

# CT and MRI in Acute Hemorrhagic Stroke

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## Key Words

Magnetic resonance imaging · Hemorrhage ·  
Diffusion-weighted imaging

## Abstract

We report the case of a woman presenting with an acute onset of hemiplegia. MRI was performed and showed a hyperintense mass on diffusion-weighted imaging, with areas of high signal intensity on T2\* imaging and T2/PD imaging. T1-weighted imaging showed a more inhomogeneous lesion with a central isointense part corresponding to the acute clot, as well as a peripheral crescent of hypointense plasma; since another mass lesion could not be acutely excluded, computed tomography was performed immediately afterwards, showing a hyperdense hematoma. The patient was taken to the neurosurgical operating theater, where she underwent decompressive craniotomy and evacuation of the hematoma.

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## Introduction

With the advent of potentially efficacious treatments for stroke, it is necessary to screen patients for therapy [1, 2]. Computed tomography (CT) can demonstrate early

signs of infarction [3] or exclude intracerebral hemorrhages, while magnetic resonance imaging (MRI) has been found to be unspecific in the early stages of bleeding. Echo-planar MRI can demonstrate hematomas [4–7] and diffusion-weighted MRI (DWI) can show early ischemia [8].

## Case Report

A 45-year-old female patient presented with acute hemiplegia and underwent early MRI and CT for suspected stroke, where it was not entirely possible to determine the hemorrhagic nature of the lesion based on the acute MRI findings alone. She had an unremarkable medical history except for migraine attacks. In the early afternoon, she suffered an acute onset of a left-sided hemiplegia. An MRI of the brain was performed 2.5 h after onset on a 1.0-T system (Siemens Harmony) with a gradient strength of 20 mT/m, and DWI was acquired with b values at 0 and 900 s/mm<sup>2</sup>. For DWI, the settings were: TR: 2,682.0 ms and TE: 135 ms. T1-weighted images (WI) were performed (TR: 519.0 ms, TE: 12.0 ms; 5-mm-thick slices), as well as T2/PD-WI (TR: 2,800.0 ms, TE: 85.0 ms, 14.0 ms; 5-mm-thick slices) and T2\*-WI (TR: 674.0 ms, TE: 26.0 ms; 5-mm-thick slices). DWI showed an inhomogeneous mass in the right basal ganglia and corona radiata with central hyperintense values (fig. 1A), corresponding to diminished diffusion, along with an anteriorly located hypointense crescent, corresponding to increased diffusion. T2\*-WI showed the mass to be hyperintense and inhomogeneous; frontally, there was a large crescent-like hyperintensity which was not clearly delineated from the white matter (fig. 1B). The mass posteriorly was less hyperintense, contained only a few hypointensities in its center and had sharper borders posteriorly than frontally, also seen

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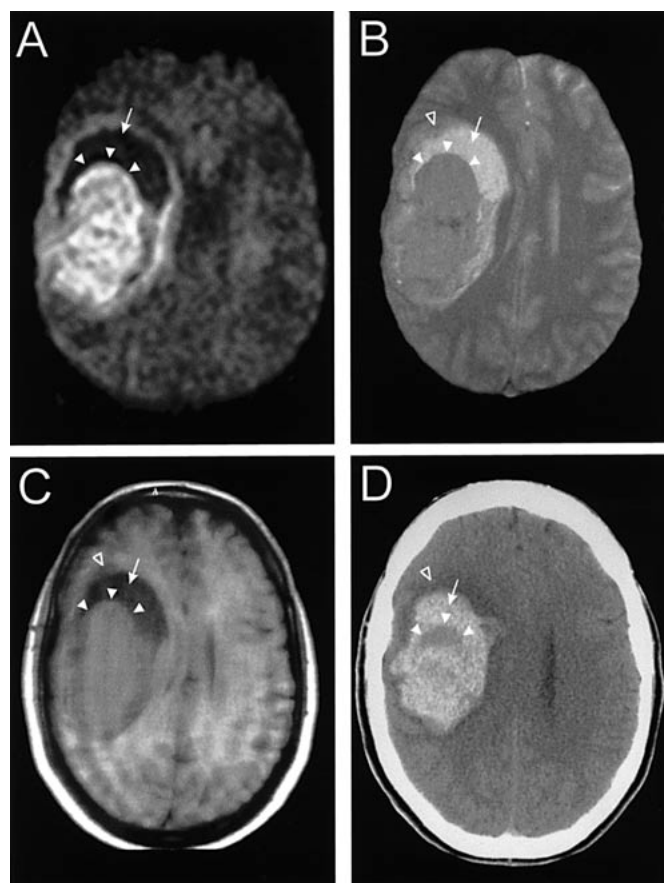
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on T2/PD-WI. T1-WI also revealed an inhomogeneous mass; there was a frontal crescent-like hypointensity (CSF-like values) with a more posterior mass-like isointense lesion, and frontally there was a slight hypointensity in the adjacent white matter (fig. 1C). Based on these findings, it could not be definitively concluded that this was a hematoma and not a mass lesion such as a tumor or an abscess. Therefore, the patient was directly transferred to the CT scanner. CT of the brain was performed and showed a large right-sided hematoma which was almost fully hyperdense, with a few less dense areas centrally in the region of the central mass seen on MRI (fig. 1D). There was also a small peripheral rim of hypodensity corresponding to edema. Carotid angiography did not show any bleeding source. The hematoma was surgically evacuated. The patient subsequently showed a left-sided hemisindrome with a distal motor paresis.

