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Acute subdural hematomas: atypical CT findings.

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Acute Subdural Hematomas:
Atypical CT Findings

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Seventy-one patients with acute subdural hematomas were examined by CT within 72 hr of a documented head injury. Lesions often did not have the classical appearance of a homogeneous, high-density extracerebral collection of blood in a crescentic configuration. Specifically, 28 patients (39%) had mixed-density subdural hematomas (MDSDH) with various degrees of low-density blood within the subdural space. In 10 of these 28 patients, the hematoma had a relatively localized mass effect with a convex inner margin, occasionally mimicking the appearance of an epidural hematoma. The MDSDH group differed from the typical homogeneous high-density subdural hematomas in that they were larger (average maximal thickness was 18.1 mm versus 8.0 mm), had more midline shift, and had a higher mortality rate (50% versus 26%). Four patients with MDSDH demonstrated an unusual pattern of ventricular compression with trapping of cerebrospinal fluid in the body of the ipsilateral ventricle and compression of the body of the contralateral ventricle. This pattern has to our knowledge not been previously described. Possible causes of the low-density regions within the hematomas include unclotted blood in an early stage of hematoma development, serum extruded during the early phase of clot retraction, or cerebrospinal fluid within the subdural space due to an arachnoid tear.

Acute subdural hematomas (SDH) have been classically described on CT as homogeneous, high-density, extracerebral collections of blood with a crescentic configuration [1–3]. If a subdural collection has mixed density with areas of both high and low attenuation, it is often thought to be a chronic SDH, with the high density resulting from an episode of acute rebleeding. The observation of several patients, in whom mixed-density subdural hematomas (MDSDH) identified on CT within hours of an acute head injury proved to be acute SDHs at surgery, prompted us to review our experience with acute SDHs. Specifically, we evaluated how often low-density regions were present in these lesions and whether they were clinically significant.

Subjects and Methods

The CT scans and medical records of 71 consecutive patients with acute subdural hematomas were retrospectively evaluated. Patients were included in the study only if review of their histories could adequately document the time of a single acute head injury and if the patients had been examined by CT within 72 hr of the trauma. The selection criteria, by requiring documentation of the time of an acute injury, probably biased this group toward more severe head injuries (e.g., those caused by auto accidents).

CT scans were reviewed to note the appearance of the SDH and particularly to see whether areas of low density were present in the subdural space. The maximal thickness of the SDH and the degree of midline shift were measured. Other findings were noted, including the prevalence of an interhemispheric or tentorial component to the SDH, the presence of pneumocephalus, and the frequency of an unusual pattern of ventricular compression.

In all cases charts were reviewed and coagulation studies as well as operative and autopsy reports were evaluated.

TABLE 1: **Patterns of Subdural Hematomas**

Acute Subdural Hematomas	No. of Patients (%)
HDSDH[1]	43 (61)
MDSDH[2]	28 (39)
Total	71 (100)
Patterns of MDSDH	
Marginal	9
Irregular	16
Laminar	9

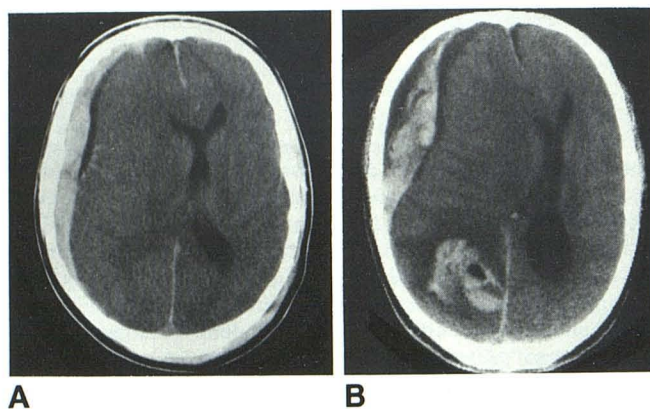[1] HDSDH = High-density subdural hematoma.
[2] MDSDH = Mixed-density subdural hematoma.

