Susceptibility weighted imaging: Clinical applications and future directions

Ahmet Mesrur Halefoglu, David Mark Yousem

Ahmet Mesrur Halefoglu, Department of Radiology, Sisli Hamidiye Etfal Training and Research Hospital, University of Health Sciences, Istanbul 34371, Turkey
David Mark Yousem, Division of Neuroradiology, Department of Radiology, Johns Hopkins Medical Institution, Baltimore, MI 21287, United States
ORCID number: Ahmet Mesrur Halefoglu (0000-0002-2054-3550); David Mark Yousem (0000-0002-1222-6643).

Author contributions: All authors equally contributed to this paper with conception and design of the study, literature review and analysis, drafting, critical revision and editing, and final approval of the final version.

Conflict-of-interest statement: No potential conflicts of interest.

Open-Access: This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/4.0/

Manuscript source: Unsolicited manuscript

Correspondence to: Ahmet Mesrur Halefoglu, MD, Professor, Department of Radiology, Sisli Hamidiye Etfal Training and Research Hospital, University of Health Sciences, Birlik sok, Parksaray ap, No: 17/4, Istanbul 34371, Turkey. halefoglu@hotmail.com
Telephone: +90-212-3735000
Fax: +90-212-2415015

Received: March 17, 2018
Peer-review started: March 17, 2018
First decision: April 4, 2018
Revised: April 8, 2018
Accepted: April 20, 2018
Article in press: April 20, 2018
Published online: April 28, 2018

Abstract

Susceptibility weighted imaging (SWI) is a recently developed magnetic resonance imaging (MRI) technique that is increasingly being used to narrow the differential diagnosis of many neurologic disorders. It exploits the magnetic susceptibility differences of various compounds including deoxygenated blood, blood products, iron and calcium, thus enabling a new source of contrast in MR. In this review, we illustrate its basic clinical applications in neuroimaging. SWI is based on a fully velocity-compensated, high-resolution, three dimensional gradient-echo sequence using magnitude and phase images either separately or in combination with each other, in order to characterize brain tissue. SWI is particularly useful in the setting of trauma and acute neurologic presentations suggestive of stroke, but can also characterize occult low-flow vascular malformations, cerebral microbleeds, intracranial calcifications, neurodegenerative diseases and brain tumors. Furthermore, advanced MRI post-processing technique with quantitative susceptibility mapping, enables detailed anatomical differentiation based on quantification of brain iron from SWI raw data.

Key words: Quantitative susceptibility mapping; Brain; Ischemia; Magnetic resonance imaging; Susceptibility weighted imaging

© The Author(s) 2018. Published by Baishideng Publishing Group Inc. All rights reserved.

Core tip: Susceptibility weighted imaging has a variety of applications in neuroradiology practice and should be included in routine protocols. It can detect micro- and macrohemorrhages and delineate cerebral microvasculature and can also reveal low-flow vascular malformations. It has been proven as a complementary, valuable imaging sequence in the management of stroke patients. It provides differentiation of calcium from hemorrhage in the brain. It plays an important role in the evaluation of traumatic brain injury patients and aids in the characterization and grading of cerebral tumors.
Quantitative susceptibility mapping can be applied on many neurodegenerative disorders by assessing brain iron content.

Halefoglu AM, Yousem DM. Susceptibility weighted imaging: Clinical applications and future directions. World J Radiol 2018; 10(4): 30-45 Available from: URL: http://www.wjgnet.com/1949-8470/full/v10/i4/30.htm DOI: http://dx.doi.org/10.4329/wjr.v10.i4.30

INTRODUCTION

Susceptibility weighted imaging (SWI) is an magnetic resonance imaging (MRI) technique that exploits the magnetic susceptibility differences of various compounds, such as blood, iron, and diamagnetic calcium, thus enabling new sources of MR contrast[1-3].

SWI has been shown to provide clinically useful complementary information to conventional spin-echo MRI sequences. Additionally, through post-processed quantitative susceptibility mapping (QSM) SWI sequences allow data driven research to evaluate compounds that alter the magnetic field of the brain, a strategy most useful in neurodegenerative disorders. In this review, we highlight many clinical applications of SWI in the evaluation and differential diagnosis of diverse central nervous system (CNS) pathologic conditions. We close with an assessment of the future of SWI and QSM.

PRINCIPLES AND TECHNICAL ASPECTS OF SWI

Local magnetic field heterogeneity leading to T2 shortening may be induced by paramagnetic, diamagnetic and ferromagnetic substances and results in signal loss on T2* weighted gradient-echo (GE) sequences (proton relaxation enhancement). The susceptibility effect is most visible in non-refocused GE techniques using long echo times (TE), short flip angles, and high field strengths. Although SWI relies on GE sequences, it has enhanced susceptibility sensitivity compared with conventional T2* weighted GE sequences because it is based on a high-resolution, long TE, flow-compensated, 3D GE imaging technique containing filtered phase information in each voxel[4]. In SWI, the magnitude and phase MR data are brought together and a phase mask is created. Multiplying these with the original magnitude images result in a final magnitude SWI dataset. Both magnitude and phase information are essential for proper tissue characterization, and are brought together to create an SWI image[5]. Finally, these images are further processed with a minimum intensity projection algorithm (minIP) to obtain 3-10 mm thick high signal to noise minIP slabs. These minIP images thereby reveal the continuity of tortuous veins across the slices while attenuating the signal coming from the brain tissue[4]. On magnitude images, the longer TE of the SWI sequence (e.g., 40 ms at 1.5 tesla) compared with conventional GE sequences (TE 25 ms) allows for more phase dispersion and T2 shortening of the protons in the local inhomogenous magnetic field. Thus SWI highlights small changes in susceptibility across a voxel as signal intensity loss. A low flip angle can keep the CSF brighter than the surrounding parenchyma. Consequently, the magnitude image highlights areas with short T2* and leads to lower signal in major veins due to the presence of deoxyhemoglobin[6]. SWI sequence parameters for 1.5 and 3 Tesla Siemens (Erlangen, Germany) and Philips (the Netherlands) magnets are shown in Tables 1-4.

Paramagnetic materials have at least one unpaired electron in the system, but diamagnetic materials have all their electrons paired. Diamagnetic materials are repelled by a magnetic field; an applied magnetic field creates an induced magnetic field in them in the opposite direction, causing a repulsive force. In contrast, paramagnetic and ferromagnetic materials are attracted by a magnetic field. Oxyhemoglobin is diamagnetic in nature, whereas deoxyhemoglobin is paramagnetic. The paramagnetic deoxyhemoglobin serves as an intrinsic contrast agent on SWI sequences, and is low in signal. This causes magnetic field inhomogeneity due to two effects: A reduction of T2* and a phase difference between the vessel and its surrounding tissue. This property also forms the basic principle for blood oxygen level dependent functional and venographic imaging.

