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MAGNETIC RESONANCE

Focal hemodynamic patterns of status epilepticus detected by susceptibility weighted imaging (SWI)

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Abstract
Objective To investigate pathological findings in the susceptibility weighted imaging (SWI) of patients experiencing convulsive (CSE) or non-convulsive status epilepticus (NCSE) with focal hyperperfusion in the acute setting.
Methods Twelve patients (six with NCSE confirmed by electroencephalogram (EEG) and six patients with CSE with seizure event clinically diagnosed) underwent MRI in this acute setting (mean time between onset of symptoms and MRI was 3 h 8 min), including SWI, dynamic susceptibility contrast MR imaging (DSC) and diffusion-weighted imaging (DWI). MRI sequences were retrospectively evaluated and compared with EEG findings (10/12 patients), and clinical symptoms.
Results Twelve out of 12 (100 %) patients showed a focal parenchymal area with pseudo-narrowed cortical veins on SWI, associated with focal hyperperfused areas (increased cerebral blood flow (CBF) and mean transit time (MTT) shortening), and cortical DWI restriction in 6/12 patients (50 %). Additionally, these areas were associated with ictal or postical EEG patterns in 8/10 patients (80 %). Most frequent acute clinical findings were aphasia and/or hemiparesis in eight patients, and all of them showed pseudo-narrowed veins in those parenchymal areas responsible for these symptoms.
Conclusion In this study series with CSE and NCSE patients, SWI showed focally pseudo-narrowed cortical veins in hyperperfused and ictal parenchymal areas. Therefore, SWI might have the potential to identify an ictal region in CSE/NCSE.
Key Points
- *The focal ictal brain regions show hyperperfusion in DSC MR-perfusion imaging.*
- *SWI shows focally diminished cortical veins in hyperperfused ictal regions.*
- *SWI has the potential to identify a focal ictal region in CSE/NCSE.*

Keywords Magnetic resonance imaging · Perfusion imaging · Susceptibility-weighted imaging (SWI) · Status epilepticus · Non-convulsive status epilepticus

Introduction

The definition of a status epilepticus (SE) varies slightly among authors but is commonly accepted as a single unremitting seizure extraordinarily prolonged lasting longer than 5 min ('impending SE') or 30 min ('established SE') or repetitive clinical seizures without an interictal return to the baseline clinical state [1, 2]. SE may lead to a progressing and deepening impairment of consciousness and may become life threatening (mortality rates up to 18 % have been described [3]). While several typical clinical findings can occur in convulsive SE (CSE), i.e. prolonged focal or generalized myoclonus, a non-convulsive status epilepticus (NCSE) may present with much subtler and less specific signs. Indeed, NCSE is a heterogeneous disorder with multiple subtypes, e.g. absence status epilepticus, simple partial and complex partial status