## Discussion

The T1 isointense lesion probably corresponded to the acute clot, with aggregation of erythrocytes and fibrin. The T1 hypointense crescent, which was also T2 hyperintense, must have been extravasated plasma, since it had all the MRI features of liquid (CSF, water). The fact that it was still hyperdense on CT must have been due to the extreme sensitivity of the method to the iron in the few erythrocytes which were still floating freely. The T2 hyperintensity was larger and encompassed both compartments, as well as the small rim of frontally located edema. It was not possible to distinguish the mass seen on T1-WI from the liquid crescent. Such a lesion cannot realistically be differentiated on imaging grounds alone from a mass lesion such as a tumor or an abscess, and therefore the postulation that hyperacute hemorrhage can be 'unequivocally recognized' by MRI findings alone should be somewhat toned down [4]. More experience is still needed in our opinion. When blood is extravasated, hemoglobin becomes deoxyhemoglobin, which is paramagnetic and induces a signal loss on T2\*-WI. Also, even though the theoretic basis for the susceptibility effects induced by ferritin and hemosiderin is not fully understood, higher magnetic fields increase susceptibility effects [9]. The results seen here are, however, in agreement with those seen in a recently published series of patients with hyperacute hemorrhage [7]; we also offer confirmation with concomitant use of CT, angiography and exploration. Some other studies have shown slightly different results, with more significant image drops in the area of hemorrhage both on T2\*-WI and DWI; this might be due in part to the imaging being performed slightly later than in this case and the series of Linfante et al. [7]. Patel et al. [6] found large areas of signal loss on susceptibility-weighted images in a series of 6 patients imaged between 2.5 and 6 h after clinical



**Fig. 1.** **A–C** MRI of the brain, performed 2.5 h after the onset of symptoms. **D** CT performed afterwards. **A** Axial DWI showing a mass in the right basal ganglia and corona radiata ( $b = 900\text{s/mm}^2$ ). The posterior part is hyperdense (white arrowheads), whereas frontally there is an area of higher diffusion (white arrow). **B** Axial T2\*-WI shows the mass to be hyperintense and inhomogeneous; frontally, there is a large crescent-like hyperintensity (white arrow) which is not clearly delineated from the white matter due to a small area of edema (open arrowhead). The mass posteriorly is less hyperintense and contains only a few hypointensities in its center (white arrowhead). Posteriorly, the borders are sharper than frontally. **C** Axial T1-WI shows the mass to be inhomogeneous; there is a frontal crescent-like hypointensity (CSF-like values, white arrow) with a more posterior mass-like isointense lesion (white arrowhead). Frontally, there is a slight hypointensity in the adjacent white matter, corresponding to edema (open arrowhead). **D** CT of the brain performed afterwards which shows a large right-sided hematoma; it is almost fully hyperdense, also peripherally (white arrow), with a few less dense areas centrally in the region of the clot (white arrowhead). There is also a small rim of hypodensity frontally (open arrowhead).

onset of symptoms. This series was imaged at field strengths between 1.0 and 1.5 T. Linfante et al. [7], in a series imaged between 23 and 120 min after symptom onset, found the lesions to have bright signal intensities centrally on T2- and T2\*-WI and hypointensities peripherally on T2- and T2\*-WI, along with a rim of T1 hypointensity and T2 hyperintensity. These findings correspond to those in our case. Interestingly, the center and peripheral parts as described by Linfante et al. [7] and seen in this case are not to be distinguished from one another on CT, showing MRI to be sensitive to the different parts of the hematoma. The peripheral rim of T1 hypointensity that they attributed to vasogenic edema can be very well visualized on the CT scan in our case. Schellinger et al. [4], in a series of patients imaged slightly later on a 1.5-T system (3–5.75 h), reported somewhat different findings; they found that T2\* imaging showed no or little area of

hyperintensity. Ebisu et al. [5], using a 1.5-T system, found that DWI demonstrated areas of mixed hyper- and hypointensity corresponding to the hematoma. The inhomogeneous signal observed on DWI in our case is partly due to the echo-planar nature of the sequences, which causes high susceptibility effects locally with areas of both hypo- and hypersignal. This report is encouraging, in that there is a correlation between acute CT and MRI findings, and it shows that even at a lower field strength, with the use of more powerful gradient systems, we can demonstrate acute bleeding with a combination of conventional MRI, echo-planar diffusion imaging and susceptibility-weighted sequences. However, it is necessary to carefully evaluate the data, since there is still not full agreement in the literature on the MRI findings alone. Therefore, more data are needed in order to finally confirm these initial findings.

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