Case Reports: Susceptibility-Weighted Imaging (*syngo* SWI) at 3T

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Introduction

This is a pictorial review of susceptibility-weighted imaging (*syngo* SWI) using a MAGNETOM Trio system with software version *syngo* MR B15 and a 32-channel head coil at The Geelong Hospital, Victoria, Australia.
syngo SWI is a 3D FLASH sequence that is flow compensated in slice, read and phase directions. The data received contains a combination of phase and magnitude information. The susceptibility-weighted images are produced by first filtering the phase images of unwanted field inhomogeneities and then weighting the magnitude images with this phase mask. Two maps are automatically calculated; phase mask multiplied magnitude images and SWI minIP (minimum intensity projection of 8 images on a sliding scale). In addition, the phase and magnitude images can also be produced by modifying the reconstruction tab card.
The SWI images are T2*-weighted and are enhanced by flow compensation and phase masking, so there is exquisite detail of areas of susceptibility due to venous blood, haemorrhage and iron storage.

The phase images can be windowed to see contrast between iron deposition and normal tissue and also to visualize gyral pattern to anatomically orientate lesions more accurately. The SWI sliding minIP is useful to visualize change in tissue susceptibility caused by structures such as veins that cross many slices.

SWI sequence details for all case studies: swi3d1r, transverse plane, TR 28 ms, TE 20 ms, flip angle 15, bandwidth 120 Hx/px, FOV 220 (FOV phase 84.4%), resolution 199 x 256, slice thickness 3 mm, 48 slices, voxel size 0.9 x 0.9 x 3 mm, 1 average, acquisition time 2:19 min.

Since SWI is more sensitive to haemorrhage than conventional T2* gradient echo imaging, we replaced the T2* gradient echo sequence with *syngo* SWI in all of our brain protocols. In order to do this without increasing scan time, the SWI sequence as provided by the standard protocol tree with the software version *syngo* MR B15 was modified by increasing the voxel size from 0.8 mm x 0.7 mm x 1.2 mm (resolution 256 x 384 and 1.2 mm slice thickness) to 0.9 mm x 0.9 mm x 3 mm (resolution 199 x 256 and slice thickness 3 mm), giving us lower resolution but allowing us to image the whole brain rather than only a section of it, in half the time of the standard sequence. The 3 mm slice thickness also correlates to our other brain sequences allowing direct comparison to be made.

The resolution is high enough to diagnose clinically relevant lesions and the sequence short enough to include in all protocols that would benefit from this new technique, without a time penalty. Whole brain coverage of our sequence means that lesions in unexpected locations would not be missed due to lack of coverage.

