Alexandria Journal of Medicine

ISSN: 2090-5068 (Print) 2090-5076 (Online) Journal homepage: https://www.tandfonline.com/loi/tajm20

Importance of susceptibility weighted imaging (SWI) in management of cerebro-vascular strokes (CVS)

Abd Elaziz Elnekeidy, Ahmed Yehia & Amr Elfatatry

To cite this article: Abd Elaziz Elnekeidy, Ahmed Yehia & Amr Elfatatry (2014) Importance of susceptibility weighted imaging (SWI) in management of cerebro-vascular strokes (CVS), Alexandria Journal of Medicine, 50:1, 83-91, DOI: 10.1016/j.ajme.2013.05.006

To link to this article: https://doi.org/10.1016/j.ajme.2013.05.006

Alexandria Journal of Medicine (2014) **50**, 83–91

Alexandria University Faculty of Medicine

Alexandria Journal of Medicine

www.sciencedirect.com

Importance of susceptibility weighted imaging (SWI) in management of cerebro-vascular strokes (CVS)

Abd Elaziz Elnekeidy [a,*], **Ahmed Yehia** [b], **Amr Elfatatry** [c]

[a] *Department of Radio-Diagnosis, Faculty of Medicine, Alexandria University, Egypt*
[b] *Department of Neurosurgery, Faculty of Medicine, Alexandria University, Egypt*
[c] *Department of Neurology, Faculty of Medicine, Alexandria University, Egypt*

Received 3 March 2013; accepted 16 May 2013
Available online 22 June 2013

KEYWORDS

Susceptibility weighted imaging;
Hemorrhagic infarction;
Microbleeds;
Dural sinus thrombosis

Abstract *Introduction:* Susceptibility-weighted imaging offers information about any tissue that has a different susceptibility from its surrounding structures such as deoxygenated blood, hemosiderin, ferritin, and calcium. It can detect minute hemorrhages, old microbleeds as well as thrombosed veins or sinuses.
Purpose: The aim of this work was to evaluate the importance of susceptibility 'weighted imaging (SWI) in management of cerebro-vascular strokes.
Patients and methods: The study was conducted upon 50 patients presenting with cerebro-vascular strokes within 72 h after the onset of the neurological symptoms. All patients were subjected to non-contrast CT of the brain as well as MRI of the brain including susceptibility weighted MRI.
Results: Among the 50 studied patients, 46 had infarctions (92%), while three patients had hematomas (6%) and one patient (2%) had no infarctions or hematomas but transverse sinus thrombosis. Among the 46 infarctions CT detected hemorrhagic transformation in two patients (4.3%) while the SWI detected hemorrhagic transformation in ten patients (21.7%). Both the CT and SWI detected the hematomas in three patients. SWI detected old micro-bleeds in eight patients out of 50 (16%), while CT could not detect any of these micro-bleeds. These eight patients included four

Abbreviations: SWI, susceptibility weighted imaging; CVS, cerebro-vascular strokes; MRI, magnetic resonance imaging; DWI, diffusion weighted imaging; ADC, apparent diffusion coefficient; CVST, cerebral venous sinus thrombosis.

* Corresponding author. Tel.: +20 1110129441.
E-mail addresses: elnekidy@gmail.com, elnekidy@yahoo.com (A.E. Elnekeidy), ahmedyehyamd@yahoo.com (A. Yehia), amrelfatatry@gmail.com (A. Elfatatry).

Peer review under responsibility of Alexandria University Faculty of Medicine.

ELSEVIER Production and hosting by Elsevier

http://dx.doi.org/10.1016/j.ajme.2013.05.006

with non-hemorrhagic infarctions, three with hematomas and one with early hemorrhagic transformation.SWI detected sinus thrombosis in two cases out of 50 (4%), one had hemorrhagic infarction, and the other had no visible brain insults on CT. Management was planned for each group according to the presence or absence of hemorrhagic transformation of infarction or associated old microbleeds.

Conclusions: Susceptibility-weighted imaging is an important technique that allows accurate detection of early hemorrhagic transformations within acute infarctions as well as detecting old microbleeds thus alarming the treating physician about the devastating complication of anticoagulant therapies. Also SWI can early detect cerebral venous thrombosis. Thus SWI should be a routine sequence in the protocol of stroke imaging.

1. Introduction

Susceptibility-weighted imaging (SWI) is an MRI sequence aiming to enhance contrast in MR imaging. The susceptibility information adds valuable data to what is already available with conventional spin density, T1-, and T2- weighted imaging. SWI offers information about any tissue that has a different susceptibility from its surrounding structures such as deoxygenated blood, hemosiderin, ferritin, and calcium.[1]

Cerebro-vascular ischemia from thromboembolism or arteriosclerotic stenosis leads to acute infarct with or without hemorrhage.[2] Studies have shown that SWI is more sensitive in detecting hemorrhage inside acute infarct lesions than CT and 2D gradient recalled-echo (2D−GRE = $T2^{*}$) weighted imaging. SWI is so sensitive that it can detect minute amount of hemorrhage, putting in consideration that early detection of hemorrhage inside acute infarction restricts the use of thrombolytic drugs and minimize the development of large hematoma that need to be evacuated surgically.[3]

The susceptibility sign referred to intra-arterial thrombus and is defined as the presence of marked hypointensity within the intracranial arteries with apparent enlargement of vessel compared to the contra-lateral vessel diameter due to ''blooming'' artifact.[4] This is similar to what is known in CT as hyperdense middle cerebral artery sign.[5]