Paramagnetic substances display positive phase shift in left-handed MR systems such as the Avanto system of Siemens magnets (Erlangen, Germany). Hence, the phase images are particularly useful for differentiating between paramagnetic susceptibility effects of blood products such as deoxygenated hemoglobin, intracellular methemoglobin, hemosiderin and ferritin (positive shift) and diamagnetic effects of calcium (negative or no shift)[7,8]. Unfortunately, ferrocalcinosis may lead to confusing signal intensity patterns as even basal ganglia “calcification” is often a mixture of paramagnetic iron and diamagnetic calcium. Yamada et al[9] demonstrated that all basal ganglia calcifications show a paramagnetic susceptibility effect, whereas other calcifications located outside the basal ganglia (such as choroid plexus or dural calcifications) exhibit exclusively a diamagnetic susceptibility effect. Iron accumulation in brain not only occurs in aging but is also encountered in diverse neurodegenerative diseases.

SWI sequences have some intrinsic disadvantages. Undesirable magnetic susceptibility sources that cause artifacts occurring at air-tissue interfaces such as the areas adjacent to the temporal bone and sinuses limit investigation of these regions. Also the blooming artifact, a useful sign for detecting sources of field inhomogeneity, may sometimes lead to extreme tissue signal cancellation and loss of anatomical borders[8].
Cerebral amyloid angiopathy

Cerebral amyloid angiopathy (CAA) is a small vessel disease characterized by amyloid β protein deposition within the cerebral arterioles leading to fibrinoid necrosis and vessel fragility. CAA causes microhemorrhages in and around the arteriole vessel wall extending into the parenchyma. CAA is a major cause of primary lobar intracranial hemorrhage and cerebral microhemorrhages in the elderly. CT and routine MRI techniques are usually not able to detect cerebral microbleeds; however, SWI with its unique sensitivity to susceptibility effects, clearly demonstrates lobar and microhemorrhages predominantly in the frontal and parietal cortical and subcortical regions (Figure 1). In contrast, microhemorrhages resulting from hypertensive or atherosclerotic microangiopathy have a predilection of deep gray matter or the infratentorial location (Figure 2). CAA is also manifested by white matter hyperintensities on MRI sequences which may be accompanied clinically by cognitive impairment. Detection of two or more lobar hemorrhages of any duration, high signal intensity changes in the white matter and multiple cerebral microbleeds at the corticomedullary junction are highly suspicious for CAA (10) (Figure 1). Linn et al. have highlighted the presence of hemosiderosis on the pial surface of the brain, likely from the leakage of blood products from repetitive superficial hemorrhages in as much as 35% of CAA cases.

Hypertensive cerebral angiopathy

Hypertensive cerebral angiopathy may also be characterized by multiple silent cerebral microhemorrhages. Unlike CAA, cerebral microhemorrhages associated with chronic systemic hypertension are more commonly found in the thalamus, basal ganglia, cerebellum and pons (10) (Figure 2). These hypertensive cerebral microhemorrhages are a risk factor for development of a subsequent intracerebral macrohematomas/lobar hemorrhages (13). The number of cerebral microhemorrhages corresponds also with blood pressure levels. Such micro- and macrohemorrhages are exquisitely well-demonstrated by SWI particularly with acute deoxyhemoglobin or chronic hemosiderin.

TABLE 1 Susceptibility weighted imaging sequence parameters for 1.5 T Siemens Magnetom Avanto syngo magnet

| Slab group 1 | Slabs | 1 |
| --- | --- | --- |
| Dist. factor | 20% |
| Position | L0.0 A16.0 H37.8 |
| Orientation | T > C-6.9 |
| Phase enc. dir. | R >> L |
| Rotation | 90.00 deg |
| Phase oversampling | 0% |
| Slice oversampling | 23.10% |
| Slices per slab | 104 |
| FoV read | 230 mm |
| FoV phase | 75% |
| Slice thickness | 1.50 mm |
| TR | 28 ms |
| TE | 20.00 ms |
| Averages | 1 |
| Concatenations | 1 |
| Filter | Prescan normalize |
| Matrix size | 256 × 256 |
| TA | 4.44 |
| PAT | 2 |
| Voxel size | 1.0 mm × 0.9 mm × 1.5 mm |
| Flip angle | 15 deg |
| Dimension | 3D |
| Bandwidth | 120 Hz/Px |
| Slice resolution | 100% |
| Coil elements | HE1-4 |

Table 2 Susceptibility weighted imaging sequence parameters for 3 T Siemens Magnetom TrioTim syngo magnet

| Slab group 1 | Slabs | 1 |
| --- | --- | --- |
| Dist. factor | 20% |
| Position | L0.0 A16.0 H37.8 |
| Orientation | T > C-6.9 |
| Phase enc. dir. | R >> L |
| Rotation | 90.00 deg |
| Phase oversampling | 0% |
| Slice oversampling | 23.10% |
| Slices per slab | 104 |
| FoV read | 230 mm |
| FoV phase | 75% |
| Slice thickness | 1.50 mm |
| TR | 28 ms |
| TE | 20.00 ms |
| Averages | 1 |
| Concatenations | 1 |
| Filter | Prescan normalize |
| Matrix size | 256 × 256 |
| TA | 4.44 |
| PAT | 2 |
| Voxel size | 1.0 mm × 0.9 mm × 1.5 mm |
| Flip angle | 15 deg |
| Dimension | 3D |
| Bandwidth | 120 Hz/Px |
| Slice resolution | 100% |
| Coil elements | HEA; HEP; NE1, 2 |

CLINICAL APPLICATIONS

Cerebral amyloid angiopathy

Diffuse axonal injury (DAI) is a type of traumatic brain injury, in which torsional forces generated by rapid acceleration or deceleration of the head cause shearing of axons. Areas most vulnerable to shear injury include the cerebral gray-white matter junction, splenium of the corpus callosum, basal ganglia and dorsolateral brainstem. The extent of the axonal injury has been shown to correlate with a poor prognosis, as do parenchymal hemorrhages. Recent studies have shown that SWI is more sensitive than CT or GE sequences in terms of detecting suspected hemorrhagic DAI. Most DAI patients have small punctate hemorrhages located in the deep subcortical white matter (Figure 3). Tong et al. and Babikian et al. demonstrated that SWI is 3–6 times more sensitive than T2* GE sequences in terms of detecting the number, size, volume and distribution of hemorrhagic lesions seen in DAI cases.
Because of this, the previously held concept of "non-hemorrhagic shearing injury" has largely been debunked. Those formerly "bland lesions" of DAI are now shown to have microbleeds on SWI. Brain stem involvement in DAI patients is also a very important predictor that determines the long-term outcome.[20] Mittal et al.[16] demonstrated that SWI was more helpful in detecting traumatic lesions occurring in the brainstem than any other MRI sequence, and revealed intraventricular and subarachnoid hemorrhage, invisible on CT. Long term hemosiderosis from recurrent traumatic bleeds (which may occur in non-accidental trauma) is also best detected on SWI. Epidural and subdural hematomas can also be demonstrated well on SWI sequences as long as air-bone interfaces do not lead to masking artifacts.