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epilepticus or subtle status epilepticus [4]. Electroencephalogram (EEG), the diagnostic gold standard, may not be available in emergency situations in due time. Therefore the clinical diagnosis remains challenging, and NCSE is known to be underdiagnosed [5]. Recently, magnetic resonance imaging (MRI) and computed tomography (CT) have emerged as promising tools to help in diagnosing CSE and NCSE in the acute setting. In a few studies, a focal hyperperfusion of the affected cerebral region was demonstrated [6–11]. Additionally, the affected regions partly showed a diffusion restriction in some studies, presumably because the compensatory mechanism of hyperperfusion is insufficient to prevent the stimulation of anaerobic glycolysis due to ictal activity leading to apparent diffusion coefficient (ADC) reductions [10, 12, 13]. However, susceptibility-weighted imaging (SWI) has not yet been evaluated in detail for SE. As a useful diagnostic sequence, SWI has been widely applied, e.g. for detecting or evaluating intracranial haemorrhage, calcification, cerebral venous thrombosis, tumour evolution [14, 15] or haemorrhagic transformation of stroke [16–18], using the parametric properties of deoxyhaemoglobin. SWI has been described inter alia to be helpful in detecting occult calcified lesions or vascular malformations that may be responsible for seizures [19]. The role of SWI has not yet been evaluated in the acute ictal setting with respect to alterations in cortical vein appearance due to perfusion changes. It is assumed that the deoxyhaemoglobin content in veins correlates with their prominence and darkness in SWI due to their paramagnetic properties. Focusing on cortical veins in SWI, their prominent and dark appearance has been described in stroke patients in perfusion-disturbed regions after thromboembolic occlusion [17, 18, 20, 21], owing to increased deoxyhaemoglobin levels, secondary to an increased extraction fraction of oxygen by the ischemic parenchyma. Further, prominent-appearing cortical veins have been described in patients with hemiplegic migraine correlating with hypoperfusion [22, 23]. Since focal parenchymal areas are expected to be hyperperfused during seizure activity, an opposite effect with correlating pseudo-narrowing of cortical veins would be expected in case of a lower deoxyhaemoglobin content, and therefore decreased paramagnetic properties. To our knowledge only one case report by Lee et al. [24] has described this phenomenon. Our retrospective study was undertaken to investigate whether pseudo-narrowed or pseudo-diminished cortical veins in SWI are a finding that could be diagnostically useful for detecting ictal activity. Further, we evaluated a presumed correlation between the location of pseudo-narrowed cortical veins in SWI with findings in DWI, DSC, EEG and clinical symptoms.

Materials and methods

Patient data

The study was approved by the local ethics committee. Inclusion criteria for this retrospective study were a clinically or electrophysiologically confirmed CSE or NCSE (NCSE selected cases were all by definition EEG-proven). Mandatory MRI sequences were DSC, DWI and SWI sequences according to the domestic emergency/stroke protocol and showing a focal decreased mean transit time (MTT) in DSC in the colour-coded maps. Patient data were excluded if image quality was poor, e.g. as a result of motion artefacts. A total of 12 patients (eight female and four male; age range, 1–87 years; mean, 34.5 years) fulfilled these criteria. Ten of 12 subjects had received EEG in between 47 min and 31 h 38 min after symptom onset, and prior or after MRI (time range 4 h 15 min before and 21 h 8 min after MRI acquisition).

Patients were referred to our neuroradiology department from the emergency department where they had been evaluated by neurologists. They were referred for acute neurological symptoms and/or to rule out stroke. Five of 12 patients were intubated (four children and one adult).

Data acquisition

Imaging studies were performed using a 1.5-T and a 3-T Siemens system (Magnetom Avanto, Magnetom Trio, respectively; Siemens Medical Solution, Erlangen, Germany) with a 12-channel head coil. The MRI protocol performed was our local emergency/stroke protocol, which includes the following sequences: axial DWI, axial T2 SE, ToF angiography, perfusion imaging, contrast-enhanced angiography of the cervical and intracranial arteries, and an axial T1 SE post-contrast. For the 1.5-T MRI the SWI parameters were TR 49 ms, TE 40 ms, number of averages 1, FoV read 230 mm, FoV phase 81.3 %, voxel size 1.1×0.9×1.8 mm, flip angle 15°, acquisition time 2:59 min. Perfusion imaging parameters (DSC) were TR 1,410 ms, TE 30 ms, number of averages 1, FoV read 230 mm, FoV phase 100 %, voxel size 1.8×1.8×5.0 mm, flip angle 90°, acquisition time 2:00 min. DWI parameters were TR 3,000 ms, TE 89 ms, number of averages 4, FoV read 230 ms, FoV phase 100 %, voxel size 1.2×1.2×5.0 mm, acquisition time 1:35 min. For the 3-T scanner the SWI parameters were as follows: TR 27 ms, TE 20 ms, number of averages 1, FoV read 230 mm, FoV phase 75.0 %, voxel size 0.9×0.9×2.0 mm, flip angle 15°, acquisition time 2:59 min. A standard perfusion imaging sequence was used with following parameters (DSC): TR 1,400 ms, TE 29 ms, number of averages 1, FoV read 230 mm, FoV phase