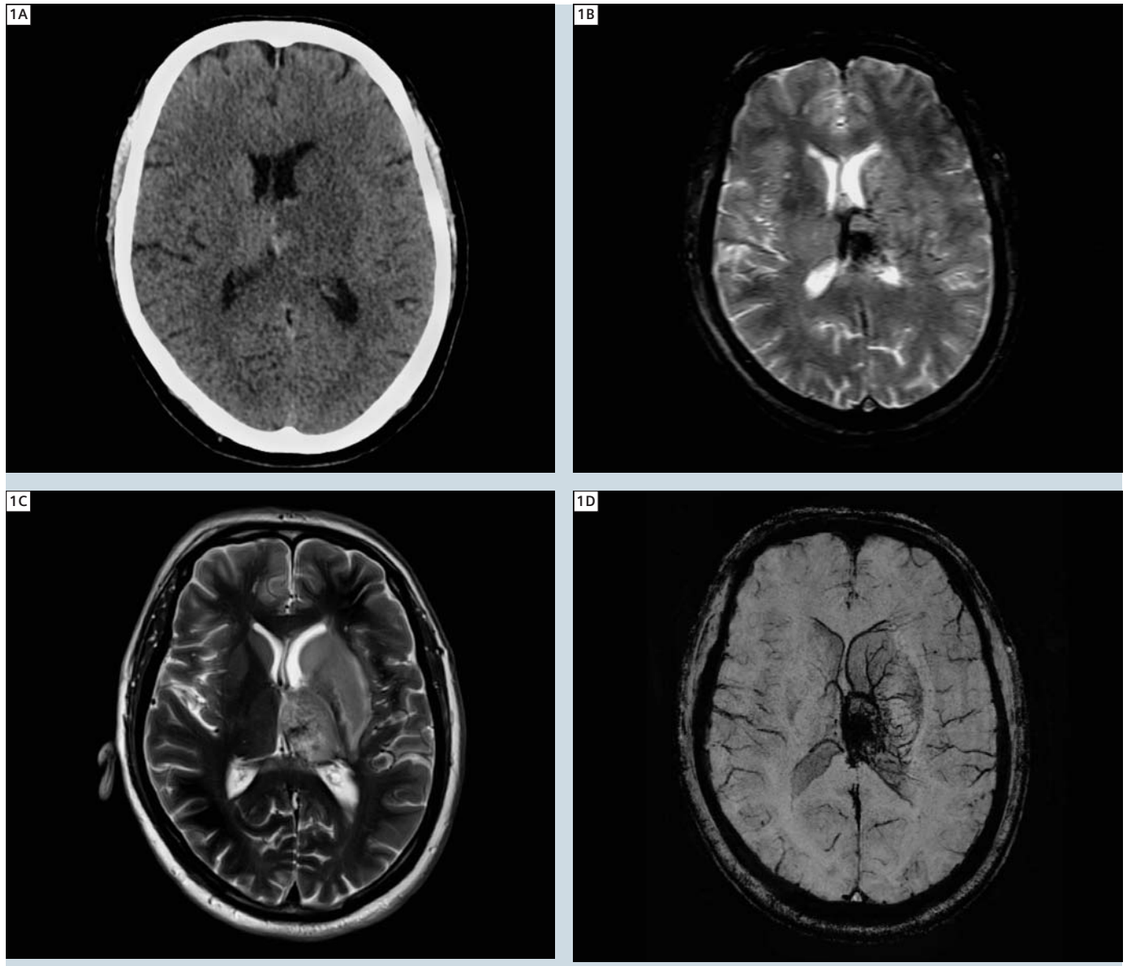
Case 1: Thrombosis and Associated Venous Infarct

Patient history

A 65-year-old male presented to our emergency department with dysphagia, word-finding difficulty and right sided weakness.

Imaging findings

Non-contrast CT identified a hypodense mass lesion in the left thalamus with a hyperdense border. Contrast CT and CT venogram demonstrated a segment of non-filling likely due to thrombosis in the left internal cerebral vein with associated venous infarct in the left thalamus. MRI was obtained to confirm the vein thrombosis and extent of infarction. Initial MRI on our Philips Edge 1.5T system confirmed a non-filling section of the left internal cerebral vein in keeping with thrombosis, extending to the vein of Galen. There was an area of susceptibility artefact in the gradient echo images in the left thalamus representing haemorrhage. There were 2 small foci of restricted diffusion in the left centrum semiovale likely related to the venous infarction, but no definite restricted diffusion involving the left thalamus or the left basal ganglia. MR spectroscopy of the basal ganglia region showed an increased lactate peak suggestive of ischaemia.