**CNS VASCULAR MALFORMATIONS**

True arteriovascular malformations (AVMs) usually are present at birth and can become large with time. These AVMs are characterized by their high-flow and therefore can usually be detected by conventional MRI/MR angiography techniques. In contrast, low-flow vascular malformations including cerebral cavernous malformations (CCMs), developmental venous anomalies (DVAs) and CaTes (capillary telengiectasias) may be inapparent on fast spin echo (FSE) MRI/MR angiography techniques because they mainly contain slow-flow small vessels. Although T2* weighted GE imaging is capable of detecting small venous structures and hemosiderin deposition in cavernomas and CaTes, the incorporation of the magnitude and phase information in SWI provides improved sensitivity for identifying low-flow vascular malformations that are undetectable on GE sequences.[21,22] Lee et al.[21] showed that SWI is the ideal imaging sequence for screening patients who have a high clinical suspicion of low-flow vascular malformations. These lesions may be responsible for cryptogenic

---

**Table 3** Susceptibility weighted imaging sequence parameters for 1.5 T Philips Achieva magnet

| Parameter       | Value                  |
|-----------------|------------------------|
| FoV read        | 230 mm                 |
| Slice thickness | 5 mm                   |
| Gap             | 0                      |
| TR              | 35 ms                  |
| TE              | 50 ms                  |
| Matrix size     | 256 x 512              |
| TA              | 5.15                   |
| Flip angle      | 15 deg                 |
| Coil elements   | 8 channel SENSE head coil |

**Table 4** Susceptibility weighted imaging sequence parameters for 3 T Philips Achieva magnet

| Parameter       | Value                  |
|-----------------|------------------------|
| FoV read        | 230 mm                 |
| Slice thickness | 5 mm                   |
| Gap             | 0                      |
| TR              | 23 ms                  |
| TE              | 20 ms                  |
| Matrix size     | 256 x 512              |
| TA              | 4.04                   |
| Flip angle      | 10 deg                 |
| Coil elements   | 8 channel SENSE head coil |

---

**Figure 1** A 68-year-old man with cerebral amyloid angiopathy. A: Axial FLAIR image demonstrates periventricular confluent hyperintense regions; B: Axial T1 weighted SE image shows high signal intensity subacute hemorrhage in the left occipital lobe; C and D: On SWI minIP images, hemorrhage is depicted as a hypointense signal intensity lesion and, in addition to the left occipital lobe hemorrhage, one can see multiple microhemorrhagic lesions in the cortical and subcortical white matter from cerebral amyloid angiopathy. SWI: Susceptibility weighted imaging; minIP: Minimum intensity projection algorithm; SE: Spin echo.

**Figure 2** A 45-year-old woman with long standing chronic hypertension. SWI minIP images depicts numerous microhemorrhages in the deep basal ganglia, thalami, and subcortical white matter regions, typical of hypertensive microangiopathy. SWI: Susceptibility weighted imaging.
epilepsy, recurrent subarachnoid hemorrhage and/or hemosiderosis, and hemorrhagic injury to cranial nerves. Because the incidence of hemorrhage from occult cerebrovascular malformations is contingent on whether they have ever bled, using SWI to detect previous bleeding helps prognosticate on future risk[23].

CCMs are composed of abnormally enlarged capillary cavities surrounded by a single layer of endothelium without intervening with brain parenchyma and comprise 10%-20% of all cerebrovascular malformations[24]. The MRI findings of CCMs are variable, depending on the presence of calcification and hemorrhage within the lesions, but they typically show a mixed signal intensity, usually recognized as “popcorn-like” with a central reticulated core surrounded by a peripheral rim of hemosiderin[15] (Figure 4). Since recurrent microhemorrhages occur in the CCM lesions, they may contain deoxyhemoglobin acutely or hemosiderin chronically, both dark on SWI[25,26] (Figure 5). Symptomatic, growing and recurrently hemorrhagic CCMs are considered for surgical resection.

DVAs are the most common type of cerebral vascular malformations (≤60%) and are often discovered incidentally during routine MRI examinations[27]. A DVA consists of radially arranged venous structures converging to a centrally located venous trunk, which drains normal brain parenchyma[28]. They are mainly asymptomatic lesions, do not often bleed, and neurosurgical intervention is largely contraindicated due to the risk of venous infarction[29]. However, they have a high association with other vascular malformations, especially CCMs. SWI better shows the collector (head of Medusa) and deep medullary veins (snake hair of Medusa) than T1W MR image. The bilateral subdural hematomas are nearly as dark as the cortical bone; F: Phase contrast SWI image, hemorrhagic lesions show a bright/positive shift effect on phase image, due to paramagnetic susceptibility effect. SWI: Susceptibility weighted imaging; miniIP: Minimum intensity projection algorithm; SE: Spin echo.

Figure 3 A 37-year-old man who had an accident was in coma after traumatic brain injury. A: On the non-contrast CT image, bilateral frontal subcortical and right basal ganglia hyperdense hemorrhagic foci with surrounding hypodense edema are seen consistent with diffuse axonal injury. Also left parieto-temporal subdural hemorrhage is present. Post-op changes are present on the right with a tiny frontal subdural hematoma; B: SE T1W image, can only reveal hyperintense right basal ganglia hemorrhagic lesion with surrounding hypointense edema and left subdural hemorrhage, but can not demonstrate the other parenchymal lesions; C: Diffusion weighted image (DWI) reveals hyperintense caudate lesions; D: Apparent diffusion coefficients (ADC) map demonstrates restricted diffusion within the lesions; E: SWI miniIP image, clearly depicts multiple frontal cortical and subcortical and also right basal ganglia microhemorrhages better than those of CT and T1W MR image. The bilateral subdural hematomas are nearly as dark as the cortical bone; F: Phase contrast SWI image, hemorrhagic lesions show a bright/positive shift effect on phase image, due to paramagnetic susceptibility effect. SWI: Susceptibility weighted imaging; miniIP: Minimum intensity projection algorithm; SE: Spin echo.
venous collaterals. SWI has been found to be superior to post gadolinium-enhanced T1WI in characterizing calcification, abnormal periventricular and transmedullary veins, cortical gyriform hypointensities and gray-white matter abnormalities (Figure 8)\[22\], but enhanced T1W images depict the leptomeningial angiomatosis and enlarged choroid plexus more clearly.