Table 1 MRI parameter overview

	TR (ms)	TE (ms)	FOV (mm)	Voxel size (mm)	Flip angle (°)	Acquisition time (min)
Sequence @1.5 T						
SWI	49	40	230	1.1×0.9×1.8	15	02:59
DSCE	1,410	30	230	1.2×1.2×5	90	02:00
DWI	3,000	89	230	1.2×1.2×5	no	01:35
Sequence @3 T						
SWI	27	20	230	0.9×0.9×2	15	02:59
DSCE	1,400	29	230	1.8×1.8×5	90	01:59
DWI	3,500	89	230	1.8×1.8×4	no	01:15

100 %, voxel size 1.8×1.8×5.0 mm, flip angle 90°, acquisition time 1:59 min. DWI parameters were TR 3,500 ms, TE 89 ms, number of averages 4, FoV read 230 ms, FoV phase 100 %, voxel size 1.8×1.8×4.0 mm, acquisition time 1:15 min (Table 1). For both scanners tracer concentration–time curves of the perfusion sequence were analysed using Siemens workstations to obtain parametric colour-coded maps of MTT, time-to-peak (TTP), relative cerebral blood flow (rCBF) and relative cerebral blood volume (rCBV). Further, the SWI and minimum intensity projections (mIP) images were generated automatically by the scanner software.