A   **B**

Fig. 1.—Marginal pattern. CT scan obtained 4 hr after an auto accident (**A**) illustrates a right-sided MDSDH with the low-density component similar in density to CSF along the inner margin of the hematoma, representing the marginal pattern. An acute subdural hematoma was found at autopsy. **B**, This right-sided MDSDH was identified approximately 1 hr after the patient fell, striking his head on the floor. Both the irregular and the marginal patterns are present, with relatively sharp margination between the high-density blood and the CSF density material within the subdural space. A right parietooccipital intraparenchymal hematoma is also present. Autopsy confirmed the presence of an acute SDH.

A   **B**

Fig. 2.—**A**, This scan was obtained 26 hr after the patient fell, striking his head. Somewhat ill-defined areas of low density, representing the irregular pattern, are present within the otherwise high-density, left-sided SDH. An arterial bleeding site was identified at surgery. **B**, This CT scan, taken 1½ hr after the patient fell from a second-story balcony, shows a right-sided MDSDH with the irregular pattern. An anterior interhemispheric component to the SDH is noted. Surgery confirmed the presence of an acute hematoma.

Fig. 3.—Laminar pattern. This scan was performed approximately 1 hr after the patient fell, striking his head on a cement floor. The somewhat linear areas of low density within this right-sided hematoma are typical of the laminar pattern of MDSDH. At surgery, a venous bleeding site was identified, and portions of the hematoma were noted to be unclotted.

All CT scans were performed with a General Electric 8800 or 9800 scanner, and all were performed without intravenous contrast material.

Results

Of the 71 patients with documented acute SDHs, 28 (39%) demonstrated a component of low density within the subdural space. Twenty-seven of these 28 MDSDHs were confirmed, either at surgery or autopsy, to be in the acute phase; that is, there was no membrane formation or breakdown of blood products. The patient population with MDSDHs was similar to the homogeneous high-density (HDSDH) group. The average age in the two groups was 43; the male-to-female ratio was approximately 3:1 in both groups.

Three distinct patterns of MDSDH were identified (Table 1). In nine patients, the low density was at the inner and/or outer margin of the hematoma, (marginal pattern) (Fig. 1). In seven of these nine patients, the low density was similar to cerebrospinal fluid (CSF) density by visual inspection; in the other two, it was of greater density than CSF but hypodense relative
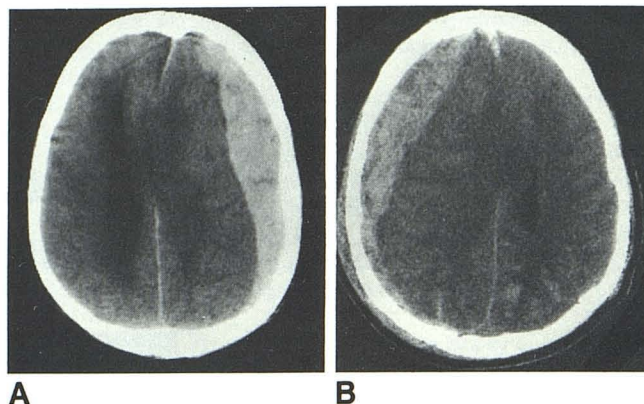
to gray matter. (Actual measurements in Hounsfield units were not available in most cases.)

Both the other patterns demonstrated areas of low density within the hematoma itself. Sixteen patients had an irregular pattern of low densities within the hematoma (Fig. 2); and nine had a laminar pattern with somewhat linear low densities within the hematoma (Fig. 3). In only one case did the irregular pattern have a density similar to CSF. In all other lesions with either of these two patterns, the low-density component visually appeared isodense with gray matter; however, actual measurement of CT densities in several cases showed them to be between 10–15 H higher than gray matter. Three patients with both the irregular and the laminar pattern also demonstrated the marginal pattern.

