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# High-Field MRI of Hemorrhagic Cortical Infarction

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High-field MRI is capable of differentiating acute, subacute, and chronic hemorrhagic cortical infarctions. In eight of nine patients, hemorrhage occurred in a vascular watershed zone. Acute hemorrhagic cortical infarction produces mild cortical low intensity on T2-weighted images outlined by subcortical edema (high intensity) and isointensity with normal cortex on T1-weighted images. Subacute hemorrhagic cortical infarction shows cortical high intensity first on T1-weighted images and later on T2-weighted images; it is also associated with subcortical edema. In the chronic stage, there is a marked persistent cortical low intensity on T2-weighted images. This is most prominent in the deeply infolded cortical gyri. The low intensity noted in acute and chronic hemorrhagic cortical infarction with T2 weighting appears to be related to two separate underlying histochemical states. The characteristic cortical low intensity observed on T2-weighted images in acute and chronic hemorrhagic cortical infarction is proportional to the square of the magnetic field strength.

We recently described characteristic intensity patterns on high-field MR images that permit staging of intraparenchymal hemorrhage [1]. This report extends these observations to hemorrhagic cortical infarction (HCI) and correlates our observations with the underlying pathophysiology.

## Subjects and Methods

Nine patients with CT evidence of HCI were imaged using spin-echo pulse sequences on a Signa 1.5-T MR scanner (General Electric, Milwaukee, WI). T1-weighted images used repetition time (TR) = 600 msec and echo time (TE) = 25 msec. T2-weighted images had TR = 2500 msec and TE = 30–80 msec. One patient was studied serially.

There were seven males and two females ranging in age from 3 to 86 years. All patients had unenhanced CT performed on either a GE 8800 or 9800 scanner. Two patients also had enhanced CT.

All patients presented with strokes. Five patients had a history of hypertension and one had been anticoagulated after an initial embolic stroke.

Patients had high-field MR scans 5–37 days after clinical presentation. CTs were performed within 3 days before MR in four patients, within 5–8 days before MR in three patients, and within 16–20 days before MR in two patients. Clinical detection and staging of HCI was difficult because hemorrhage does not necessarily occur at the time of acute infarction and may not be associated with clinical exacerbation [2]. The diagnosis of acute HCI was made on unenhanced CT by the presence of cortical high density. Subacute and chronic HCI was diagnosed and staged by MR. The presence of deoxyhemoglobin as detected by MR was defined as acute HCI. The presence of methemoglobin was defined as the subacute stage of HCI, and the presence of hemosiderin as the chronic stage.

## Results

All hemorrhagic cortical infarcts seen on CT were seen on high-field MR. We separated HCI on high-field MR into three stages (Table 1). More than one stage

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TABLE 1: MR Characteristics of Hemorrhagic Cortical Infarction

	Time Between Ictus and MR	Intensities on T1-Weighted Images	Intensities on T2-Weighted Images
Acute HCI (deoxyhemoglobin)	5–18 days	Isointense	Decreased intensity (pH and pO <sub>2</sub> dependent)
Subacute HCI (methemoglobin)	5–24 days	Increased intensity	Isointense; proceeds to increased intensity
Chronic HCI (hemosiderin)	5 years	Isointense	Markedly decreased intensity
Edema	5–24 days	Isointense	Increased intensity

