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Contrast-Enhanced CT of Acute Isodense Subdural Hematoma

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Originally, all acute subdural hematomas were thought to be hyperdense with respect to brain on CT [1]. But over the last 10–15 years, cases of acute isodense subdural and epidural hematomas have been reported [2–6]. Although contrast-enhanced CT has aided in the visualization of chronic isodense subdural hematomas [7], the use of contrast material was not efficacious in two of the acute isodense hematoma cases reported [4, 5], and was not performed in four others [2–4, 6].

This report illustrates the usefulness of contrast-enhanced CT in the evaluation of a case of acute head trauma with unexplained midline shift. In our case, an acute isodense subdural hematoma enhanced, as did the CSF cisterns and the isodense cerebral contusions. The recognition of this constellation of radiologic signs on unenhanced and enhanced CT in the setting of acute head trauma has an important hematologic implication and suggests the underlying presence of disseminated intravascular coagulation (DIC).

Case Report

A 45-year-old man, previously in good health, was admitted with massive head trauma after falling off the trunk of a slow-moving car. Unenhanced head CT scan obtained within 2 hr of the injury, demonstrated a right to left midline shift (Figs. 1A and 1B). There was no evidence of major intracerebral hemorrhage or of a hyperdense extracerebral hematoma that could account for the mass effect. Multiple bilateral occipital and temporal bone skull fractures were present. A contrast-enhanced CT scan demonstrated a homogeneously enhancing acute isodense right subdural hematoma (Figs. 1C and 1D). On the unenhanced CT, the CSF in the cisterns was poorly visualized (Fig. 2A). Contrast-enhanced CT showed cisternal, aqueductal, subarachnoid, and tentorial incisura enhancement, as well as enhancement of isodense intracerebral contusions (Fig. 2B). The patient had a hematocrit (Hct) of 43% and hemoglobin (Hg) of 14.7 g/dl. Laboratory confirmation of DIC was obtained with prothrombin time (PT) > 60 sec (11–13 N), partial thromboplastin time (APTT) > 120 sec (18–30 N), fibrinogen < 10 mg/dl (170–410 N), fibrinogen degradation products (FDP) 512 μ g/ml (0–10 N), platelet count

136,000 ($150\text{--}400 \times 10^3$ N). An emergency right craniotomy was performed with evacuation of an acute, nonclotted subdural hematoma. Postoperatively, the patient had elevated intracranial pressure and continued to bleed profusely from his head wounds, despite the administration of cryoprecipitate, fresh-frozen plasma, and blood. He died 12 hr after sustaining his injuries.

Discussion

Acute isodense subdural hematomas are rare. This case demonstrates the efficacy of contrast enhancement in the CT diagnosis of an acute isodense extracerebral hematoma.

Anemia has been reported as a cause of the isodensity of blood in some acute subdural hematomas [4]. Blood with an Hg concentration of 8–10 g/dl is isodense with brain [4]. Mild DIC, creating a liquid clot, has been suggested as the cause of an acute isodense subdural hematoma in a patient with an Hg of 12.3 g/dl [3]. In a recent report [8], DIC was present in four of 13 patients with acute head trauma who had either an acute subdural or epidural hematoma. Despite having low hematocrits (15–30%), none of these four patients had completely isodense extracerebral hematomas on CT scans obtained within 2 hr of the injury, as in our case. Contrast enhancement was not needed, since each extracerebral hematoma had a hyperdense area allowing for its visualization. The regions of isodensity in the hematomas seen on the CT scan correlated with areas of incoagulability found at surgery [8]. Others have suggested that admixture with CSF also contributes to the phenomenon of isodensity [3]. The various causes of isodensity in subdural hematomas have not been completely defined.