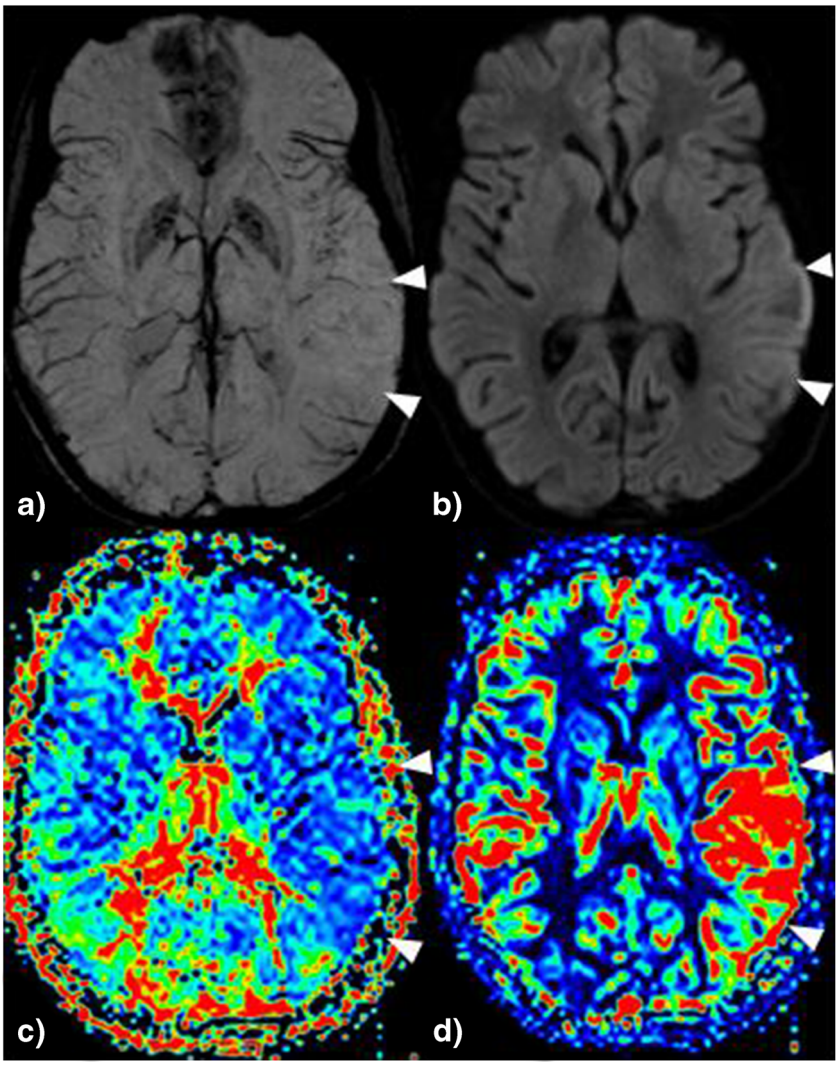

Fig. 1 22-year-old woman with MELAS syndrome and acute global aphasia. **a** In SWI pseudo-diminished cortical veins are seen temporal left. **b** In association subtle left cortical diffusion restriction and a focal hyperperfusion with **c** MTT shortening and **d** increased rCBF (see Table 2, patient 3)

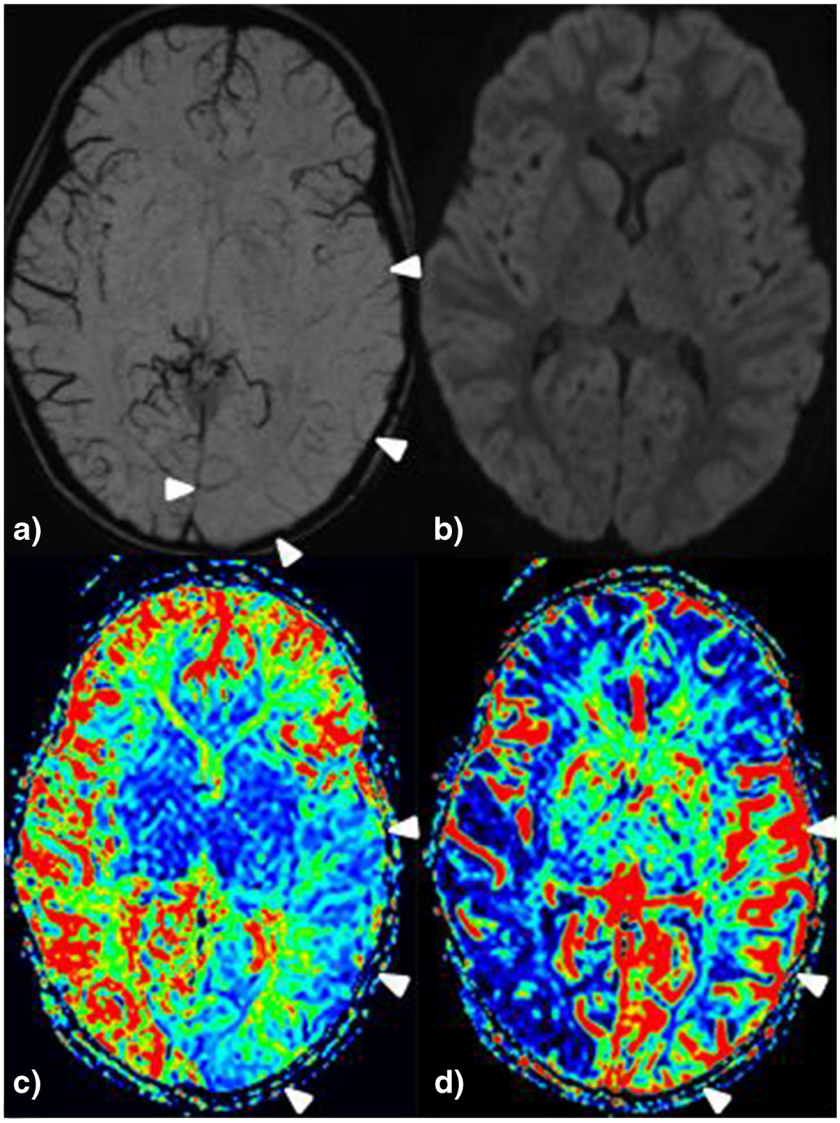

Fig. 2 4-year-old girl with fever seizure and clinical status epilepticus. **a** Pseudo-narrowed or pseudo-diminished cortical veins are found temporal-parietal and occipital left without diffusion restriction (**b**, but hyperperfusion in MTT (shortened) and rCBF (increased), see **c** and **d**; (see Table 2, patient 5)

