Organized Intracerebral Hematoma with Acute Hemorrhage: CT Patterns and Pathologic Correlations

Ya-Yen Lee¹
 Richard Moser²
 Janet M. Bruner³
 Pamela Van Tassel¹

Five cases of pathologically proven organized intracerebral hematoma with recent hemorrhage are reported. The hemorrhages were spontaneous, were located in deep structures of the brain, and presented without a clinical history of hypertension or trauma. No underlying causes of the hematomas were identified histologically. Various computed tomographic (CT) patterns of recurrent hemorrhage in an organized hematoma were observed. A hyperdense, well demarcated mass with minimal contrast enhancement but no surrounding edema was observed when rehemorrhage was confined within a late-organizing hematoma. A "target" sign of a ring-enhancing mass was observed when the new hemorrhage developed within an early-organizing hematoma. When a new hemorrhage occurred outside a solid-enhancing organized hematoma, the CT appearance suggested a possible tumoral hemorrhage. Close clinical and CT follow-up is desirable for initial management of a suspicious rehemorrhaging organized hematoma situated in the deep-seated or superficial sensitive structures if the symptomatology has stabilized or improved. Tissue diagnosis and surgical evacuation are indicated when the clinical course fluctuates or progresses and CT demonstrates an interval increase of lesion.

Intracerebral hemorrhage frequently presents clinically as an acute devastating event with severe neurologic deficit that usually leads to immediate and proper medical attention. On some occasions, small hemorrhages may recur spontaneously at the same site resulting in a perplexing computed tomographic (CT) appearance and clinical course. Frequently, these small recurrent hematomas are located in deep-seated and vital structures such as the basal ganglia, thalamus, and brainstem, where improper treatment under a presumptive diagnosis without tissue pathology may lead to severe sequelae. In this communication, we present the clinical courses and sequential CT scans of five deep-seated organized hematomas with recent hemorrhage and describe their various CT patterns.

This article appears in the May/June 1986 issue of *AJNR* and the July 1986 issue of *AJR*.

Received May 13, 1985; accepted after revision July 1, 1985.

¹ Department of Diagnostic Radiology, University of Texas M. D. Anderson Hospital and Tumor Institute, 6723 Bertner, Houston, TX 77030. Address reprint requests to Y.-Y. Lee.

² Department of Head and Neck Surgery, Section of Neurosurgery, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, TX 77030.

³ Department of Pathology, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, TX 77030.

AJNR 7:409-416, May/June 1986
 0195-6108/86/0703-0409

© American Society of Neuroradiology

Case Report

Case 1

A 12-year-old boy first presented in December 1980 with a sudden onset of nausea, vomiting, diplopia, right facial palsy, and right gaze paralysis. There was no history of head trauma or hypertension. CT of the brain 2 days after the episode revealed a small, well marginated, hyperdense lesion in the right posterior brainstem with minimal contrast enhancement but no surrounding edema (Fig. 1A). With the presumed diagnosis of a brainstem glioma, he was referred to our institute for possible radiotherapy. Vertebral arteriography revealed no abnormality. A suboccipital craniotomy revealed a bluish intrinsic mass in the brainstem, projecting into the floor of the fourth ventricle. Upon entering the lesion, a small amount of dark bloody fluid was aspirated, and a few blood clots were removed. Examination of the tissue revealed fresh and organized hematomas without evidence of vascular malformation or neoplasia. Postoperatively, he recovered uneventfully with complete resolution of the neurologic deficits within 3 months.

Fig. 1.—Case 1. **A**, Unenhanced CT scan 2 days after episode. Small well demarcated hyperdense mass in posterior brainstem, near slightly deformed floor of fourth ventricle, with small central hypodensity (arrow). Craniotomy revealed organized hematoma with recent hemorrhage. Pre- (**B**) and post- (**C**) enhancement CT scans 1 week after next episode 2 years later. Again, a small hyperdense mass with minimal contrast enhancement is seen in operative site. Note absence of surrounding edema. Pre- (**D**) and post- (**E**) enhancement CT

scans 1 week after most recent episode and 4+ years after initial episode. Larger hyperdense enhancing mass recurred at same location, without surrounding edema. **F**, Organized hematoma with superimposed more recent hemorrhage. Well established fibrous tissue bands (**F**). Abundant granular hemosiderin pigments within macrophages and free in extracellular tissue (arrows), features of capsule stage, on right. Intact and degenerating erythrocytes representing acute and early subacute stages are identified on left.

