Case Report / *Olgu Sunumu*

# Magnetic Resonance Imaging Features of Hyperacute Intracranial Hemorrhage Within the First Minutes

## *Hiperakut İntrakraniyal Kanamanın İlk Dakikalardaki Manyetik Rezonans Görüntüleri*

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**Abstract**

Hyperacute active intracranial hemorrhage is described which occurred while the patient was undergoing MR imaging. *(The Medical Bulletin of Haseki 2010; 48: 50-2)*

**Key Words:** Hyperacute intracranial hemorrhage, MRI

**Özet**

Burada manyetik rezonans görüntülemesi yapılırken gelişen hiperakut intrakraniyal kanama olgusu sunulmuştur. *(Haseki Tıp Bülteni 2010; 48: 50-2)*

**Anahtar Kelimeler:** Hiperakut intrakraniyal kanama, MR

### Introduction

Hyperacute intracranial hemorrhage is an infrequent finding on MR imaging and there are only a few studies on MR imaging of hyperacute cerebral hemorrhage (1,2). After intraparenchymal hemorrhage, oxyhemoglobin, a diamagnetic substance, passes from arterial blood to the tissue with low oxygen concentration and becomes deoxygenated. Owing to its unpaired electrons, deoxyhemoglobin is a paramagnetic substance and causes signal intensity loss with T2-weighted sequences, especially on T2*-weighted images, due to susceptibility differences between diamagnetic tissue and paramagnetic deoxyhemoglobin (1-4). Although a few data exist about the time of transition to deoxyhemoglobin, it is mostly stated that it does not become apparent until several hours (3). We report active intracranial hemorrhage which occurred while the patient was undergoing MR imaging.

### Case Report

A 61-year-old woman who was on medication for breast cancer was referred to MR department for cranial MR. She underwent MR imaging (Philips gyroscan intera 200. Netherlands.) at 17:16 with the following sequences: axial diffusion (echo-planar spin-echo at 17:17, TR: 4393msec, TE: 81 msec, b=0 and 1000 seconds/cm$^2$, slice thickness: 5.0 mm gap: 1 mm, epi factor: 77, NSA:1, matrix size: 77x256, FOV: 230 mm), coronal FLAIR (at 17:18, TR: 6000 TE: 100 TI: 2000 slice thickness: 5 mm gap:1.5 mm NSA: 1 matrix size: 203x256 FOV: 230 mm), axial T1 (spin echo, at 17:22 TR: 450 TE: 12 slice thickness: 5 mm gap: 1.5 mm NSA: 1 matrix size: 179x256 FOV: 230 mm), axial dual echo T2 (turbo spin echo, at 17:24 TR: 2200 TE: 20 TSE factor: 14 slice thickness: 5 mm gap: 1.5 mm NSA: 2 matrix size: 193x512 FOV: 230 mm), sagital T2 (turbo spin echo, at 17:28 TR: 4057 TE: 100 TSE factor: 15 slice thickness: 5 mm gap: 1 mm NSA: 2 matrix size: 292x512 FOV: 230 mm) and postcontrast axial, sagital and coronal T1 with the same parameters before contrast injection, except for SPIR on coronal sequence. A mass lesion of approximately 4x4x4 cm dimensions was seen on the right frontal lobe with hyperacute hemorrhage (Figure 1).

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## Discussion

Appearance of intracerebral hemorrhage on MR images has a complicated appearance depending on its evolution over time and location which leads to the changes in the signal intensity pattern in the evolving hemorrhage due to the products of iron metabolism and to the integrity of the red blood cells (1,3).

What happens in intracranial hemorrhage is that oxyhemoglobin is diamagnetic substance and as oxyhemoglobin passes from arterial blood with high oxygen tension to tissue with low oxygen concentration, the molecule becomes deoxygenated. Due to its unpaired electrons, deoxyhemoglobin is a paramagnetic substance and produces a nonuniform magnetic field. Susceptibility differences between diamagnetic tissue and paramagnetic deoxyhemoglobin, confined within erythrocytes, result in rapid spin dephasing that leads to signal intensity loss with T2-weighted sequences; this finding is more obvious on T2*-weighted images (1,2,4).

Intracranial hemorrhage less than 24 hours old has been postulated to be in oxyhemoglobin phase and would be isointense on T1-weighted and slightly hyperintense on T2-weighted images, however, in 1996, Patel et al. (3) reported in a study of 6 patients that susceptibility sequences were sensitive for hyperacute hemorrhage within 2.5-5 hours of onset of clinical symptoms as regions of marked signal loss due to susceptibility effects. They also added that either due to a rim of storage iron or due to a region of dephasing at the boundary of brain and blood, a peripheral rim of hypointensity is seen in acute phase on both T1-and T2-weighted images.