Hyperdensity of the subdural hematoma would have been expected in our patient with an Hct of 43% and Hg of 14.7. However, our patient also had acute DIC, which caused the incoagulable subdural hematoma found at surgery and which contributed to its isodensity. DIC also contributed to the isodensity of the cerebral contusions. Figure 3 is a schematic representation of the mechanism of the consumptive coagu-

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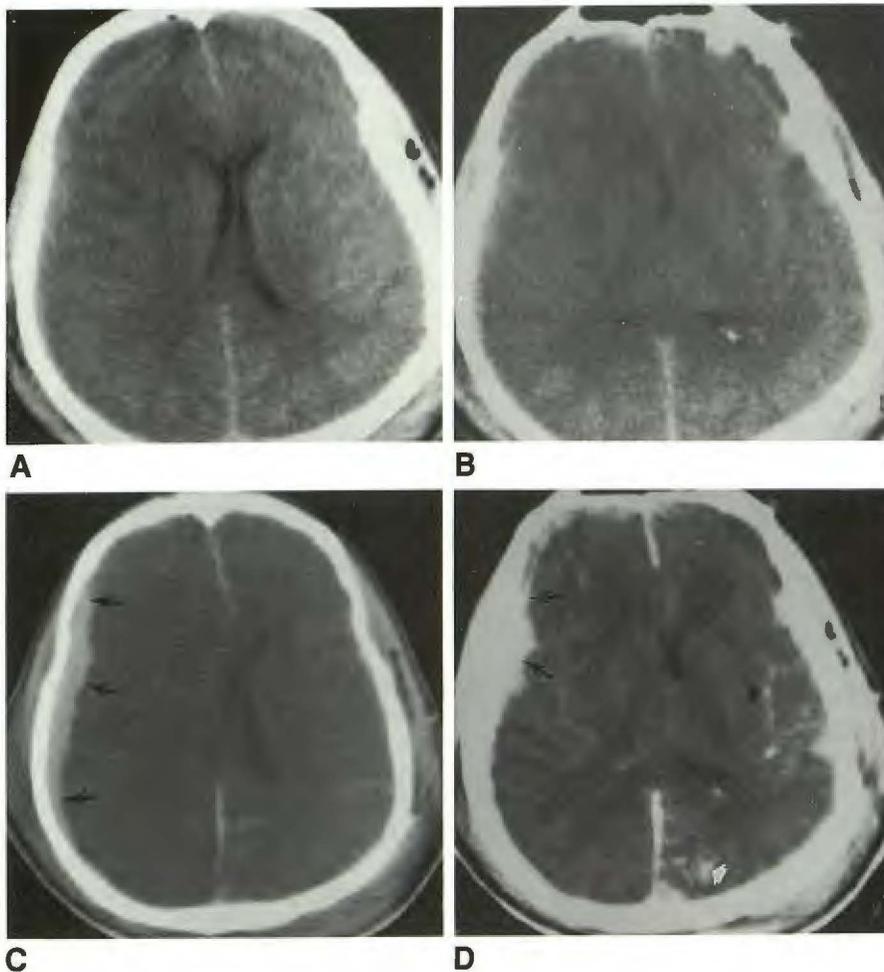


Fig. 1.—A and B, Noncontrast CT scans show right to left midline shift without an identifiable extracerebral fluid collection.

C and D, Contrast-enhanced CT scans show homogeneous enhancement of an isodense acute right subdural hematoma (arrows). There is diffuse subarachnoid enhancement (black arrowhead in D) and enhancement of an isodense left cerebral contusion (white arrowhead in D).

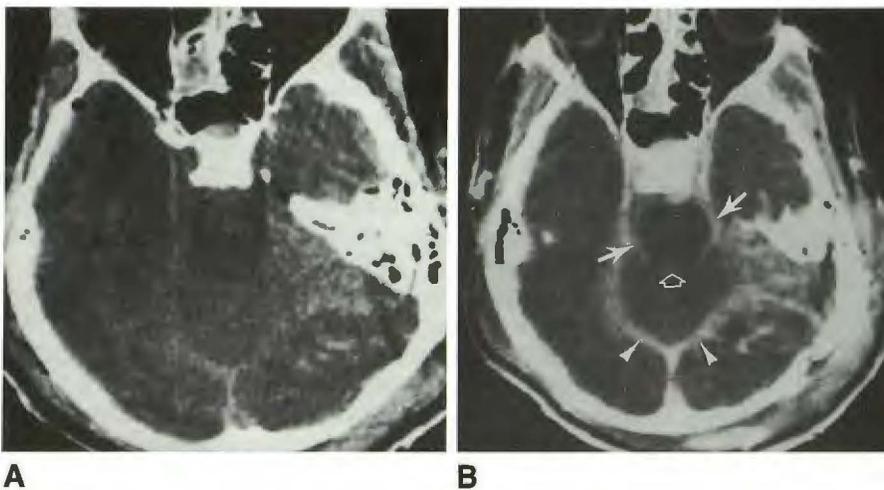


Fig. 2.—A, Noncontrast CT scan shows poor visualization of CSF in perimesencephalic cistern.