Cerebral venous sinus thrombosis and venous infarction

Patients with cerebral venous sinus thrombosis (CVST) may present with headaches or non-specific signs due to increased intracranial pressure (ICP). Remaining undiagnosed, it can become deadly if it progresses to malignant increased ICP\[32\]. An acute CVST shows deoxygenated hemoglobin in the involved veins, seen as prominent hypointense signal intensity areas with “blooming” artifact on SWI (Figure 9). SWI may demonstrate engorgement of the venous system as an early sign of CVST and can also show the associated parenchymal hemorrhage which occurs in 73% of venous infarctions\[33\].

ARTERIAL STROKE

Acute cerebral infarct with or without hemorrhage occurs due to thromboembolism or atherosclerotic stenosis of a vessel. Vascular occlusion causes a susceptibility change
by decreasing the arterial flow, and increases pooling of deoxygenated blood, thus leading to a high concentration of deoxyhemoglobin\(^{[34]}\). In the setting of acute stroke, such conversion to deoxyhemoglobin can occur as early as 2 h after the onset of symptoms.

In the setting of stroke, SWI assists in identifying:
1. Hemorrhages within the infarct region, thus enabling the differentiation of a hemorrhagic from a bland ischemic stroke. Many studies have also proven that SWI is more sensitive in revealing hemorrhage within the acute infarct regions than CT and 2D GE T2* weighted sequences\(^{[35,36]}\). SWI can also detect acute subarachnoid hemorrhage and is very sensitive to subacute and chronic subarachnoid hemorrhages, sometimes missed by CT and FLAIR\(^{[3]}\) (Figure 10); (2) Prominent hypointense draining veins within areas of impaired perfusion (Figure 11). The visualization of these prominent veins allows for the identification of diffusion-perfusion mismatch representing perumbral brain tissue in a different fashion than current perfusion weighted imaging techniques\(^{[16]}\). The oxygen extraction fraction (OEF) which reflects the ratio of deoxyhemoglobin to

---

### Figure 7
A 65-year-old man with incidentally discovered capillary telangiectasia in the pons. A: Axial FSE T2W image shows a hyperintense lesion located in the central pons; B: Axial contrast-enhanced T1W image reveals very little contrast enhancement in the lesion; C: SWI image demonstrates a markedly hypointense lesion in the pons indicating a capillary telangiectasia based on its location and size. SWI: Susceptibility weighted imaging; FSE: Fast spin echo.

### Figure 8
A 3-year-old girl with Sturge-Weber syndrome. A: Non-contrast CT image shows hyperdense tram-track calcifications along the left frontal gyri; B: Axial MIP TOF MRA shows a normal cranial angiogram; C: Axial SWI minIP image, hypointense gyral calcification is clearly depicted, also deep abnormal transmedullary veins are visible; D: SWI phase image confirms these calcifications as low signal intensity areas. SWI: Susceptibility weighted imaging; minIP: Minimum intensity projection algorithm.
oxyhemoglobin in the capillaries and veins, is significantly increased in the penumbra following occlusion of the artery. This high OEF in cortical veins is presumably responsible for the increased conspicuity in the infarct region[30,36]; (3) Acute intra-arterial thrombus: The susceptibility vessel sign (SVS) is defined as the presence of hypointensity from acute deoxyhemoglobin thrombus within the intracranial arteries in which the diameter of the hypointense vessel exceeds the contralateral vessel diameter[27,39] (Figures 12 and 13). Lingegowda et al[40] found an 82% sensitivity and 100% specificity for the SVS in the determination of all acute major intracranial occlusions. They also showed that SVS is more sensitive and specific than the hyperdense artery sign on CT[40,41] and the hyperintense vessel sign on FLAIR images for intracranial artery occlusions. Huang et al[42] found that patients with negative prominent veins and positive susceptibility vessel sign exhibited poor outcomes; and (4) Hemorrhagic transformation of acute ischemic infarction: Approximately 20%-40% of patients bleed
within the first week after a stroke\textsuperscript{43} (Figure 14). Old microhemorrhages in a stroke patient may presage the vulnerability of the vascular system\textsuperscript{44}. In patients with a small number of microhemorrhages (< 5), thrombolytic therapy can be applied safely, whereas patients with large numbers of microhemorrhages (> 5) have a great risk for potential hemorrhagic transformation from thrombolytic therapy\textsuperscript{45-49}. Huang et al\textsuperscript{42} showed that microhemorrhages were significantly associated with later hemorrhagic transformation. SWI is able to detect microhemorrhages within the infarct region more accurately than T2* weighted GE sequences.

**NEURODEGENERATIVE DISEASES**

It is widely accepted that iron deposition in the brain increases with normal aging, particularly in the basal ganglia region primarily in the form of ferritin and ferrocalcinosis (Figure 15). Increased iron levels in the CNS are encountered in a variety of neurodegenerative
diseases, superimposed on the normal senescent iron increase\[50,51\] in the globus pallidum, substantia nigra, red nucleus, subthalamic nucleus and dentate nucleus. Increased iron deposition is found in Parkinson’s disease, Huntington’s disease, Alzheimer’s disease, Multiple sclerosis (MS), Amyotrophic lateral sclerosis, Hallervorden-Spatz syndrome, Wilson’s Disease (copper) and Pantothenate kinase-associated neurodegeneration (PKAN)\[52\] (Table 5). In neurodegenerative diseases, the ability to measure the amount of ferritin in the brain may help predict prognosis, disease progression and treatment outcomes.

SWI shows differences in stable and progressive MCI\[57\]. Fourteen percent of controls, 33% of patients with stable MCI and 54% of those with progressive MCI had microhemorrhages. Furthermore, the iron content in the right pallidum and right substantia nigra was greater in progressive MCI than stable MCI.

Barnaure et al\[58\] studied 328 cognitively normal control subjects and 71 patients with MCI using SWI on a 3T magnet to investigate the presence and distribution of cerebral microbleeds. They found no difference be-
Between the two groups in terms of cerebral microbleed prevalence, distribution and severity. The patients’ cognitive decline over an 18 mo period did not correlate with microbleeds. They concluded that microbleeds do not predict cognitive decline in advanced age.

MS affects both brain and spinal cord and is typically imaged with FLAIR and contrast-enhanced T1W images. The sensitivity of MRI in depicting MS lesions in the brain is demonstrated to be high, but its specificity remains low. SWI helps by revealing the perivenular distribution of the demyelinating lesions by showing the MS plaque surrounding the small veins [59]. This has been used to distinguish MS from SAPHO (synovitis, acne, pustulosis, hyperostosis and osteitis) syndrome white matter lesions.

Rudko et al [61] have shown that the levels of iron deposition in patients with MS correlates better with disability than MS plaque volume. They have also shown increased iron content in patients with clinically isolated syndrome (CIS).

### BRAIN TUMORS

SWI can help in the grading of cerebral tumors because it provides identification of both hemorrhagic and calcified foci inside the tumors and also allows assessment of the detailed internal angioarchitecture of the tumors. High grade tumors like glioblastomas usually contain a hemorrhagic component (Figure 16) [3,62-64].

