment of lesion by gray matter.

around the hematoma, although compressed, was not penetrated by the hematoma (fig. 2D). Other brain findings included areas of petechial hemorrhage throughout the brain and infarction of the medial left temporal lobe.

Case 3

A 74-year-old man was being treated for squamous cell carcinoma of the tongue when he fell, striking his left occiput. A left hemiplegia developed. CT (fig. 3A) showed an extensive right parieto-occipital contusion and adjacent subdural hematoma. The contused brain exhibited rounded islands of blood, which extended to the brain surface. Postmortem examination revealed a 2 cm wide, right parietal subdural hematoma. Localized hematomas expanded multiple gyri in the right frontoparietal and temporal areas. Most of the hematomas disrupted the apex of the gyrus and were seen on the brain surface. Partial gray matter containment of the hematomas, however, could be seen in several gyri (fig. 3B). White matter localization of a hematoma was also noted.

Case 4

A 76-year-old woman had previous neurosyphilis. After a fall, she was found on admission to Peter Bent Brigham Hospital, Boston, Mass. to be stuporous, with a mild left hemiparesis. Nonenhanced CT defined a right subdural hematoma as well as several subcortical hemorrhages (fig. 4A). The hematoma was drained, but neurologic function continued to deteriorate, and she died.

Postmortem examination showed compression of the hemispheric gyri, more on the right than the left. A small remaining right frontotemporal subdural hematoma was present. Beneath the subdural hematoma was both cortical and subcortical contusion (figs. 4B and 4C). Blood collections of varying size in the white matter of the right frontal and temporal lobes were found. Some of these communicated with the ventricular system.

Case 5

A 24-year-old man was in an automobile accident and had multiple injuries, including severe head trauma and facial fractures. Initial CT (fig. 5A) without contrast material showed a prominent subcortical collection of blood with associated edema in the right postero-occipital region. He was treated with mannitol and Decadron, with some initial improvement of his neurologic findings.

His neurologic status deteriorated 2 days after admission and repeat CT (fig. 5B) showed a large hematoma in the midright

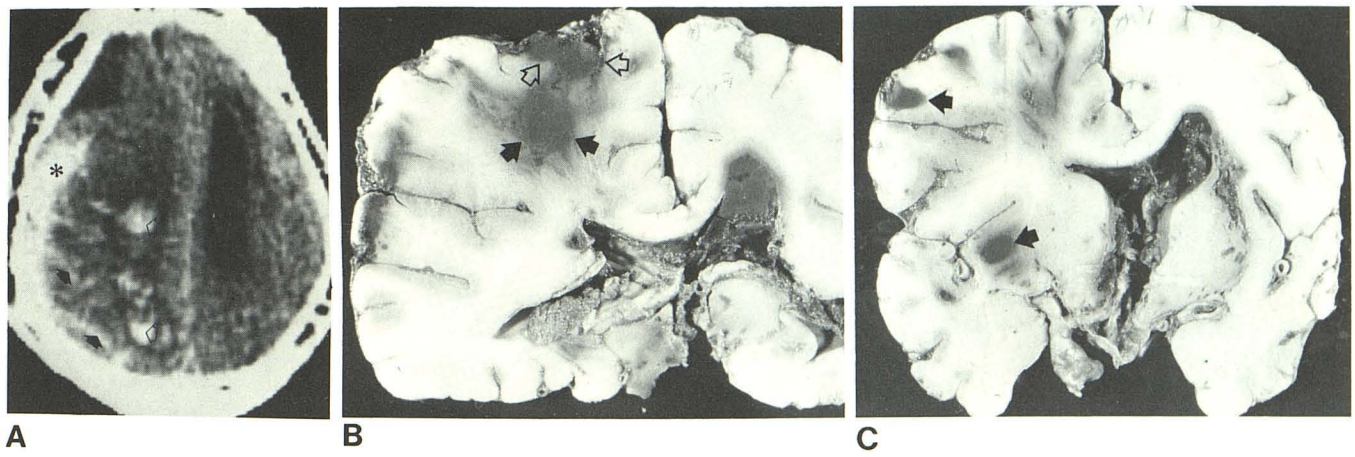


Fig. 4.—Case 4. **A**, Right subdural hematoma (*asterisk*) and subcortical hematomas with surrounding edema in medial right parietal lobe (*open arrows*). Blood (*solid arrows*) in gyri or surface of brain. **B**, Subcortical collection of blood and edema (*solid arrows*) in right parietal region. Hemorrhage extends to brain surface (*open arrows*). Blood in left lateral ventricle. **C**, Small subcortical hematomas (*arrows*) in right posterior frontal lobe. More superior lesion involves adjacent gyrus.

