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Quantitative Susceptibility Mapping of Intracerebral Hemorrhages at Various Stages

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Abstract

Purpose—To investigate the magnetic susceptibility of intracerebral hemorrhages (ICH) at various stages by applying quantitative susceptibility mapping (QSM).

Materials and Methods—Blood susceptibility was measured serially using QSM after venous blood withdrawal from healthy subjects. Forty-two patients who provided written consent were recruited in this institutional review board approved study. Gradient echo MRI data of the 42 patients (17 females; 64±12 yrs) with ICH were processed with QSM. The susceptibilities of various blood products within hematomas were measured on QSM.

Results—Blood susceptibility continually increased and reached a plateau 96 hours after venous blood withdrawal. Hematomas at all stages were consistently hyperintense on QSM. Susceptibility was 0.57 ± 0.48 , 1.30 ± 0.33 , 1.14 ± 0.46 , 0.40 ± 0.13 , and 0.71 ± 0.31 parts per million (ppm) for hyperacute, acute, early subacute, late subacute and chronic stages of hematomas respectively. The susceptibility decrease from early subacute (1.14ppm) to late subacute (0.4ppm) was significant ($p < 0.01$).

Conclusion—QSM reveals positive susceptibility in hyperacute hematomas, indicating that even at their hyperacute stage, deoxyhemoglobin may exist throughout the hematoma volume, not just at its rim as seen on conventional T2* imaging. QSM also reveals reduction of susceptibility from early subacute to late subacute ICH, suggesting that methemoglobin concentration decreases at the late subacute stage.

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Keywords

Magnetic Susceptibility; QSM; Intracerebral Hemorrhage; CT

Introduction

The blood degradation process in intracerebral hemorrhage (ICH) has been characterized qualitatively but not quantitatively on MRI using standard T1 weighted (T1w), T2 weighted (T2w), and T2* weighted gradient echo (T2*w) imaging (1–3). It is generally assumed that blood degradation products in intracerebral hematomas are composed primarily of oxyhemoglobin (oxyHb) in the hyperacute stage (<24h), deoxyhemoglobin (deoxyHb) in the acute stage (1–3 days), intracellular methemoglobin (metHb) in the early subacute stage (3–7 days), extracellular metHb in the late subacute stage (7–14 days), and hemosiderin in the chronic stage (>14 days). During blood degradation, the molar magnetic susceptibility monotonically increases, from slightly negative relative to CSF in oxyHb to highly positive in deoxyHb, metHb, and hemosiderin.

Quantitative Susceptibility Mapping (QSM) has recently been applied to quantify cerebral microbleed burden (4), hematoma volume (5), blood leakage in cerebral cavernous malformation (6,7), and to differentiate hemorrhages from calcifications (8,9). Because QSM directly quantifies the magnetic susceptibility of tissue, it provides an opportunity to quantitatively characterize hematomas by using MRI, especially at the hyperacute/acute phase where treatment is critically needed. Here, we apply QSM to investigate the magnetic susceptibility of hematomas at various stages.

Materials and Methods

The susceptibility of hematomas was investigated using QSM of blood drawn from healthy subjects and using QSM performed in ICH patients at various stages. All magnetic susceptibility reported in this study were measured in SI units relative the cerebral spinal fluid (CSF) in subjects and relative to water in phantoms.

Blood experiment

The blood experiment was approved by our institutional review board to study the change in blood susceptibility over time following venous blood withdrawal from healthy subjects. A phantom was constructed with 5mL transfer pipets (Thermo Scientific, Waltham, MA) embedded in a plastic container with 1% agar solution to support the blood tube. The same procedure was repeated on five healthy volunteers. For each subject, 5 ml venous blood was drawn and immediately injected into the tube. The plastic container was kept in a 37°C water bath when not being scanned. The first scan started immediately after blood withdrawal. The blood phantom was scanned every hour during the first 8 hours, then at 16 hours, 24 hours, and every day thereafter up to 7 days.