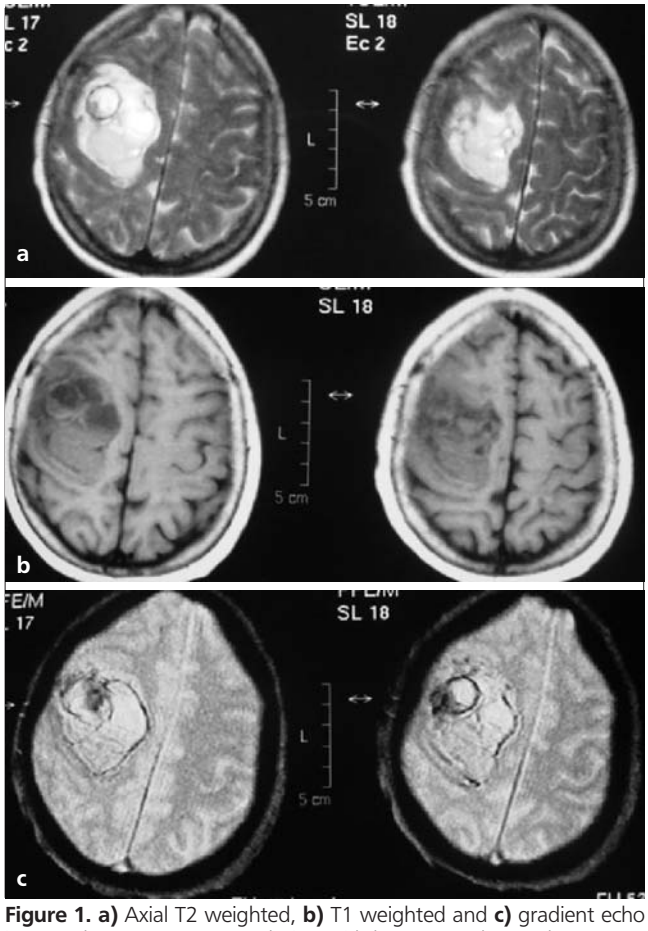


**Figure 1. a)** Axial T2 weighted, **b)** T1 weighted and **c)** gradient echo images demonstrate a mass lesion with hyperacute hemorrhage

Signal loss on long TR/short TE, long TR/long TE and gradient-echo images is the earliest published change observed in a rat model of intracranial hemorrhage (5).

In 1999, Linfante et al. (4) examined 5 patients between 23 minutes and 2 hours after onset of symptoms of cerebral hemorrhage and found out that EPI T2*-weighted imaging was the most useful image modality in detecting hyperacute phase of intracranial hemorrhage. They stated that hyperacute hematoma appears to be composed of 3 distinct areas: a center, a periphery and a surrounding rim. The center is isointense to hyperintense heterogeneous signal on susceptibility-weighted and T2-weighted imaging and isointense to hypointense on T1-weighted imaging and becomes smaller over time. The periphery shows hypointensity (susceptibility effect) on susceptibility-weighted and T2-weighted imaging and not apparent on T1-weighted imaging. A surrounding rim of hypointense on T1-weighted imaging and hyperintense on T2-weighted imaging represents vasogenic edema encasing the hematoma. They proposed that hypointensity on T2-weighted images progresses with time from the periphery of the hematoma towards the center. This observation has been described both in animal models (5) and in humans (3), which was explained by the presence of deoxyhemoglobin being highest in the periphery of the hematoma (5). Although the meaning of increased signal intensity seen on susceptibility-weighted imaging in the center of the acute intracranial hemorrhage, which is higher than in normal tissue but lower than in edema or cerebrospinal fluid, is not clear, Linfante et al. (4) hypothesized that fresh blood in the center of a hyperacute intracranial hemorrhage has signal characteristics of a proteinaceous solution since proton relation times are not yet significantly influenced by the presence of deoxyhemoglobin.

Weisman et al. (2) examined 7 patients at 3.5-24 hours after onset of symptoms of hyperacute hemorrhage and reported that intracranial hemorrhage was clearly discernible on EPI-T2*-weighted and diffusion-weighted images in all cases and that all images showed the hematomas as either discrete, deeply hypointense homogeneous lesions or as lesions of mixed signal intensity containing hypointense areas.

Schellinger et al. (6) reported that intracranial hemorrhage could be identified on all T2-weighted, FLAIR, diffusion- and perfusion-weighted sequences in a study of 9 cases of 3-6 hours from symptoms onset. They stated that the typical appearance of intracranial hemorrhage on the MR images was a heterogeneous focus of high and low signal intensities, with increasing susceptibility weight, the central area of hypointensity becoming more pronounced (7).

We conclude that MR imaging is diagnostic in hyperacute intracerebral hemorrhage, even in the first minutes.

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