Susceptibility Weighted Imaging: Physics and Clinical applications in Neuroimaging at 3 Tesla

Award: Cum Laude
Poster No.: C-1472
Congress: ECR 2014
Type: Educational Exhibit
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Keywords: Ischemia / Infarction, Hemorrhage, Arteriovenous malformations, Technical aspects, Imaging sequences, MR, Image manipulation / Reconstruction, Neuroradiology brain, MR physics, CNS
DOI: 10.1594/ecr2014/C-1472

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Learning objectives

Susceptibility-weighted imaging (SWI) is a high-spatial-resolution 3D gradient-echo MR imaging technique to enhance contrast by accentuating the paramagnetic properties of blood product.

The aim of this pictorial review is to outline the basic physics of Susceptibility weighted imaging and to describe different applications of Susceptibility-weighted imaging in neuroimaging.

Background

Susceptibility weighted imaging (SWI), originally called BOLD venographic imaging is an imaging technique developed by Haacke et al in 2004. SWI is a fully velocity compensated high-resolution 3D gradient-echo sequence that uses magnitude and filtered-phase information to create a different form of contrast.

This technique exploits the susceptibility differences in tissues such that signal from substances with different susceptibilities than their neighbouring tissues (such as venous blood or haemorrhage) will become out of phase. This special data acquisition and image processing produces an enhanced contrast magnitude image which is exquisitely sensitive to venous blood, haemorrhage and iron storage. SWI offers the potential for:

- better diagnosis of disease and effect on patient management and prognosis.
- better follow up for longitudinal studies
- to be involved in clinical research

SWI has been found to provide additional clinically useful information that is often complementary to conventional MR imaging sequences.

Various applications of SWI:

1. Small hemorrhagic shearing lesions in DAI are only visible by using SWI.
2. More sensitive in detecting hemorrhage inside acute infarct and identifying the microangiopathic changes in Cerebral amyloid angiopathy (CAA), CADASIL and cerebral fat embolism.
4. Distinguishing hemorrhage from tumoral veins if SWI is used both before and after administration of a contrast agent.
5. Venous connectivity and evidence of iron deposition in Multiple sclerosis lesions.
6. Differentiating calcification from hemorrhage.
7. Increased iron deposition as in Parkinson disease, Multiple sclerosis lesions and hallervorden-spatz syndrome.
8. Depicting the small veins of the brain, phase Images of dentate Nucleus and the midbrain giving images analogous to the anatomical sections.

Findings and procedure details

Basic Physics of Susceptibility-weighted imaging (SWI)

Compounds which have paramagnetic, diamagnetic and ferromagnetic properties all interact with the local magnetic field distorting it and thus altering the phase of local tissue which in turn results in loss of signal. Paramagnetic compounds include deoxyhaemoglobin, ferritin and hemosiderin and diamagnetic compounds include bone minerals and dystrophic calcifications.

This new method exploits the susceptibility differences between tissues. SWI uses a fully velocity compensated, three dimensional, rf spoiled, high-resolution, 3D gradient echo scan. Signal from substances with different susceptibilities than their neighbouring tissues (such as venous blood, or haemorrhage) can manifest contrast in following ways:

First, tissues with different susceptibilities can be differentiated on phase filtered SWI images alone.

second, the signal from objects smaller than a voxel like veins will be less(hypointense) than that of its neighbour. This forms a magnitude image which is exquisitely sensitive to venous blood, haemorrhage and iron storage. Larger signal-intensity loss occurs than the actual T2* of the tissue because the signal intensity from the vein can cancel the signal intensity from the gray matter or white matter causing a complete loss of signal intensity at the appropriate TE.
third; phase and magnitude can be combined to absorb both effects.

The term "SWI" implies full use of magnitude and phase information and is not simply a T2* imaging approach.