Patients

This study was approved by our institutional review board and written consent was obtained from patients or their families. From September 2010 to December 2012, patients suspected of hemorrhagic strokes were prescribed either MRI or CT and were recruited for this study. After their conditions stabilized, a follow-up CT or MRI scan that was complementary to the initial scan was performed. If the initial scan was an MRI scan and the hematoma was chronic, the follow-up CT scan was sometimes omitted. 42 patients (25 males and 17 females; 41~89 years old, mean $\pm$ standard deviation, 64 ± 12) with hematomas at various stages were scanned consecutively during this time period and included in this study. The interval between CT and MR imaging ranged from less than an hour to 8 days, but only seven of them were longer than 25 hours, resulting in a mean interval of 37 hours and a median of 2 hours. At the time of the initial scan, the symptom duration was less than 24 hours for 4 patients, 1~3 days for 10 patients, 3~7 days for 5 patients, 7~14 days for 11 patients, and more than 14 days for 12 patients. After screening the images, one patient in the >14 days category was excluded due to corrupted image quality in QSM.

Imaging Protocol and Data Reconstruction

Blood experiment—3D multi-echo spoiled gradient echo (SPGR) were acquired on a 3.0 Tesla MR system (Signa HDx, GE, United States) using an 8-channel brain coil. The imaging parameters were: flip angle = 25° , first TE=2.1 ms, echo spacing=2.8 ms, 11 echos, TR=33.4 ms, voxel size= $1 \times 1 \times 1 \text{ mm}^3$, scan time = 4 mins; QSM images were calculated from gradient echo complex images using the morphology enabled dipole inversion method (3,10,11).

Patients—MRI was performed on a 3.0 Tesla MR system (Magnetom Verio, Siemens, Erlangen, Germany) using a 12-channel head coil with the following pulse sequences: (a) T1-weighted (T1w) spin echo (TR = 431ms, TE = 13ms) (b) T2-weighted fluid-attenuated inversion recovery (T2w-FLAIR, TR = 6.4s, TE = 110ms, TI = 2s) or T2-weighted images (T2w, TR = 9s, TE = 100ms), and (c) 3D T2*-weighted spoiled multiecho gradient echo (T2*w). Imaging parameters for the multi-echo GRE sequence were as follows: flip angle, 15° ; repetition time, 58 msec; number of echoes, 8; first echo time, 5.7 msec; echo time spacing, 6.7 msec; pixel bandwidth, 500Hz; field of view, 24 cm; matrix, 384×288 ; section thickness, 2 mm; parallel imaging acceleration factor, 2; and total acquisition time, about 6 minutes. T2*w magnitude images were reconstructed by taking the square root of sum of squares of all the echoes. The QSM was reconstructed from the multiecho GRE data using the morphology enabled dipole inversion method (10–12).

CT was performed using a 64-detector CT scanner (Lightspeed VCT; GE Healthcare, Milwaukee, WI) with the following parameters: 260 mAs, 120 kV, 5mm slice thickness, 1.375:1 pitch, and 512×512 matrix.

Image Analysis

Blood experiment—Regions of interest (ROIs) covering the entire clot were manually drawn and used to measure the mean and standard deviation of susceptibilities at different time points.

Patients—Two neuroradiologists with at least 10 year-experience classified the ages of the hematomas as hyperacute, acute, early subacute, late subacute, and chronic stages, and corresponding blood products, based on characteristic signal intensity patterns on T1w, T2w-FLAIR, T2*w MRI and CT (2,13). A hematoma might have multiple ROIs (Fig. 1). Susceptibility values were measured from QSM on ROIs of the same blood product. Cerebrospinal fluid (CSF) susceptibility values in the lateral ventricles without evidence of hemorrhages were used as the susceptibility reference. For each type of blood product, the mean and standard deviation of the blood products' susceptibility values were recorded.

Statistical Analysis

QSM ROI measurements were grouped based on their hematomas stages and the corresponding blood products in statistical analysis. One-way analysis of variance (ANOVA) was performed to assess if the susceptibility differences among the five stages were significant. One-sample t-tests with a significance level of 5% were performed to assess if measured susceptibility values in each stage were significantly above zero (relative to CSF). Two-sample unpaired t-tests with intracluster correlation adjustments for multiple ROIs within the same patient (14) and a significance level of 5% were performed to assess if the susceptibility changes between two consecutive stages were statistically significant: hyperacute vs acute, acute vs early subacute, early subacute vs late subacute, and late subacute vs chronic.