Sehgal et al [65] demonstrated that in the majority of cases, SWI was equivalent to T1W contrast-enhanced images in the grading of gliomas. The criteria used for this comparison were tumor visibility, boundaries, edema, vessels, blood products, internal architecture and image quality. Mittal et al [66] have showed that high rCBV values on PWI and high choline-creatine ratios on MR spectroscopy found in tumors exhibit a good correlation with evidence of blood products demonstrated within the tumor using SWI.

In assessing brain tumors, calcification is considered as a very important indicator. Calcification is diamagnetic, whereas hemorrhage is paramagnetic, therefore resulting in opposite signal intensities on SWI phase images [66]. For the determination of calcification in brain tumors such as oligodendrogliomas, Zulfikar et al [67] found that adding SWI sequences led to a statistically significant improvement in the sensitivity for the detection of intratumoral calcification by 53% (from 33% to 86%) but no change in specificity.

SWI can also be used to distinguish vestibular schwannomas from cerebellopontine angle meningiomas.

| Entity                        | Increased iron location                           | Ref. |
|-------------------------------|--------------------------------------------------|------|
| Parkinson’s disease           | SN, pars compacta, brainstem                      | [53] |
| Alzheimer’s disease           | Hippocampus, GP                                  | [54] |
| PKAN                          | GP, SN sparing DN                                | [55] |
| Infantile neuroaxonal dystrophy | GP, SN, DN                                | [55] |
| Neuroferritinopathy           | GP, P, DN with cavitation                         | [55] |
| Acrularplasminemia            | BC, thalami with no cavitation                    | [55] |
| Huntington’s disease          | CN, P                                            | [56] |
| Progressive MCI versus stable MCI | Right GP, SN                                   | [57] |
| Multiple sclerosis            | CN, P and thalamic pulvinar                       | [60] |

CN: Caudate nucleus; GP: Globus Pallidus; P: Putamen; BG: Basal Ganglia; SN: Substantia Nigra; DN: Dentate nucleus; PKAN: Pantothenate kinase-associated neurodegeneration.
Microhemorrhages occur with schwannomas, not found in the meningiomas\(^\text{68}\) (Figure 17). The dual rim sign (hypointense inner, hyperintense outer) from the respiratory burst of bacteria converting hemoglobin to methemoglobin has been shown to differentiate abscesses (present) from glioblastoma (absent) in the face of a ring enhancing mass\(^\text{69}\).

QSM

QSM is a recently developed sophisticated postprocessing technique and numerically solves the inverse source-effect problem to quantify local tissue magnetic susceptibility from the major magnetic field distribution, reflected in the phase images of SWI\(^\text{70}\). The mapping of iron can play a crucial role in the setting of many important neurologic disorders. In early Parkinson’s disease (PD), iron elevation in the specific brain regions including the substantia nigra is a pathognomonic feature of the disease and this can be measured by QSM\(^\text{71}\). He et al\(^\text{72}\) evaluated 44 early PD patients; susceptibility values within the substantia nigra and red nucleus contralateral to the most affected limb were elevated compared to a healthy control group. Bilateral substantia nigra magnetic susceptibility showed a positive correlation with disease duration\(^\text{72}\).

Acosta-Cabrero et al\(^\text{73}\) examined 66 patients with idiopathic PD and found increased R2\(^*\) and susceptibility values in the substantia nigra, red nucleus, thalamus and globus pallidus. QSM additionally correlated with disease severity in these patients.

Moon et al\(^\text{74}\) investigated the presence and pattern of brain iron accumulation in vascular dementia (VaD) and Alzheimer’s disease (AD) patients by means of QSM. Both in VaD and AD patients significantly higher susceptibility values were found in the caudate nucleus, putamen and thalamus compared to normal control subjects. Age and cognitive disease severity of both patient groups were not correlated with increased iron accumulation in their basal ganglia (Figure 18).

Dominguez et al\(^\text{75}\) measured iron accumulation in the basal ganglia in both premanifest and symptomatic Huntington’s disease (HD) patients with QSM. Both groups of patients demonstrated substantially elevated iron content in the caudate nucleus, globus pallidus and putamen compared to normal control subjects. In-
creased iron levels showed significant correlation with disease severity (Figure 19). QSM is also capable of quantifying elevated iron levels in the motor cortex of amyotrophic lateral sclerosis patients. Furthermore, in Wilson’s disease, iron accumulation and quantification can be demonstrated by QSM\(^{[71]}\). Iron chelation therapy in PD can be monitored by QSM\(^{[71]}\). Walsh et al\(^{[76]}\) have shown that the deficits of patients with MS more strongly correlate with QSM values of brain iron content than the MS plaque volume (Figure 20).

**CONCLUSION**

SWI is a very useful imaging tool with a variety of ap-

---

Figure 18 Example orthogonal views of quantitative susceptibility mapping images of a 41-year-old female premanifest HD patient showing the basal ganglia where increased tissue magnetic susceptibility can be observed in iron-rich gray matter structures such as the caudate nuclei and putamen. Extra iron overload in the striatum in these patients as compared to age-matched controls is believed to be associated with HD pathophysiology. Gray scale is in [-0.2, 0.2] ppm.

Figure 19 Example quantitative susceptibility mapping image of a 55-year-old female relapsing-remitting MS patient, shows hyperintense susceptibility MS lesions, and the corresponding FLAIR, T1 and R2* images. Multiple contrast analysis using R2* and QSM may be helpful identifying different pathological changes in MS lesions. Gray scale is [-0.12, 0.12] ppm in QSM image and [0, 80] Hz in R2* image. MS data was acquired using similar 7T protocol as the HD study. QSM: Quantitative susceptibility mapping.

Figure 20 Increased iron accumulation of caudate and putamen is noted in patients with Alzheimer’s disease and vascular dementia as compared with normal subjects.
plications in neuroradiology practice and should be included in routine protocols. As demonstrated, it is very helpful in detecting micro-and macro-hemorrhages and delineating cerebral microvasculature and low-flow vascular malformations. It is regarded as a complementary, valuable imaging sequence in the management of stroke patients. It facilitates differentiation of calcium from hemorrhage in the brain. It is helpful in the evaluation of traumatic brain injury patients and aids in the characterization and grading of cerebral tumors. QSM can shed light on many neurodegenerative disorders in a more rigorous statistical way by assessing brain iron content. Further investigations are needed for expanding the roles of SWI and QSM in neuroradiology clinical and research arenas.

ACKNOWLEDGMENTS

Our thanks to Xu Li, PhD, from Johns Hopkins Medical Institution, Radiology and Radiological Sciences, Baltimore, Maryland, United States, for his support in providing the QSM part figures (18 and 19) of our article. We are also grateful to the Korean Journal of Radiology for their permission of Figure 20.