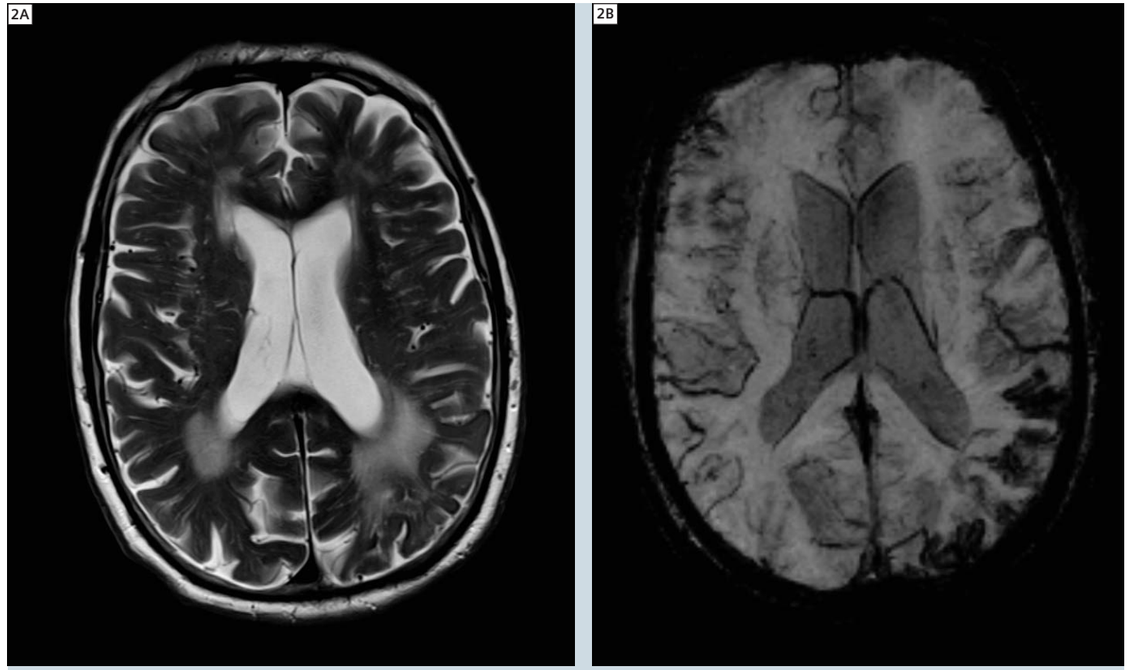
1 **A)** Native CT scan. **B)** T2* GRE at 1.5 Tesla. **C)** T2w TSE with *syngo* BLADE at 3 Tesla. **D)** Corresponding *syngo* SWI at 3 Tesla.

The patient was recalled to our Siemens 3T MAGNETOM Trio scanner the following day. The sequences performed included axial T2w, T1w, Diffusion-Weighted Imaging (DWI), Susceptibility-Weighted Imaging (*syngo* SWI) and MR venography. This imaging confirmed the left internal cerebral vein thrombosis and associated venous infarct.

Discussion

SWI nicely demonstrated the venous tributaries of the left internal cerebral vein with signal dropout due to the presence of deoxyhaemoglobin in the vessels. Signal dropout is also seen in the thrombosed internal cerebral vein and within the thalamic haemorrhage, demonstrating the high sensitivity but low specificity of this sequence.

Case 2: Amyloid Angiopathy



2 All images acquired at 3 Tesla. **A)** T2w TSE. **B)** *syngo* SWI.

Patient history

An 83-year-old male presented for MRI from the memory clinic query fronto-temporal dementia versus Alzheimers Disease with frontal features.

Sequence details

The standard dementia protocol was performed: T1 volume, axial T2, FLAIR, *syngo* SWI, DWI whole brain images with PRESS 30 MR spectroscopy of the parietal grey matter.

