

Case Report: Cerebral Amyloid Angiopathy (CAA) using Susceptibility-Weighted Imaging (*syngo* SWI)

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Background

With the development of a 3D gradient-echo (GRE) based susceptibility-weighted imaging sequence (*syngo* SWI), a neuroimaging MR technique is now available in clinical routine which maximizes tissue magnetic susceptibility and makes use of these differences to generate a unique contrast, different from that of proton density, T1, T2, and conventional T2* imaging we are used so far in clinical routine. Compared to other imaging techniques *syngo* SWI – a long TE flow compensated gradient echo imaging providing enhanced contrast with the combination of phase and magnitude information – has already provided superior results in clinical studies in detecting intracranial bleeding but also in depicting minute intracranial vascular malformations.

Cerebral Amyloid Angiopathy

Cerebral amyloid angiopathy (CAA) is a small vessel disease which is characterized by deposition of amyloid β protein within the cerebral arterioles. It is known that there is a clear association of CAA with the following aging, dementia, Alzheimer's disease, postradiation necrosis, and spongiform encephalopathies. But so far, no in vivo imaging

technique is available which enables either the direct visualization or quantification of the amyloid deposits. But as an indirect sign, typically microhemorrhages within and around the arteriole vessel wall lobar microbleeds are found and related to CAA. Usually CAA is involving the cortex and subcortical white matter within the frontal and parietal lobes. In contrast, hypertensive or atherosclerotic microangiopathy shows microhemorrhages in a deep or infratentorial location.

Sequence details

A 68-year-old patient with suspicion of TIA (transient ischemic attack) has been referred to our institution for imaging and to rule out further diseases of the brain. All images were acquired at 3 Tesla using a MAGNETOM Verio with the standard 12-channel head coil. Sequence parameters for shown images were:

T1 SE: TR 500 ms, TE 8.4 ms, FOV 230, matrix 256 / 95 % (interpolated to 512), SL 5 mm, TA 1:53 min:s, voxel size 1.0 x 0.9 x 5 mm

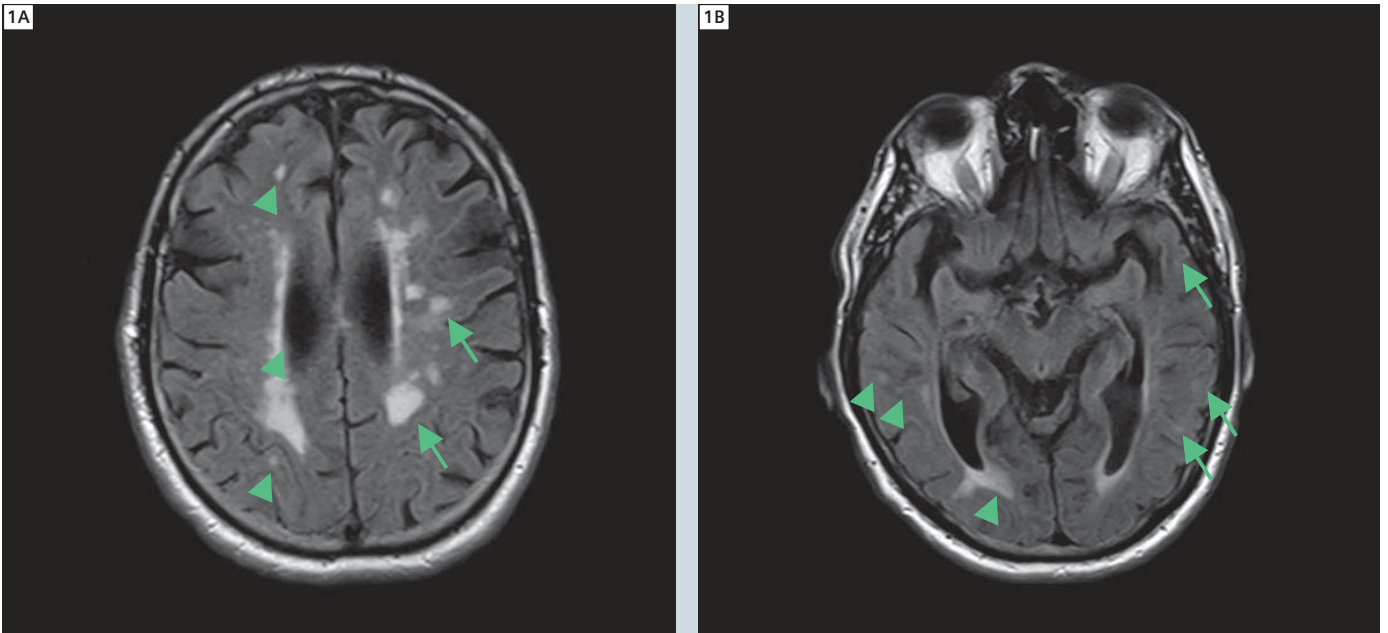
***syngo* SWI:** TR 27 ms, TE 20 ms, FOV 230 / 75 %, Matrix 256 / 95 % (interpolated to 512), SL 2.5 mm, TA 2:48 min:s, voxel size 0.9 x 0.9 x 2,5 mm. Phase and