REFERENCES

1. Reichenbach JR, Venkatesan R, Schilling DJ, Kido DK, Haake EM. Small vessels in the human brain: MR venography with deoxyhemoglobin as an intrinsic contrast agent. Radiology 1997; 204: 272-277 [PMID: 9205259 DOI: 10.1148/radiology.204.1.9205259]
2. Reichenbach JR, Haake EM. High-resolution BOLD venographic imaging: a window into brain function. NMR Biomed 2001; 14: 453-467 [PMID: 11746938 DOI: 10.1002/nbm.722]
3. Thomas B, Somasundaram S, Thamburaj K, Kesavadas C, Gupta AK, Bodhey NK, Kapilamoorthy TR. Clinical applications of susceptibility weighted MR imaging of the brain - a pictorial review. Neuroradiology 2008; 50: 105-116 [PMID: 17929005 DOI: 10.1007/s00234-007-0316-z]
4. Gasparotti R, Pinelli L, Liserre R. New MR sequences in daily practice: susceptibility weighted imaging. A pictorial essay. Insights Imaging 2011; 2: 335-347 [PMID: 22347957 DOI: 10.1007/s13244-011-0086-3]
5. Haake EM, Xu Y, Cheng YC, Reichenbach JR. Susceptibility weighted imaging (SWI). Magn Reson Med 2004; 52: 612-618 [PMID: 15334582 DOI: 10.1002/mrm.20198]
6. Haake EM, Mittal S, Wu Z, Neelavalli J, Cheng YC. Susceptibility-weighted imaging: technical aspects and clinical applications, part 1. AJNR Am J Neuroradiol 2009; 30: 19-30 [PMID: 19039041 DOI: 10.3174/ajnr.A1400]
7. Rauscher A, Sedlack J, Barth M, Mentzel HJ, Reichenbach JR. Magnetic susceptibility-weighted MR phase imaging of the human brain. AJNR Am J Neuroradiol 2005; 26: 736-742 [PMID: 15814914]
8. Yamada N, Imakita S, Sakuma T, Takamiya M. Intracranial calcification on gradient-echo phase image: depiction of diamagnetic susceptibility. Radiology 1996; 198: 171-178 [PMID: 8539737 DOI: 10.1148/radiology.198.1.8539737]
9. Greenberg SM, Eng JA, Ning M, Smith EE, Rosand J. Hemorrhage burden predicts recurrent intracerebral hemorrhage after lobar hemorrhage. Stroke 2004; 35: 1415-1420 [PMID: 15073385 DOI: 10.1161/01.STR.0000126807.69758.0e]
10. Bitstein MK, Tung GA. MRI of cerebral microhemorrhages. AJR Am J Roentgenol 2007; 189: 720-725 [PMID: 17715222 DOI: 10.2214/AJR.07.2249]
11. Smith EE, Gurrol ME, Eng JA, Engel CR, Nguyen TN, Rosand J, Greenberg SM. White matter lesions, cognition, and recurrent hemorrhage in lobar intracerebral hemorrhage. Neurology 2004; 63: 1606-1612 [PMID: 15534243 DOI: 10.1212/01. WNL.0000142966.22886.20]
12. Linn J, Halpin A, Damerau P, Ruhland J, Giese AD, Dichgans M, van Buchem MA, Bruckmann H, Greenberg SM. Prevalence of superficial siderosis in patients with cerebral amyloid angiopathy. Neurology 2010; 74: 1346-1350 [PMID: 20421578 DOI: 10.1212/ WNL.0b013e3181d6d05]
13. Tsushima Y, Tanizaki Y, Aoki J, Endo K. MR detection of microhemorrhages in neurologically healthy adults. Neuroradiology 2002; 44: 31-36 [PMID: 11942497 DOI: 10.1007/s002340066649]
14. Medana IM, Esiri MM. Axonal damage: a key predictor of outcome in human CNS diseases. Brain 2003; 126: 515-530 [PMID: 12566274 DOI: 10.1093/brain/awg061]
15. Schaefer PW, Huisman TA, Sorensen AG, Gonzalez RG, Schwamm LH. Diffusion-weighted MR imaging in closed head injury: higher correlation with initial Glasgow coma scale score and score on modified Rankin scale at discharge. Radiology 2004; 233: 58-66 [PMID: 15304663 DOI: 10.1148/radiol.2332031173]
16. Mittal S, Wu Z, Neelavalli J, Haake EM. Susceptibility-weighted imaging: technical aspects and clinical applications, part 2. AJNR Am J Neuroradiol 2009; 30: 232-252 [PMID: 19131406 DOI: 10.3174/ajnr.A1461]
17. Tong KA, Ashwal S, Holshouser BA, Shutter LA, Herigault G, Haake EM, Kido DK. Hemorrhagic shearing lesions in children and adolescents with posttraumatic diffuse axonal injury: improved detection and initial results. Radiology 2003; 227: 332-339 [PMID: 12732694 DOI: 10.1148/radiology.227202176]
18. Tong KA, Ashwal S, Holshouser BA, Nickerson JP, Wall CJ, Shutter LA, Osterdock RJ, Haake EM, Kido D. Diffuse axonal injury in children: clinical correlation with hemorrhagic lesions. Ann Neurol 2004; 56: 36-50 [PMID: 15236400 DOI: 10.1002/ana.20123]
19. Babikian T, Freier MC, Tong KA, Nickerson JP, Wall CJ, Holshouser BA, Burley T, Riggs ML, Ashwal S. Susceptibility weighted imaging: neuropsychologic outcome and pediatric head injury. Pediatr Neurol 2005; 33: 184-194 [PMID: 16139733 DOI: 10.1016/j.peditneurol.2005.03.015]
20. Mannion RJ, Cross J, Bradley P, Coles JP, Chatfield D, Carpenter A, Pickard JD, Menon DK, Hutchinson PJ. Mechanism-based MRI classification of traumatic brainstem injury and its relationship to outcome. J Neurotrauma 2007; 24: 128-135 [PMID: 17263676 DOI: 10.1089/neu.2006.0127]
21. Lee BC, Vo KD, Kido DK, Mukherjee P, Reichenbach J, Lin W, Yoon MS, Haakec M. MR high-resolution blood oxygenation level-dependent venography of occult (low-flow) vascular lesions. AJNR Am J Neuroradiol 1999; 20: 1239-1242 [PMID: 10472978]
22. Barnes SR, Haakec EM. Susceptibility-weighted imaging: clinical angiographic applications. Magn Reson Imaging Clin N Am 2009; 17: 47-61 [PMID: 19364599 DOI: 10.1016/j.mric.2008.12.002]
23. Voumès DM, Flamm ES, Grossman RI. Comparison of MR imaging with clinical history in the identification of hemorrhage in patients with cerebral arteriovenous malformations. AJNR Am J Neuroradiol 1989; 10: 1151-1154 [PMID: 2512776]
24. Lehnhardt FG, von Smeak UL, Rückriem B, Stenzel W, Neveling M, Heiss WD, Jacobs AH. Value of gradient-echo magnetic resonance imaging in the diagnosis of familial cerebral cavernous malformation. Arch Neurol 2005; 62: 653-658 [PMID: 15824268 DOI: 10.1001/archneur.62.6.653]
25. Cooper AD, Campagne NG, Meissner I. Susceptibility-weighted imaging in familial cerebral cavernous malformations. Neurology 2008; 71: 382 [PMID: 18663188 DOI: 10.1212/01.WNL.0000319659.86629.e8]
26. Abla A, Wait SD, Uschold T, Lekovic GP, Spetzler RF. Developmental venous anomaly, cavernous malformation, and capillary telangiectasia: spectrum of a single disease. Acta Neurochir (Wien)
Halefoglu AM et al. SWI of brain diseases