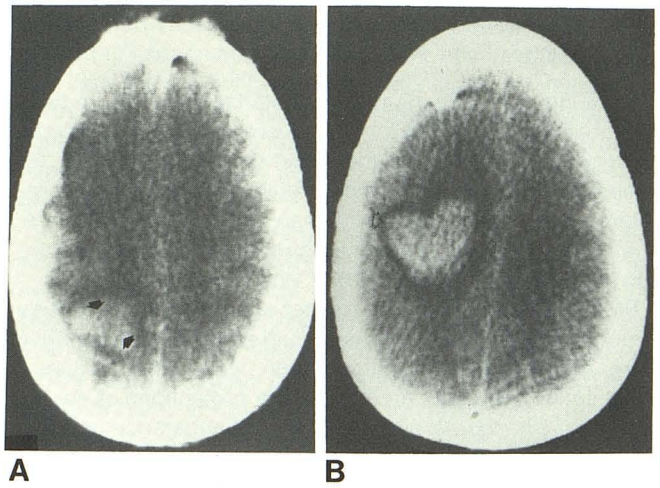


Fig. 5.—Case 5. **A**, Right parieto-occipital subcortical hematoma (*arrows*) seen immediately after trauma. Adjacent increased density in cortex due to extravasated or static blood secondary to contusion. **B**, CT study 2 days later. Large delayed hemorrhage and edema in right centrum semiovale. Lesion extends to medial and lateral (*arrows*) subcortical borders of right parietal lobe.

centrum semiovale extending to the posterior parietal subcortical region. This lesion was not present on CT 2 days before and was interpreted as delayed subcortical hemorrhage. It was evacuated surgically. Postoperatively, the patient had a marked left hemiparesis, which showed only moderate improvement over a 6 month period.

Case 6

A 46-year-old man was being treated for dermatomyositis as well as hypertension. Pulmonary embolization 1 year before admission led to maintenance anticoagulant therapy. At 1 day before death, he was unresponsive. CT (figs. 6A and 6B) showed a 5 cm diameter hematoma in the left temporal lobe. The inferior aspect of the hematoma was ring-shaped. Operative evacuation of the hemorrhage was attempted. Postmortem examination revealed residual hematoma in the inferior and central part of the temporal lobe. Although difficult to assess because of surgery and necrosis, the hematoma appeared to be in white matter around gyri at the inferior aspects of the lobe (fig. 6C). Prominent left ventricular hypertrophy, suggesting sustained hypertension, was found.

Discussion

In 1922, Apfelbach [4] noted hematoma formation in white matter adjacent to the cortical margins of gyri (fig. 7). This observation was derived from an analysis of over 500 traumatized brains. He suggested that most brain "bruises" were the result of bleeding due to rupture of arteries at or near the junction of white and gray matter. Apfelbach also proposed that subarachnoid cerebrospinal fluid at the site of maximal injury cushioned the cortical surface of the brain. However, the force of the blow was "transmitted through the brain from a medium of lesser density (gray matter) to one of greater density (white matter)—producing the greatest commotion at or near their junction."

Apfelbach did not have the advantage of CT scanning to observe that gray matter is denser than white. However, the notion that "commotion" at the junction of gray and white matter is the cause of blood vessel rupture in trauma has considerable support. Shearing stress [1, 5–8] at the time, particularly in marked rotational injury of the head, could produce subcortical blood. The work of Pudenz and Shelden [8], wherein the brain surface of a monkey could be observed through a Lucite calvarial window, is of special interest. High-speed cinematography during and immediately after a blow to the head showed prominent convolutional gliding and deformation, particularly of the parietal and occipital lobes.

In a series of laboratory experiments, Ommaya [9] found a high incidence (nine of 12 animals) of petechial hemorrhages at gray-white matter interfaces on both sides of the brain. The monkey heads had been subjected to varying degrees of rotational acceleration. In an animal concussed