Data analysis

To evaluate MRI sequences, two neuroradiologists were blinded to patient history, except for the information of a current clinically and/or electroclinically confirmed SE and a focal parenchymal area with decreased MTT, without knowledge of the exact location or the focal neurologic signs if present. All MRI sequences were evaluated separately by the neuroradiologists on our picture archiving and communication system (PACS) in a standardized order, first SWI, then rCBF and MTT images, and last DWI images. If cortical veins in SWI were undetectable or less visible in a parenchymal area compared to the cortical veins of the opposite hemisphere the area was noted as positive.

After visual evaluation of DSC data (colour-coded perfusion maps), areas of shortened MTT, increased rCBF and finally areas with diffusion restriction in DWI images were noted.

EEG results of the ten patients with EEG were analysed, and if epileptiform signals (epileptiform discharges (ED), periodic lateralized epileptiform discharges (PLED) or signs compatible with postictal changes (focal or general slowing)) occurred, the region with these alterations was noted in the same manner. Since all findings were positive (pathological) or negative (normal) we used percentage values for description in this small sample without statistical analysis.

Finally all the observed pathological SWI, DWI and DSC findings and the EEG findings were compared to each other and the clinical findings.

Results

Twelve patients fulfilled the inclusion criteria. In six patients NCSE was confirmed with EEG, all with focal ictal findings.

Among the remaining six patients with CSE, four underwent EEG within a mean interval time of 21 h 8 min, one showed ictal and three postictal EEG changes. In all 12 patients, a focal area with side asymmetrical calibre of cortical veins in the SWI sequence was found, indicating pseudo-narrowing. These areas were associated in all 12 patients with focal hyperperfusion (shortened MTT and increased rCBF, see Fig. 1). Six patients out of 12 (50 %) showed a cortical diffusion restriction within the same anatomical territory (see Figs. 2 and 3 as example), five of them with corresponding clinical symptoms (hemiparesis or aphasia). The other six patients had no cortical diffusion restriction. Two patients showed an additional diffusion restriction in the pulvinar of the thalamus, with no correlation in the SWI or perfusion sequences.

Six patients had EEG confirmed NCSE (four with epileptiform discharge, ED; and two with periodic lateralized epileptiform discharges, PLED). The localization of all areas with epileptiform signals correlated with the focal disturbed areas in SWI and DSC.

Two of the six patients with clinically confirmed CSE did not undergo an EEG examination. The other four underwent EEG. Epileptiform discharges were only detected in one patient (PLED), which was regionally associated with pseudo-narrowed veins in SWI. The remaining three showed postictal findings in EEG with generalized slowing in one patient and focal slowing in two patients. In one of the two patients with focal wave slowing, postictal changes were again in the same localization of pseudo-narrowed cortical veins and hyperperfusion in SWI and DSC images, respectively. Besides seizure in CSE, the most frequent neurological symptoms were hemiparesis found in five patients and/or aphasia found in three patients. Again, areas of pathological SWI and DSC findings were associated with the parenchymal areas responsible for these two symptoms in all eight patients (Table 2).

Discussion

Pathophysiological changes in seizures are well described. SE causes excessive neuronal activity in the early stage and concomitant perfusion increases as a compensatory

