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Literature Review

# Detection and Appearance of Intraparenchymal Hematomas of the Brain with MRI on Gradient Echo Pulse Sequence and Diffusion Weighted Images (DWI)/ADC Maps: The T2 Blackout Effect

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## Abstract

The pattern of evolving intraparenchymal brain hematomas on conventional magnetic resonance imaging (MRI) has been well documented in multiple publications but remains somewhat complex. Its interpretation also remains quite complicated when evaluated in MRI diffusion weighted imaging (DWI) and ADC map images.

***Keywords:*** *Magnetic Resonance Imaging (MRI); T2 Shine-Through, T2 Blackout; T2 Washout Effects*

Three interesting phenomena have been described in brain MR-DWI. They are T2 shine-through, T2 blackout and T2 washout effects.

### T2 shine-through

This phenomenon seems well understood and it is demonstrated as a hyperintense region on diffusion-weighted images; this is a result of T2 prolongation effects, which can be present in cases of subacute to chronic infarction for instance. When an decreased apparent diffusion coefficient may be present this may result in the accentuation of hyperintensity on diffusion-weighted images causing the lesion be apparently brighter.

### T2 washout

The term "T2 washout" denotes isointense area in lesions observed on diffusion-weighted images; this is thought to be caused by a balance between hyperintensity on T2-conventional MRI weighted images and an increased apparent diffusion coefficient inside the lesion. This phenomenon can be seen in areas of vasogenic edema.

### T2 blackout

This phenomenon implies hypointensity on diffusion-weighted images caused by hypointensity observed on T2-weighted images. T2 blackout has been commonly detected in intraparenchymal brain hematomas. The cause of T2 blackout is predominantly caused by susceptibility effects independent of tissue diffusibility.

Unfortunately, DW imaging is not a simple map of diffusion of water molecules but is a combination of contributions from diffusion of molecules inside the tissues as well as tissue relaxation characteristics related to T1 and T2 effects, hence T2 shine through and T2 washout can be detected. Because of these mixture of effects in the same image, the DW images should be interpreted with reference to images obtained with other sequences in conventional MRI, such as T1, T2-weighted sequences as well as images originated from the calculation of ADC map.

DWI evaluation of hemorrhage should be interpreted cautiously and in conjunction with T2- and T2*-weighted MRI since susceptibility artifacts from blood products contribute to the appearance of hemorrhage on DWI and may influence the apparent diffusion coefficient (ADC) calculations.

The appearance of hematoma on DWI and ADC maps varies with the "age" of the hematoma. Hyperacute hematomas are hyperintense on T2-weighted imaging and DWI because of the T2 shine-through phenomenon associated with brain edema and T2 effects. T2- blackout effect may occur in the acute and early subacute stage when hematomas are hypointense on T2-weighted imaging. These hematomas can be equally hypointense on DWI and ADC map which is related to the T2-blackout effect.

In a study, DWI was accurate for the detection of hyperacute, medium, large sized acute, early and late subacute, subdural, hemorrhagic components of arterial and venous infarction, intraventricular hemorrhage. In this study subarachnoid and small intraparenchymal hemorrhage were described on DWI as areas of low sensitivity.

In hyperacute, acute, early and late subacute hematoma the ADC measurements were statistically equivalent and were significantly less than that of late subacute hematoma. ADC values are moderately but consistently diminished during the first three stages of hematoma; this indicated that hematomas are almost always hypointense in ADC images.

Oxyhemoglobin evolves sequentially in hemorrhage: first to deoxyhemoglobin, then to methemoglobin, and finally to hemosiderin or ferritin. Oxyhemoglobin is a diamagnetic component and as such has been reported to show hyperintensity on T2WI and diffusion-weighted images with a decreased ADC measurement. Deoxyhemoglobin, being paramagnetic, as well as intracellular methemoglobin, hemosiderin, and ferritin may cause hypointensity on T2WI and diffusion weighted images. A reduced ADC in hemorrhage with intact red blood cell membranes and an increased apparent diffusion coefficient after lysis of red blood cell membranes have been reported. The latter can be related to dilution of methemoglobin in the extracellular fluid.

In a study, using gradient strengths with b values of 1000 s/mm$^2$, it was found that in the core of the hematoma the ADC values can be moderately but significantly reduced during the initial three stages of hematoma, without any substantial variation throughout the course of the hematoma.

In another study, the core of acute and early subacute hematomas was found to be markedly hypointense on DWI as well as on T2WI conventional MRI. This hypointensity can be attributed to the magnetic field inhomogeneity caused by paramagnetic intracellular deoxyhemoglobin in acute hematomas. The paramagnetic intracellular methemoglobin of acute and early subacute brain hematomas showed reduced ADC values compared with normal white matter. Late subacute hematoma was hyperintense on DWI, its ADC value was lower than that of the adjacent normal white matter.

At the chronic stage of brain hematomas, paramagnetic hemosiderin and ferritin are found in the periphery; this can be seen on DWI and on T2-weighted images as a dark halo surrounding the hematoma. The ADC value rises as the lesion approaches a cystic cavity which means that the core of the lesion in a chronic hematoma may appears isointense at the early chronic stage and hypointense at the late chronic stage on DWI.