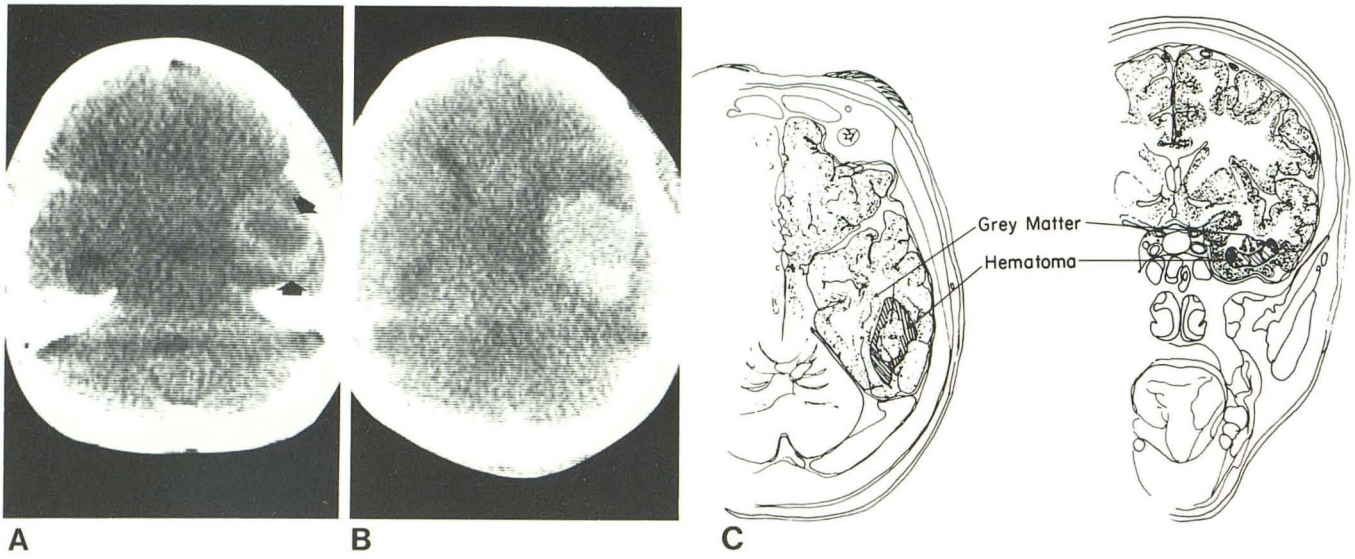


Fig. 6.—Case 6. Hypertensive patient. **A**, Ring formation of blood (*arrows*) at inferior aspect of left temporal lobe. **B**, Next higher section. Solid hematoma in temporal lobe. **C**, Possible explanation for ring hematoma. Blood in white matter extends around cortical substance of gyri of inferior part of temporal lobe.

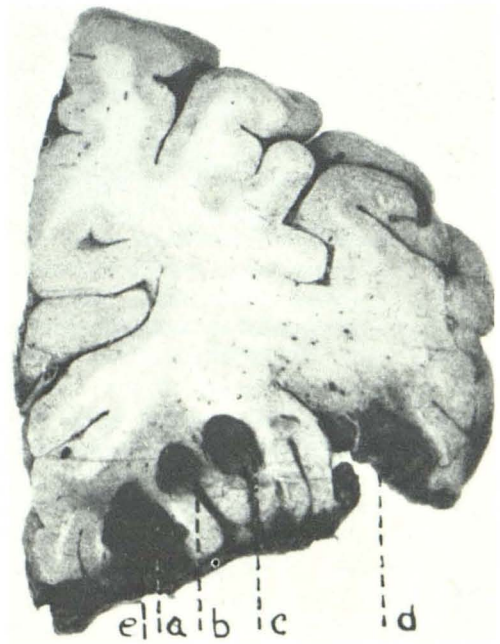


Fig. 7.—Pathologic display of subcortical hematoma formation from work of Apfelbach [4]. (Copyright 1922, American Medical Association).

in a rectilinear fashion, a large hemorrhage developed in the white matter of the occipital lobe and was contained by the gray matter. Ommaya suggests in this work that in acceleration trauma of the head, "cortical-subcortical disconnection phenomena" are more prominent in the periphery of the brain and are enhanced at sites of structural inhomogeneity.

Noting these observations, it is reasonable to suggest that gray matter develops a greater momentum during these movements due to its heavier vascularization. This, in turn, is probably responsible for the severance of either arterial or venous structures at the gray-white junction (fig. 8). Vessels that have become more fragile secondary to atherosclerosis or hypertension may also break at this interface. It is interesting that extensive subcortical hemorrhage occurred in cases 3 and 4, both of whom were elderly.

Blood can also move with greater ease in the more loosely structured white matter. Edema fluid also appears to move more readily through white matter. CT scans of tumor, abscess, or infarction show fingerlike projections of edema in white matter bordered by the cortical substance of gyri.