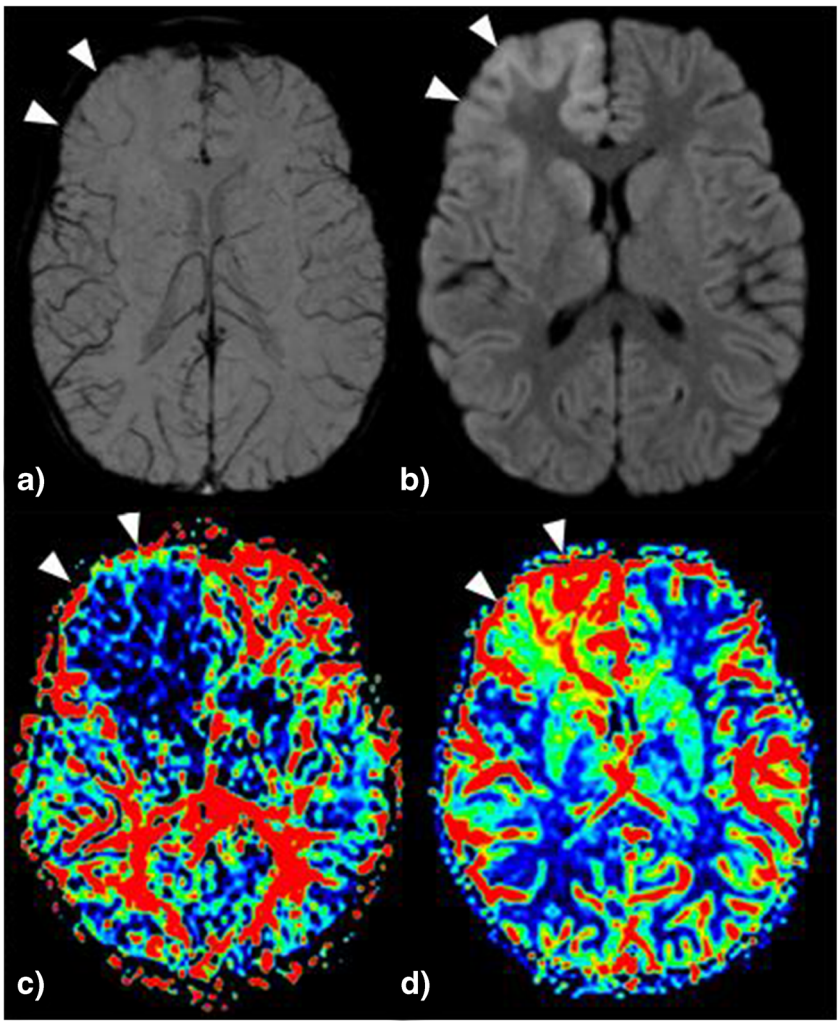

Fig. 3 4-year-old girl with fever seizure, Todd paresis left hemisyndrome. **a** Pseudo-narrowed or pseudo-diminished cortical veins are found frontal right with **b** correlating diffusion restriction and **c** hyperperfusion in the MTT (shortened) and **d** CBF maps (increased) (see Table 2, patient 4)