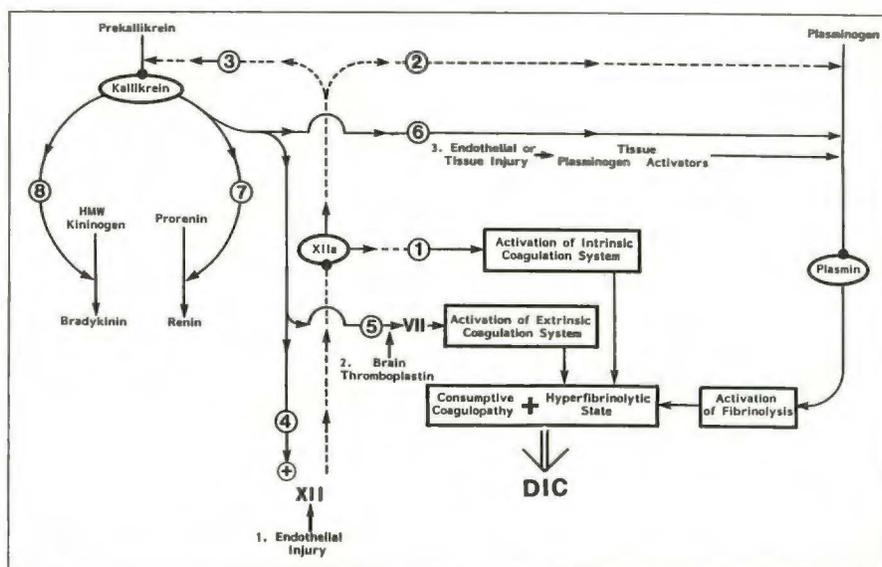
B, Contrast-enhanced CT scan shows cisternal (closed arrows) and aqueductal (open arrow) enhancement with marked enhancement of tentorial incisura (arrowheads).

lopathy and the hyperfibrinolytic state of head trauma-induced DIC (refer to [9] for more detailed information).

The homogeneous contrast enhancement of our patient's isodense subdural hematoma and isodense cerebral contusions most likely was related to increased blood brain barrier

permeability, partly induced by the activation of the kallikrein system and generation of bradykinin (Fig. 3) [10]. Explanations for cisternal enhancement include active bleeding, meningeal hyperemia, chemical irritation of the meninges by blood, and increased blood brain barrier permeability, with leakage

Fig. 3.—Schematic representation of mechanism of disseminated intravascular coagulation (DIC) activation in acute head trauma. Endothelial injury causes the conversion of factor XII to XIIa, which activates pathway 1 (intrinsic coagulation system), pathway 2 (fibrinolysis), and pathway 3 (kallikrein). Kallikrein serves as a positive feedback to activate more of factor XII (pathway 4), as well as pathways 5, 6, 7, and 8. Brain thromboplastin direct activation of factor VII and the extrinsic coagulation system also contribute to the consumptive coagulopathy component of DIC. The activation of plasmin by pathways 2 and 6, as well as tissue injury, leads to the hyperfibrinolytic state of DIC.



of contrast material through the leptomeninges, pial vessels, or venules [11].

MR imaging has been shown to play a role in diagnosing chronic isodense extracerebral fluid collections [12], but its feasibility for use in the acute, severely injured patient has not been demonstrated.

It has been stated that the use of contrast material in acute head trauma is unnecessary [13]. In our case, contrast administration demonstrated a homogeneously enhancing acute isodense subdural hematoma. We believe that contrast enhancement is important in the evaluation of unexplained midline shift and may also help to suggest the presence of DIC prior to laboratory verification.

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