The average maximum thickness of the 28 MDSDHs was 18.1 mm, and the average midline shift was 16.5 mm. In contrast, the average maximal thickness in the high-density group was 8.0 mm, with an average midline shift of 4.6 mm. In 10 of the patients in the MDSDH group, the hematoma remained relatively localized in the subdural space rather than
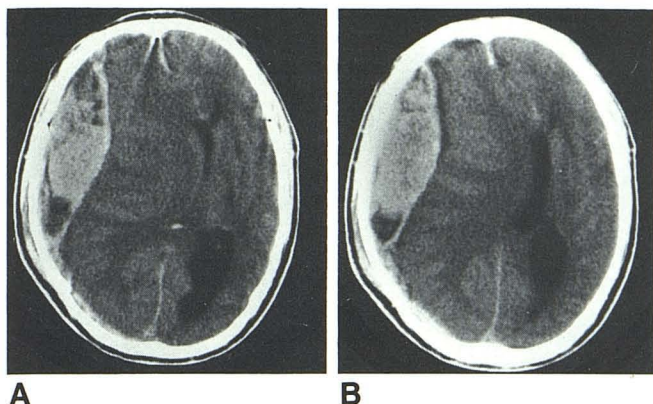
A   **B**

Fig. 4.—CT scans obtained approximately 1 hr after this elderly patient fell, striking his head. This autopsy-proven acute SDH is relatively localized in the subdural space on the right and has a convex inner margin suggestive of an epidural hematoma (**A**). The irregular and marginal patterns of MDSDH are present. An unusual pattern of ventricular compression is demonstrated in **B**. The ipsilateral ventricle remains filled with CSF while compressing CSF out of the contralateral ventricle as it crosses the midline (see text).

Fig. 5.—CT scan obtained approximately 7 days after an acute head injury. A right-sided MDSDH is identified with a relatively indistinct margin between the high-density fresh clot and the low-density liquefied chronic SDH. Surgery confirmed the presence of a subacute hematoma superimposed on a chronic SDH.

TABLE 2: **Time Interval between Head Injury and CT**

Hours	No. of Patients	HDSDH (%)	MDSDH
0–1.5	14	4 (29)	10 (71)
1.5–3	14	12 (86)	2 (14)
3–6	16	9 (56)	7 (44)
6–12	6	4 (67)	2 (33)
12–24	7	4 (57)	3 (43)
24–72	14	10 (71)	4 (29)
Totals	71	43 (61)	28 (39)

extending along the whole hemisphere (Fig. 4). This seemed to correlate with an early phase of the hematoma, as five of these 10 patients were scanned within 1½ hr of injury, and two others were scanned within 6 hr of injury. This finding was associated with a worse prognosis, as 70% of these patients died; and it was frequently associated with the marginal pattern, which was present in six of the 10 patients.

Our original hypothesis was that this mixed-density pattern was seen in the early or "hyperacute" phase of an SDH. With that in mind, we evaluated the time interval between injury and CT and correlated this with the mixed-density and typical high-density patterns (Table 2). Of the 14 patients with acute SDHs scanned within 1½ hr of injury, 71% had the mixed-density pattern. This pattern was present in only 14% of the 14 patients examined between 1½ and 3 hr of injury. The remaining patients were grouped into several time intervals between 3 and 72 hr, with percentages of MDSDH in these groups varying from 17% to 44%. The statistical correlation between the HDSDH pattern and the injury-to-CT interval of less than 1½ hr approaches significance using the chi square test with a p value = 0.06.

Review of coagulation studies identified three of the 28 patients in the MDSDH group who had abnormal coagulation. Two patients were on Coumadin (crystalline warfarin sodium,

USP) and one had idiopathic thrombocytopenic purpura. One of the 43 patients in the MDSDH group had laboratory evidence of coagulopathy due to Coumadin therapy. Twelve other patients in the MDSDH group and 13 in the HDSDH group were either known alcoholics or were inebriated at the time of injury.

Eighteen of the 28 patients with MDSDH were operated on; the other 10 were considered hopeless. Of the 18, eight had active bleeding at surgery, and in four of these, an arterial bleeding site was identified. On two occasions, the operative report mentioned the presence of an area of unclotted blood in the region of the low-density abnormality seen on CT.

The mortality rate was significantly different in the two groups, being 50% in the mixed-density group and 26% in the high-density group. No significant difference in mortality was noted among the three patterns of MDSDH. Mortality with both the marginal and irregular patterns was 56%, and in the laminar pattern it was 45%.