Table 2 Detailed information of patients

#	Age	Sex	Clinical history	Underlying disease as potential cause for the seizures	CBF	MTT	DWI	SWI	EEG findings	Time between symptoms onset and MRI	Time between MRI and EEG	Time between symptom onset and EEG
1	55	M	NCSE with aphasia, right motor hemisyndrome, NIHSS 10	No	Temporo-parieto-occipital left	Temporo-parieto-occipital left	Temporo-parieto-occipital left	Left hemisphere	Left temporo-occipital, FS, PLED	0 h 45 min	4 h 26 min	5 h 10 min
2	22	F	NCSE with global aphasia since 2 days	MELAS syndrome	Temporo-parietal left	Temporo-parietal left	Temporo-parietal left	Temporo-parietal left	Left temporo-occipital, FS, ED	2 h 35 min	1 h 48 min	0 h 47 min
3	4	F	CSE with seizure followed by Todd paresis (left hemisyndrome)	Fever	Frontal right and parietal left	Frontal right and parietal left	Frontal right	Frontal right and parietal left	NA	1 h	NA	NA
4	4	F	CSE with seizure	Fever	Temporo-occipital left	Temporo-occipital left	0	Temporo-occipital left	GS	10 h 30 min	21 h 08 min	31 h 38 min
5	1	F	CSE with seizure followed by unclear clinical picture	Fever	Temporo-parietal right, frontal right and left	Temporo-parietal right, frontal right and left	Temporo-parietal right, frontal right and left	Temporo-parietal right, frontal right and left	GS, left temporo-parietal PLED, two left temporo-parietal focal SZ	2 h 45 min	Minus 1 h 50 min	0 h 55 min
6	67	F	NCSE with right hemiparesis	No	Temporo-parieto-occipital left	Temporo-parieto-occipital left	Temporo-parieto-occipital left and thalamus left	Temporo-parieto-occipital left	Left fronto-temporal FS, ED	1 h 40 min	16 h 35 min	18 h 15 min
7	70	M	CSE with seizure followed by apathy	No	Left hemisphere	Left hemisphere	0	Temporo-occipital left	NA	2 h 25 min	NA	NA
8	10	F	CSE with seizure followed by right hemisyndrome and coma	No	Left hemisphere	Left hemisphere	0	Left hemisphere	right temporo-occipital FS	2 h 25 min	20 h 05 min	22 h 30 min
9	87	F	NCSE motor aphasia NIHSS 4	No	Temporo-parietal left	Temporo-parietal left	Temporo-parietal left and thalamus left	Left hemisphere	Left temporo-occipital FS, PLED	2 h 45 min	8 h 40 min	11 h 25 min
10	4	M	NCSE with loss of consciousness, gaze deviation to the right, tremor left	No	Right fronto-temporo-parietal	Right fronto-temporo-parietal	Right fronto-temporo-parietal	0	Right predominant intermittent generalized ED	5 h 10 min	Minus 4 h 15 min	0 h 55 min
11	6	F	CSE with seizure followed by hemisyndrome right and gaze deviation to the left	No	Left hemisphere	Left hemisphere	0	Left hemisphere	Right hemispheric FS	3 h 15 min	4 h 04 min	7 h 19 min
12	84	M	NCSE with complex focal status epilepticus	Small cell lung carcinoma with one brain metastasis frontal right	Parietal right	Parietal right	0	Parietal right	Right hemispheric ED	2 h 30 min	0 h 48 min	3 h 18 min

GS general slowing, *FS* focal slowing, *PLED* periodic lateralized epileptiform discharges, *ED* epileptiform discharges, *SZ* seizures, *NA* not available, *CSE* convulsive status epilepticus, *NCSE* non convulsive status epilepticus

mechanism to the increased metabolic demand, which results in higher levels of oxygenated haemoglobin [25–28].

Consequently this underlying pathophysiological mechanism leads to a net increase of oxyhaemoglobin supply, exceeding the demand, resulting in a net decrease in deoxyhaemoglobin content in the cortical veins. In studies investigating SWI, it is assumed that changes in venous deoxyhaemoglobin levels are proportional to changes of paramagnetic venous properties, resulting in pseudo-calibre changes of the affected cortical veins [17, 20, 21].

The observations of this study suggest an association between lower deoxyhaemoglobin levels in cortical veins and increased focal cerebral perfusion. In our retrospective analysis, pseudo-narrowing of cortical veins and ictal or postictal findings in EEG were in the same parenchymal area in eight out of ten patients (six patients with focal epileptogenic discharges and two with postictal focal wave slowing). The remaining two showed a generalized EEG slowing and a focal EEG slowing in the opposite hemisphere, respectively. Since EEGs were performed more than 4 h before and 20 h after MRI examination, and SE and NCSE are evolving disorders with highly dynamic changes affecting both clinical and electroencephalographic features [1], we assume that the epileptiform discharges had been widespread or multifocal in these subjects (see Table 2). In eight of eight patients with neurological symptoms of hemiparesis or aphasia, the responsible parenchymal areas correlated with the areas showing focal pseudo-narrowed cortical veins in SWI and focal hyperperfusion in DSC.

Overall, these results suggest that SWI has the potential for detecting ictal areas in early SE by the appearance of pseudo-narrowed or pseudo-diminished cortical veins in the affected areas, since pseudo-narrowed cortical veins in SWI were associated in all patients with parenchymal hyperperfusion by location in terms of focal decreased MTT and increased rCBF.

Diffusion restriction was present in only six of 12 patients, so that in our small sample, SWI had a higher detection rate than DWI for a supposed ictal region (for summary see Table 3).