SWI can identify clinically silent microbleeds representing hemosiderin deposits that are related to microangiopathy. It has been suggested that these microbleeds may be a marker for patients at risk for intra-cranial hemorrhage (ICH).[6,7]

Lobar microbleeds are related to cerebral amyloid angiopathy (CAA), that usually involves the cortex and subcortical white matter within the frontal and parietal lobes, whereas micro-hemorrhages in a deep or infra-tentorial location typically result from hypertensive or atherosclerotic micro-angiopathy. In addition to recurrent microbleeds, CAA can lead to intracranial lobar macrohemorrhages that need surgical evacuation.[8]

Cerebral venous sinus thrombosis (CVST) is an infrequent neurologic condition, which is difficult to diagnose because of its nonspecific clinical presentation.[9] CVST can be fatal if it remains undiagnosed and if thrombolytic treatment is not started rapidly.[10,11] SWI has become a useful method to detect CVST early 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 or blooming on SWI.[2]

2. Aim of the work

The aim of this work was to evaluate the added value of susceptibility weighted imaging (SWI) in management of cerebro-vascular strokes.

3. Patients

The study was conducted upon 50 patients presenting with cerebro-vascular strokes within 72 h after the onset of the neurological symptoms. The fifty patients included 32 males and 18 females, their ages ranged between 32 and 79 years with a mean age of 54 years. Informed written consents were taken from the patients or their relatives.

4. Methods

All patients were subjected to full history taking and thorough clinical and neurological examination and neuro-imaging including non-contrast CT of the brain and MRI which was performed by a 1.5 Tesla magnet (Avanto Siemens and GE).

The following MRI sequences were performed, including (a) axial and coronal T2 weighted turbo spin echo (T2 TSE) utilizing the following parameters: a repetition time (TR) of 3000 ms, an echo time (TE) of 120 ms, a slice thickness of 3 mm, a gap of 0.3 mm, 256×256 acquisition matrix, FOV = 180 mm given an acquisition time of 2 min 51 s (b) axial and sagittal T1 weighted spin echo (T1 TSE) utilizing the following parameters: TR of 550 ms, a TE of 15 ms, a 256×256 acquisition matrix, a field of view of 180 mm, a slice thickness of 3 mm and a gap of 0.3 mm, given an acquisition time of 3 min 23 s (c) axial FLAIR utilizing the following parameters: TR of 8000 ms, a TE of 138 ms, TI 2000 ms a field of view of 180 mm, a slice thickness of 3 mm and a gap of 0.3 mm (d) susceptibility weighted MRI utilizing the following parameters: TR of 6400 ms, a TE of 30 ms, a 256×256 acquisition matrix, a field of view of 280 mm, a slice thickness of 2.4 mm and a gap of 0.2 mm, given an acquisition time of 3 min, with minIP reconstruction for the images (e) diffusion weighted axial MRI imaging with B0 and B1000 values as well as apparent diffusion coefficient (ADC) map reconstruction.

Table 1 SWI versus CT detection of hemorrhagic transformation within the infarctions.

46 patients with infarctions		
36 without hemorrhagic transformation	10 with hemorrhagic transformation	
All were negative for hemorrhage by both CT and SWI	10 were positive for hemorrhage by SWI	2 were positive for hemorrhage by CT

Then the therapeutic protocols were chosen according to the type of the stroke.

5. Results

Our study included 46 patients with infarctions (92%) (one of them was venous infarct due to superior sagittal sinus thrombosis), three patients with hematomas (6%) and one patient (2%) with transverse sinus thrombosis still without infarctions or hematomas.

Among the 46 infarctions, CT detected hemorrhagic transformation in two patients (4.3%) while SWI detected hemorrhagic transformation in ten patients (21.7%) (Table 1). Both CT and SWI clearly detected the large hematomas in three patients.

SWI detected old micro-bleeds in eight patients out of 50 (16%), while CT could not detect any of these micro-bleeds. The eight patients with microbleeds included four patients having non-hemorrhagic infarctions, three patients with hematomas and one patient with infarction showing early hemorrhagic transformation.

SWI detected sinus thrombosis in two cases out of 50 (4%), one had hemorrhagic infarction, and the other had no infarctions.

Forty-nine patients presented with signs and symptoms of CVS according to the involved part of the brain while one patient presented with severe headache without neurological deficit due to transverse sinus thrombosis. The 50 cases were categorized into five groups according to the presence of microbleeds or hemorrhagic elements which modified their therapeutic protocols (Table 2).

Group I included 32 patients in whom MRI revealed acute infarction. SWI showed no foci of blooming within the infarctions, excluding hemorrhagic transformation. SWIs showed neither micro-bleeds, large foci of hemosiderin deposition due to previous hemorrhagic lesions nor sinus thrombosis (Fig. 1). SWI revealed blooming along the left vertebral artery in one patient within this group, denoting its thrombosis, a finding that was proven by CT angiography.

Group II included ten patients in whom MRI detected acute infarctions. SWI showed foci of blooming within the infarctions denoting hemorrhagic transformation. In two of these ten patients, CT showed fine streaks of hyper density within the infarctions matching with hemorrhagic foci while in the other eight patients the CT showed no hyper densities (Figs. 2 and 3).

One of the patients in group II had micro-bleeds detected only by SWI not by CT. Another patient in this group showed blooming along the superior sagittal sinus and cortical veins denoting their thrombosis.