Shearing forces also affect the central parts of the brain. Zimmerman et al. [10] reported CT findings of hematoma in the corpus callosum in seven or eight cases after trauma. Blood was also seen in the walls of the third ventricle as well as the tectum in some of these patients.

Delayed intracerebral hemorrhage is a well recognized sequel of trauma. Courville and Blomquist [11] pointed out that bleeding could occur superficially or in the deeper structures of the brain. Necrosis, hypercapnia, and vascular congestion in white matter produce a setting for delayed vessel rupture [12]. As suggested, penetrating cortical vessels would appear to have increased vulnerability to rupture secondary to shearing. Although the vessel may be intact immediately after trauma, mounting pathologic change around it could cause rupture. Reference to CT recordings [13, 14] of delayed hemorrhage shows that the preponderance of hematomas are subcortical or cortical in position.

Subcortical hemorrhages are often found in both hypertensive and normotensive patients [2]. The residua of these hemorrhages appear as slitlike cavities and have pigmented walls [2]. Pant and Dreyfuss [15] reported a patient with a 10 year migraine history who had over 100 old and recent hemorrhages at autopsy. Many of these, including a 5 cm

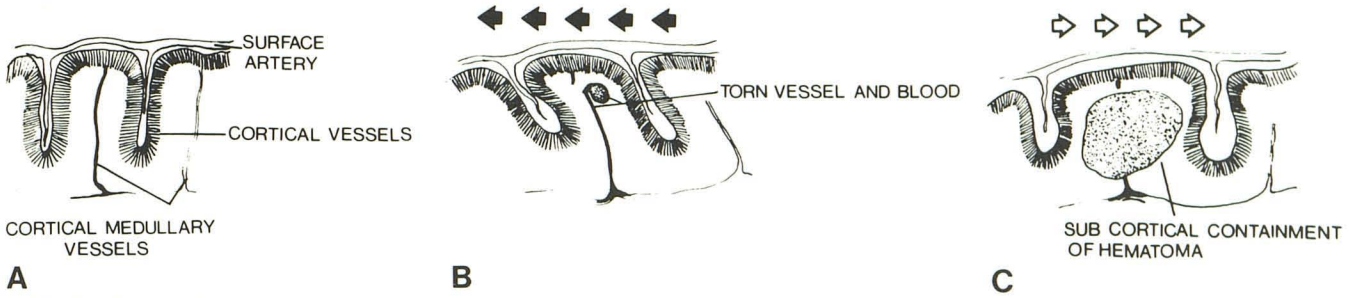


Fig. 8.—**A** and **B**, Possible explanation for subcortical hematoma formation. Inertial movement of denser gray matter during trauma greater than white matter, causing rupture of corticomedullary vessels. **C**, Hematoma is contained by gray matter in subcortical position. Overlying gyrus becomes more convex.

diameter lesion, were subcortical. Microscopic sections of cortical arteries and veins showed hyaline degeneration, which may have made these vessels more susceptible to rupture.

Similar changes are seen in cortical vessels in longstanding hypertension. These degenerative changes can lead to vessel widening and rupture [16]. The reason for disruption of diseased vessels at the subcortical level is unclear. Romanul [17] speculated that vasomotor spasm affecting only the penetrating cortical arteries accounts for subcortical infarction. Possibly, sustained spasm in these vessels in hypertensive patients may, over the long-term, produce degenerative change, making them more susceptible to rupture.

It is possible that the hemorrhage in case 6 was secondary to the use of anticoagulants. This therapeutic regimen had been used for over 1 year without a change in dosage. The patient's hypertension had been difficult to control, however, and most likely the hemorrhage can be attributed to it.

The ring distribution of fresh blood in case 6 was difficult to understand at the time of CT interpretation. There had been no trauma to suggest temporal lobe necrosis in the center of the lesion. Assessment of the temporal lobe at autopsy was difficult due to partial surgical evacuation of the hematoma and resulting necrosis. A possible explanation could be curvilinear extension of blood in white matter around superiorly extending gray matter of the inferior temporal and fusiform gyri (fig. 6C). Associated edema in the encircled gray matter could also add to the central lucency.

Subcortical hematomas will be recognized with increasing frequency as the gray-white matter junction is defined with late model CT scanners. Assessment of the extent of these lesions can serve as an index of the severity of brain stem injury [9]. Containment of the lesion in white matter, either in trauma or hypertension, raises the question of the degree of preserved cortical activity in overlying gray matter. Evaluation of this issue in animal models would be of interest.

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