Results

Blood experiment

Blood coagulated about 9 min after drawing. Hematoma retraction started immediately after coagulation and stabilized within 1–2 hours. Figure 2 shows the time course of blood clot susceptibilities ($n = 5$). Susceptibility monotonically increased from 456 ± 91 ppb (parts per billion) at the start to a plateau of 1509 ± 60 ppb beginning at 96 hours.

Patients

There were 47 hematomas identified in this study: 4 hyperacute, 5 acute, 15 early subacute, 16 late subacute, and 7 chronic. Susceptibility measurements on a total of 78 ROIs were: 0.57 ± 0.48 parts per million (ppm) from 11 ROIs denoting oxyHb mixed with deoxyHb from 11 patients, 1.30 ± 0.33 ppm from 27 ROIs denoting deoxyHb from 26 patients, 1.14 ± 0.46 ppm from 13 ROIs denoting intracellular metHb from 13 patients, 0.40 ± 0.13 ppm from 15 ROIs denoting extracellular metHb from 15 patients, and 0.71 ± 0.31 ppm from 12 ROIs denoting hemosiderin from 10 patients (Fig. 3). All values were significantly greater than zero ($p < 0.01$). The susceptibility differences among the five stages were significant ($p < 0.01$, ANOVA). Figure 4 depicts a typical case of a hyperacute hematoma having positive susceptibility throughout its volume on QSM and hypointensity at its rim on T2w-FLAIR and T2*w. Figure 5 illustrates a typical case of an acute hematoma having a core that is hyperintense on QSM but hypointense on T2w-FLAIR and T2*w, and isointense on T1w. Figure 6 shows typical early and late subacute hematomas, demonstrating a reduction of susceptibility in the late subacute stage.

The susceptibility value at the hyperacute stage (0.57 ppm) was significantly greater than zero ($p < 0.01$), suggesting an oxy-deoxyHb mixture. Compared to hyperacute, a significant increase of 0.73 ppm ($p < 0.05$, $df = 14$, 32 patients with 5 patients have ROIs in both stages) in hematoma susceptibility was found in the acute stage. A statistically insignificant ($p = 0.12$, $df = 18$, 32 patients with 7 patients have ROIs in both stages) decrease in susceptibility of -0.17 ppm was found in early subacute (intracellular methHb) stage when compared to that in the acute stage (deoxyHb). There was an unexpected significant decrease of -0.74 ppm ($p < 0.01$, $df = 14$, 25 patients with 3 patients have ROIs in both stages) in susceptibility from early subacute (intracellular methHB) to late subacute stage (extracellular methHb; Figs. 3 & 6). There was a significant increase of 0.31 ppm ($p < 0.01$, $df = 14$, 23 patients with 2 patients have ROIs in both stages) in susceptibility from late subacute (extracellular methHB) to chronic stage (hemosiderin).

Discussion

Data from this QSM study of intracerebral hematomas of various stages suggest the following two findings. 1) The susceptibility of hematomas are consistently positive relative to CSF even during the hyperacute stage, challenging the traditional model that hyperacute hematomas contain mostly oxygenated arterial blood which has slightly negative susceptibility (15,16). 2) The susceptibilities of hematomas decrease substantially from the acute to subacute stage, thus furthering the MRI literature (1,13) about the late subacute stage with new insight for a quantitative model to explain the hemoglobin degradation process.