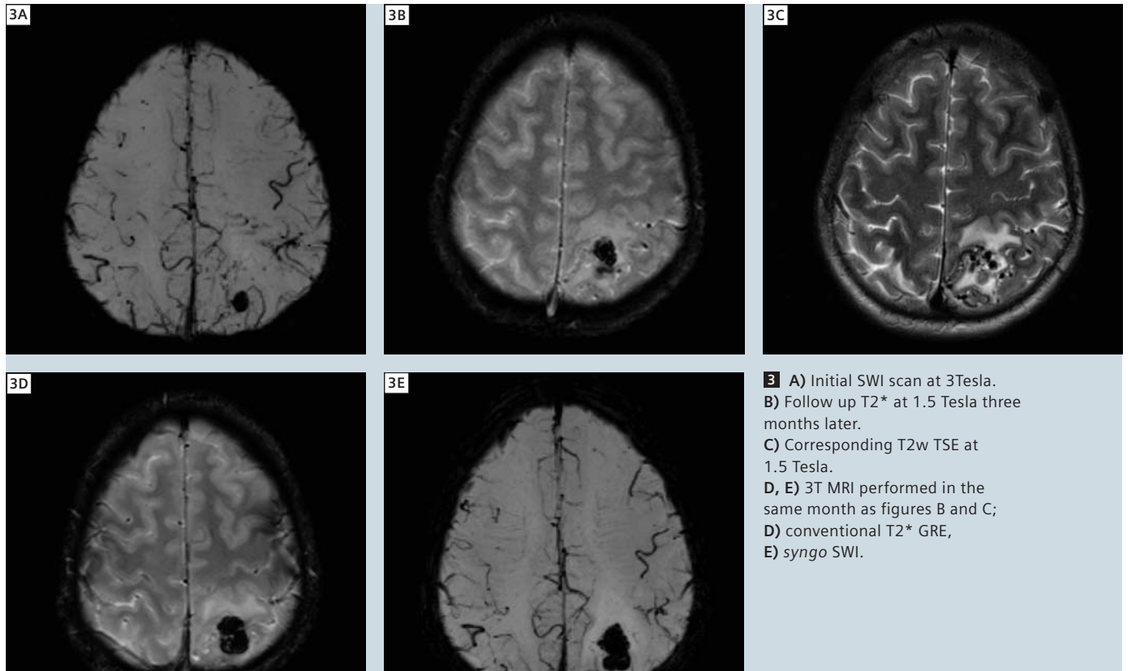
Imaging findings

Haemosiderin staining over the cortical surface of the frontal and parietal lobes was evident on the SWI, consistent with previous subarachnoid haemorrhage, most likely secondary to amyloid angiopathy.

Discussion

The SWI demonstrated signal loss due to haemorrhage which was not appreciable on the routine imaging. Micro haemorrhages in the arterioles of the grey matter may lead to vascular dementia associated with amyloid angiopathy. *syngo* SWI may provide useful information in the imaging of dementia.

Case 3: Cerebral haemorrhage in case of AVM



3 **A)** Initial SWI scan at 3Tesla.
B) Follow up T2* at 1.5 Tesla three months later.
C) Corresponding T2w TSE at 1.5 Tesla.
D, E) 3T MRI performed in the same month as figures B and C;
D) conventional T2* GRE,
E) *syngo* SWI.

Patient history

A 33-year-old male with a known brain arterio-venous malformation (AVM) presented to our emergency department with a history of 5 minutes of motor problems in his right hand. MRI was performed to rule out cerebral haemorrhage.

Sequence details

T1 volume, axial T2, FLAIR, field-echo whole brain images, 3D Time-of-Flight (TOF) and contrast-enhanced MR angiography and MR venography sequences were performed on our Siemens 1.5T MAGNETOM Avanto system.

Imaging findings

A collection of serpiginous flow-voids was evident within the left superior parietal lobule, similar in appearance to the patient's previous study. However there was a region of hypointense signal present within the region of the vascular malformation that was not visible on the SWI from a previous study performed on the patient 3 months prior. This was suspicious for acute haemorrhage.
The patient was recalled for SWI at 3 Tesla, so we could have a direct comparison with the previous imaging that was also performed on our 3T scanner. This demonstrated the development of a region of hypointensity situated centrally within the vascular malformation within the left parietal lobe, measuring 2.0 x 1.5 x 3.0 cm in size. On the previous imaging from 3 months prior, a small focus of hypointensity at this site was evident measuring 1 x 1 x 1 cm in diameter.

Discussion

The SWI appearance indicated the development of haemorrhage into the vascular malformation within the left parietal lobe, which had occurred since the previous study. The signal dropout on the SWI shows the margin of the haemorrhage and the associated anomalous vessels more accurately than other routine sequences.

Case 4: Traumatic haemorrhage

Patient history

48-year-old female presented to our emergency department with vomiting and headache after previously discharging herself following a diagnosis of cortical vein thrombosis.