Other features noted in the 71 patients included a tentorial component to the SDH in nine cases, an interhemispheric SDH in three cases, and varying degrees of pneumocephalus in four cases.

An unusual pattern of ventricular compression was identified in four patients with MDSDH. The typical pattern consists of compression of the ipsilateral lateral ventricle with shift of the midline to the opposite side while the contralateral lateral ventricle remains filled with CSF. In these four patients, rather than the hematoma compressing CSF out of the ipsilateral ventricle, this ventricle remained filled with CSF and was pushed across the midline, compressing CSF out of the body of the contralateral ventricle (Fig. 4B).

Discussion

Since the development of high-resolution CT, very little has been published about the appearance of subdural hematomas. With improved resolution, the structure of subdural hematomas can be studied, and it can be seen that they often do not have the classical appearance, specifically that they often have areas of low density within them. This pattern can occasionally mimic the appearance of a chronic subdural hematoma with an episode of acute rebleeding, as shown in Figure 5 (compare this with the acute MDSDH shown in

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Fig. 6.—CT scan shows predominantly isodense right-sided MDSDH approximately 1 hr after patient was involved in a pedestrian–motor vehicle accident. A high-density inner rim is present. The patient had a normal hemoglobin on admission and nothing unusual was noted at surgery to explain this predominantly low-density appearance.

A   B

Fig. 7.—**A**, CT scan obtained approximately 6 hr after patient fell from a ladder, striking his head on cement. Both the irregular and marginal patterns are identified in this left-sided MDSDH. **B**, Scan obtained approximately 20 hr postmortem. While the irregular pattern of low densities within the hematoma are no longer present, the marginal low density persists, again similar in density to the CSF within the ventricles.

Figure 1B). The patient in Figure 5 was surgically confirmed to have a fresh clot within a chronic liquified SDH. In several medicolegal cases, the age of an SDH has been an issue, making it important to recognize that areas of low density in an SDH do not necessarily imply a chronic lesion.

In general, it is not difficult to differentiate an acute MDSDH from rebleeding into a chronic subdural hematoma: The interface between the high and low densities is quite sharp in patients with acute lesions, while this interface is more indistinct in patients with rebleeding into a chronic SDH. A horizontally layered appearance or a meniscus with the high-density blood in the dependent position is also characteristic of a chronic SDH unless the patient has a coagulopathy or is on anticoagulants.

In virtually all our patients with MDSDH, the lesions were predominantly of high density. The notable exception is shown in Figure 6. This scan was obtained approximately 1 hr after this young woman was involved in a pedestrian–motor vehicle accident. CT demonstrates an SDH that is predominantly isodense with brain, with a high-density inner margin. Isodense acute subdural hematomas have been described in patients who are anemic [4, 5]; however, this patient had a normal hemoglobin of 14.1, and nothing unusual was noted at surgery to explain the low-density appearance.

The recognition of MDSDHs is of clinical significance in that they are more serious lesions than are the typical high-density SDHs. The lesions are large, have more mass effect, and have a higher mortality rate than the typical HDSDH. The etiology of the low-density component is uncertain. Zimmerman and Bilaniuk [6] have described this appearance in epidural hematomas, suggesting that the low densities represent active bleeding. Cohen et al. [7] have suggested that the low densities represent active bleeding due to a coagulation disorder. It has been shown by New and Aronow [5] that the attenuation of clotted blood is approximately 20 H higher than that of whole blood and that whole blood is approximately 10 H higher than that of gray matter. Although in the majority of our cases the low density within the SDH was visually similar to gray matter, measurement of absolute CT numbers in several cases indicated that it was approximately 10–15 units higher than gray matter. Its visual similarity to gray matter presumably represents an optical illusion due to the higher density surrounding clotted blood. It seems
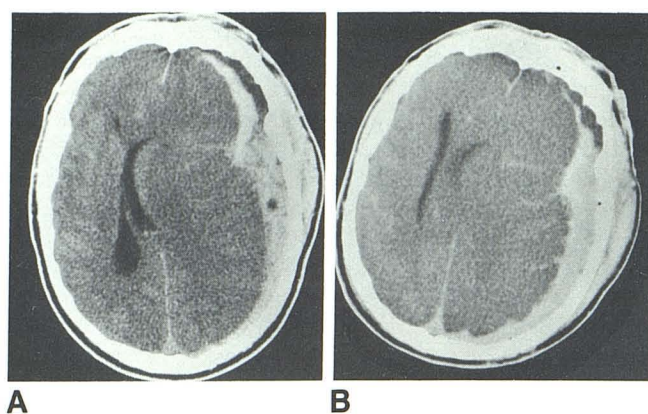
reasonable to conclude that the low densities in these cases merely represent whole blood which is as yet unclotted. Patients with active bleeding certainly would be expected to have some unclotted blood; however, we feel the presence of the low density does not necessarily imply active bleeding at the time of the scan. In fact, active bleeding was present at surgery in fewer than half the operated cases with MDSDH. (The average time interval between CT and surgical intervention was 30 to 45 min in these cases.)