In July 1982 he experienced a similar episode. A CT scan 1 week later again showed a hyperdense, mildly enhancing lesion at the original site (Figs. 1B and 1C). No surrounding edema or significant mass effect was associated. His neurologic signs and symptoms resolved in 3 weeks without treatment.

In January 1985 he experienced a sudden onset of confusion, disorientation, slurring of speech, and a right facial weakness that partially resolved over the next 3 days without treatment. A CT scan 1 week after this episode revealed findings similar to the previous two studies (Figs. 1D and 1E). Angiography again was unremarkable. Via a suboccipital craniotomy, aspiration of a few blood clots and partial resection of an organized hematoma were performed. The pathologic examination again revealed organized hematoma with a superimposed more recent hemorrhage (Fig. 1F). The postoperative course was uncomplicated, with complete resolution of symptoms in 1 month.

Case 2

A 36-year-old man was referred to our institute for evaluation of a right basal ganglion mass in early March 1983. His history was

remarkable for a grand mal seizure 10 years previously. Cerebral arteriography at that time was normal. He had remained seizure-free on Dilantin therapy. Two years before admission he experienced a severe bifrontal headache, but CT and cerebral arteriography were negative. During the 2 months before admission he experienced several episodes of severe bifrontal headache associated with decreased coordination on the left side and clumsiness in walking. There was no history of hypertension or head trauma. A CT scan demonstrated a mass in the right basal ganglia. He was referred to our institution for a possible stereotaxic needle biopsy to rule out a deep-seated glioma.

Physical examination revealed a left hemiparesis with a left circumducting gait disturbance. The blood pressure was normal. An unenhanced CT scan showed a sharply marginated hyperdense round mass in the right basal ganglia associated with a more laterally located fresh hematoma and surrounding edema (Fig. 2A). The biopsy was postponed, and cerebral arteriography revealed only an avascular mass effect in the right basal ganglia without demonstration of an arteriovenous malformation, aneurysm, or other vascular anomaly. A postangiogram CT scan showed heterogeneous enhancement of the

Fig. 2.—Case 2. **A**, Unenhanced CT scan 2 months after episode. Sharply demarcated hyperdense round mass in right basal ganglia and more laterally located fresh hematoma with hemoglobin-layering phenomenon. Surrounding edema. **B**, Postangiography contrast-enhanced CT scan 1 day later. Heterogeneous contrast enhancement of medially located hyperdense lesion with central hypodensities (arrow). Laterally located fresh hematoma does not enhance. **C**, Contrast-enhanced CT scan 3½ months after initial episode. Nodular pattern of medial hyperdense mass has changed into "target" pattern,

with classical ring enhancement of lateral resolving hematoma. No surrounding edema is associated. **D**, Section from edge of medial lesion. Well established collagen bands (C). Adjacent gliotic brain comprises granular hemosiderin pigment (arrows) and reactive astrocytes (arrowheads). Extensive acute hemorrhage with intact erythrocytes on right within fibrous capsule of organized hematoma. **E**, Contrast-enhanced CT scan 1 year after surgical evacuation. Calcium deposit in resolved hematoma (arrow) is identified with postoperative hypodensity. Patient was asymptomatic.

medial hyperdense mass with central hypodensities (Fig. 2B). Steroid therapy for the associated edema was begun, and he was discharged with some improvement of neurologic deficits.

