

Added combined value of Pulse Arterial Spin Labelling (PASL) and Susceptibility Weighted Imaging (SWI) in cerebral vascular occlusion, stroke and recovery

M. VIALON¹, A. Federspiel², S. Altrichter¹, A. Syrogiannopoulou¹, R. Sztajzel³, M-I. Vargas¹, J. Delavelle¹, and K. O. Lövblad¹

¹Radiology, Hôpital Universitaire de Genève, GENEVA, Switzerland. ²Psychiatric Neurophysiology, University Hospital of Psychiatry, BERN, Switzerland, ³Neurology, Hôpital Universitaire de Genève, GENEVA, Switzerland

Introduction: MR protocols using a combination of diffusion, MRA and perfusion sequences have become the standard of care for patients with cerebrovascular diseases (1). But the assessment of perfusion status as well as parenchymal injury via diffusion imaging is not sufficient since the degree of tissue injury and hence the potential to save viable tissue depends also on the duration and degree of ischemia, the presence of micro-hemorrhages within the infarct zone, the presence of collaterals, and then the location of the occlusion. Advanced MRI techniques such as arterial spin-labelling (ASL) (2,3) and susceptibility-weighted imaging (SWI) have considerably be improved with the implementation of high field (3T) MR units (4). ASL was successfully used to detect decreases in CBF associated with brain pathology, relying on arterial blood water as an endogenous tracer. Compared with dynamic contrast enhance perfusion weighted imaging (ce-PWI), it is not only safer and more economical but ASL is still a valid technique to study perfusion even when the integrity of the BBB is broken, being potentially more robust to track CBF changes in longitudinal studies post ischemic events and recovery. We wanted to address if ASL and SWI added to a clinical stroke protocol could lead to improved diagnosis, prognosis and patient management.

Methods: 30 patients (13 female, 17 male; ages 40 to 88) with a diagnosis of cerebrovascular diseases patients were examined. 24 Acute or subacute stroke patients with new neurological deficits were imaged from 2 to 15 days after onset of symptoms. Six patients had severe carotid stenoses. Imaging was performed on a 3.0 T MAGNETOM TRIO (Siemens, Erlangen, Germany) using a 8 channel head coil. **SWI** 3D acquisition with in plane resolution of 1x1x1mm and Perfusion Weighted Imaging using a **PASL** were added to clinically indicated MRI scans: axial T2 TSE, coronal T2 FLAIR, diffusion weighted imaging was derived from a 30 directions DTI scan (in line calculation of Trace, FA and ADC), MRA, cePWI (voxel size=1.7x1.7x5.0mm) after intravenous injection of 0.1mmol/kg Gadovist at 5ml/sec, and finally T1 GE post Gadolinium. For the **PASL** sequence, we used a **Fair QUIPSSII** perfusion mode (4) and the following parameters: 16slices, voxel size=3.4x3.4x6.0 mm, TA=5:55 min, lambda = 0.9 mL/g, alpha = 95%, TE/TR/T11/T12/T1(blood,3T)[ms] = 15/5000/700/1800/1496,19. RelCBF maps for ASL were calculated in-line by the MRI scanner, and off-line for ce-PWI using the Syngo Perfusion(MR) software (Siemens Medical Solutions).

Results: In the patients with acute stroke (n =18), ASL showed hypoperfusion in the same areas as cePWI did. In three cases there was additional hyperperfusion on ASL that cePWI did not detect. In the patients with a suspicion of stroke but no stroke (n =6) on DWI or PWI, ASL was also negative. In the patients with carotid stenosis, there was also a coherence between ASL and cePWI in all cases (n =6). SWI demonstrated loss of natural contrast (GM/WM) in the corresponding vascular territories (n=3), important signal loss in gliotic area related to tissue loss and increased vascular fraction. SWI depicted micro/macro-hemorrhages within the infarct area (n=8) much better than conventional T2* weighted sequence.

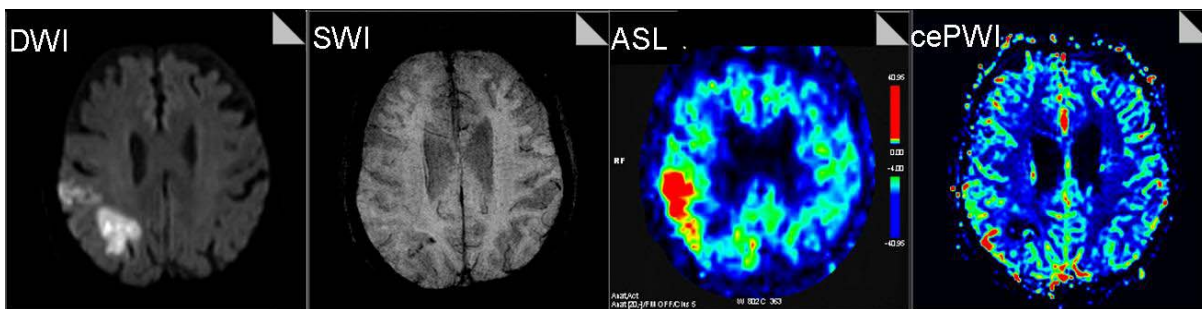


Figure1:Example of Ischemic lesions 5 days after stroke, hypoperfusion is found around lesion 1 (strong hypersignal on DWI, b=1000) on both cePWI and ASL, with localized microhemorrhage in SWI; lesion2 (moderate hypersignal in DWI) correspond to hyperperfusion in ASL non evidenced by cePWI, SWI evidenced loss of GM/WM contrast in corresponding area. No bbb leakage was evidenced on T1 post gadolinium.

Conclusions: ASL showed hypoperfusion in areas where a stroke was evidenced by cePWI, while not having the spatial resolution of cePWI. Areas of cortical reperfusion due to collaterals were better assessed by ASL that seems to demonstrate hyperperfusion that might signal recanalization and/or collaterals. Moreover ASL correctly measure CBF even when BBB breaks down, which commonly occurs after a few days in stroke. SWI identifies micro-hemorrhagic changes better than conventional T2* imaging. Thus performing this extended protocol allows to better include/exclude patients for treatment: impacting on prevention by demonstrating hypoperfusion in carotid stenosis and improving treatment of ischemia by showing hemodynamic compromise. Finally due to increased concerns related to nephrogenic systemic fibrosis, performing perfusion studies without contrast are of great interest.

References:

1.Lövblad KO. Eur Radiol.;16(6):1253-69 (2006) 2.Detre JA, Alsop DC. EJR 30 115– 124 (1999) 3.Gunther M. MRM 56:671– 675 (2006) 4.Wang J et al. JMRI 18:404-413 (2003)

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