SWI is a unique combination of the following features (Fig. 1 and 2):

- High resolution 3D gradient echo imaging
- Full flow compensation in all three spatial directions to reduce signal losses of blood due to flow-induced dephasing, leaving us with the desired susceptibility-induced phase.
- Thin slices to remove low-spatial frequency components of the background field.
- Filtering of phase images to reduce unwanted field effects.
- Creating a phase mask image.
- Multiplying the magnitude image with the phase mask.
- Taking a minimum intensity projection over neighbouring slices.
- Processed SWI data

Interpreting SWI Data

There are 3 components to interpret in the SWI data to make a clinical diagnosis

- Original magnitude image
- Phase image.
- Final processed SWI

Magnitude image

Two essential points when interpreting the magnitude data

1. No large blooming effects are seen because the resolution is high.
2. Clearly reveals areas with either short T2* or an oscillatory signal intensity in the magnitude image caused by the presence of deoxyhemoglobin in the major veins.

On magnitude images the veins have a bright signal intensity inside with a hypointense ring around it. Nevertheless, following phase-mask multiplication the whole vein appears as a hypointense region in the processed SWI image.
Phase image

First let us understand the Left- and Right-handed coordinate systems (Fig. 3).

Positions in space can be represented in either a left- or right-handed coordinate system, where one system is a mirror image of the other. In a right-handed, the first dimension (often referred to as the x-direction) increases from left to right, the second dimension goes from posterior to anterior (back to front) and the third dimension increases from inferior to superior (bottom to top).

For a right-handed system, veins will look dark on the phase image (negative phase) because the deoxygenated blood is paramagnetic relative to its surrounding tissue and calcium will look bright (positive phase) because calcium is diamagnetic relative to the brain tissue. The reverse is true for a left handed system.

Final processed SWI

This takes full advantage of T2* signal-intensity losses in the magnitude image and iron increases in the phase images to highlight both types of contrast in a single image.

Clinical Applications of Susceptibility-weighted imaging in Neuroimaging

1. Trauma in Adults and Children

Diffuse Axonal Injury (Fig. 4, 5 and 6)

The detection of micro-haemorrhages, shearing and diffuse axonal injury (DAI) in trauma patients is difficult on conventional imaging. SWI is particularly helpful for the evaluation of diffuse axonal injury (DAI) associated with punctate haemorrhages in the deep subcortical white matter, corpus callosum and brainstem.

SWI is 3-6 times more sensitive than conventional T2*-weighted gradient-echo (T2*GE) sequences in detecting the size, number, volume, and distribution of haemorrhagic lesions in DAI. The number and volume of SWI haemorrhagic lesions correlate with specific neuropsychological deficits. The involvement of the brainstem in TBI is a very important predictor of long term outcome.
Intra-ventricular and Subarachnoid Haemorrhage (Fig. 7 and 8)

SWI also shows intra-ventricular haemorrhage and subarachnoid haemorrhage, sometimes even better than CT. By following the resolution of haemorrhage in the brain, along with spectroscopic imaging, it may be possible to better monitor the internal improvement of trauma induced coma patients.

2. Stroke

Diffusion weighted imaging offers a powerful means to detect acute stroke. Although it is well known that gradient echo imaging can detect haemorrhage, it is best detected with SWI. SWI is a complementary sequence and provides additional information by the following:

- **Detecting a haemorrhagic component within the region of infarction:** The presence of haemorrhage in acute infarct is one of the contraindications of thrombolytic therapy. SWI has higher sensitivity than CT and GRE in the detection of haemorrhage in acute ischemic infarct (Fig.9).

- **Demonstrating hypoperfusion by prominent draining veins and directing the necessity of Perfusion imaging (Fig. 10):** Uncoupling between oxygen supply and demand in hypoperfused tissue causes a relative increase of deoxyhemoglobin levels and a decrease of oxyhemoglobin in the tissue capillaries and the draining veins. As deoxyhemoglobin is a paramagnetic substance that induces inhomogeneity of the local magnetic field, it will result in signal reduction within the draining veins on SWI.

SWI shows prominent hypointense signals in the draining veins within areas of impaired perfusion. The areas with more prominent veins on SWI are in excellent agreement with the abnormal area on the MTT map. So we can combine DWI and SWI to predict the "mismatch" of diffusion-perfusion.

