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Pictorial Review

Diffusion-weighted signal patterns of intracranial haemorrhage

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The signal pattern of intracranial haemorrhage on diffusion-weighted imaging (DWI) as it evolves over time is rarely discussed due to the sensitivity of T2*-weighted sequences and the specificity of classic signal patterns on T1 and T2-weighted sequences. The DWI signal is strongly affected by the magnetic susceptibility of paramagnetic blood products and, therefore, is markedly hypointense in the same phases that demonstrate hypointensity on T2*-weighted sequences; however, hyperacute haemorrhage (oxyhaemoglobin-predominant clot) and late subacute haemorrhage (extracellular methaemoglobin) do not demonstrate T2* hypointensity. Moreover, T2*-weighted sequences are less sensitive to the presence of extra-axial haemorrhage than to intraparenchymal haemorrhage. At these stages of evolution, haemorrhage demonstrates high DWI signal in association with low ADC values, which may be more pronounced than even its corresponding fluid-attenuated inversion recovery (FLAIR) signal. DWI is useful for identifying hyperacute subarachnoid haemorrhage and as a problem-solving tool in challenging cases.

Introduction

Hypointensity on T2*-weighted sequences, such as gradient echo (GRE) and susceptibility-weighted imaging (SWI), has been ingrained into radiologists' minds as synonymous with the presence of blood or calcium. This strong association is due in part to the relative sensitivity of such sequences to paramagnetic effects, which cause spin dephasing (a concept with a more straightforward explanation than the complex magnetic interactions underlying the T1-weighted and T2-weighted signal of haemorrhage).

For the sake of simplicity, we often fail to emphasise the fact that not all blood is magnetically susceptible and, therefore, that not all blood can be detected on T2*-weighted sequences. Although most parenchymal haemorrhage is composed of mixed blood products in different stages of evolution, resulting in T2* hypointensity for at least a portion of the haematoma (and fairly reliable detection), such sequences are frequently insensitive to extra-axial haemorrhage.[1–3] For example, small subdural haematomas can be missed on T2* imaging due to difficulty distinguishing the haematoma from the blooming produced by adjacent bone.[4] For this reason, other sequences, such as fluid-attenuated inversion recovery (FLAIR) as well as diffusion-weighted (DW), may be important in evaluating for extra-axial haemorrhage.

Although DW imaging is a standard sequence in most magnetic resonance imaging (MRI) of the brain, many

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radiologists remain unfamiliar with the appearance of haemorrhage on this sequence. In this review, we discuss the temporal evolution of DW signal in various forms of intracranial haemorrhage, with emphasis on its utility as an adjunct to T2*-weighted sequences in detecting haemorrhage.

Major physical principles underlying DW signal in haemorrhage

Signal on DW imaging is derived from multiple factors intrinsic to the tissue being excited as well as its local microscopic environment. In its most clinically relevant applications, DW signal is dominated by the cancellation of signal from water molecules, which are restricted from diffusing freely within their local microenvironment[5]; however, in tissue without restricted diffusion or free diffusion (such as cerebrospinal fluid), DW signal receives varying contributions from the T2 relaxation time, T1 relaxation time (in general less significant than the T2 effect), and T2* effects intrinsic to the tissue.[6,7]

The DW signal of haemorrhage at each stage of blood-product evolution is summarised in Table 1 and each stage is discussed in detail below. For many forms of haemorrhage, the T2* effect comprises the dominant contribution to DW signal due to the presence of paramagnetic haemoglobin by-products.[6] Deoxyhaemoglobin and methaemoglobin are considered weakly paramagnetic, the iron atoms at their cores having four and five unpaired electrons, respectively.[8] Haemosiderin, a macromolecular aggregate of haemoglobin by-products, has tens of thousands of unpaired electrons.[9]

The mere presence of a paramagnetic substance is, by itself, insufficient to produce MRI-detectable spin dephasing. The compartmentalisation of such paramagnetic molecules within the intracellular compartment of the red blood cell or the macrophage/microglia is a key factor in generating the local heterogeneity of the magnetic field (Fig 1b and 1d), which is the most proximate cause of spin dephasing and signal loss on GRE/SWI sequences.[10] Such blood products demonstrate marked DW hypointensity as the T2* effects predominate over the diffusion characteristics and intrinsic T1 and T2 signal of the haematoma.[11,12]

In contrast, extracellular methaemoglobin distributed in relatively homogeneous fashion throughout the extracellular space (Fig 1c) does not result in significant magnetic field heterogeneity[8] and, therefore, does not demonstrate magnetic susceptibility on GRE or SWI sequences. Oxyhaemoglobin and carboxyhaemoglobin, seen in hyperacute haemorrhage, are weakly diamagnetic substances, having no unpaired electrons.[8,9] This results in minimal distortion of the magnetic field and no significant signal loss from magnetic susceptibility (Fig 1a). In hyperacute and late subacute haemorrhage, therefore, the DW signal is free of significant T2* effects and is instead largely determined by what may represent true restricted diffusion and the intrinsically long T2 time of such haematomas.[1,7,13]

Relationship of location and environment to blood-product evolution

A few location-specific factors that can affect the temporal evolution of blood merit additional consideration when analysing the MRI signal pattern of haemorrhage, including its DW signal: oxygen tension, the presence of tissue macrophages, and the concomitant presence of tumour cells.

High oxygen tension inhibits the transition from oxyhaemoglobin to deoxyhaemoglobin as well as promoting the oxidation of the iron atom at the centre of the haem molecule during the transition from deoxyhaemoglobin to methaemoglobin.[8,14] When compared to parenchymal haemorrhage, therefore, the relatively high oxygen environment of epidural, subdural, and subarachnoid haemorrhage lends itself to a prolonged hyperacute phase (as oxyhaemoglobin is deoxygenated less quickly). The oxidation of deoxyhaemoglobin to methaemoglobin should be accelerated by the presence of high oxygen levels; however, this effect is counteracted by the oxygen-driven action of methaemoglobin reductase, which returns the haem to the deoxyhaemoglobin state. The net effect, in fact, may be a

Table 1
Magnetic resonance imaging signal characteristics of different stages of haemorrhage.

Stage	Hyperacute	Acute	Early subacute	Late subacute	Chronic
Type of blood product	Oxyhaemoglobin	Deoxyhaemoglobin	Intracellular Methaemoglobin	Extracellular Methaemoglobin	Haemosiderin
T1-weighted signal intensity	Intermediate	Intermediate	High	High	Low
T2-weighted and FLAIR signal intensity	High	Low	Low	High	Low
Diffusion-weighted signal intensity	High	Low[a]	Low[a]	High	Low
Apparent diffusion coefficient signal intensity	Low-intermediate	Low[a]	Low[a]	Low-intermediate	Low
T2* Gradient signal intensity	Intermediate	Low	Low	Intermediate	Low

FLAIR, fluid-attenuated inversion recovery.
[a] T2 blackout effect.