The ADC values can be higher than those of normal white matter in chronic hematomas at the late chronic stage.

Hyperintensities could be explained by the presence of traces of methemoglobin causing proton - electron dipole - dipole (PEDD) interaction. This PEDD is the main cause of hyperintensity in a late subacute infarct on T1 and T2 weighted images [1]. The findings on DWI and ADC images have been reported to be characteristic of a chronic hematoma [2]. The marked hypointense ring observed on DWI

and GRE images is explained by the presence of paramagnetic hemosiderin and ferritin [3]. The hyperintense halo observed around the hematoma on T2, FLAIR, and ADC images is produced by resolving vasogenic edema. This hyperintensity may not be observed on the DWI, which indicates that there is free movement of water due to vasogenic edema [2]. This finding may rule out the diagnosis of acute infarct [2,4-7].

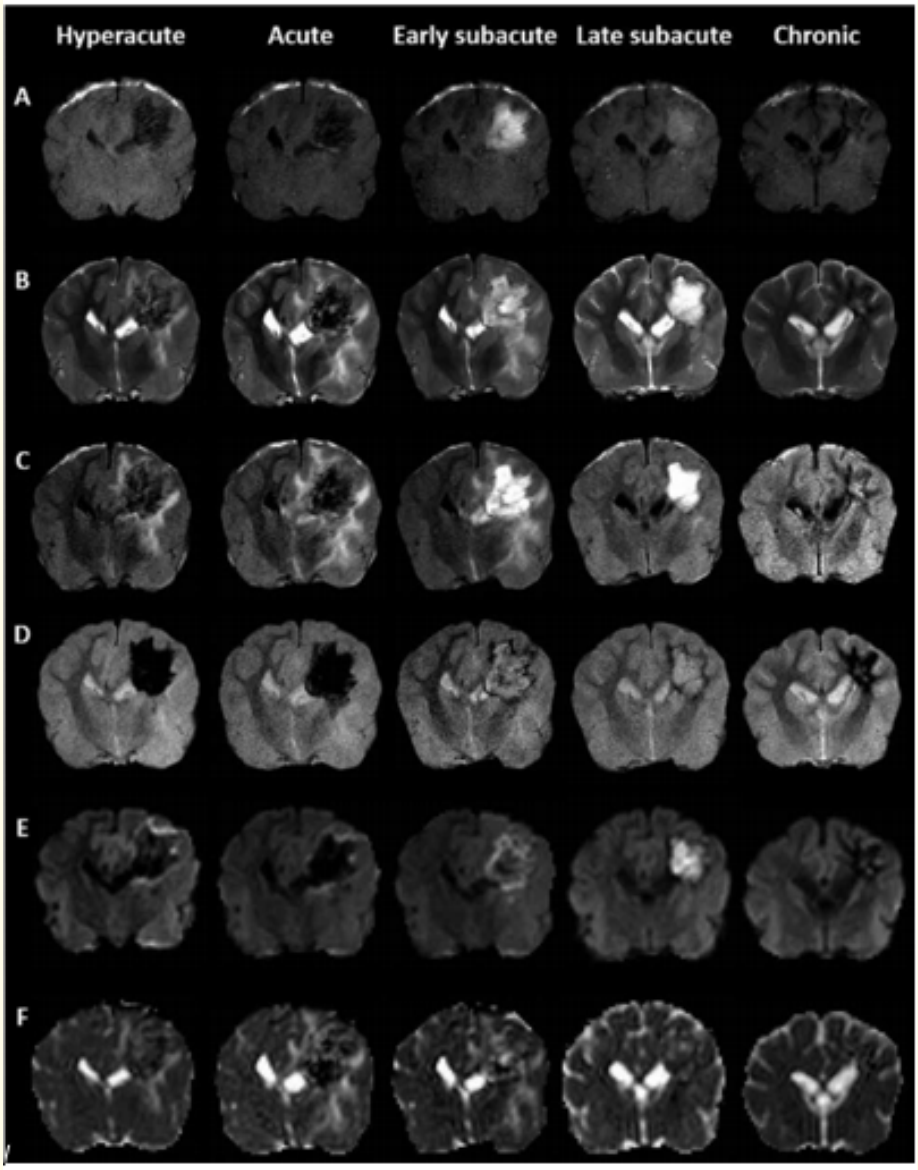


***Figure 1:*** *Representative (A) T1WI, (B) T2WI, (C) FLAIR, (D) GRE, (E) DWI, and (F) ADC maps illustrating the temporal evolution of hemorrhagic lesions according to the evolving stages. Hematomas in the right side of the parietal lobe underwent rapid and reversible changes in signal intensity, which varied with the pulse sequence. From: Daegi An., et al [8].*
*There is a persistent black out effect noted in ADC map images.*