In mid-April 1983 his symptoms worsened, and CT showed a new "target" appearance of the enhancing hyperdense mass and typical ring enhancement around the associated hematoma (Fig. 2C). Cerebral arteriography again showed no vascular abnormality except for a minimal avascular mass effect in the right basal ganglia. Because of the fluctuating clinical course, it was elected to perform an explor-

atory operation to rule out possible tumoral hemorrhage. A frontotemporal craniotomy was carried out and the lesion was exposed through splitting of the right sylvian fissure. The laterally located hematoma was first encountered and evacuated followed by a partial resection of the deeper mass. Examination of the deeper lesion revealed a chronic organized hematoma and superimposing recent hemorrhage, without evidence of vascular malformation or neoplasia (Fig. 2D). The cultures for microorganisms were negative. The postoperative course was uneventful and his symptoms improved steadily. He was asymp-

Fig. 3.—Case 3. Pre- (A) and post- (B and C) enhancement CT scans. Oval-shaped hyperdense mass lesion in left superior thalamus with heterogeneous contrast enhancement and small eccentric hypodensity (*arrow*). No surrounding edema is associated. Patient was asymptomatic. D and E, Contrast-enhanced study 2 weeks after episode and 2 months after initial CT study, before

stereotaxic needle biopsy. Interval enlargement of mass lesion with partial rim enhancement and minimal surrounding edema. Small hypodensities are within mass medially. F, Follow-up contrast-enhanced CT scan 14 months after biopsy. Persistent but smaller resolved hematoma with calcium deposits (*arrow*). Patient was asymptomatic.

tomatic on the last follow-up 1 year after surgery, with CT showing calcium deposits in the resolved hematoma (Fig. 2E).

Case 3

A 29-year-old man presented in April 1983 with a 5-month history of temporal lobe seizures. Thirteen years before admission, he had a resection of an esthesioneuroblastoma from the left maxillary antrum and ethmoid and sphenoid sinuses, followed by radiotherapy with 6000 rad (60 Gy) in 6 weeks. Subsequently he had the left eye enucleated due to a radiation complication, but remained tumor-free. An admission CT scan of the brain revealed a poorly defined hypo-

dense calcified lesion with moderate heterogeneous contrast enhancement in the left inferior temporal lobe, consistent with radiation damage. In addition, an unexpected round, hyperdense mass, without edema or calcification, was seen in the left superior thalamus impinging on the third ventricle; heterogeneous contrast enhancement of this lesion was observed with a small central hypodensity (Figs. 3A–3C). Cerebral angiography, neurologic examination, and electroencephalography were all unremarkable. He denied a history of head trauma or hypertension. He remained asymptomatic, with seizures controlled for 2 months. In mid-June 1983 he suddenly experienced numbness of the right lower jaw as well as incoordination of the right extremities. Over the next 2 weeks the symptoms diminished and eventually resolved. A contrast-enhanced CT scan 2 weeks after this

Fig. 4.—Case 4. Pre- (A) and post- (B) enhancement CT scans. "Target"-appearing hyperdense mass in right basal ganglia with contrast enhancement. No surrounding edema. C, Unenhanced CT scan 1 year later. "Target" CT

pattern is replaced by nodular pattern and margin has become slightly irregular. There are small eccentric hypodensities (arrow). Patient was asymptomatic.

acute episode revealed a significant enlargement of the left thalamic lesion, which remained heterogeneously enhanced and demonstrated new rim enhancement in its upper lateral portion and minimal surrounding edema (Figs. 3D and 3E).

Stereotaxic needle biopsy for possible tumor hemorrhage was carried out and yielded several milliliters of old hemorrhagic fluid. Pathology revealed an old organized hematoma with evidence of recent hemorrhage. The cultures were negative for microorganisms. The postoperative course was uneventful. The last follow-up in August 1984 revealed the patient to be asymptomatic, and a CT scan showed calcium deposits in the left thalamic resolved hematoma (Fig. 3F).

Case 4

A 30-year-old woman suddenly developed clumsiness of the left hand and weakness of the left leg while running in February 1984. She had a long history of nonspecific headache, but denied experiencing headache while the episode was developing. There was no history of head trauma or hypertension. The symptoms progressed and a CT scan of the brain 1 week after the episode revealed a discrete, noncalcified, "target"-appearing hyperdense mass lesion in the right basal ganglia with minimal contrast enhancement (Figs. 4A and 4B). Minimal mass effect was noted without surrounding edema. In the next 2 weeks, the weakness of both left extremities progressed slowly and the patient began experiencing numbness of the left side of the face. She was transferred to our institute for a stereotaxic needle biopsy in March 1984.