magnitude images and the finally post-processed SWI are available for image analysis. Also, a thick-slice MPR (multiplanar reconstruction) which is generated Inline is available.

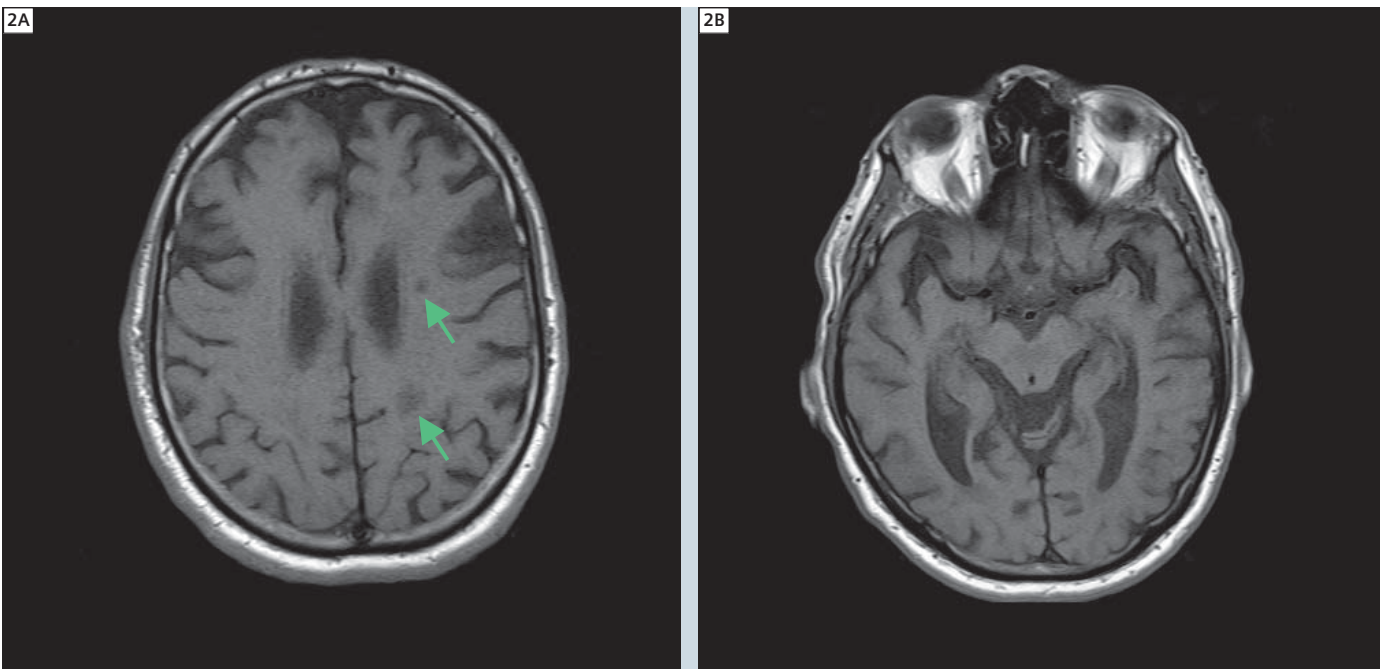
DarkFluid (FLAIR): TR 9000 ms, TE 94 ms, FOV 230 / 84 %, Matrix 256 / 95 % (interpolated to 512), SL 5 mm, TA 2:26 min:s, voxel size 1.0 x 0.9 x 5 mm