2008; 150: 487-489; discussion 489 [PMID: 18351283 DOI: 10.1007/s00701-008-1570-5]
27 Abe T, Singer RJ, Marks MP, Norbash AM, Crowley RS, Steinberg GK. Coexistence of occult vascular malformations and developmental venous anomalies in the central nervous system: MR evaluation. AJNR Am J Neuroradiol 1998; 19: 51-57 [PMID: 9432157]
28 Töpper R, Jürgens E, Reul J, Thron A. Clinical significance of intracranial developmental venous anomalies. J Neurol Neurosurg Psychiatry 1999; 67: 234-238 [PMID: 10407000 DOI: 10.1136/jnnp.67.2.234]
29 Tong KA, Ashwal S, Obenaus A, Nickerson JR, Kido D, Haacke EM. Susceptibility-weighted MR imaging: a review of clinical applications in children. AJNR Am J Neuroradiol 2008; 29: 9-17 [PMID: 17925363 DOI: 10.3174/ajnr.A0786]
30 Comi AM. Update on Sturge-Weber syndrome: diagnosis, treatment, quantitative measures, and controversies. Lymphat Res Biol 2007; 5: 257-264 [PMID: 18370916 DOI: 10.1089/lrb.2007.1016]
31 Hinman JM, Provenzano JM. Hypointense thombo on T2-weighted MR imaging: a potential pitfall in the diagnosis of dural sinus thrombosis. Eur J Radiol 2002; 41: 147-152 [PMID: 11809544 DOI: 10.1016/S0720-048X(01)00365-5]
32 Mammen EF. Pathogenesis of venous thrombosis. Chest 1992; 102: 640-644 [DOI: 10.1378/chest.102.6_Supp 640S]
33 Khandelwal N, Agarwal A, Kochhar R, Babaraj SR, Singh P, Prabhakar S, Suri S. Comparison of CT venography with MR venography in cerebral sinovenous thrombosis. AJR Am J Roentgenol 2006; 187: 1637-1643 [PMID: 17114562 DOI: 10.2214/AJR.05.1249]
34 Viallon M, Altrichter S, Pereira VM, Nguyen D, Sekoranja L, Federspiel A, Kuelzer S, Stajzel R, Ouread R, Bonvin C, Pfeuffer J, Löblad KO. Combined use of pulsed arterial spin-labeling and susceptibility-weighted imaging technique compared to computed tomography: a retrospective study. J Magn Reson Imaging 2004; 20: 372-377 [PMID: 15352242 DOI: 10.1002/jmri.20130]
35 von Kummer R. MRI: the new gold standard for detecting brain hemorrhage? Stroke 2002; 33: 1748-1749 [PMID: 12105345 DOI: 10.1161/01.STR.0000019882.06696.D6]
36 Wintermark M, Sanelli PC, Albers GW, Bello JA, Derdeyn CP, Hiett SW, Johnson MH, Kidwell CS, Lev MH, Liebeskind DS, Rowley HA, Schaefer PW, Sunshine JL, Zaharchuk G, Meltzer CC; American Society of Neuroradiology; American College of Radiology; Society of Neurointerventional Surgery. Imaging recommendations for acute stroke and transient ischemic attack patients: a joint statement by the American Society of Neuroradiology, the American College of Radiology and the Society of Neurointerventional Surgery. J Am Coll Radiol 2013; 10: 828-832 [PMID: 23948676 DOI: 10.1016/j.jacr.2013.06.019]
37 Haacke EM, Cheng NY, House MJ, Liu Q, Neelavalli J, Ogg RJ, Khan A, Ayaz M, Kirsch W, Obenaus A. Imaging iron stores in the brain using magnetic resonance imaging. Magn Reson Imaging 2005; 23: 1-25 [PMID: 15733784 DOI: 10.1016/j.mri.2004.10.001]
38 Hecht MJ, Mellin C, Schmid A, Neundörfer B, Fellner FA. Cortical T2 signal shortening in amyotrophic lateral sclerosis is not due to iron deposits. Neurology 2005; 47: 805-808 [PMID: 16175348 DOI: 10.1007/s00234-005-1421-5]
39 Martin WR, Wieler M, Gee M. Midbrain iron content in early Parkinson disease: a potential biomarker of disease status. Neurology 2008; 70: 1411-1417 [PMID: 18172063 DOI: 10.1212/01.wnl.0000286384.31050.b5]
40 Schenck JF, Zimmerman EA, Li Z, Adak S, Saha A, Tandon R, Fish KM, Belden C, Gillen RW, Barba A, Henderson DL, Neil W, O’Keefe T. High-field magnetic resonance imaging of brain iron in Alzheimer disease. Top Magn Reson Imaging 2006; 17: 41-50 [PMID: 17198986 DOI: 10.1097/01.mri.0000435399.02747.42]
41 McNeill A, Birchall D, Hayflick SJ, Gregory A, Schenck JF, Zimmerman EA, Shang H, Miyajima H, Chinnery PF. T2* and FSE MRI distinguishes four subtypes of neurodegeneration with brain iron accumulation. Neurology 2006; 70: 1614-1619 [PMID: 18443312 DOI: 10.1212/01.wnl.0000313998.40011.d6]
42 van Bergen JM, Hua J, Unschuld PG, Lim IA, Jones CK, Margolis RL, Ross CA, van Zijl PC, Li X. Quantitative Susceptibility Mapping Suggests Altered Brain Iron in Premanifest Huntington
Disease. AJNR Am J Neuroradiol 2016; 37: 789-796 [PMID: 26680466 DOI: 10.3174/ajnr.A4617]

57 Haller S, Bartisch A, Nguyen D, Rodriguez C, Emch J, Gold G, Løvblad KO, Giannakopoulos P. Cerebral microhemorrhage and iron deposition in mild cognitive impairment: susceptibility-weighted MR imaging assessment. Radiology 2010; 257: 764-773 [PMID: 20923870 DOI: 10.1148/radiol.10100612]