Physical examination on admission was unremarkable except for a dense left hemiparesis with increased reflexes. A repeat unenhanced CT scan revealed no interval change in the lesion. Due to progression of symptoms and the possibility of tumoral hemorrhage, a stereotaxic needle biopsy was performed. On entering the lesion, 3 ml of dark, brownish semiliquid material was aspirated. Pathologic examination of the biopsy revealed an organized hematoma and

superimposed more recent hemorrhage. Cultures for microorganisms were negative. Postoperatively her symptoms persisted due to re-hemorrhage into the hematoma cavity. These resolved gradually, and she was asymptomatic 4 months later. On a follow-up CT scan in January 1985 the lesion remained hyperdense and sharply demarcated, but had become somewhat irregular with small central hypodensities (Fig. 4C).

Case 5

A 43-year-old man presented in January 1985 with a 2-week duration of generalized headache and numbness and clumsiness of the right side of his body. He had experienced a similar episode of less severity 4 years before that had resolved spontaneously after 4 months. There was no history of head trauma or hypertension. A CT scan of his brain 2 days after the onset of symptoms showed a well defined, round, hyperdense lesion with minimal contrast enhancement in the brainstem near the left quadrigeminal cistern (Figs. 5A and 5B). The lesion had no definite surrounding edema and had only minimal mass effect. Cerebral angiography on the same day was unremarkable. With an impression of a possible tentorial meningioma, he was placed on steroid therapy, which partially relieved his symptoms, and was referred to our institute for further management.

Physical examination on admission revealed mild weakness of his right extremities with hypesthesia and slightly increased reflexes. Right dysmetria was detected with a difficulty in performing tandem gait. A repeat CT scan about 2 weeks after the initial symptoms showed the lesion had become less well defined with small central hypodensities (Figs. 5C and 5D). Again, no surrounding edema or severe mass effect was seen, and there was only minimal contrast enhancement of the lesion. A left subtemporal craniotomy with a transtentorial approach revealed brownish discoloration of the brainstem at the level of the pontomesencephalic tegmentum. After aspiration of 1–2 ml of dark, brownish liquid material, a cystic mass was identified and multiple samplings of its wall were obtained for biopsy.

Fig. 5.—Case 5. Pre- (A) and post- (B) enhancement CT scans 2 days after episode. Solid hyperdense mass in posterior brainstem, with minimal contrast enhancement. Absence of surrounding edema. Pre- (C) and post- (D) enhancement CT scans 2 weeks after episode. Eccentric hypodensities within hyperdense mass lesion cannot be isolated from tentorial notch. No associated edema. E, Unenhanced CT scan 2 months after partial surgical evacuation. Only minimal residual hyperdense hematoma remains (arrow), without mass effect or surrounding edema. Patient was symptom-free.

The pathologic examination revealed foci of recent hemorrhage in an old organized hematoma, without definite evidence of vascular malformation or neoplasia. The postoperative course was uneventful, with significant improvement of symptoms. Two months after surgery he was asymptomatic, and CT showed a small residual hyperdense hematoma (Fig. 5E).

Discussion

Spontaneous intracerebral hemorrhage occurs most often in the basal ganglia and thalamus with an incidence of 43%–53% [1, 2]. Microaneurysmal rupture associated with hypertension is thought to be the most common underlying etiology [3]. Occasionally metastatic neoplasm, vascular malformation, or blood dyscrasia is responsible. Sometimes, however, an underlying cause cannot be identified, particularly in the

limited surgical specimen from the sensitive structures. Except for case 5, we could not exclude the possibility of a bleeding source, such as a cryptic AVM or cavernous hemangioma, in the periphery or wall of the hematomas in the other four cases. Spontaneous hemorrhage usually presents as an acute devastating clinical event that leads to immediate medical attention. On some occasions, the hematoma is small, and the symptomatology produced is so subtle that it may be ignored by the clinician or even the patient himself. However, in most cases surrounding edema in the subacute stage of a resolving hematoma may be severe enough to elicit medical attention.