Group III included four patients in whom MRI revealed acute infarction. SWI showed no foci of blooming within the infarctions themselves excluding hemorrhagic transformation. However SWI showed numerous foci of blooming denoting micro-bleeds distributed mainly in the basal ganglia and to a lesser extent at the subcortical white matter suggestive of chronic hypertensive encephalopathy. None of these micro-bleeds could be detected by the corresponding CTs of these patients.

Group IV included three patients presented with CVS. In two patients, small cortical hematomas were detected by MRI expressing T1 iso-to hyper-intense and T2 low signal.

Table 2 50 patients distributed into five groups according to the presence of microbleeds or hemorrhagic elements and the chosen therapeutic protocols.

Group	Number of patients	MRI, SWI and CT findings	Management
I	32	Infarctions without hemorrhagic transformation or micro-bleeds on SWI or CT	This group was safely treated with full dose anti-platelet and anticoagulant drugs, only three patient developed hemorrhagic transformation on follow up
II	10	Infarctions with early hemorrhagic transformation a. All were diagnosed by SWI b. Only 2 were diagnosed by CT c. One patient had microbleeds seen by SWI, not by CT d. One patient had superior sagittal sinus thrombosis	This group was managed by the lowest dose anti-platelets and anti-thrombotic drugs with control of their high blood pressures. One of these patients developed a large hematoma on follow up leading to increase intracranial pressure with marked mass effect and was evacuated surgically
III	4	Infarction without hemorrhagic transformation but micro-bleeds detected by SWI not CT	This group was managed by the lowest dose anti-platelets and anti-thrombotic drugs with control of their high blood pressures
IV	3	Hematomas + micro-bleeds detected by SWI not CT	This group was managed surgically in two patients by evacuation of the hematomas, while the 3rd patient received supportive measurement in ICU
V	1	Venous thrombus without infarction or hematomas	This patient was managed by the conventional dose anti-platelets and anti-thrombotic drugs
Total	50		

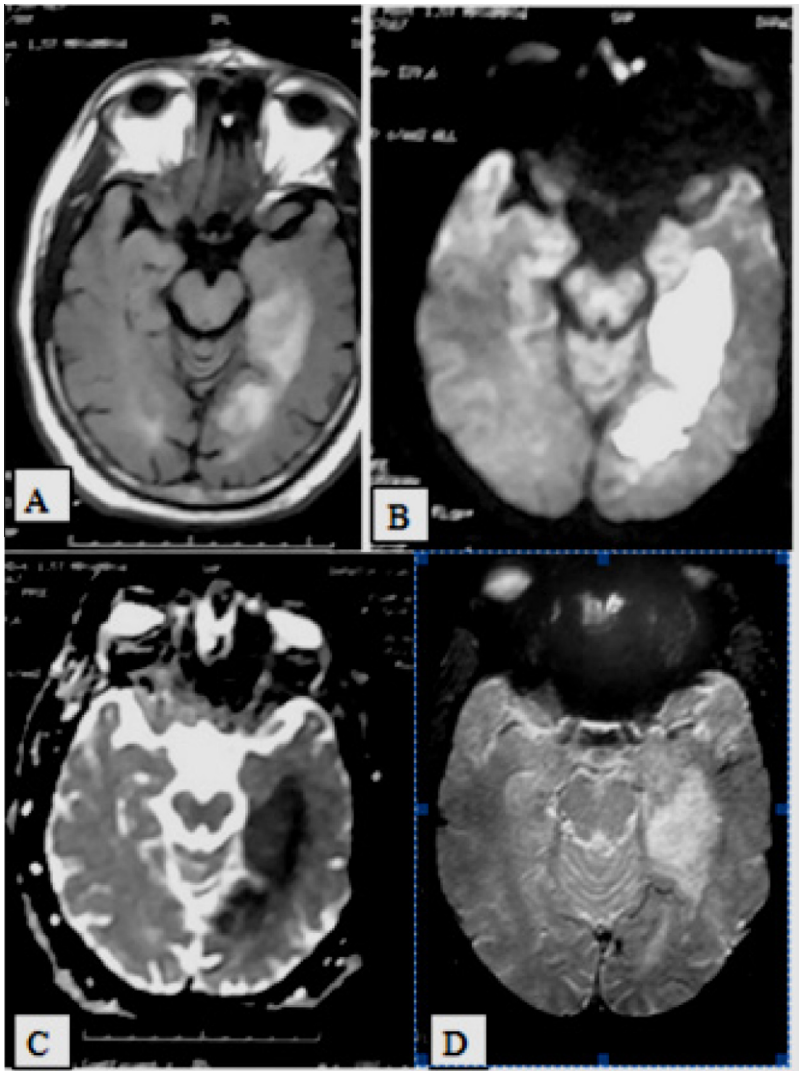

Figure 1 53 years old male patient presenting with disturbed sensorium. Axial FLAIR (A), DWI (B) and ADC map (C) revealed acute infarct involving the posteromedial aspect of the left temporal lobe. SWI minIP (D) images revealed no foci of hemorrhagic transformation within the infarct.MRI diagnosis: left temporal infarction without hemorrhagic transformation.