Table 3 Summarized data of patients

Number of patients	12
Mean age (years)	34.5
Sex (female/male)	8/4
SWI (%)	100
MTT (%)	100
CBF (%)	100
DWI (%)	50
EEG findings (10 patients) (%)	80

SWI focal pseudo-narrowed cortical veins due to hyperperfusion, *MTT* associated focal hyperperfusion (MTT decrease) in colour-coded perfusion-maps, *CBF* associated focal hyperperfusion (CBF increase) in colour-coded perfusion maps, *EEG* associated focal ictal or postictal findings

Both diffusion restriction and the neurological symptoms of hemiparesis and aphasia are typical findings in acute stroke, whereas a prominent cortical vein appearance in one vascular territory can be found in SWI [18, 21]. NCSE is often underdiagnosed [5] and can clinically mimic an acute stroke or hemiplegic migraine and vice versa. Therefore, SWI may serve as a practical tool to further differentiate between these two pathologies, since the appearance of cortical veins may be the opposite: pseudo-diminished in case of hyperperfusion versus pseudo-prominent cortical veins in case of hypoperfusion. Additionally, in SE the pathological appearance of cortical veins may be in more than one vascular territory.

Our findings corroborate with findings in a patient recently described in a case report [24], where the whole right hemisphere was hyperperfused and cortical veins in SWI were pseudo-diminished during assumed NCSE.

Apart from this case report of Lee et al., very few data have been published about the phenomenon of pseudo-narrowed or pseudo-diminished cortical veins in SWI [29, 30]. SWI is a very sensitive technique for the detection of intravascular venous deoxygenated blood due to ischemia [31, 32]. Especially in stroke patients, pseudo-prominent cortical veins can be frequently seen in hypoperfused areas [17, 18, 20], caused by a disproportion between oxygen supply and demand resulting in a higher intravenous deoxyhaemoglobin content. Since in our study mainly small calibred or diminished cortical veins were detected in hyperperfused areas, this corroborates the thesis of an association between the extent of cortical vein appearance and deoxyhaemoglobin levels, inversely proportional to cerebral blood flow. This study has some limitations. First, the number of subjects, 12, is small, making further studies necessary with a larger number of patients to confirm our results. A prospective study design, where EEG is performed temporally close to MRI examination would additionally help in validating our results. Furthermore, we selected patients with SE or NCSE having a focally decreased MTT in perfusion imaging. This might lead to selection bias, since potential patients with postictal focal changes like hypoperfusion and increased MTT were eliminated. Gelfand et al. reported that MTT can be prolonged focally after seizure indicating a postictal condition with hypoperfusion [33]. As a consequence deoxyhaemoglobin levels are higher with prominent cortical vein appearance. A recently published case report [34] described three children in whom this phenomenon of focally prominent cortical veins in SWI was associated with epileptic focus localization in acute stage of epileptic encephalopathy. The authors suspected a higher deoxyhaemoglobin concentration. Therefore, caution is advised with the differential diagnosis of ischemic stroke, where a similar perfusion pattern can occur. Future studies should enrol patients with electroclinically confirmed SE/NCSE without regard to focal hyperperfusion. Another bias might be that five of 12 patients were intubated, since oxygen supply can generally reduce

appearance of cortical veins [35]. However, since in both intubated and non-intubated patients the area with focally reduced cortical vein appearance was surrounded by normal cortical vein appearance, this effect should be negligible.

In summary this study demonstrates an association of focal pseudo-narrowed or pseudo-diminished cortical veins in SWI and focal hyperperfusion in DSC with ictal and postictal signals in EEG, clinical findings and focal diffusion restriction in DWI in this small sample of patients presenting with SE or NCSE. The SWI findings can be explained by a decreased amount of deoxygenated blood and therefore lowered paramagnetic properties due to hyperperfusion of the ictal region. Therefore as a non-contrast sequence, SWI might be useful for detection of a focal ictal area in SE/NCSE.

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