Ring enhancement of the resolving hematoma on CT has been well recognized and does not create much diagnostic difficulty [4–7]; however, if episodes of small hemorrhage recur at the same location where the original hematoma has

evolved into the stage of organization, the CT appearance becomes perplexing and may lead to a delay in diagnosis. Furthermore, the most common sites of hemorrhage—basal ganglia, thalamus, and brainstem—are difficult to explore surgically. Recurrent hematomas in these locations may become a diagnostic dilemma, particularly when the clinical course fluctuates or progresses, and without a tissue diagnosis they may be treated inappropriately as a growing neoplasm.

The evolution of intracerebral hemorrhage using a canine model was thoroughly investigated by Enzmann et al. [8]. They categorized the evolution of intracerebral hemorrhage into four stages: acute (days 1–3), subacute (days 4–8), capsule (days 9–13), and organization (days 13 and after). Pathologically, the acute stage exhibits intact red blood cells in the hematoma, with progressive crenation. There may be mild perivascular inflammatory reaction consisting mostly of lymphocytes and a narrow zone of neuron death, and white-matter edema surrounds the hematoma. In the subacute stage, the red blood cells become ghostlike and hemosiderin-laden macrophages appear. Inflammation peaks with the appearance of foamy macrophages and fibroblasts peripherally, and reactive astrocytes become apparent in the surrounding brain. During the capsule stage, macrophages and fibroblasts increase, and vascular proliferation begins to encroach on the hematoma from the periphery. Concurrently, inflammation regresses and reactive astrocytosis becomes pronounced. The final stage, organization, involves the formation of a dense collagenous capsule at the edge of the hematoma. Macrophages are densely filled with hemosiderin, and less well organized collagen proliferates in the center of the hematoma.

The sequence of CT changes is closely correlated with neuropathologic findings [8]. Ring enhancement on CT correlates closely with the perivascular inflammatory reaction (cerebritis) in the brain tissue surrounding the acellular hematoma and first appears in the subacute stage. When the hematoma evolves into the capsule and early organization stages, the ring enhancement becomes more intense and less smooth, while its diameter decreases; clusters of neovascular proliferation in the capsular wall now replaces cerebritis as the responsible factor for contrast enhancement on CT. Eventually, the enhancement of hematoma changes from a ring into a solid nodular pattern when a vascularized collagenous matrix completely replaces the acellular hematoma in the late organization stage.

Acute ICH is clearly recognized on the preenhancement CT scan as a well marginated hyperdense mass. Ring enhancement of a resolving hematoma may be confusing when seen for the first time. Close clinical correlation and short-term CT follow-up usually will suggest the diagnosis and lead to a proper management. The CT pattern of a resolving hematoma in the late organization stage, however, has not been emphasized in the literature and is less well recognized clinically. The inward filling of the acellular hematoma by a vascularized matrix not only obliterates the more familiar ring enhancement but also creates a hyperdensity on the noncontrast scan (Figs. 4C and 5E). The nodular enhancement of a hematoma

at this stage may mimic a neoplasm. The central relative hypodensity, as demonstrated in all five of our cases, most likely represents the residual acellular portion and may be misinterpreted as necrosis. No surrounding edema is associated. Minute dystrophic calcifications sometimes can be found in the chronic organized hematoma (Figs. 2E and 3F). When located near the tentorium the lesion may be mistaken for a meningioma (Figs. 5C and 5D), despite avascularity on arteriography.

The CT appearance becomes more confusing if new hemorrhage develops. When the new blood clot is confined within a late-organizing hematoma, which is now filled with a vascularized matrix, the entire lesion may appear as a hyperdense, well marginated mass with minimal homogeneous enhancement and no surrounding edema, as seen in cases 1 and 5 (Figs. 1D, 1E, 5A, and 5B). If the same process occurs in the acellular center of an early-organizing hematoma, a "target" appearance may be observed as cases 2 and 4 demonstrate (Figs. 2C, 4A, and 4B). A further perplexing CT appearance will exist when the new hemorrhage develops outside the old hematoma. The increased symptoms due to the new hematoma and its associated edema, along with nodular enhancement of the organized hematoma on CT, will strongly suggest a tumoral hemorrhage, as in cases 2 and 3 (Figs. 2A and 3D).