Sequence details

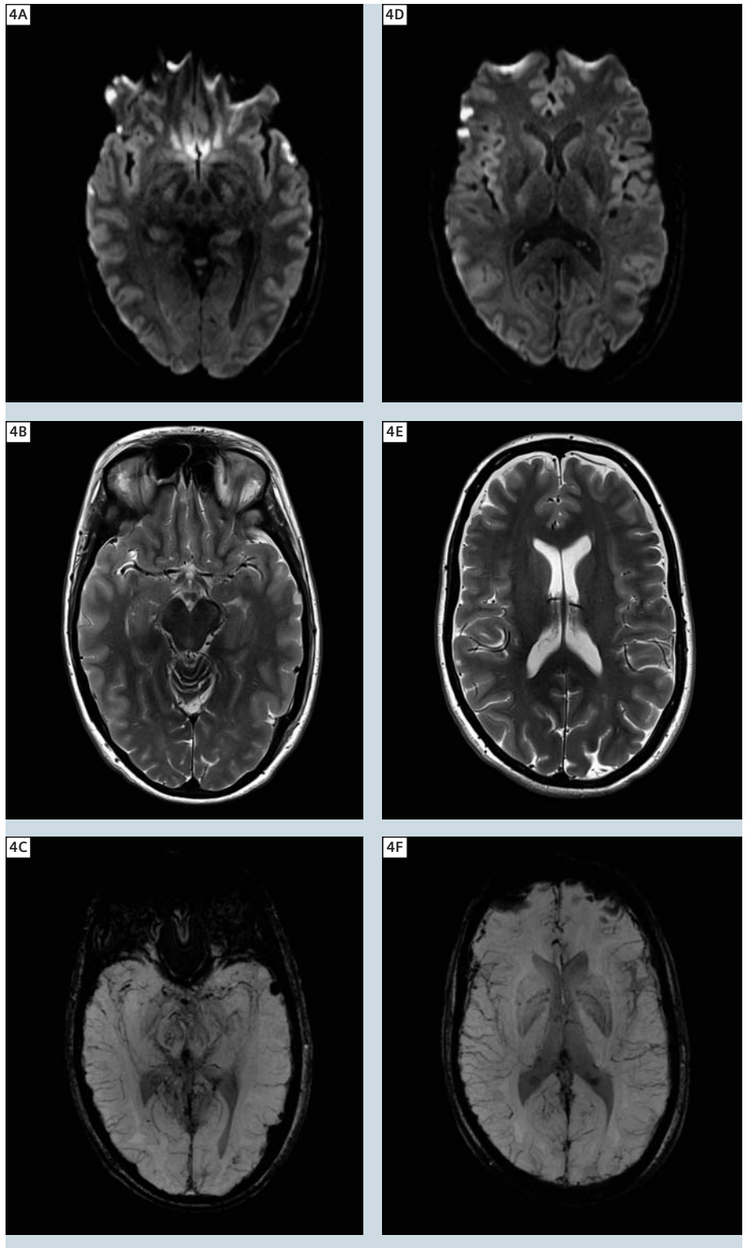
Pre and post contrast T1 whole brain images, axial T2, DWI, *syngo* SWI whole brain images with MR venogram.

Imaging findings

syngo SWI demonstrated a number of hypointense foci within the sulci of the frontal lobes bilaterally and a number of extra-axial locations. These were associated with a number of small foci of restricted diffusion within the cerebral cortex. The history of recent head trauma, subsequently elicited from the patient, indicated that the appearance was most likely due to regions of extra-axial haemorrhage and small cortical contusions.

