The contrast-enhanced CT scan in the diagnosis of isodense subdural hematoma

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The value of computerized tomography (CT) in the evaluation of head trauma is undisputed. During their natural course, subdural hematomas (SDH) have been shown to decrease in attenuation. At some undetermined time, their density may become equal to that of brain. These “isodense” SDH’s are frequently difficult to diagnose. Certain findings, thought to be characteristic of isodense SDH, have proven to be nonspecific. Contrast-enhanced CT scans have, in our experience, proven to be valuable in this diagnosis, thereby virtually eliminating the necessity for cerebral angiography.

Summary of Cases

We reviewed a total of 195 cases of SDH’s examined by CT, and found 32 cases of surgically proven isodense SDH. Three cases of isodense SDH were excluded from the study because contrast-enhanced CT scans were not performed. Of the total of 29 patients with isodense SDH examined by both non-contrast and contrast-enhanced CT scans, three underwent angiography because of suspected non-traumatic origin of their SDH. The 29 patients underwent CT scanning at intervals ranging from 2 days to 2 months after head trauma. All cases were examined on the EMI CT-1010 scanner, with 160 x 160 matrix.

Contrast-enhanced CT scans were performed during the administration of 300 cc of 30% Reno-M-Dip (diatrizoate meglumine): 200 cc was infused rapidly before the scan and the remainder infused more slowly so as to maintain adequate blood levels during the scanning procedure. We prefer to place the intravenous line before non-contrast scanning, in order to minimize patient motion between the non-contrast and contrast-enhanced scans. Obtaining equivalent “slices” during the non-contrast and contrast-enhanced phases of the examination is important when investigating subtle abnormalities.

Three morphological types of isodense SDH were identified, as follows:

Type I: Extra-axial collections homogeneously isodense with adjacent brain (16 cases)
Type II: Isodense collections in association with acute or chronic subdural collections (nine cases)
Type III: Gravitational layering of isodense material in the dependent portions of the subdural collection (four cases).

Illustrative Cases

Type I

Three of the 16 cases of homogeneously isodense SDH are presented briefly.

Case 1. This 30-year-old man sustained head trauma as the result of a fall. Six weeks later, CT scanning was performed (Fig. 1). The non-contrast
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scan revealed displacement of the midline structures toward the left side. Thin curvilinear and punctate areas of enhancement along the inner margin of the extra-axial collection were noted after administration of contrast. At surgery, a subacute SDH with a moderately organized membrane was evacuated.

**Case 2.** This 45-year-old man presented for evaluation of head trauma with subsequent lethargy. He had undergone a right frontal craniotomy several years before following a previous head trauma. Since then, he had had a mild left hemiparesis. He underwent CT scanning 12 days after his latest injury (Fig. 2). The non-contrast scan revealed an area of focal atrophy in the right frontal region, with shift of the midline structures from left to right. Following contrast administration, a curvilinear area of enhancement was demonstrated along the internal aspect of a subdural collection in the left frontoparietal region. At surgery, a subacute SDH was evacuated.

**Case 3.** This 25-year-old man developed confusion and lethargy following a fall. No focal signs were noted. Soft-tissue swelling involved the right face and head. The non-contrast scan demonstrated displacement of midline structures toward the left (Fig. 3 left). The diagnosis of isodense SDH was entertained until completion of the contrast-enhanced scan. Enhancement within the substance of the brain was demonstrated (Fig. 3 center). The linear pattern of enhancement so typical of SDH was absent. A repeat CT scan 5 days later confirmed the diagnosis of concussion (Fig. 3 right). No evidence of extra-axial hematoma was noted.

**Type II**

Three of the nine cases with isodense collections associated with acute or chronic SDH are summarized.

**Case 4.** This 40-year-old alcoholic woman developed lethargy, confusion, and bifrontal headaches following a fall. Physical examination revealed increased muscular tone on the left side of the body. Two weeks after her injury she underwent CT scan-
FIG. 3. Case 3. Isodense cerebral contusion. Left: The non-contrast scan demonstrates a shift of the lateral ventricles from right to left. Center: An amorphous area of enhancement is seen in the right Sylvian region after contrast administration. Right: Scan 5 days later is characteristic of a resolving cerebral contusion.

