Lack of Incidental DWI Hyperintense Lesions in Healthy Elderly Individuals

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INTRODUCTION

In MRI examinations of the brain in normal elderly controls, non-specific periventricular and subcortical T_2 -hyperintense lesions are a common finding, occurring in about 30% of patients over 60 years of age. The nature and clinical significance of these white matter hyperintensities is still controversial. Among various explanations for these findings are perivascular demyelination, ischemia as well as gliosis [1-2]. DWI is sensitive to acute tissue changes in cerebral ischemia and has also been shown to sensitive to other acute pathologies that cause reduction of the apparent diffusion coefficient (ADC) or T_2 -shine through effects. DWI is commonly seen as a means to differentiate acute from chronic pathology. Given the high prevalence of T_2 -hyperintense lesions we were interested whether incidental hyperintense DWI lesions may occur in elderly normal controls. We investigated 72 elderly individuals aged 65 years or more with no previous history of neurological dysfunction and normal results in neurological examination, who volunteered to take part in this study.

METHODS

MRI was performed with a clinical 1.5T MRI systems (Siemens VISION) according to the following protocol:

1. Transverse, coronal, and sagittal localizing sequences followed by transverse oblique contiguous images (slice thickness 5 mm) aligned with the inferior borders of the corpus callosum (sequences 2.-5.). 2. Proton density (PD)- and T_2 -weighted (TSE 2620 ms/14 ms/85 ms, FOV 180×240 mm2, matrix size 192x256). 3. FLAIR (9000 ms/105 ms/2340 ms) 4. T1 - weighted SE (530 ms/ 12 ms, FOV 180×240 mm2, matrix size 192x256) 5. DW EP-SE (TR 4000 ms/TE 110 ms, b = 0/1000 s/mm2, FOV 240 mm2, matrix size 128x128, sequential application of 3 separate diffusion sensitizing gradients in perpendicular directions). Maps of the ADC were obtained by a linear least-squares fit on a pixel-by-pixel basis after averaging of the direction-dependent DW images. The directionally independent trace of the diffusion tensor (ADC/3) was determined. WML T₂-lesion load was specified and graded according to the scheme developed by Fazekas into punctate (grade 1), early confluent (grade 2), and confluent (grade 3) abnormalities [2]. The DWI scans were analyzed independently by two different experts (K.S., A.G.). Trace DWI and single direction DWI were evaluated for the presence of hyperintense lesions. Areas that were difficult to interpret in regard to the presence of hyperintense abnormal signal vs. normal signal variation were recorded.

RESULTS

In the 72 patients, the WML load was graded 1 in 29 cases, 2 in 27 cases, while grade 3 in 16 patients. None of these patients showed hyperintense DWI lesions, neither on trace images nor on single diffusion direction images. However, slight signal variation on DWI images was noted on single diffusion direction images in areas of high anisotropy (e.g. splenium of the corpus callosum) or in areas of the combination of high anisotropy and T_2 shine-through from subcortical chronic lesions. On systematic analysis these areas were not suspicious of acute lesions and in all cases lack of hyperintensity on trace DWI confirmed this interpretation.

DISCUSSION and CONCLUSIONS

In a large cohort of asymptomatic elderly normal controls with incidental WML grades 1-3, no hyperintense signal abnormality was identified on DWI. Both, single diffusion direction and trace DWI images showed no areas suspicious of acute signal change. The knowledge of anisotropy effects and T_2 shine through phenomena is important and trace DWI may be necessary to confirm findings from intrinsically heterogeneuos signal on single diffusion direction images, that appear more difficult to interpret. On the basis of these results, the occurrence of hyperintense lesions on DWI should appear as a much more specific finding indicating acute pathology, as it has been identified in many stroke studies. Furthermore the lack of DWI hyperintense lesions in this cross-sectional study may be viewed as an indication that subcortical periventricular lesions are not the result of focal ischemia leading to confluent lesions, but could be slowly evolving tissue degeneration not presenting with reductions of the ADC.

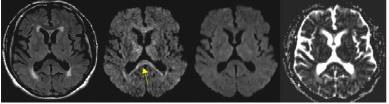


Figure 1: FLAIR images (a) of an 80-year old male with Grade 2 WML lesions. Single diffusion direction DWI (b), DWI trace image (c) and ADC map (d) show no signal abnormalities. Yellow arrow shows anisotropy artifact of the corpus callosum, not visible on trace image.

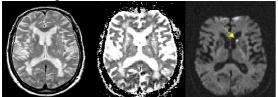


Figure 2: Example of a small ischemic acute lesion, that was the main target searched for in this study. T_2 -weighted image (a), ADC map (b), and trace DWI (c) in a patient with sudden onset abulia. Extensive chronic white matter and basalganglia lesions are noted on the T_2 -weighted image. A small acute ischemic lesion in the head of the caudate is noted on the ADC map and DWI (yellow arrow) well explaining the acute syndrome. References

[1] Fazekas F, Niederkorn K, Schmidt R, Offenbacher H, Horner S, Bertha G, Lechner H: White matter signal abnormalities in normal individuals: Correlation with carotid ultrasonography, cerebral blood flow measurements and cerebrovascular risk factors. Stroke 1988;19:1285-1288. [2] Schmidt R, Enzinger C, Ropele S, Schmidt H, Fazekas F; Austrian Stroke Prevention Study.Lancet. 2003 Jun 14;361(9374):2046-8. Progression of cerebral white matter lesions: 6-year results of the Austrian Stroke Prevention Study.

[3] Fazekas F, Kleinert R, Offenbacher H, et al. Pathological correlates of incidental MRI white matter signal hyperintensities. Neurology 1993; 43: 1683-89.