- **Detecting acute thrombus in occluded arteries:** In acute stroke, the demonstration of arterial clot with an accurate determination of its location helps to direct thrombolytic treatment. Acute thrombus contains deoxyhemoglobin therefore, acute arterial thrombosis can be identified on SWI images by what is known as the susceptibility sign. This comprises of a focal hypointense signal in the vessel and apparent enlargement of its diameter, due to blooming, related to its paramagnetic properties (Fig. 11,12 and 13).
• Predicting the probability of potential haemorrhagic transformation: SWI can also detect microhemorrhages, which is a risk factor of haemorrhagic transformation with thrombolytic therapy in acute ischemic stroke.

• Detecting hemorrhagic complication after intraarterial thrombolysis: SWI is sensitive to deoxyhemoglobin and can detect haemorrhagic transformation after an acute infarct.

3. Brain Tumors

SWI allows improved contrast and detection of both the venous vasculature and haemorrhage within tumors, which cannot be seen with conventional imaging methods.

Detection of Haemorrhage in Tumors with SWI (Fig. 16)

High-grade tumors such as glioblastomas often have a haemorrhagic component, which may be useful for staging. High rCBV values on perfusion imaging and high choline-creatine ratios on MR spectroscopy in tumors is seen in most of the tumors with haemorrhage detected within the tumor matrix on SWI (Fig. 14).

SWI also shows haemorrhage within metastatic lesions. Many cerebral parenchymal metastatic lesions, including those from renal cell carcinoma, melanoma, and bronchogenic carcinoma, frequently show intratumoral hemorrhage.

Identifying Tumor Boundaries with SWI (Fig. 14)

SWI clearly shows not only the boundary of the tumor but also different parts of the tumor and hemorrhage inside the tumor.

Intratumoral Calcification (Fig. 15)

Calcification is a very important indicator in diagnosis and differential diagnosis of brain tumors. Calcification cannot be definitively identified by conventional MR imaging because its signals are variable on spin-echo T1- or T2- weighted images and cannot be differentiated from hemorrhage by gradient images because both will appear hypointense. However, phase images can help differentiate calcification from haemorrhage because calcification is diamagnetic, whereas haemorrhage is paramagnetic, resulting in opposite signal intensities on SWI phase images.
4. Occult Vascular Disease such as Cavernomas and Venous Angiomas

Cavernoma (Fig. 17, 18)

The MR imaging findings of cavernomas is variable, depending on the presence of haemorrhage and calcifications, but lesions typically appear as mixed-signal intensities, often described as "popcorn-like" in appearance, with a central reticulated core surrounded by a peripheral hypointense rim representing deposition of hemosiderin.

Sensitivity of SWI in identifying the number of lesions is significantly higher than that of T2-weighted fast spin-echo (FSE) and GRE sequences. Also, because of its high degree of sensitivity, SWI can help confirm the diagnosis of cavernous malformations in patients presenting with cerebral haemorrhage.

Venous Angioma (Developmental Venous Anomaly)

SWI can demonstrate conspicuously both numerous deep medullary veins and collector veins that are hardly visible on T2-weighted images or proton-attenuation images. SWI allows better visualization of DVAs without requiring contrast media (Fig. 19,20).

5. Sturge Weber Syndrome (Fig. 21,22)

SWI is superior to T1-Gadolinium enhanced MRI in characterizing abnormal transmedullary veins, abnormal periventricular veins, cortical gyriform hypointensities. The enlarged regional transmedullary veins have elevated deoxyhemoglobin concentration due to venous stasis and reduced perfusion.

SWI demonstrates presence of abnormally enlarged veins in, which are thought to provide collateral drainage for venous blood toward the deep cerebral venous system and can become increasingly prominent as surface veins occlude progressively. The ability to show a redistribution of the venous system by SWI could assist in making decisions about therapeutic intervention.