The third patient showed a large deeply seated hematoma expressing T1 iso to hyper-intense and T2 low signal. All these hematomas showed marked blooming on SWI. Also the SWI showed numerous foci of blooming denoting micro-bleeds distributed mainly at the sub-cortical white matter suggestive of amyloid micro-angiopathy in the first two patients (Fig. 4). The other patient showed numerous foci of blooming denoting micro-bleeds distributed mainly in the basal ganglia and to a lesser extent at the subcortical white matter suggestive of chronic hypertensive encephalopathy (Fig. 5). None of these micro-bleeds could be detected by CT, but the large hematomas were clearly identified. This group was managed surgically in two patients by evacuation of the hematomas, while the 3rd patient received medical supportive measurement.

Group V included a female patient, presented with severe headache. MRI detected no infarction or foci of restricted diffusion with lost signal void on T1 and T2 WI within the left transverse sinus. SWI showed no foci of blooming within the brain but showed blooming along the left transverse sinus itself denoting its thrombosis (Fig. 6). Non enhanced CT was unremarkable.

Management was planned for each group according to the presence or absence of hemorrhagic transformation of infarction or associated old microbleeds as detailed in Table 2. The presence of microbleeds or hemorrhagic transformation of infarctions as in group II and III prevented the use of conventional dose of anti-platelets and anticoagulants aiming to avoid serous hemorrhagic. Surgical intervention was performed if large hematomas are diagnosed as in group III to avoid the hazards of their mass effects.

6. Discussion

Susceptibility-weighted imaging (SWI) is a magnetic resonance technique that exploits the magnetic susceptibility differences of various tissues, such as de-oxy-hemoglobin, met-hemoglobin, ferritin, hemosiderin and calcification.[2] SWI is not a new concept, but recent advances have allowed the technique to be refined with a lot of clinical applications.[12]

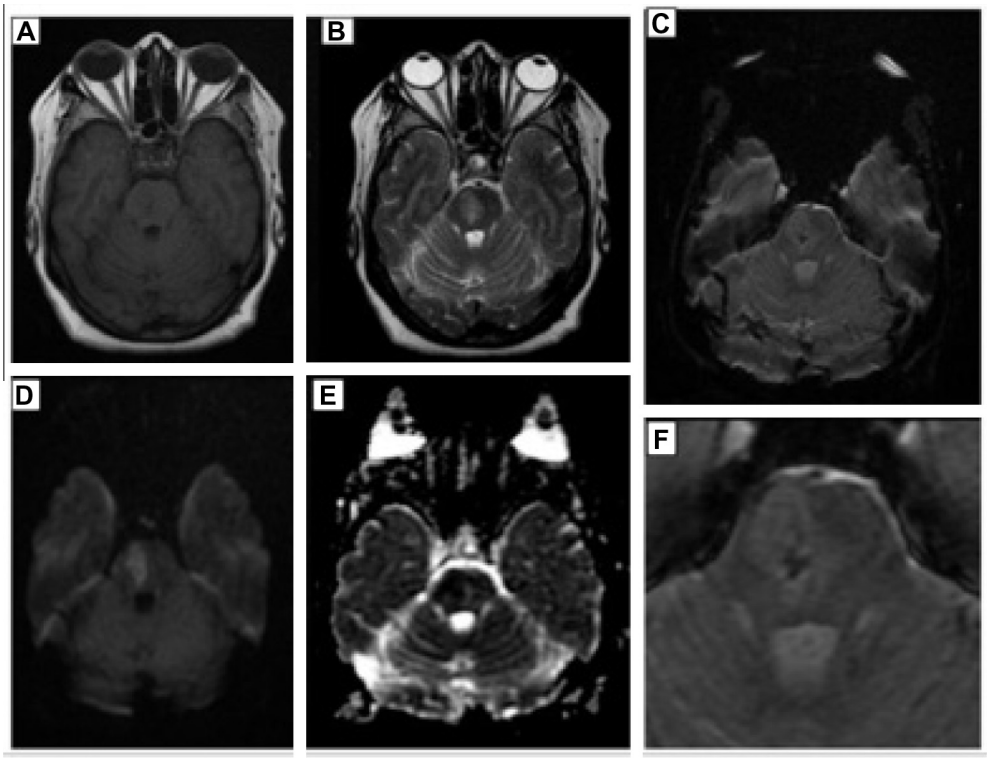

Figure 2 55 years old male patient in presenting with disturbed sensorium.T1 (A),T2 (B) and DWI (C) and ADC (D) images revealed infarction involving the right side of the pons. SWI minIP (E) images and zooming (F) reveals foci of hemorrhagic transformation within the infarct Radiological diagnosis. MRI diagnosis: pontine infarction with hemorrhagic transformation.

Although patient's symptoms and clinical examinations may suggest the diagnosis of acute stroke, only brain imaging studies can confirm the diagnosis and differentiate hemorrhage from ischemia with high accuracy, and this distinction is extremely important as the treatment decisions are dependent on this. Cerebro-vascular ischemia from thromboembolism or arteriosclerotic stenosis leads to acute infarct with or without hemorrhage. Accurate early detection of bleeding is crucial because hemorrhage is a contraindication to the use of anticoagulant and thrombolytic agents in the acute stroke setting.[2]

In the current study, early detection of hemorrhagic transformation was confirmed by SWI in 10 out of 46 patients (21.7%), while the CT detected hemorrhagic transformation in 2 out of these 46 patients (4.3%). This was in agreement with multiple studies that have shown that SWI is more sensitive in detecting hemorrhage inside acute infarct lesions than CT and 2D gradient recalled-echo ($T2^*$)-weighted imaging.[3] Haemorrhagic transformation of stroke is observed in approximately 20–40% of all stroke patients within the first week of onset.[13] This can be a devastating complication especially, if the patient is considered for revascularization therapies. Conventional MRI often fails to detect these bleeds in the early stages of stroke. SWI, which is exquisitely sensitive to magnetic field inhomogeneity, can detect very small bleeds within the infarct and make it more conspicuous than $T2^*$W GRE sequences.[3]

Based on our results, we are in agreement with Wycliffe et al.[14] and Santhosh et al.[3] in that SWI is useful in the evaluation of acute stroke patients being extremely sensitive to even traces of hemorrhage and has potential diagnostic, as well as therapeutic, implications in acute stroke patients, and this technique should be included in the routine evaluation of stroke patients where it is essential to rule out the presence of hemorrhage within the infarct with a high degree of accuracy as thrombolysis and even full dose of anticoagulants in such patients can be catastrophic.