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FIG. 4. Case 4. Type II isodense subdural hematoma. Left: A biconvex, high-density extra-axial hematoma is seen over the right frontoparietal region on the non-contrast scan. Right: Following contrast administration, a serpiginous zone of enhancement (arrows) separated from the acute hematoma is noted, demarcating the internal border of an isodense collection.

Case 5. This 38-year-old alcoholic man was admitted because of progressive headaches over a 2-month period. He denied a history of head trauma. The non-contrast CT scan (Fig. 5 left pair) demonstrated displacement of the midline structures toward the right side. No abnormal density was identified. After contrast administration (Fig. 5 right pair), a zone of curvilinear and punctate enhancement was noted along the medial border of an isodense extra-axial collection. Closer inspection revealed an area of increased density adjacent to the inner table of the skull, separated from the cerebral cortex by a less dense collection. At surgery, two distinct subdural fluid collections were found, the outer one containing fresher blood than the one adjacent to the cortex. There was a very thin membrane separating these two collections.

Case 6. This 35-year-old man fell and sustained head trauma. He lost consciousness for several minutes, after which he suffered persistent confusion and mild left-sided weakness. Two days after injury, CT scanning was performed (Fig. 6). The non-contrast scan demonstrated an area of increased density in the right frontal lobe associated with shift of the frontal horns toward the left. In addition, the calcified choroid of the right ventricle was shifted anteromedially, suggesting an additional right parieto-occipital mass. After contrast administration, visualization of a thin subdural membrane demonstrated a mixed-density SDH extending posteriorly over the parieto-occipital area. The findings were confirmed at surgery.

Type III

One of the four cases with gravitational layering of isodense material is described.
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Case 7. This 38-year-old man was found unconscious and brought to our medical center by the police. He had been seen at the hospital previously because of problems related to alcoholism. The patient was comatose and unresponsive. His pupils were fixed and dilated to light. The non-contrast CT scans revealed a left cerebral hemispheric mass. After contrast enhancement, a linear zone of enhancement (black and open arrows) demarcates the internal border of this mixed-density SDH. The higher-density subdural collection is noted just under the calvaria (white arrows).

Discussion

The literature neglects the importance of contrast-enhanced CT scans in the evaluation of head trauma. Many authors even discourage its use, stating that the low yield of information from the use of contrast-enhanced scans in head trauma does not warrant the additional risk or the time required for this extra examination. Cerebral angiography and radionuclide brain scanning have been mentioned as necessary adjunctive procedures in the evaluation of head trauma — especially in cases of isodense SDH.

Our previous experience demonstrated evidence of contrast enhancement in 45% of cases of head trauma. It is frequently helpful for confirmation of suspected lesions and demonstration of occult lesions. It has been most helpful in the diagnosis of isodense SDH.

Homogeneously isodense collections (Type I) were the most common type of isodense SDH that we encountered. A definitive diagnosis of Type I isodense SDH is seldom possible on the unenhanced CT scan alone. The demonstration of curvilinear and punctate areas of enhancement, parallel to but separated from the inner table of the skull, is characteristic after contrast administration and was seen in all cases of isodense SDH. This linear pattern of enhancement may range from a very fine, interrupted line to a dense, thick, continuous line (compare Case 7 with Case 1). Correlation of CT findings with surgical findings indicates that contrast accumulation most likely occurs within the subdural membrane, cortical gyri,

Fig. 5. Case 5. Type II isodense subdural hematoma (SDH). Left Pair: The non-contrast scans reveal a left cerebral hemispheric mass. Right Pair: After contrast enhancement, a linear zone of enhancement (black and open arrows) demarcates the internal border of this mixed-density SDH. The higher-density subdural collection is noted just under the calvaria (white arrows).

Fig. 6. Case 6. Type II isodense subdural hematoma (SDH). Left: Non-contrast scan showing a poorly demarcated high-density lesion in the right frontal region with a shift of the midline structures. Right: After contrast administration, enhancement is seen along the internal border of this mixed-density SDH (arrows).
and cortical vessels. The presence of contrast enhancement in cases of acute, high-density SDH has been previously reported by us. The mechanism of this phenomenon differs, however. Both high-density and isodense SDH show enhancement in a linear fashion along the medial border of the SDH. In acute, high-density lesions this appears to be associated with underlying areas of cortical contusion, however.