The location and pattern of the gyriform hypointense lesions in the cortex and the hypointensities at the junction of gray/white matter on SWI images are consistent with the pattern and location of CT findings of calcifications.
6. Cerebral Venous Sinus Thrombosis (Fig. 23,24)

SWI has become a useful method to evaluate CVST by demonstrating venous stasis and collateral slow flow. **Dural sinus thrombosis causes an increase of deoxyhemoglobin concentration in the involved veins. This appears as a prominent hypointense signal intensity on SWI.**

If the radiologist had missed the direct signs of the thrombosis itself in T1- or T2-weighted imaging, the engorged veins can alert the radiologist to look for the cause. Venous hypertension can be detected at the early stage of CVST before infarction or haemorrhage occurs. Thus, SWI can assist in directing the radiologist to look for CVST while the patient is in the acute stage.

Thrombosed sinuses have deoxyhemoglobin, which can be readily detected on SWI by the presence of hypointensity and blooming. **Venous infarcts are frequently hemorrhagic and SWI assists in detecting even small hemorrhages in venous infarcts.**

7. Mineralization, Calcification and Haemorrhage in the Basal Ganglia (Fig. 25, 26 and 27)

The standard method to evaluate mineralization in the basal ganglia has been using Computed Tomography (CT). Now SWI offers a more sensitive means to pick up abnormalities in iron and calcium in these regions.

8. Vascular Dementia and Cerebral Amyloid Angiopathy (CAA) Fig. 27, 28

CAA typically results in micro haemorrhages in and around the arteriole vessel wall. Lobar microbleeds are related to CAA (usually involving the cortex and subcortical white matter within the frontal and parietal lobes), whereas micro haemorrhages in a deep or infratentorial location typically result from hypertensive or atherosclerotic microangiopathy. Conventional gradient-echo imaging is a well-established technique for detecting cerebral micro haemorrhages. However, **25% of patients with CAA did not have evidence of microbleeds on conventional T2*GE images. SWI is a more sensitive diagnostic tool, which may permit more precise assessment of the natural history of CAA and response to therapy.**

9. Cerebral Fat Embolism (Fig. 29)
Fat embolism syndrome (FES) is generally associated with displaced long-bone fractures of the lower extremities, which occur in 0.5-3.5% of cases. Fat emboli reach the brain through a right-to-left cardiac shunt. The clinical features of FES include respiratory insufficiency, neurologic symptoms, and a petechial skin rash, confusion to coma and seizures.

Though MRI findings are definite in a given clinical setting they alone are not specific and can be observed in diffuse axonal injury (DAI), and demyelination. In FES symptoms usually develop 1-3 days after the event.

FLAIR and conventional T2W sequences show multiple diffuse foci of hyperintensity in white matter, subcortical, periventricular, and centrum semiovale regions. In the majority of cases restricted diffusion foci giving characteristic "star field pattern" are seen in the centrum semiovale.

Susceptibility-weighted imaging (SWI) reveals several punctate foci of low signal intensity representing the micro haemorrhages in the bilateral cerebral and cerebellar white matter, predominantly in the corticomedullary junction and splenium of the corpus callosum.

10. Neurodegenerative Disorders

Iron content in the brain increases with age, particularly in the basal ganglia, and that abnormal levels of iron in the central nervous system (CNS) are seen in various neurodegenerative diseases. Increased iron deposition occurs in Parkinson disease, Huntington disease, Alzheimer disease, multiple sclerosis and Hallervorden-Spatz syndrome. Currently, phase measured from filtered MR images, is the most sensitive tool for measuring relative changes in iron content in the subcortical structures of the brain.

Hallervorden-spatz disease (Fig. 30)

Hallervorden-Spatz syndrome, now known as pantothenate kinase-associated neurodegeneration (PKAN), is an autosomal recessive disorder causing involuntary spasticity and progressive dementia. It is a subset of neurodegeneration with brain iron accumulation (NBIA).

MRI findings on T2-weighted MRI images often demonstrate hyperintense changes in anteriomedial part of globus pallidus and hypodense lateral part of globus pallidus and
pars reticulata of substantia nigra. **SWI / T2* : shows susceptibility artefact (low signal with blooming) in corresponding areas from iron deposition.** The "eye of the tiger" sign refers to a central T2 relatively hyperintense spot within the hypointense globi pallidi.