Imaging findings

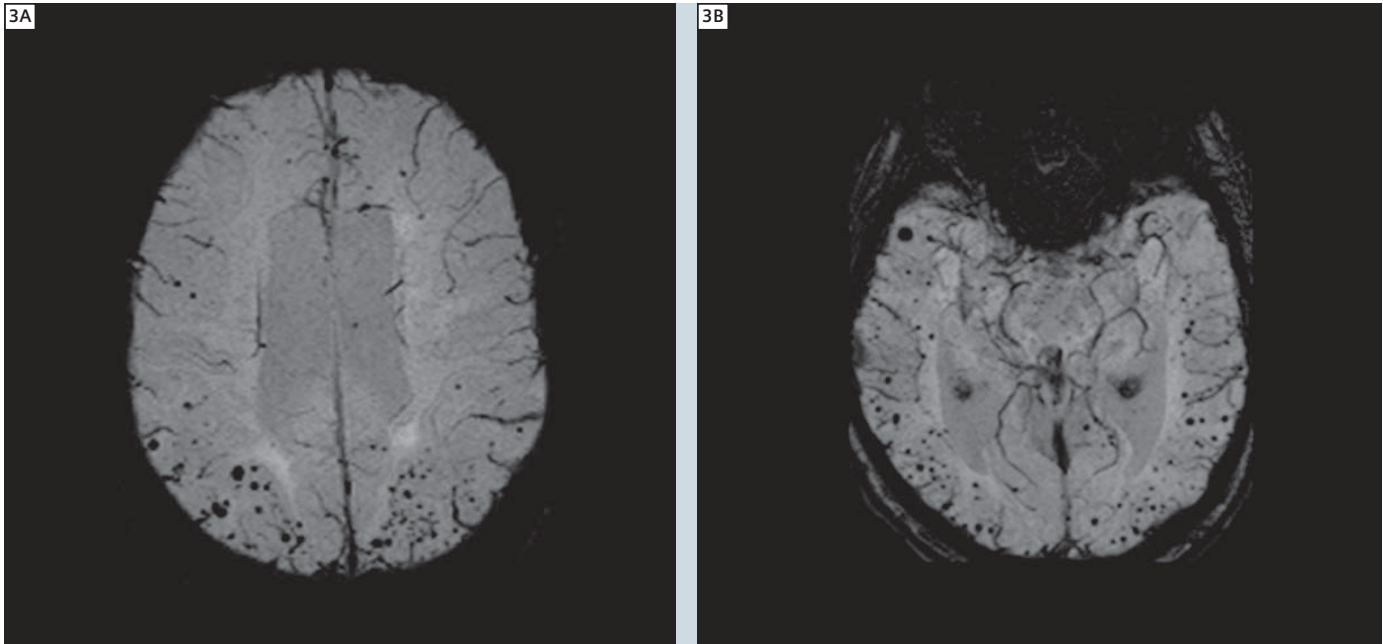
Multiple T2w hyperintense isolated foci in the periventricular white matter are shown on DarkFluid (FLAIR) images (arrows figure 1A). In addition, dorsal of the posterior horn and lateral ventricle converging hyperintense periventricular T2w hyperintense areas are shown, which can be interpreted as age-related periventricular gliosis (arrowheads figure 1). However, also in the temporal lobe cortical and subcortical T2 hyperintense spots with only slightly increased signal can be visualized by DarkFluid (FLAIR) imaging (arrows figure 1B). In addition, there is a widening of the internal and external cerebral fluid interspaces. On native T1w MRI, no hyperintense signal can be demonstrated; only in the case of the largest periventricular white-matter foci, a corresponding



1 DarkFluid (FLAIR) images of a patient with cerebral amyloid angiopathy.



2 Corresponding native T1-weighted images.



3 syngo SWI showing multiple cortical and subcortical bleedings.

hypointense lesion can be found. However, SWI looked completely different: multiple smallest cortical and subcortical bleedings were visualized in the temporal, parietal and less prominent in the frontal lobe (figure 3).

In conclusion the findings in our patient are a mixture of unspecific vascular / age related findings (periventricular gliosis, reduced brain volume, microinfarcts) and CAA. However, extent and severity of CAA is only visualized by *syngo* SWI in detail and would have been clearly underestimated based on conventional MRI only.

Conclusion

syngo SWI has shown in this case to be a sensitive tool for precise assessment of CAA. In general, SWI can provide useful additional information in the evaluation of various pediatric and adult neurologic conditions and can be incorporated easily into the routine imaging assessment. It is known that SWI is more sensitive in detection of small bleedings and small vascular malformations than conventional T2* imaging and that it is an imaging technique which is highly sensi-

tive to iron accumulation in the brain; this is observed in ageing process, reflection of brain damage, diseases of iron metabolism and haemorrhages. Iron involvement is already accepted in Hallervorden-Spatz disease, neuroferritinopathy, aceruloplasminemia, Friedreich's Ataxia. However, larger studies are still needed to determine the role of SWI in iron measuring especially in neurodegenerative diseases (Alzheimer's disease, Parkinson, ALS, and in Multiple Sclerosis).

References

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