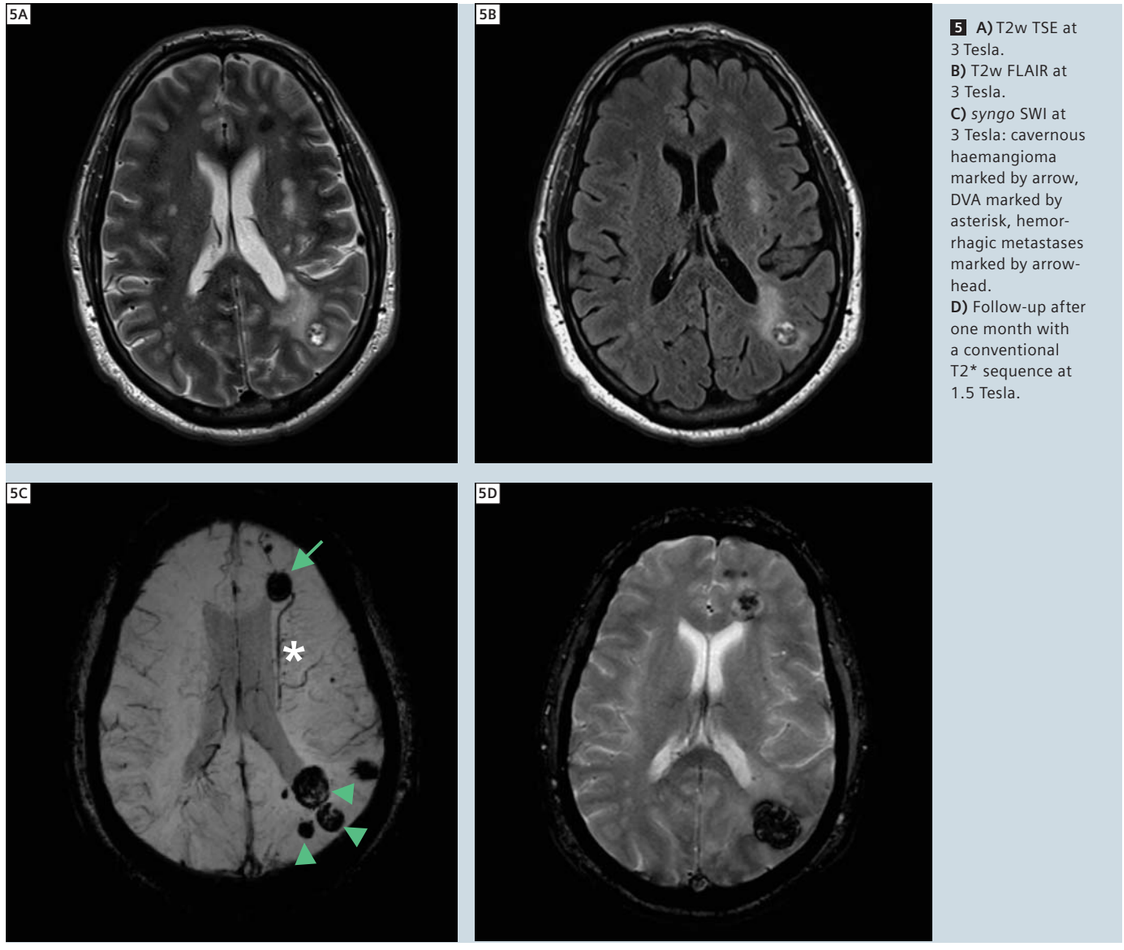
Discussion

SWI is more sensitive to very small areas of traumatic haemorrhage because of its higher resolution and better sensitivity to blood products than the routine sequences.



4 All images acquired at 3 Tesla. **A, D)** DWI **B, E)** T2w TSE 4 **C, F)** *syngo* SWI.

Case 5: Cerebral metastases in case of oesophageal adenocarcinoma



5 **A)** T2w TSE at 3 Tesla.
B) T2w FLAIR at 3 Tesla.
C) *syngo* SWI at 3 Tesla: cavernous haemangioma marked by arrow, DVA marked by asterisk, hemorrhagic metastases marked by arrowhead.
D) Follow-up after one month with a conventional T2* sequence at 1.5 Tesla.

Patient history

A 48-year-old male with oesophageal adenocarcinoma presented with right retro orbital pain for 8 weeks and was scanned for query cerebral metastases.

Sequence details

Pre- and post contrast T1 volume, axial T2, FLAIR, DWI, *syngo* SWI whole brain images, coronal T1, fat sat T2, post contrast fat sat T1 images of orbits and paranasal sinuses.