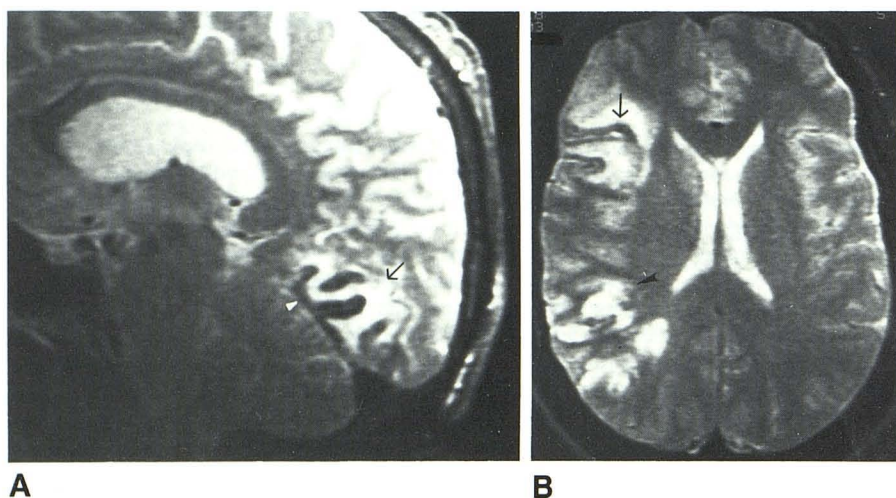


Fig. 1.—A, 77-year-old hypertensive man with acute hemorrhagic cortical infarction on CT 8 days before MR. Edema (arrow) highlights low intensity of deoxyhemoglobin (arrowhead). Surface coil image, SE 2500/80. B, Acute hemorrhagic cortical infarction in distribution of right middle cerebral artery (arrow) and its posterior watershed zone (arrowhead), T2-weighted image, SE 2500/80. Intensity of infarcted cortex is decreased to approximately that of normal white matter due to presence of deoxyhemoglobin.

was seen in three patients. In the acute stage, all but one HCI was of mildly low intensity on T2-weighted images and isointense with normal cortex on T1-weighted images. One HCI was very low intensity on T2-weighted images. The associated subcortical edema, which is of high intensity on T2-weighted images, highlighted the cortical low intensity (Fig. 1). In the subacute stage, cortical high intensity was initially noted on T1-weighted images (Fig. 2) and later on T2-weighted images (Fig. 3). On CT, edema without cortical high density was seen in the subacute stage of the high-field MR-detected HCI. In all cases, edema was better seen on high-field MR. Cortical atrophy on CT was seen in the chronic stage of high-field MR-detected HCI. Chronic HCI had very low intensity on T2-weighted images and was isointense with normal cortex on T1-weighted images (Fig. 4). Atrophy may be present in the chronic stage and can be separated from edema by the absence of mass effect and by the T2-weighted intensity. On T2-weighted images, edema converted to high intensity before cerebrospinal fluid did, whereas atrophic regions paralleled the intensity of cerebrospinal fluid.

Acute HCI was observed in seven patients, subacute HCI in six, and chronic HCI in one. In three patients, acute and subacute stages of HCI could be seen simultaneously in one infarct region. In one patient, two HCIs in different stages were noted. Multiple regions of HCI in a watershed distribution were observed in two patients (Figs. 4 and 5). The five patients with acute HCI on high-field MR had been scanned 12–18



Fig. 2.—3-year-old boy with right frontal hematoma after head injury and tentorial herniation, producing right posterior cerebral territory hemorrhagic cortical infarction. MR performed 37 days after trauma, 24 days after clinical exacerbation. High signal on T1-weighted image, SE 600/25, indicates methemoglobin. Subacute hemorrhagic cortical infarction.



Fig. 3.—69-year-old hypertensive man with hemorrhagic cortical infarction complicating cerebral arteriogram for carotid vascular disease. MR performed subacutely 13 days after stroke is bright on T1-weighted (A) and T2-weighted (B) images, consistent with extracellular cortical methemoglobin.