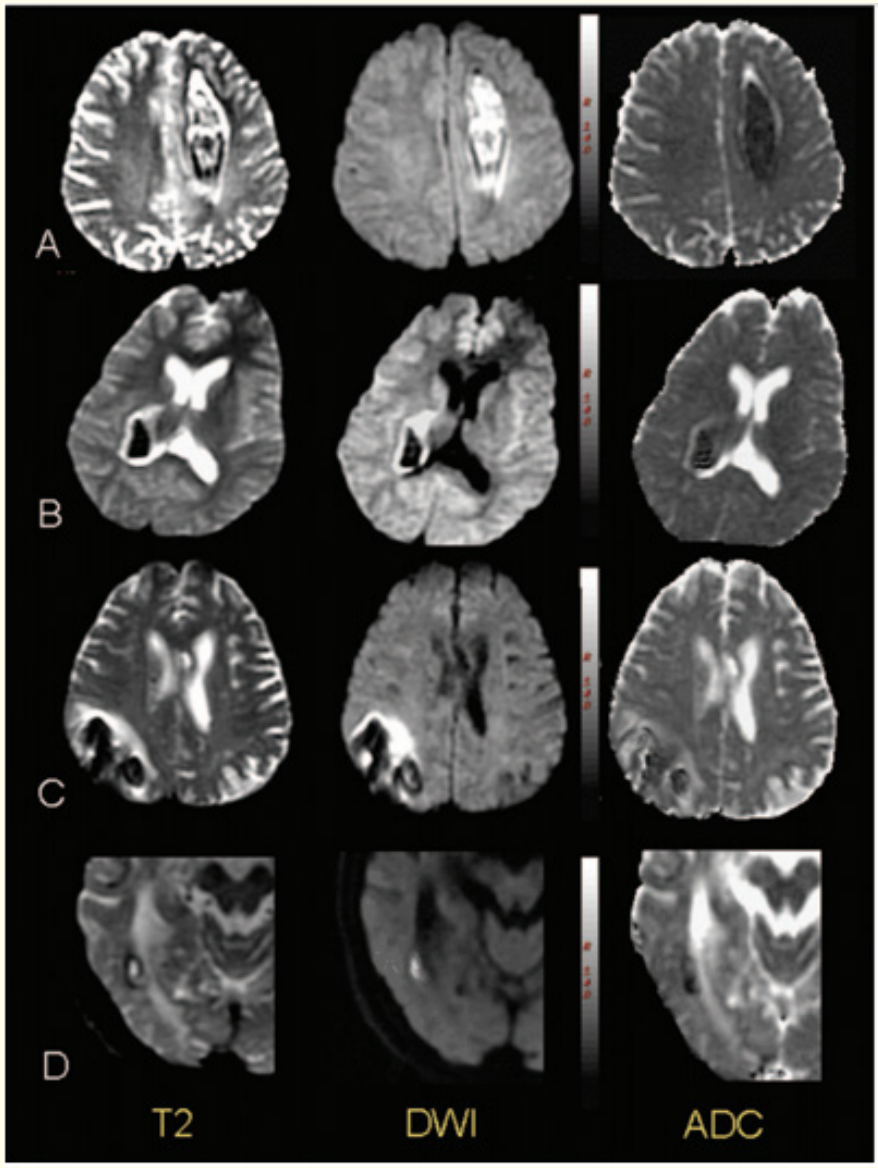


***Figure 2:*** *T2 shine-through (A, D) and T2 blackout effects (B, C) in intracerebral hematomas. A, Hyperacute hematoma in a 28-year-old man. The frontal lobe hematoma is isointense on a T1-weighted, hyperintense on a fluid-attenuated inversion recovery (not shown), T2-weighted, and DW image acquired with low ADC values. B, Acute hematoma in a 19-year-old woman. The right capsule-thalamic hematoma is isointense on a T1-weighted (not shown), hypointense on a T2-weighted, and hypointense on a DW image with low ADC values. C, Early subacute hematoma in a 54-year-old man. The right parietal lobe hematoma is hyperintense on a T1-weighted (not shown), hypointense on a T2-weighted, and hypointense on a DW image acquired with low ADC values. D, Late subacute hematoma in a 73-year-old man. The right temporo-occipital hematoma is hyperintense on a T1-weighted (not shown), hyperintense on a T2-weighted, and hyperintense on a DW image acquired with low ADC values. From: Stephane Silvera., et al [9]. There is a persistent black out effect noted in ADC map images.*

## Conclusion

Information obtained from DWI/ADC images may be of great contribution for the diagnosis of intraparenchymal brain hematomas/hemorrhage through the T2 black out phenomenon; still additional evaluation of the images from conventional MRI including post contrast images may be necessary. Persistent T2 black out effect of various intensities or degree of blackness can be noted in ADC map images. Hypointensities detected on diffusion-weighted images due to T2 shortening and magnetic susceptibility effects (with T2*/gradient echo images) occur in a variety of conditions related to iron deposition including intraparenchymal brain hemorrhage, primary lesions with associated hemorrhagic areas like some granulomatous lesions and tumors, like metastasis from hemangiosarcoma.

GRE/T2* images are typically more sensitive than b0 images in the detection of hemorrhages including pinpoint microbleed events and should be included in emergency brain MR studies for acute infarction.

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