**Parkinson disease (Fig. 31)**

**Lateral substantia nigra pars compacta** abnormalities are seen in early Parkinson disease which shows **increased iron deposition.**

**Multiple Sclerosis (Fig. 32, 33)**

SWI enables precise in vivo visualization of the venous architecture of the CNS and can help improve our understanding of the pathophysiology by showing typical periventricular distribution of MS lesions.

**Chronic MS lesions may show evidence of iron deposition, which may be better detected by SWI than by conventional GRE.**

SWI can show the perivenular distribution of the demyelinating lesions in multiple sclerosis. This is useful in large tumefactive demyelinating lesions that mimic tumors.

SWI appears to add new perspectives to imaging MS. Specifically, SWI sees lesions:

- connected by vessels
- with dark centers
- not seen with FLAIR
- with iron deposition
- of the gray matter

A **significantly reduced visualization of periventricular white matter venous vasculature has been found in patients with MS compared to control subjects.** These findings of decreased visibility of veins in periventricular white matter in MS is
a result of decreased oxygen utilization in the chronic diseased tissue state of MS and suggests there are to decreased levels of venous deoxyhemoglobin or, conversely, higher levels of oxygenated venous haemoglobin.

Alzheimer's Disease

It is thought that iron around beta amyloid plaque or other sources of iron may be an indication of the early development of Alzheimer's disease. Abnormal iron deposition is seen in the motor cortex in early Alzheimer's disease.

11. Infections

Neurocysticercosis (Fig. 34)

The SWI phase image associated with the SWI magnitude image shows a 1-to-1 correspondence with the calcifications in the CT scan. the scolex is readily detected on phase images compared to conventional sequences.

Fungal Cerebritis (Fig. 35)

Haemorrhagic cerebritis secondary to aspergillus in immunocompromised patients can be seen. The angioinvasive nature of the aspergillus can cause haemorrhages and infarctions. The haemorrhage is well seen on susceptibility weighted images.

12. Ruptured Intracranial Dermoid (Fig. 36)

SWI can show blooming of the fat containing lesion and the disseminated fat droplets in ruptured intracranial dermoid. Blooming of fat on the susceptibility-weighted images remains speculative. The causes of blooming on SWI images are not well-established in literature. Chemical shift artifact as a cause of blooming has been suggested.

13. Radiation-Induced Changes (Fig. 37)

SWI also appears to be useful by showing haemorrhage at the treatment sites or radiation induced telangiectasia.
14. Effect of Oxygen on visibility of veins (Fig. 38)

Breathing normal air has a better visualization of the veins than breathing oxygen because of increase in venous blood oxygenation which leads to a reduction of field inhomogeneities around the vessels. As a result the contrast between veins and surrounding parenchyma is reduced.

15. Anatomical

Phase Images of the Mid-Brain and dentate nucleus (Fig. 39, 40)

Increase iron deposition is seen in neurodegenerative diseases. The iron deposition may correspond to hemosiderin from the ongoing presence of micro-hemorrhages in the aging process. The anatomy of the midbrain and dentate nucleus is also well visualised compared to the conventional MR sequences analogous to the anatomical sections.

Labeling the Small Veins of the Brain Small Vessel Imaging (Fig. 41)

With SWI one can visualize veins on the order of a few hundred microns with 1 mm3 resolution and image vessels smaller than a voxel, as small as deep white matter vessels. Using 3.0T imaging time can be reduced or resolution increased to create images competitive to anatomical brain images.