As evidence against the active bleeding theory, we recently encountered a patient (not included in this study) with a mixed-density subdural hematoma with both the marginal and irregular patterns (Fig. 7A). Owing to the severity of the patient's injury, this lesion was not surgically removed and the patient died. A repeat CT scan (Fig. 7B) performed 1 day postmortem continued to demonstate a marginal pattern of low density. While the irregular pattern of low density was no longer present, probably due to clotting of previously unclotted blood, the persistent marginal pattern was clearly not due to active bleeding.

With regard to the hypothesis that the low densities are due to a coagulation disorder, only three of our 28 patients had laboratory evidence of an abnormal clotting mechanism. Admittedly, several additional patients were alcoholic and may have had abnormal platelet function, which was not specifically studied. In none of these patients, however, was any difficulty with hemostasis encountered during surgery.

While unclotted blood can explain the low densities in the majority of our cases, seven of nine patients who demonstrated the marginal pattern had low-density regions that appeared similar to CSF. We feel this is due to a different mechanism than unclotted blood. The attenuation of serum has been demonstrated by New and Aronow [5] to measure approximately 24 H, and we suggest that these areas of low density may represent serum extruded during an early phase of clot retraction or possibly CSF within the subdural space as a result of an arachnoid tear.

Since the subdural space typically presents no barriers to the spread of a hematoma, SDHs are usually panhemispheric and crescentic in shape. Ten of our 28 patients, however, demonstrated a relatively localized hematoma, occasionally with biconvex margins, mimicking the appearance of an epidural hematoma. Seven of these 10 patients were scanned within 6 hr of injury, suggesting that it may take a period of several hours for a subdural hematoma to diffuse and spread over the cerebral hemisphere, particularly when the hematoma is very large.

Another atypical feature we noted was the presence of an unusual pattern of ventricular compression in four patients with MDSDH (the patient in Figure 7 is the fifth example). Three of the four were scanned within 1½ hr of injury and all were seen to have very large hematomas, measuring greater than 23 mm in maximal thickness. We hypothesize that this pattern is due to rapid development of severe local mass effect causing kinking or compression of the ipsilateral foramen of Monro before all the CSF can be compressed out of this ventricle. The trapped CSF causes this ventricle to be displaced across midline, where it compresses CSF out of the body of the contralateral ventricle.

Conclusions

With the development of high-resolution CT, the structural appearance of acute subdural hematomas is more accurately represented. As many as 39% of these lesions have areas of low density within them. As this usually relates to the presence of unclotted blood within the lesion, areas of low density are more frequently seen the earlier the patient is scanned after head injury. Occasionally, areas of low density are present which are similar to CSF in density. These areas cannot be explained by unclotted blood and may be due either to extrusion of serum during an early phase of clot retraction or possibly CSF in the subdural space from an arachnoid tear. It is important to recognize this appearance not only to avoid confusion with rebleeding into a chronic subdural hematoma, but also because these lesions have a worse prognosis than the typical high-density SDH. This is not to suggest that there is anything particularly ominous about having unclotted blood in an SDH, but merely that it is more frequently seen in the larger, more severe lesions.

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