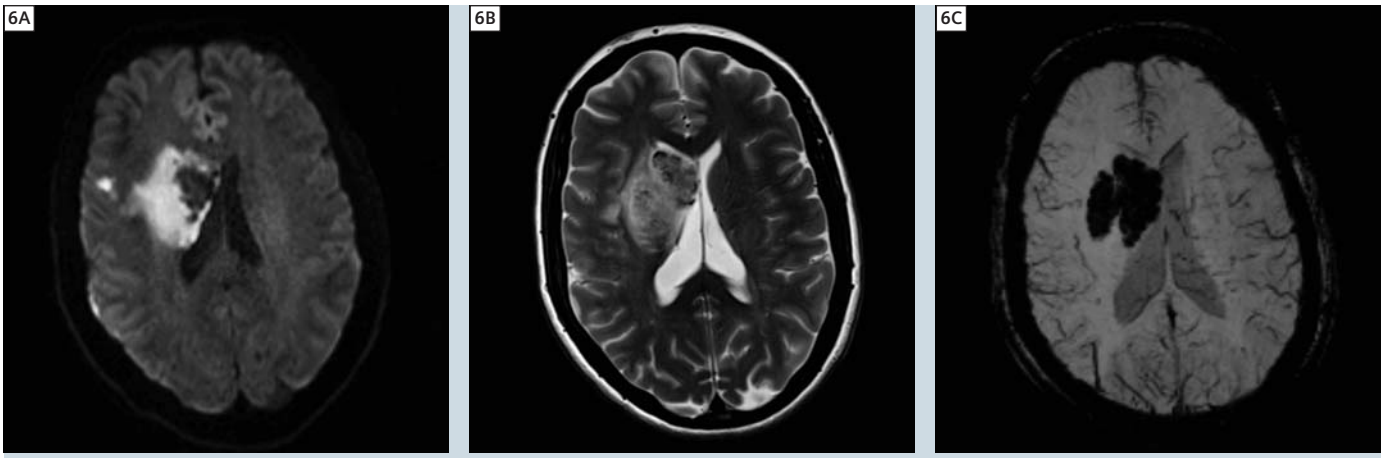
Imaging findings

No evidence of orbital mass or mass within the paranasal sinuses was demonstrated.
Numerous T2 hypointense lesions with marked signal dropout on SWI were evident throughout the left cerebral hemisphere. However, some of these were unaltered in appearance from the previous study from 2 years earlier and were consistent with cavernous haemangiomas. The others represent haemorragic metastases.

Discussion

The patient returned for a follow-up scan on our 1.5T MAGNETOM Avanto scanner 1 month later and standard T2* gradient echo imaging was performed. Compared to the 3T SWI, the standard gradient echo imaging at 1.5T is not as sensitive to the multiple haemorrhagic areas, failing to show some of the smaller lesions evident on the 3T SWI sequence.

Case 6: Haemorrhagic component of MCA infarction



6 All images acquired at 3 Tesla. **A)** DWI **B)** T2w TSE **C)** *syngo* SWI

Patient history

A 48-year-old female presented to our emergency department with sudden onset of left face, arm and leg weakness. CT brain was reported as right middle cerebellar artery infarction. MRI was performed to confirm this finding.

Sequence details

Pre- and post contrast volume T1, axial FSE T2, FLAIR, *syngo* SWI, DWI images of the whole brain and 3D TOF MRA circle of Willis.

Imaging findings

Abnormal signal was seen within the right caudate head and lentiform nucleus with significant susceptibility artefact within these structures that was most consistent with the presence of blood products. The pathology is contained within the middle cerebral artery distribution and appearances on *syngo* SWI are most consistent with a cerebral infarction with haemorrhagic transformation.

Discussion

The SWI sequence demonstrated the full extent of the haemorrhagic component of the infarction better than any of the routine sequences. The presence of haemorrhage with stroke is important to demonstrate as it changes treatment options.

Case study discussion

syngo SWI has allowed smaller susceptibility lesions to be demonstrated than previously possible, in cases of vascular malformation, tumor, stroke, trauma and dementia.
In many cases cited in the literature, SWI was the only imaging sequence to show the abnormality due to its increased sensitivity to iron content.
In all 6 of our cases the SWI sequence demonstrated increased detail of the pathology compared with the routine imaging sequences. In cases 2, 4 and 5, some lesions appeared to be too small to see on other imaging sequences, indicating how the sensitivity of *syngo* SWI may benefit diagnosis.
The increased signal and susceptibility effects at 3T enhance the use of *syngo* SWI, allowing full brain coverage in a short amount of time.

References

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