In addition, current guidelines for thrombolytic therapy indicate that a prior history of intra-cerebral hemorrhage is also a contraindication to thrombolytic therapy.[15] These guidelines, however, refer to clinically manifest prior hemorrhages and hemorrhages diagnosed by head CT; they do not address the presence of clinically silent micro-bleeds that are only detected with advanced magnetic resonance imaging (MRI) sequences such as gradient echo and susceptibility-weighted imaging (SWI). These micro-bleeds are most commonly caused by hypertension, cerebral amyloid angiopathy, or other causes of small-vessel vasculopathy.[16,17]

In the current study, SWI detected old micro-bleeds in 8 patients (16%) out of the 50 while CT could not detect any of these micro-bleeds. These 8 patients included 4 with non-hemorrhagic infarctions, 3 with hematomas and one with early hemorrhagic transformation. This was in agreement with Thomas et al.[18], Santhosh et al.[3] and Mittal et al.[2] who reported that SWI, with its higher sensitivity, identifies numerous microbleeds unidentified on routine images.

Studies have demonstrated the presence of microbleeds in more than half of the patients with primary cerebral bleeds, one fourth of the patients with ischaemic stroke and less than 10% of the healthy population.[19] It has been suggested that these microbleeds may be a marker for patients at risk for ICH.[6,7]

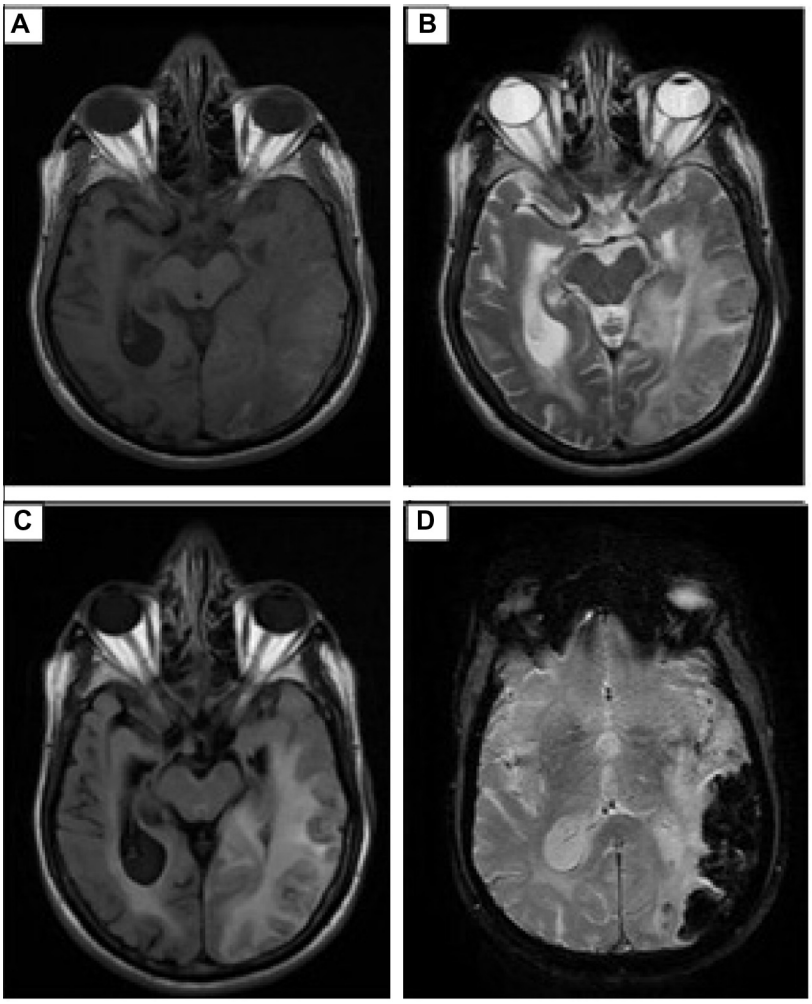

Figure 3 79 years old male patient in presenting with disturbed sensorium. T1 (A) images show hypointense left temporal infarct with hyperintense foci, T2 (B) and FLAIR (C) show hyperintense signal. SWI (D) shows sizable area of blooming within the infarct confirming and revealing higher degree of hemorrhagic transformation. MRI diagnosis: sizable infarction in the left temporal lobe with significant hemorrhagic transformation.

In the current study, SWI detected old micro-bleeds associated with hematomas in three patients, two of them having typical pattern of amyloid angio-pathy with cortical hematomas, while one patient had a deeply seated hematoma with micro-bleeds having the distribution of chronic hypertensive encephalopathy.