Images for this section:
Fig. 1: Sequence of events in 3D long TE gradient echo MR imaging with flow compensation in all three directions. In addition to the conventional first-order gradient moment nulling in the read (GR) and section (slice) select (GS) directions, the phase encoding (GP) and partition encoding gradients are also flow compensated with respect to the echo. After sampling the data points, these gradients are rewound, whereas the readout gradient remains to ensure dephasing of the spins in this direction.
**Fig. 2:** Steps of image reconstruction in susceptibility weighted imaging. (Concept of Image-Matsushita T et al Acta Med Okayama. 2008;62:159-68)

**Fig. 3:** Left- and right-handed coordinate systems. The left- (and right-) hand systems are so called because they can be derived using the left (and right) hand, with the thumb, first and second finger representing the x-, y- and z-axes respectively.
**Fig. 4:** Diffuse Axonal Injury. Axial nonenhanced CT scan showing subtle hypodensity in the splenium on right side. Axial FLAIR image shows hyperintense lesions in the splenium and right parietal region. Corresponding SWI image showing punctate and linear hemorrhages in the splenium. Associated left subdural hemorrhage is also seen.

**Fig. 5:** Diffuse Axonal Injury. Same Patient as in Fig. 4. Axial nonenhanced CT scan showing no significant lesion. Axial FLAIR image shows hyperintensity in the dorsal pons and right temporal lobe. Corresponding SWI images showing multiple punctate hemorrhages in the dorsal pons, midbrain and the right temporal lobe. There are more lesions on SWI and they are also larger than those shown on FLAIR sequence. The shape of hemorrhagic lesions also reflects shearing effect indicating axonal injury.
Fig. 6: Diffuse Axonal Injury. Same patient as in Fig. 4 and 5. SWI shows more lesions than on nonenhanced CT and MRI. SWI images showing multiple haemorrhages in the subcortical fronto-parietal white matter, body of corpus callosum, right cerebellar
hemisphere and right temporal lobe. In addition intraventricular haemorrhage is also seen which was not seen on nonenhanced CT. Left subdural haemorrhage is noted.

**Fig. 7:** Intraventricular haemorrhage. Nonenhanced CT and T1 Weighted images shows no intraventricular haemorrhage. The SWI image clearly show hemorrhage layering within the occipital horns of both the lateral ventricles.

**Fig. 8:** Subarachnoid haemorrhage. CT scan shows high-attenuation within the sulci of left parietal region indicating traumatic subarachnoid haemorrhage. T1 Weighted image shows corresponding sulcal hyperintensity. SWI clearly shows haemorrhage within the left parietal sulci with a sharper contrast in agreement with CT and T1 Weighted images.
**Fig. 9:** Haemorrhage within Infarct. Axial DWI, FLAIR, T1 Weighted and SWI images showing acute infarct in the right temporal lobe. No obvious haemorrhage seen on T1W images with multifocal haemorrhages clearly seen as blooming on SWI.
**Fig. 10:** Hypoperfusion in acute infarct. DWI, ADC and T1 Weighted images (top row). DWI/ADC images show acute infarct in right corona radiata. T1 Weighted images
showing intra-arterial hyperintensity in the sylvian branches of right MCA. MRA, SWI images (middle row). MRA shows lack of time-of-flight signal intensity in the distal M2 segment and cortical branches of the right MCA. SWI images show hypointense signal with blooming in the right M2 segment, representing the acute thrombus. SWI also demonstrates prominently hypointense cortical veins within the right MCA territory, suggesting relatively increased deoxyhemoglobin in the draining veins within the acutely ischemic region. CBF,CBV,MTT maps (lower row). The CBF is moderately reduced with small area of reduced CBV representing large penumbra. The MTT map shows delayed transit times in the right MCA territory, matching the draining veins on SWI.

**Fig. 11:** Susceptibility Sign. Acute infarct in the right parietal region. No obvious thrombosed cortical vessel is seen on axial T1 and T2 W images(above row) On SWI images focal hypointense signal is seen in the right MCA and its cortical branches with apparent enlargement of its diameter due to blooming representing the acute thrombus. Corresponding coronal CT angiography image shows abrupt cut off in the right MCA representing thrombosed vessel.
**Fig. 12:** Susceptibility sign in Left Posterior Cerebral Artery. Acute thrombus seen on SWI/PHASE images with corresponding MR angiography TOF image showing loss of flow related enhancement in the left PCA.
Fig. 13: Basilar Artery Thrombosis. Multiple acute infarcts in the posterior circulation involving the bilateral Posterior cerebral artery (PCA), superior and anterior inferior cerebellar arteries (SCA, AICA). Acute thrombus of basilar artery seen as blooming dot.
on SWI image in the prepontine cistern. Corresponding CT image showing the basilar artery hyperdensity.