In the phantom experiment, the blood susceptibility experienced its most rapid increase (0.070 ± 0.014 ppm/hour) during the first 8 hours. In the patient data, the quantitative measurement of susceptibility at the acute stage showed a striking agreement with the literature (15) and with our theoretical prediction. Assuming deoxyHb is the dominating species in acute hematomas, the measured average susceptibility of 1.30 ppm agreed with the expected value of 1.37 ppm calculated using $Hct \times \chi_{do}$, where the hematocrit value is $Hct = 0.4$ and fully deoxygenated hemoglobin susceptibility is $\chi_{do} = 3.43$ ppm (15). The observed highly positive susceptibility values in hyperacute hematomas also suggest the presence of deoxyHb, potentially methHb in addition to oxygenated blood. Previously, deoxyHb was regarded as only appearing at the periphery of hyperacute hematoma, indicated by a hypointense rim on T2*w images but without histological confirmation (1,4,13,17,18). We saw neither a hyperintense rim nor a hypointense center on QSM images of hyperacute hematomas. QSM utilizes the phase information from all voxels surrounding the susceptibility source that act as “field observers” and not only those occupied by the source. In numerical simulations, it has been shown that MEDI is capable of accurately reconstructing the susceptibility of a source even when the magnitude signal is artificially removed (10). It has also been shown that in patients with calcifications (9) and microbleeds (4), a reconstruction of the susceptibility in those lesions is still possible with the correct sign, which is negative and positive using CSF as the reference, respectively. Our finding suggests that the degradation of oxyHb to deoxyHb in ICH may be faster than expected and may not strictly follow a periphery-central path. Indeed, the hypointense rim on T2*w images may be caused by the local field inhomogeneity at the interface between the

hematoma with highly positive susceptibility and brain tissue with slightly negative susceptibility. This rapid appearance of deoxyHb in a hyperacute hemorrhage may be explained by the ability of hemoglobin to immediately release oxygen into the extravascular space during conditions of low oxygen concentration.

An immediate clinical application of this finding is to identify rebleeding in ICH, which may appear as isointense on T1w and hyperintense on T2w images, a scenario which is visually similar to conditions of edema. The positive susceptibility values in fresh blood allow clear differentiation between rebleeding and edema, which has zero susceptibility.

The susceptibility decrease from acute to late subacute ICH is unexpected. Theoretically, susceptibility should increase from the acute to subacute stage, because iron in deoxyHb changed from Fe^{2+} (four unpaired electrons, 121ppm·L/mol) to Fe^{3+} in metHb (five unpaired electrons, 177ppm·L/mol; (19–21)). In the blood phantom experiment, we observed a monotonically non-decreasing trend. The measured susceptibility reduction from acute to late subacute hematomas reflects a lower concentration of metHb. This finding may indicate the clearance of metHb by macrophages and the dilution of metHb by interstitial fluid in the hematoma due to high water content of the lysed red blood cells and inflammation (influx of macrophages) at the late subacute stage (13). Erythrocytes, Hb after hemolysis and damaged cell debris are removed quickly by microglia/macrophages from the hematoma in immune response to minimize cytotoxic effects (22). As suggested by our findings, ICH in the late subacute stage has substantially reduced metHb and reduced cellular contents, which are consistent with the observed hyperintensities on both T1w and T2w images. The residual metHb is highly mobile and can still cause enhancement of $1/T_1$ and $1/T_2$, markedly shortening T_1 but only marginally shortening T_2 (which is typically much shorter than T_1 in contrast enhanced MRI). The hematoma fluid dilution of metHb if substantial may be detected by longitudinal monitoring of the hematoma volume change during acute and late subacute stages. Assuming that the volume is stabilized in the late subacute stage, our finding suggests active metHb clearance.

The significant increase in susceptibility (0.31ppm) was found in chronic stage ICH relative to that in the late subacute stage. This susceptibility increase might be caused by the degradation of blood products such as small magnetic domain formation in hemosiderin, as well as edema resolution, particularly if the hematoma volume on T2w decreases because this would lead to an increase in iron concentration. The removal of metHb by microglia/macrophages seems to have stopped after the late subacute stage, allowing for the resolution of the remaining metHb into final stable hemosiderin.

Despite the lower inner structure contrast of ICH and longer scan time (6 minutes) when compared to T1 and T2 weighted images, QSM provided quantitative susceptibility measurements of ICH, which have the potential to be converted into concentrations of different blood products using molar susceptibilities of blood iron products (dHb, metHb, etc.). This feature allows for better understanding of ICH pathophysiology and may be a promising monitoring tool in drug trials and in the clinical setting. In addition, since QSM images are calculated from magnitude and phase data that was acquired using a gradient echo (GRE) sequence (from which both T2* weighted and susceptibility weighted images

(SWI) can be generated), QSM does not require the addition of a specialized sequence to the standard ICH MRI protocol.