Our experience contrasts sharply with that of Amendola and Ostrum, who were able to make the diagnosis in only three of seven cases of isodense SDH examined by delayed CT scans 4 to 6 hours after contrast administration. Contrast enhancement was not seen in any of their cases when scanned immediately after administration of contrast. The method of continuous infusion of contrast material during scanning, as compared to bolus injection of the material before scanning, may account for the high rate of contrast enhancement seen in our series. In addition, the CT scanner used at our institution may also be more effective in demonstrating these areas of enhancement.

The appearance of isodense contusion on non-contrast CT examination is indistinguishable from isodense SDH. The non-contrast scans are identical in Cases 1 and 3 (Figs. 1 and 3). The contrast-enhanced scan in Case 3 does not demonstrate the typical pattern of linear enhancement seen in Case 1, however. Instead, the contusion enhanced in an irregular, amorphous pattern located deep in the cerebral cortex. A pattern of contrast enhancement similar to that seen in cases of isodense SDH has not been observed by us in 74 cases of isolated contusion studied with contrast-enhanced CT scanning.

Misinterpretation of the CT scan in Case 2 might have led to a long delay in diagnosis, had the contrast-enhanced scan not been performed. The shift of midline structures to the right could be explained by the presence of postsurgical atrophy involving the right frontal lobe. Absence of focal neurological signs referable to the left hemisphere supported this contention. The pattern of enhancement so characteristic of SDH made angiography unnecessary.

The diagnosis of an extra-axial hematoma in patients with Type II isodense SDH before contrast enhancement may be obvious because of the high-density component adjacent to the inner table. In these cases, the shift of the midline structures is usually greater than one would expect from the size of the hematoma. By demonstrating linear enhancement, the contrast-enhanced scans indicated the presence of an additional isodense subdural collection deep to the fresh hematoma. At surgery, separate subdural collections in different stages of organization were noted in the Type II isodense SDH.

Lindenberg stated that fresh bleeding can occur within the subdural membrane or within the sac of an old hematoma. Because of the friability of the membranes, the degree of trauma necessary to produce fresh bleeding may be insignificant. This rebleeding within the more organized external membranes of an older SDH probably accounts for the findings seen in Type II SDH. Rebleeding into the subdural sac could theoretically produce an appearance resembling Type I isodense SDH if adequate mixing of fresh blood and chronic subdural fluid to produce an isodense collection had occurred. Bergström, et al., have shown that the attenuation of extra-axial hematomas decreases at a predictable rate with time, provided rebleeding does not occur. On the basis of their data, isodensity should be reached in a subdural collection between 2 weeks and 1 month after bleeding. We compared historical information from patients in our series with the theoretical values to see if our contention that Type II isodense SDH was due to rebleeding was tenable.
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Histories from six patients were either unobtainable or were thought to be unreliable. Of the remaining three patients, two dated the onset of symptoms 2 and 4 days before the CT scan — a much shorter interval than one would expect had rebleeding not occurred. One patient dated the onset of symptoms 3 weeks previously.

Likewise, we compared the predicted and actual time intervals between onset of symptoms and CT scanning in our patients with Type I SDH. Of the 10 patients with reliable histories, two dated the onset of symptoms 2 and 3 days before scanning. Indeed, these two patients may have experienced rebleeding into a chronic subdural sac with homogeneous mixing of the old effusion and the fresh blood in the correct proportion to produce an isodense SDH.

Type III isodense SDH was the least common variety encountered. A horizontal border is formed between the isodense material in the dependent portion of the subdural sac and the less dense material above. As with Type II isodense SDH, the diagnosis of SDH is usually obvious on the non-contrast scan. However, the full extent of the lesion is only apparent after administration of contrast material. One of our patients gave a history of trauma occurring 3 days before scanning. Rebleeding may play a role in the production of these lesions also.

Our experience leads us to believe that the pathogenesis of isodense SDH’s may be more complicated than the simple resolution of acute hematomas. Although this may be the most common origin of isodense SDH, rebleeding probably plays a more important role than was previously suspected. Rebleeding seems to be the only satisfactory explanation for the appearance of Type II isodense SDH. The short clinical course seen in some patients with Type I and III lesions seems to support this hypothesis, also. The relationship between the pathogenesis and the morphological appearance in isodense SDH is summarized in Fig. 8.

**Conclusions**

The diagnosis of isodense SDH may be established by use of contrast-enhanced CT scans. The demonstration of linear areas of contrast enhancement along the medial boundary of the collection is characteristic, and occurs frequently enough so that angiography may be avoided.

**References**


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