All five patients (six hematoma evacuations) in our presentation showed pathologic evidence of a hematoma in the stage of late organization, with abundant or scattered hemosiderin-laden macrophages, reactive gliosis, and collagenous capsule formation. In addition, there was histologic evidence of a more recent hemorrhage in each patient. One patient (case 1) had sheets of intact erythrocytes with some intervening fibrin and early fibroblast proliferation in both hematoma evacuations, indicating the early subacute stage of resolution. Two other patients (cases 3 and 4) were in the subacute stage and exhibited perivascular inflammation with mononuclear cells or small flecks of hemosiderin and red-blood-cell degeneration. The other two patients (cases 2 and 5) demonstrated findings of the capsule stage of hematoma with foamy macrophages and proliferating small vessels.

Awareness of the possible CT patterns is essential for the proper management of a suspected rehemorrhaging organized hematoma. Angiography should be performed to exclude a possible underlying vascular malformation and avoid unnecessary invasive procedures. Surgical resection is the treatment of choice in most cases. However, we believe close clinical and CT follow-up should be the initial management of the clinically stabilized lesions, which are deep-seated or in important superficial structures such as the motor or sensory cortex. Any surgical manipulation, even a stereotaxic needle biopsy, is risky in these sensitive structures. Obtaining a tissue diagnosis, nevertheless, may become mandatory when the clinic course fluctuates or progresses, and CT demonstrates an interval increase in the size of lesion. Precise needle biopsy using stereotaxic CT guidance [9, 10] or surgical resection with intraoperative sonographic assistance is recommended [11]. Collection of specimens at multiple sites is mandatory to rule out a hemorrhagic tumor or cryptic vascular

malformation. Aspiration through the biopsy needle may further provide therapeutic benefit for the resolution of the hematoma.

REFERENCES

1. Grumme T, Lanksch W, Wende S. Diagnosis of spontaneous intracranial hemorrhage by computerized tomography. In: Lanksch W, Kazner E, eds. *Cranial computerized tomography*. Berlin: Springer, **1976**:284-290
2. Kase CS, Williams JP, Wyatt DA, Mohr JP. Lobar intracerebral hematomas: clinical and CT analysis of 22 cases. *Neurology* **1982**;32:1146-1150
3. Yates PO. Vascular disease of the central nervous system. In: Blackwood W, Corsellis J, eds. *Greenfield's neuropathology*. Chicago: Year Book Medical, **1976**:133
4. Zimmerman RD, Norman EL, Naidich TP. Ring blush associated with intracranial hematoma. *Radiology* **1977**;122:707-711
5. Laster DW, Moody DM, Ball MR. Resolving intracerebral hematoma: alteration of the "ring-sign" with steroids. *AJR* **1978**;130:935-939
6. Weisberg LA. Peripheral rim enhancement in supratentorial intracerebral hematoma. *Comput Radiol* **1980**;4:145-154
7. Takasugi S. Cause of ring enhancement in computed tomographic image of intracerebral hemorrhage—an experimental histological study. *Neurol Med Chir (Tokyo)* **1980**;20:689-700
8. Enzmann DR, Britt RH, Lyons BE, Buxton JL, Wilson DA. Natural history of experimental intracerebral hemorrhage: sonography, computed tomography, and neuropathology. *AJNR* **1981**;2:517-526
9. Hahn JF, Levy WJ, Weinstein MJ. Needle biopsy of intracranial lesions guided by computed tomography. *Neurosurgery* **1979**;5:11-15
10. Edner G. Stereotactic biopsy of intracranial space occupying lesions. *Acta Neurochir (Wien)* **1981**;57:213-234
11. Rogers JV III, Shuman WP, Lange SC, Howe JF, Burchie LK. Intraoperative neurosonography: application and technique. *AJNR* **1984**;5:755-760