In this study, a major limitation consists of uneven sampling density over various stages of ICH, especially for the hyperacute stage, and the lack of longitudinally serial imaging. Compared to MR, CT is currently preferred for imaging ICH in the hyperacute stage because of shorter scan time and better availability. After CT scans, patients are often not stable enough for immediate MRI follow ups within 24hr before hyperacute hematomas progress to acute stage. Both of these factors led to small sample size in the hyperacute stage. A second limitation of this study is its cross-sectional design. This design was chosen because it was relatively difficult to get consent for longitudinal MRI follow up after acute ICH. A third limitation is the lack of histological validation. This issue can be addressed through follow up studies of hematoma susceptibility in animal models.

In conclusion, QSM reveals positive susceptibility relative to CSF in hyperacute hematomas, indicating the existence of deoxyhemoglobin even in the hyperacute stage. QSM also reveals a significant reduction of susceptibility from acute to late subacute ICH, suggesting a decrease in methemoglobin concentration in the late subacute stage.

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FIGURE 1.

Illustration of the quantitative susceptibility measurement of various blood products. The hyperintense rim on both T1w (a) and T2w (b) images suggested extracellular methemoglobin at the periphery of the hematoma. The iso-intense core on the T1w image and the corresponding hypointensity on T2w suggested the presence of deoxy-hemoglobin in the center. The upper half of the hematoma showed hyperintensity in both T1w and T2w images, suggesting extracellular methemoglobin. The rest of the hematoma showed hyperintensity on T1w and hypointensity on T2w images, suggesting intracellular methemoglobin. Accordingly, susceptibility values measured in these regions are shown as ROIs on QSM (c).

FIGURE 2.

Susceptibility values of blood clot phantom and corresponding time points, demonstrating an increase in susceptibility in the first 4 days, followed by a plateau.

FIGURE 3.

Susceptibility values of intracerebral hematomas at various stages and corresponding blood products, demonstrating positive susceptibility at the hyperacute stage, followed by a significant increase at the acute stage from hyperacute, a significant decrease in the late subacute stage from early subacute, and an eventual significant increase at the chronic stage.

FIGURE 4.

Hyperacute ICH in a 61 year old male with MRI obtained 6 hours after symptom onset and a follow up CT performed 20 minutes later. a) CT, b)T1w, c)T2w-FLAIR, d)T2*w and e)QSM. T2w Flair (c) and T2*w (d) reveal a hypointense rim and an isointense to hypointense core typical for hyperacute ICH. QSM (e) shows a relatively isointense rim, especially in the medial part of the lesion, which suggests that the hypointense rim seen on T2w Flair and T2*w images may be susceptibility artifacts at the core of the hematoma. The core on QSM is hyperintense suggesting a mixture of oxy and deoxy Hb. This hyperacute ICH shows a high susceptibility value of 1.32ppm.

FIGURE 5.

An acute ICH is shown on a)CT, b)T1w, c)T2w-FLAIR, d)T2*w and e)QSM images. CT (a) shows a hyperdense lesion in the right thalamus, T1w (b) shows an isointense core, T2w-FLAIR (c) and T2*w (d) both show a hypointense core, and QSM shows a hyperintense core.

FIGURE 6.

An early subacute ICH is shown on a)CT, b)T1w, c)T2w-FLAIR, d)T2*w and e)QSM images, demonstrating a hyperintense periphery with an isointense core on T1w (b) but with the appearance of hypointensity on both on T2w-FLAIR (c), T2*w (d) and hyperintense on QSM (e). A late subacute ICH is shown on f) CT, g)T1w, h)T2w-FLAIR, i)T2*w and j)QSM, demonstrating a hyperintense core on all MRI images (g-j). This late subacute ICH has a low susceptibility value of 0.44ppm.