Lobar microbleeds are related to cerebral amyloid angiopathy CAA (usually involving the cortex and subcortical white matter within the frontal and parietal lobes), whereas microhemorrhages in a deep or infratentorial location typically result from hypertensive or atherosclerotic microangiopathy.[8]

The likelihood of early cerebral bleeds after ischemic stroke might be increased in patients who had the most vulnerable micro-vascular system.[20] MRI demonstration of microbleeds could gain even more clinical significance if this finding could be used to identify patients at increased risk of early cerebral bleeds. Old microbleeds provide further evidence of severe microangiopathy with a subsequent increased vascular vulnerability.[21]

The information regarding the presence of intra-arterial thrombus and its location may be useful in planning various treatment options and may help in assessing the extent of infarct and prognosis.[4,22]

The susceptibility sign denoting intra-arterial thrombus was seen in one of our cases denoting thrombosis of the left vertebral artery, proven by further assessment by CTA and associated with left cerebellar non hemorrhagic infarction. The susceptibility effect is attributed to the severe T2 shortening of high concentrations of deoxyhaemoglobin within the clot. However, susceptibility artifacts from the paranasal sinuses and base of the skull can obscure the detection of clots limited to the intracranial internal carotid arteries, and this is a potential drawback to this technique, but the SWI can detect small clots or distally located clots in the arteries away from the skull base and this may be a potential benefit of the technique.[5]

In the current study, SWI detected sinus thrombosis demonstrating blooming along the sinus in two cases out of the 50, one had superior sagittal sinus thrombosis with hemorrhagic infarction seen as foci of SWI blooming within the infarcted areas, while the other patient had left transverse sinus thrombosis without brain insults. In the last case, SWI showed left transverse sinus thrombosis, a finding that was

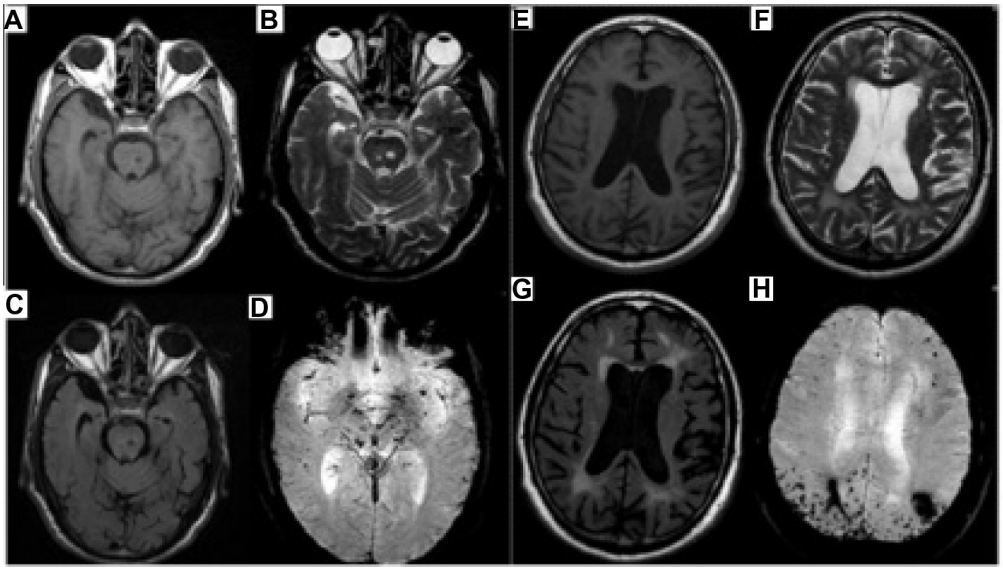

Figure 4 70 years old male non hypertensive patient. T1 (A), T2 (B)and FLAIR (C) images show a lacunar infarct in the pons, SWI image(D) revealed multiple foci of old microbleeds in the brain stem and both temporal lobes T1 (E), T2 (F) and FLAIR (G) images show Involutional brain changes, SWI MinIP (H) images reveals multiple foci of bleeding posteriorly located within both occipital lobes. MRI diagnosis: cerebral amyloid angiopathy with cortical hematomas.

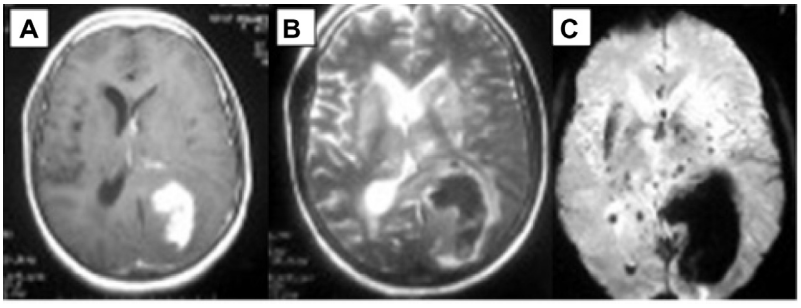

Figure 5 60 years old hypertensive male patient presenting with right hemiparesis.T1 (A),T2 (B) images show a large left parieto-occipital early sub-acute hematoma. SWI image (C) revealed multiple foci of old microbleeds in the basal ganglia and both temporal lobes. The hematoma markedly blooms on SWI with minimal blood seen within the right lateral ventricle.MRI diagnosis: cerebral hematoma with microbleeds due to chronic hypertensive encephalopathy.

not seen on conventional scans except for noticed faint hyperintense signal in FALIR images on a retrograde review. Sinus thrombosis was confirmed by complementary MRV, this is in agreement with Thomas et al.[18] who found that susceptibility-weighted imaging with both phase and magnitude information facilitates the detection of venous sinus thrombosis which is otherwise difficult to detect in conventional spin-echo T2 images.