**Fig. 14:** Glioblastoma Multiforme. T1W, T2W, DWI, T1 Contrast enhanced, rCBV map, SWI images. Heterogenous intra-axial mass in left frontal region with diffusion restriction and multiple haemorrhages seen along its margins. The rim enhancement corresponds well with the areas of high perfusion on rCBV and haemorrhages on SWI. High grade tumors usually have more haemorrhages. The tumors margin on SWI is in good agreement with postcontrast and rCBV maps.
**Fig. 15:** Intratumoral Calcification. Case of Anaplastic Oligodendroglioma Left Thalamus. Axial T2W, TIW, SWI, PHASE and CT images. The calcification is well seen on SWI/PHASE compared to conventional MRI sequences and can be differentiated from haemorrhage (calcification is black on SWI and PHASE images in left handed system as here.)
Fig. 16: Intratumoral Haemorrhage. Right cerebellopontine angle schwannoma with multiple intralesional haemorrhages.
**Fig. 17:** Giant Cavernoma with Superficial siderosis. MRI shows giant cavernoma in the right caudate head showing blood degradation products in different stages. Marked blooming of the lesion is seen on susceptibility weighted images.

**Fig. 18:** Giant Cavernoma Right Caudate Nucleus with Superficial siderosis. (Same patient as in Fig. 16) Diffuse cortical gyriform hypointensity seen on the susceptibility weighted images in both cerebral and cerebellar hemispheres likely secondary to previous subarachnoid bleed from cavernous malformation. This is not seen in the conventional MR images. In addition two more small cavernnomas are seen in bilateral medial temporal lobes on SWI not seen on the conventional MR sequences.
Fig. 19: Developmental Venous Anomaly (Venous Angioma). T1 and T2 Weighted axial images. Leash of vessels with collector vein seen in the left cerebellar hemisphere.

Fig. 20: Developmental Venous Anomaly (Venous Angioma). Contast T1W axial, MIP and SWI images. Excellent details of venous angioma seen on the MIP and SWI images compared to the conventional MR sequences.
Fig. 21: Sturge Weber Syndrome. Axial CT, T1W, T2W AND SWI images. The gyriform calcification and the enlarged deep medullary veins are more prominently seen on SWI images.
**Fig. 22:** Sturge Weber Syndrome. Axial CT, T1W,T2W AND SWI images. The gyriform calcification and the enlarged deep medullary veins are more prominently seen on SWI images.
Fig. 23: Cerebral Venous Thrombosis. Child with leukemia. Axial T1W, T2W, Coronal T2W and Postcontrast coronal mprage. Early subacute thrombus is seen in left
transverse and sigmoid sinus. No obvious haemorrhages or infarct on conventional images is seen.
**Fig. 24:** Cerebral Venous Thrombosis. Child with leukemia. Axial T1W, T2W and SWI images. No obvious haemorrhages seen on conventional images. SWI show engorged deep venous system with multifocal parenchymal haemorrhages.

**Fig. 25:** Basal Ganglia Mineralisation. Phase and SWI images. Iron deposition seen in the globus pallidus as a normal ageing process.

**Fig. 26:** Basal Ganglia Calcification. SWI, Phase and CT images.
Fig. 27: Chronic Hyertensive Bleeds. Axial T2W, SWI iamges. Multiple haemorrhages are seen only on the SWI images in basal ganglia and pons. SWI can show effects in
the order of a few cubic millimeters detecting multiple microbleeds not seen on routine MR imaging..