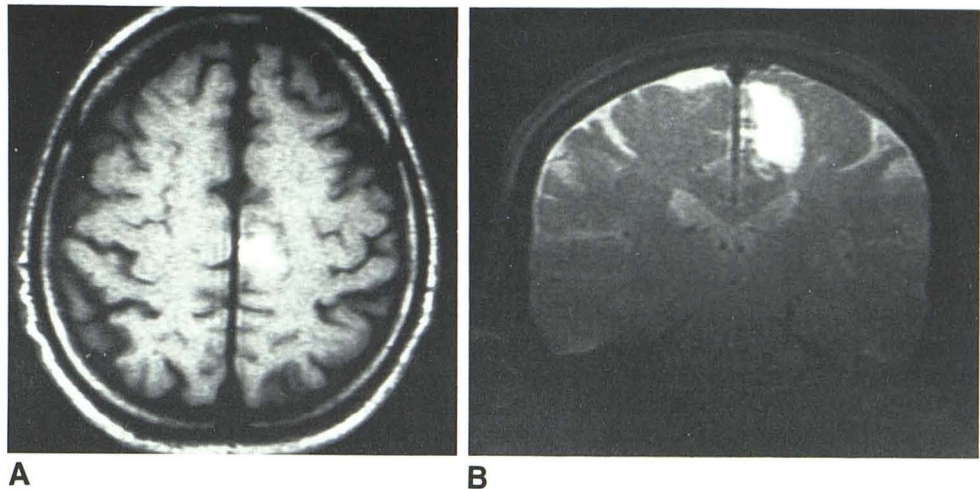
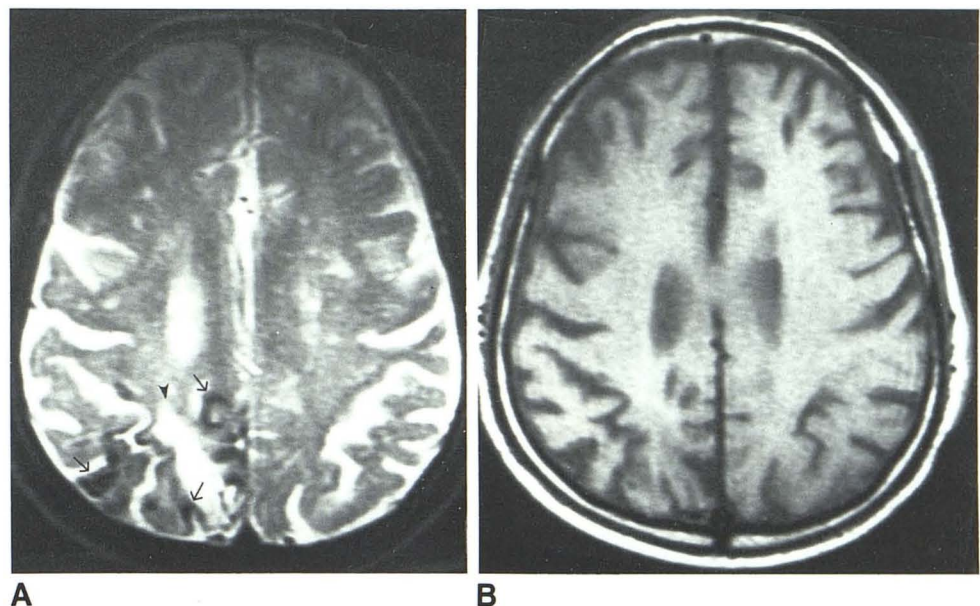


Fig. 4.—A, Incidental finding of chronic hemorrhagic cortical infarction in right occipital lobe from infarct that occurred 5 years before scan. Cortical hemosiderin (arrows) is low signal (black) and encephalomalacia (arrowhead) is high signal (white), SE 2500/80. B, T1-weighted image, SE 600/25, shows only atrophy in same location.



days after clinical stroke. The three patients with subacute HCI had been scanned 13–24 days after stroke, and the two patients with acute and subacute HCI had been scanned 5–8 days after stroke. The one patient with chronic HCI had a history of previous infarct 5 years before, corresponding to the lesion seen on MR. Bleeding may be apparent on CT immediately or from 1–21 days after clinical stroke [3, 4]. In four patients, the first CT showed no hemorrhage. In seven patients, infarcts were completely hemorrhagic and occurred in anatomic watershed zones. Regions of HCI also occurred in the periphery, corresponding to the shifted watershed of one otherwise bland infarct (Fig. 3). In one patient, cortical hemorrhage occurred adjacent to a necrotic tumor (Fig. 6).

## Discussion

Hemorrhagic infarction of the brain, described on gross pathology as red softening [5], occurs in about 20% of all

infarcts [2]. Hemorrhage is almost always confined to the cortex [5, 6]. Extravasated blood is found in the perivascular spaces, surrounding softened cortex, and usually in the overlying subarachnoid space [7]. Microscopically, scattered petechial hemorrhages may be seen in bland infarctions. However, in hemorrhagic infarction, gross hemorrhages in the cortex are numerous and the pia mater may be stained dark red or brown, indicating deposition of hemoglobin pigments (including hemosiderin). In the cerebellum, hemorrhages may become confluent, resulting in thin lakes of blood between layers of the cortex. An infarct may be only partially hemorrhagic, in which case the hemorrhage most often occurs in the periphery. The hemorrhage is most evident in the deeply infolded cortical gyri (Fig. 5). Initial hemosiderin deposition may be detected pathologically in 2–10 days [6].