58 Barnaure I, Montandon ML, Rodriguez C, Herrmann F, Løvblad KO, Giannakopoulos P, Haller S. Clinicoradiologic Correlations of Cerebral Microbleeds in Advanced Age. AJNR Am J Neuroradiol 2017; 38: 39-45 [PMID: 27686485 DOI: 10.3174/ajnr.A4956]

59 Tan HL, van Schijndel RA, Pouwels PJ, van Walderveen MA, Reichenbach JR, Manoliu RA, Barkhof F. MR venography of multiple sclerosis. AJNR Am J Neuroradiol 2000; 21: 1039-1042 [PMID: 10871010]

60 Haacke EM, Ayaz M, Khan A, Manova ES, Krishnamurthy B, Gollapalli P, Ciulla C, Kim I, Petersen F, Kirsch W. Establishing a baseline phase behavior in magnetic resonance imaging to determine normal vs. abnormal iron content in the brain. J Magn Reson Imaging 2005; 21: 545-549 [PMID: 15930584 DOI: 10.1002/jmri.20356]

61 Rudko DA, Solovey I, Gati JS, Kremenchutzky M, Menon RS. Multiple sclerosis: improved identification of disease-relevant changes in gray and white matter by using susceptibility-based MR imaging. Radiology 2014; 272: 851-864 [PMID: 24828000 DOI: 10.1148/radiol.14132475]

62 Rauscher A, Sedlacik J, Fitzek C, Walter B, Hochstetter A, Kalff R, Kaiser WA, Reichenbach JR. High resolution susceptibility weighted MR-imaging of brain tumors during the application of a gaseous agent. Rofo 2005; 177: 1065-1069 [PMID: 16021537 DOI: 10.1055/s-2005-858428]

63 Sehgal V, Delproposto Z, Haacke EM, Tong KA, Wyciffe N, Kido DK, Xu Y, Neelavalli J, Haddar D, Reichenbach JR. Clinical applications of neuroimaging with susceptibility-weighted imaging. J Magn Reson Imaging 2005; 22: 439-450 [PMID: 16163700 DOI: 10.1002/jmri.20404]

64 Hammond KE, Lupo JM, Xu D, Metcalf M, Kelley DA, Pelletier D, Chang SM, Mukherjee P, Vigneron DB, Nelson SJ. Development of a robust method for generating 7.0 T multichannel phase images of the brain with application to normal volunteers and patients with neurologic diseases. Neuroimage 2008; 39: 1682-1692 [PMID: 18096412 DOI: 10.1016/j.neuroimage.2007.10.037]

65 Sehgal V, Delproposto Z, Haddar D, Haacke EM, Sloan AE, Zamorano LJ, Barger G, Hu J, Xu Y, Prabhakaran KP, Elangovan JR, Neelavalli J, Reichenbach JR. Susceptibility-weighted imaging to visualize blood products and improve tumor contrast in the study of brain masses. J Magn Reson Imaging 2006; 24: 41-51 [PMID: 16755540 DOI: 10.1002/jmri.20598]

66 Wu Z, Mittal S, Kish K, Yu Y, Hu J, Haacke EM. Identification of calcification with MRI using susceptibility-weighted imaging: a case study. J Magn Reson Imaging 2009; 29: 177-182 [PMID: 19097156 DOI: 10.1002/jmri.21617]

67 Zulliqar M, Dumrongpisutikul N, Intraprionkul J, Yousem DM. Detection of intratumoral calcification in oligodendrogliomas by susceptibility-weighted MR imaging. AJNR Am J Neuroradiol 2012; 33: 858-864 [PMID: 22268093 DOI: 10.3174/ajnr.A2862]

68 Thamburaj K, Radhakrishnan VV, Thomas B, Nair S, Menon RS. Intratumoral microhemorrhages on T2*-weighted gradient-echo imaging helps differentiate vestibular schwannoma from meningioma. AJNR Am J Neuroradiol 2008; 29: 552-557 [PMID: 18079187 DOI: 10.3174/ajnr.A0887]

69 Toh CH, Wei KC, Chang CN, Hsu PW, Wong HF, Ng SH, Castillo M, Lin CP. Differentiation of pyogenic brain abscesses from necrotic globlastomas with use of susceptibility-weighted imaging. AJNR Am J Neuroradiol 2013; 33: 1534-1538 [PMID: 22422181 DOI: 10.3174/ajnr.A2968]

70 Reichenbach JR, Schweser F, Serres B, Deistung A. Quantitative Susceptibility Mapping: Concepts and Applications. Clin Neuroradiol 2015; 25 Suppl 2: 225-230 [PMID: 26198880 DOI: 10.1007/s00062-015-0432-9]

71 Eskreis-Winkler S, Zhang Y, Zhang J, Liu Z, Dimow A, Gupta A, Wang Y. The clinical utility of QSM: disease diagnosis, medical management, and surgical planning. NMR Biomed 2017; 30: [PMID: 27906255 DOI: 10.1002/nbm.3668]

72 He N, Ling H, Ding B, Huang J, Zhang Y, Zhang Z, Liu C, Chen K, Yan F. Region-specific disturbed iron distribution in early idiopathic Parkinson's disease measured by quantitative susceptibility mapping. Hum Brain Mapp 2015; 36: 4407-4420 [PMID: 26429218 DOI: 10.1002/hbm.22928]

73 Acosta-Cabrero J, Williams GB, Cardenas-Blanco A, Arnold RJ, Lupson V, Nestor PJ. In vivo quantitative susceptibility mapping (QSM) in Alzheimer's disease. PLoS One 2013; 8: e81093 [PMID: 24278382 DOI: 10.1371/journal.pone.0081093]

74 Moon Y, Han SH, Moon WJ. Patterns of Brain Iron Accumulation in Vascular Dementia and Alzheimer's Dementia Using Quantitative Susceptibility Mapping Imaging. J Alzheimers Dis 2016; 51: 737-745 [PMID: 26890777 DOI: 10.3233/JAD-151037]

75 Domínguez JF, Ng AC, Poudel G, Stout JC, Churchyard A, Chua P, Egan GF, Georgiou-Karistianis N. Iron accumulation in the basal ganglia in Huntington's disease: cross-sectional data from the IMAGE-HD study. J Neurol Neurosurg Psychiatry 2016; 87: 545-549 [PMID: 25952334 DOI: 10.1136/jnnp-2014-310183]

76 Walsh AJ, Blevins G, Lebel RM, Seres P, Emery DJ, Wilman AH. Longitudinal MR imaging of iron in multiple sclerosis: an imaging marker of disease. Radiology 2014; 270: 186-196 [PMID: 23925273 DOI: 10.1148/radiol.13130474]