**Fig. 28:** Cerebral Amyloid Angiopathy. Extensive confluent FLAIR hyperintense areas in bilateral supratentorial white matter with extensive subcortical microhemorrhages seen on SWI.
Fig. 29: Cerebral Fat Embolism. Axial FLAIR and SWI images. Multiple small foci of signal alteration seen in supratentorial white matter, basal ganglia, splenium of corpus callosum, posterior limb of internal capsules and thalami bilaterally. On SWI image, extensive tiny hemorrhages are seen diffusely in these regions. X ray shows fracture of the right femur. The differential diagnosis includes DAI. History of fracture femur with onset of symptoms 72 hours after trauma supports Cerebral fat embolism.
**Fig. 30:** Hallervorden-spatz disease. Axial T2W, T1W, SWI and Phase images. Symmetrical areas of abnormal hypointensity in the globus pallidi on FLAIR / T2W images due to abnormal mineral (iron) deposition, which blooms on susceptibility weighted images.
Fig. 31: Parkinson Disease with Progressive Supranuclear Palsy. Increased iron deposition in the substantia nigra more prominently seen on SWI images than T2W
images. Atrophy of the midbrain tegmentum and superior portion of tectum on sagittal T1-weighted images giving an appearance of a "hummingbird" or "penguin silhouette" sign (see inset image). On the axial T1-weighted images concavity of lateral margins of the midbrain tegmentum is also seen - Morning glory sign classically seen in Progressive supranuclear palsy (PSP).

Fig. 32: Multiple Sclerosis. Axial T2W, MIP and SWI images. The demyelinating plaque(arrow) is hypointense on SWI as an evidence of iron deposition.

Fig. 33: Multiple Sclerosis. Axial T2W and SWI images. The demyelinating plaque(arrow) is hypointense on SWI as an evidence of iron deposition. There is paucity of veins in the territory of lesions possibly representing chronic ischemia.
**Fig. 34:** Neurocysticercosis. Axial T2 Weighted, Phase subset of SWI and nonenhanced CT scan. Oval T2 hyperintense lesion in parasagittal right parietal lobe with subtle eccentric dot hypointensity. Phase subset of the SWI image clearly showing the eccentric scolex appearing as hypointense dot. Corresponding CT image showing the cystic lesion with eccentric dot calcification representing the scolex which is in excellent agreement with the phase images.

**Fig. 35:** Fungal Cerebritis (Aspergillus). Axial T2W, DWI and SWI images. Post liver transplant patient. Large heterogenous hyperintense lesion in left frontal lobe with diffusion restriction and multiple small areas of blooming representing the microhaemorrhages.
**Fig. 36**: Ruptured Intracranial Dermoid. Axial T1W, Fat suppressed FLAIR, T2W, TIW and SWI images. Fat intensity lesion seen in the suprasellar region. Multiple tiny fat droplets seen in the sylvian fissures, cortical sulci and superior vermian cistern. The fat containing dermoid and fat droplets blooms on SWI.

**Fig. 37**: Post Radiotherapy Haemorrhage. Left tentorial meningioma. Post gamma Knife surgery. Multiple intratumoral haemorrhages are seen post radiotherapy.
Fig. 38: Effect of oxygen on visibility of veins. Breathing normal air has a better visualization of the veins (left image) than breathing oxygen (right image) because of increase in venous blood oxygenation which leads to a reduction of field inhomogeneities around the vessels. As a result the contrast between veins and surrounding parenchyma is reduced (right image).
Fig. 39: Normal anatomy of the Midbrain on Phase and SWI images. The iron deposition may correspond to hemosiderin from the ongoing presence of micro-hemorrhages in the aging process. Increase iron deposition is seen in neurodegenerative diseases.
Fig. 40: Phase image of the dentate nucleus.
**Fig. 41:** Venous anatomy depicted on SWI images.

- CV - Longitudinal Caudate Vein of Schlesinger
- AS – Anterior Septal Vein
- TS – Thalamostriate Vein
- ICV – Internal Cerebral Vein
- RV – Basal Vein of Rosenthal
- HV – Hippocampal Vein
Conclusion

SWI allows improved visualisation of hemorrhage, iron deposition in gray matter and venous structures in brain improving the lesion detection with better diagnosis of disease and effect on patient management and prognosis.

Personal information

References