Cerebral venous sinus thrombosis (CVST) is an infrequent neurologic condition, which is notoriously difficult to diagnose because of its nonspecific clinical presentation.[9] Direct evidence of sinus thrombosis such as the ''triangle'' sign and the ''empty delta'' on CT and loss of the normal flow voids on MR imaging can be easily missed unless clinical suspicion is high and direct signs are actively sought.[23] Indirect signs of dural venous thrombosis are various, including cerebral edema, infarction, and hemorrhage. CVST can be potentially deadly if it remains undiagnosed and if thrombolytic treatment is not started in a timely manner.[10,11] 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.[2]

7. Conclusions

Susceptibility-weighted imaging is an important technique that allows accurate detection of early hemorrhagic transformations within acute infarctions as well as detecting old microbleeds thus alarming the treating physician about the devastating complication of anticoagulant and revascularization therapies. Also SWI can early detect cerebral venous thrombosis. Thus SWI should be a routine sequence in the protocol of stroke imaging.

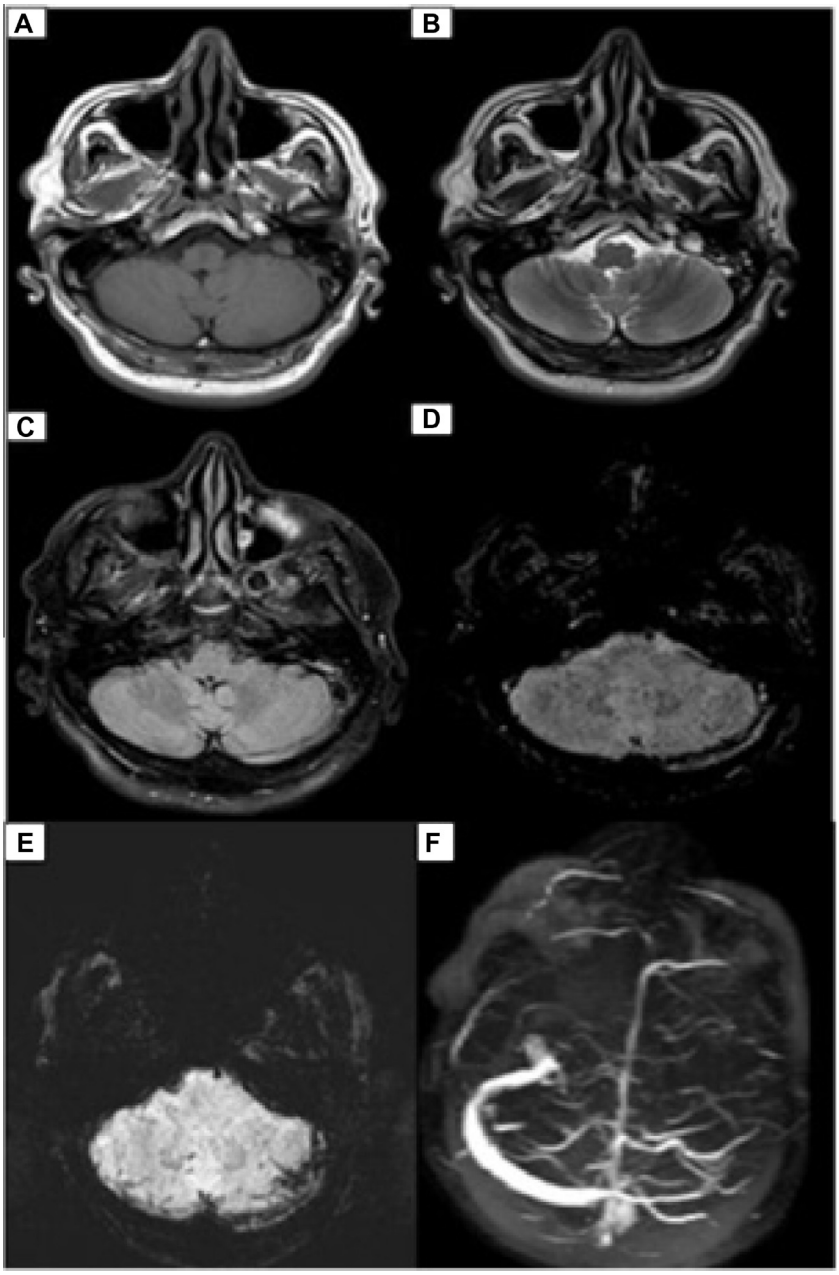

Figure 6 58 years old female patient presenting with disturbed sensorium.T1 (A) and T2 (B) images are unremarkable, FLAIR (C) images show faint hyperintense signal along the course of the left transverse sinus, SWI (D) show blooming along the sinus course In addition to blooming of the sinus, minIP images (E) revealed congested draining veins, sinus thrombosis is confirmed by MRV (F). MRI diagnosis: left transverse sinus thrombosis without infarction.

Conflict of interest

None declared.