Hemorrhage occurs in areas of ischemic brain that are reperfused, and is produced under four physiologic circumstances: (1) when an embolus, after occluding an artery, lyses



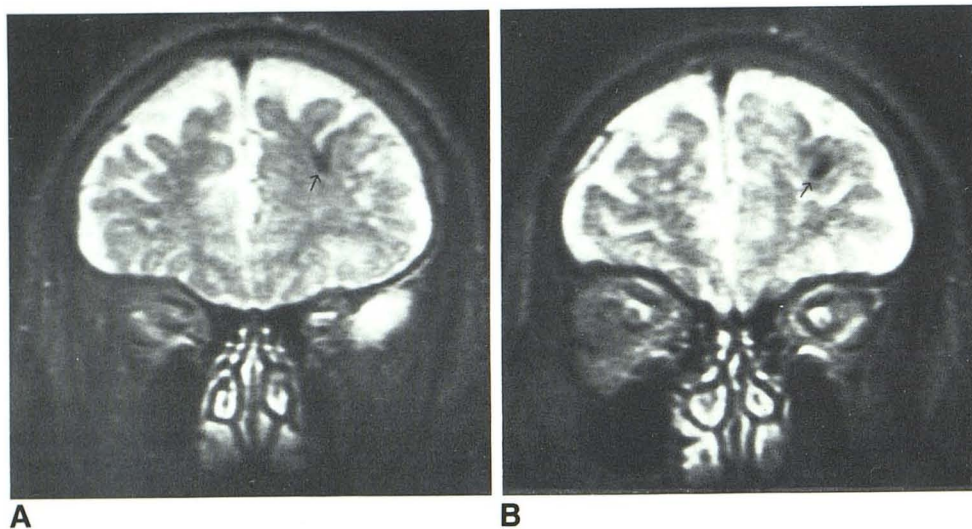


Fig. 5.—Same patient as in Figure 4. Chronic hemorrhagic cortical infarction in left frontal watershed zone with very low intensity most prominent at deeply infolded cortex (arrows). Volume averaging (A) gives hemosiderin appearance of low intensity. True depiction of very low intensity is better seen on adjacent image (B), SE 2500/80.

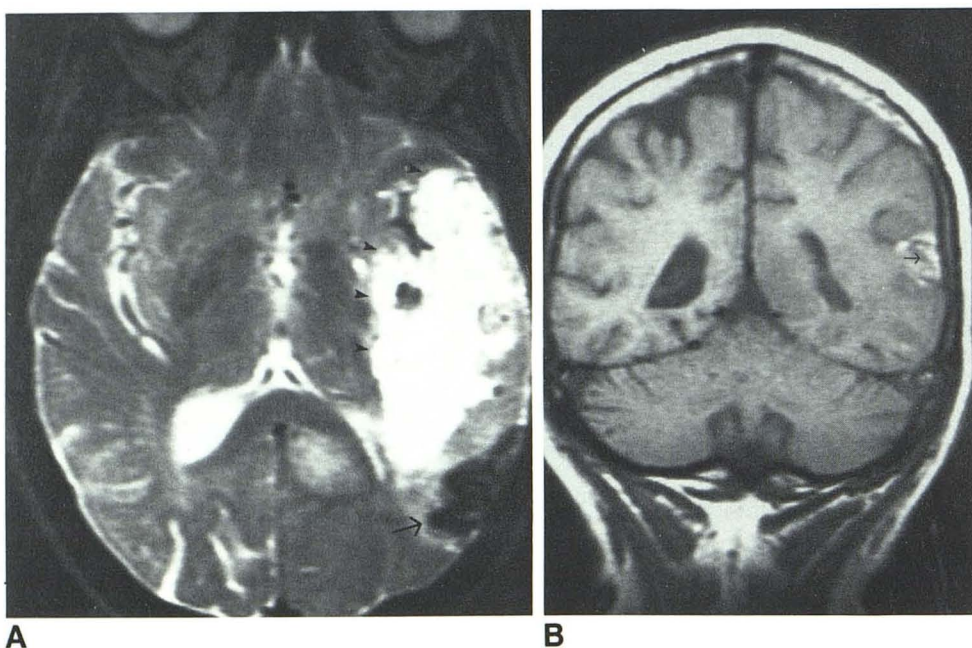


Fig. 6.—A, 72-year-old hypertensive woman. Very low intensity in acute hemorrhagic cortical infarction (arrow). High deoxyhemoglobin concentration is presumably due to local hypoxia adjacent to necrotic glioma (arrowheads), SE 2500/80. B, Subacute hemorrhagic cortical infarction in same patient. T1-weighted image, SE 600/25, shows cortical high intensity of intracellular methemoglobin (arrow).

or fragments and moves distally [7]; (2) when, after an area of brain is deprived of its blood supply by a proximal occlusion, collaterals from the adjacent vascular territory open to supply the tissue [7]; (3) when a hypotensive episode is followed by restoration of blood pressure; and (4) when there is intermittent compression of the posterior cerebral artery during tentorial herniation of the temporal lobe [8]. The first two mechanisms predispose watershed zones to HCI. In Romanul's original description of watershed infarction, five of 13 cases demonstrated hemorrhage [9]. HCI is also associated with hypertension [10, 11] and with anticoagulation [12]. Our findings are consistent with these pathologic mechanisms: HCI was observed in watershed zones (seven patients), after

anticoagulation (one patient), in hypertensive conditions (five patients), and after uncal herniation (one patient).