References

1. Haacke EM, Mittal S, Wu Z, Neelavalli J, Cheng YC. Susceptibility-weighted imaging: technical aspects and clinical applications, part 1. *AJNR Am J Neuroradiol* 2009 January;**30**(1):19–30.
2. Mittal S, Wu Z, Neelavalli J, Haacke EM. Susceptibility-weighted imaging: technical aspects and clinical applications, part 2. *AJNR Am J Neuroradiol* 2009 February;**30**(2):232–52.
3. Santhosh K, Kesavadas C, Thomas B, Gupta AK, Thamburaj K, Kapilamoorthy TR. Susceptibility weighted imaging: a new tool in magnetic resonance imaging of stroke. *Clin Radiol* 2009 January;**64**(1):74–83.
4. Rovira A, Orellana P, Alvarez-Sabin J, Arenillas JF, Aymerich X, Grive E, et al. Hyperacute ischemic stroke: middle cerebral artery susceptibility sign at echo-planar gradient-echo MR imaging. *Radiology* 2004 Aug;**232**(2):466–73.
5. Hermier M, Nighoghossian N. Contribution of susceptibility-weighted imaging to acute stroke assessment. *Stroke* 2004 August;**35**(8):1989–94.
6. Tanaka A, Ueno Y, Nakayama Y, Takano K, Takebayashi S. Small chronic hemorrhages and ischemic lesions in association

with spontaneous intracerebral hematomas. *Stroke* 1999 August;**30**(8):1637–42.
7. Fazekas F, Kleinert R, Roob G, Kleinert G, Kapeller P, Schmidt R, et al. Histopathologic analysis of foci of signal loss on gradient-echo T2*-weighted MR images in patients with spontaneous intracerebral hemorrhage: evidence of microangiopathy-related microbleeds. *AJNR Am J Neuroradiol* 1999;**20**(4):637–42.
8. Vernooij MW, van der Lugt A, Ikram MA, Wielopolski PA, Niessen WJ, Hofman A, et al. Prevalence and risk factors of cerebral microbleeds: the Rotterdam scan study. *Neurology* 2008;**70**(14):1208–14.
9. Ameri A, Bousser MG. Cerebral venous thrombosis. *Neurol Clin* 1992;**10**(1):87–111.
10. Preter M, Tzourio C, Ameri A, Bousser MG. Long-term prognosis in cerebral venous thrombosis. Follow-up of 77 patients. *Stroke* 1996;**27**(2):243–6.
11. Hinman JM, Provenzale JM. Hypointense thrombus on T2-weighted MR imaging: a potential pitfall in the diagnosis of dural sinus thrombosis. *Eur J Radiol* 2002;**41**(2):147–52.
12. Tong KA, Ashwal S, Obenaus A, Nickerson JP, Kido D, Haacke EM. Susceptibility-weighted MR imaging: a review of clinical applications in children. *AJNR Am J Neuroradiol* 2008;**29**(1):9–17.
13. Moulin T, Crepin-Leblond T, Chopard JL, Bogousslavsky J. Hemorrhagic infarcts. *Eur Neurol* 1994;**34**(2):64–77.
14. Wycliffe ND, Choe J, Holshouser B, Oyoyo UE, Haacke EM, Kido DK. Reliability in detection of hemorrhage in acute stroke by a new three-dimensional gradient recalled echo susceptibility-weighted imaging technique compared to computed tomography: a retrospective study. *J Magn Reson Imaging* 2004;**20**(3):372–7.
15. Adams Jr HP, Brott TG, Furlan AJ, Gomez CR, Grotta J, Helgason CM, et al. Guidelines for thrombolytic therapy for acute stroke: a supplement to the guidelines for the management of patients with acute ischemic stroke, A statement for healthcare professionals from a special writing group of the stroke council. *American Heart Association, Circulation* 1996;**94**(5):1167–74.
16. Roob G, Schmidt R, Kapeller P, Lechner A, Hartung HP, Fazekas F. MRI evidence of past cerebral microbleeds in a healthy elderly population. *Neurology* 1999 Mar 23;**52**(5):991–4.
17. Kwa VI, Franke CL, Verbeeten Jr B, Stam J. Silent intracerebral microhemorrhages in patients with ischemic stroke. *Amsterdam Vascular Medicine Group*. *Ann Neurol* 1998 September;**44**(3):372–7.
18. Thomas B, Somasundaram S, Thamburaj K, Kesavadas C, Gupta NK, Bodhey NK, et al. Clinical applications of susceptibility weighted MR imaging of the brain – a pictorial review. *Neuroradiology* 2008 Feb;**50**(2):105–16.
19. Kidwell CS, Saver JL, Villablanca JP, Duckwiler G, Fredieu A, Gough K, et al. Magnetic resonance imaging detection of microbleeds before thrombolysis: an emerging application. *Stroke* 2002 January;**33**(1):95–8.
20. Del Zoppo GJ VKR, Hamann GF. Ischaemic damage of brain microvessels: inherent risks for thrombolytic treatment in stroke. *J Neurol Neurosurg Psychiatry* 1998;**65**(1):1.
21. Nighoghossian N, Hermier M, Adeleine P, Blanc-Lasserre K, Derex L, Honnorat J, et al. Old microbleeds are a potential risk factor for cerebral bleeding after ischemic stroke: a gradient-echo T2*-weighted brain MRI study. *Stroke* 2002 March;**33**(3):735–42.
22. Schellinger PD, Fiebach JB, Hacke W. Imaging-based decision making in thrombolytic therapy for ischemic stroke: present status. *Stroke* 2003;**34**(2):575–83.
23. Tang PH, Chai J, Chan YH, Chng SM, Lim CC. Superior sagittal sinus thrombosis: subtle signs on neuroimaging. *Ann Acad Med Singapore* 2008;**37**(5):397–401.