High-field MR of HCI differs somewhat from our observations of intraparenchymal hematoma. In the acute stage of HCI there was less low intensity on T2-weighted images than in intraparenchymal hemorrhage, even when the observed CT densities of the blood appeared equal. We believe this observed difference in intensity in acute hemorrhage is the result of a higher local  $pO_2$  in HCI as compared with intraparenchymal hematoma because of early vascular recanalization and luxury perfusion [13]. The higher oxygen tension lowers the percentage of deoxyhemoglobin in the red blood cells. The T2 relaxation effect of intracellular deoxyhemoglobin is



proportional to the square of the percentage of deoxyhemoglobin [14] as well as to the square of the magnetic field [1, 14]. Parenchymal hematomas, in which the bulk of the red blood cells are isolated from the circulation, are more hypoxic and thus have lower intensity on T2-weighted images. One cortical hemorrhage adjacent to tumor was hypoxic enough to have very low intensity on T2-weighted images (Fig. 6). Acute HCI is distinguished from chronic HCI by edema around the acute infarct, whereas atrophy occurs in a chronic HCI. The T2 relaxation enhancement of hemosiderin is not related to  $pO_2$  but is proportional to the square of the magnetic field strength [1].

These properties enable high-field MR to detect and differentiate cortical deoxyhemoglobin (low intensity) (Fig. 1) from cortical hemosiderin (very low intensity) (Fig. 4A). The indefinite persistence of cortical hemosiderin, as in old intraparenchymal hemorrhage, allows the detection of remote HCI. Staging of HCI is thus made possible by high-field MR. Theoretically, an HCI with minimal hemorrhage or very high  $pO_2$  may be difficult to detect.

Although the autooxidation of hemoglobin to methemoglobin is sensitive to  $pO_2$  (maximal at  $pO_2 = 20$  mm Hg), its paramagnetic dipolar relaxation mechanism is not [1]. In subacute HCI, high intensity is noted initially on T1-weighted images (intracellular methemoglobin) and subsequently on T2-weighted images (free methemoglobin) [1].

A possible but unobserved phenomenon at high  $pO_2$  is a sufficient slowing of methemoglobin production so that hemosiderin will be formed before appreciable amounts of methemoglobin accumulate. In this case, the T2-weighted images in high-field MR would reveal progression from the low intensity of deoxyhemoglobin to the very low intensity of hemosiderin without the intervening high intensity of free methemoglobin.

The multiple high-field MR stages observed in one HCI in three patients may be due to multiple bleeding episodes, extension of infarction, or a variation in the rate of evolution of the hemorrhage caused by local differences in perfusion and  $pO_2$  (Fig. 6).

Both intracellular methemoglobin and deoxyhemoglobin may appear as low intensity on T2-weighted images. Only methemoglobin, however, will appear as high intensity on T1-weighted images [1].

Acute hemorrhage and chronic hemosiderin deposits occur in identical cortical distributions and have similar but not identical low-intensity patterns. They may be distinguished by edema and by the difference in degree of preferential T2 proton relaxation enhancement of acute hemorrhage (low intensity on T2-weighted images secondary to intracellular deoxyhemoglobin) and chronic hemorrhage (very low intensity on T2-weighted images secondary to intracellular hemosiderin). It is ironic that the luxury perfusion that is responsible for the higher local  $pO_2$  may also be responsible for hemor-

rhage into previously infarcted cortex; moreover, it is this same perfusion that enables us to differentiate between acute and chronic HCI [13].

## Conclusion

High-field MR can detect and stage HCI just as it can intraparenchymal hematomas. There are, however, important differences in the high-field MR appearance of these lesions that can be explained on the basis of hemoglobin oxygen saturation. Appreciation of the pathophysiology of HCI expands our understanding of the MR images and their evolution.

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