Postmortal DWI of the brain and comparison with in vivo data

Jin Yamamura[1], Tony Schmidt[1], Roland Fischer[2], Jerry Wang[3], and Gerhard Adam[1]
[1]*Diagnostic and Interventional Radiology, University Medical Center Hamburg-Eppendorf, Hamburg, Hamburg, Germany,* [2]*Children's Hospital and Research Center Oakland, Oakland, California, United States,* [3]*Department of Radiology, Children's Medical Center, University of Texas Southwestern Medical Center, Dallas, Texas, United States*

Introduction

In recent years, the diagnostic radiology has been playing an important role in the forensic medicine. Magnetic resonance imaging (MRI) was introduced in the forensic medicine as a second line tool, especially for identifying soft tissue injuries. Since the numbers of autopsies decreased in recent times, radiological imaging could play a significant role in the forensic medicine by adding important informationChanges in water diffusion can be quantified by diffusion-weighted MR imaging, e.g. to diagnose ischemic stroke. After an ischemic stroke, characteristic changes of water diffusion due to cell depolarization and cytotoxic edema can be assessed by DWI in the diagnosis of stroke and the selection of therapy. So far, there are few DWI investigations of corpses. But information about post mortem changes of ADC in the brain could be useful in forensic medicine, if they are characteristic and reproducible. In this study, we wanted to evaluate these post mortem ADC changes, to compare them to normal brain as well as stroke and to assess the role of ex vivo DWI as a forensic tool.

Material and Methods

The study protocol was approved by the local Ethics Committee, and informed consent was obtained from all relatives of the dead. Twenty-one patients, died of natural cause, were examined (13 male, 8 female; mean age: 70.5 ± 8.7 y.o.; range: 51 – 85 y.o., mean body weight 74 ± 18 kg). The corpses were stored at room temperature until the scanning. The core temperature was rectally measured throughout the MRI examination. MRI scans were started not later than 6 h post mortem and lasted for 24 h with scan intervals of one hour. To check the comparability of the ADC values a control group of 3 subjects (age: 24 – 67 y) was examined as well. All examinations were performed on a 1.5 T MRI (Magnetom Symphony; Siemens Medical Solutions, Erlangen, Germany) with an 8-channel-head-coil for brain imaging. For the morphological orientation a localizer as well as FLAIR- (TR 7900 ms, TE 105 ms, TI 2500 ms, slice thickness 0.5 cm) and HASTE-sequences (TR 1840 ms, TE 125 ms, FoV 350 mm, slice thickness 0.7cm) in transverse, coronal and sagittal orientation were performed. Based on the FLAIR images a diffusion-weighted spin echo echo-planar sequence was generated in transversal orientation to include the thalamus, cerebrum and cerebellum: TR 3000 ms; TE 87 ms; FOV 230 x 230 mm; matrix 192 x 256 mm; slice thickness 5 mm; gap 1.5 mm; voxel size 0.9x0.9x5 mm3; 20 slices. The morphological changes of the thalamus, cerebrum and cerebellum were assessed with the FLAIR images. Regions of interest (ROI) of 1.0 ± 0.03 cm2 size were manually drawn on the ADC-maps in each tissue region. Parametric statistics (arithmetic mean value ± standard deviation (SD), SEM, and Student's T-test) was used throughout this work with a significance value of $p < 0.05$ for group differences. In comparisons with our few control subjects (n = 3), the more robust discriminatory power test was used.

Results

In Figure 1, the DWI of the thalamus shows an initial decrease of the ADC from 44.5 · 10-5 mm2/s (2 h p.m.) to 30.3 · 10-5 mm2/s (4 h p.m.). Five hours post death there is an increase in ADC which reaches a value of 38.3 · 10-5 mm2/s. During the following hours the ADC decreases and reaches a minimum at 19 h p.m. (20.0 · 10-5 mm2/s). At the end of our examination, there is another increase of the ADC (35.0 · 10-5 mm2/s). In the cerebellum as well as the cerebrum there is a similar development of the ADC. After an initial decrease of the ADC, a maximum was reached five hours post death and during the following hours the ADC decreases and reaches a minimum after 19 hours with another increase of the ADC at the end of the examination.

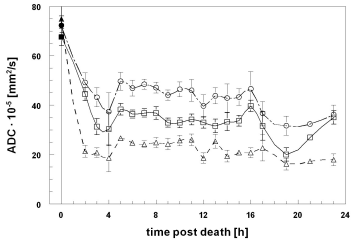

FIG 1. Time course of the ADC (mean ± SEM values) in post mortem cerebellum (circles), thalamus (squares), and cerebrum (triangles) with cubic spline functions as guidelines for the eye (dotted-dashed, solid and dashed lines, respectively) together with in vivo data from 3 normal subjects (solid symbols).

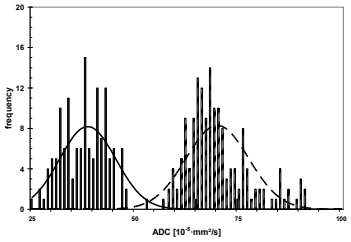

FIG 2. Pixel-wise frequency distribution of ADCs in ROIs inside ex vivo and in vivo thalamus (black and shaded bars, respectively) with fitted Gaussian curves (solid and dashed lines).

Furthermore we selected one representative corpse and pixel-wise compared the ADC distribution in the region of interest (ROI) with an in vivo patient of the control group. The mean ex vivo ADC in the thalamus was 38.5 ± 6.8 · 10-5 mm2/s. The frequency of the ADCs can be described by a Gaussian distribution. The mean in vivo ADC of the thalamus was 69.9 ± 7.6 · 10-5 mm2/s. Figure 2 shows the frequency distribution of the ADCs in ex vivo and in vivo thalamus with almost no overlap. It could be shown that there are two significantly different normal distributions ($p = 0.0146$) with a 1.8 times higher mean in vivo ADC. The range of ex vivo ADC values (25 - 75 · 10-5 mm2/s) varies more than in vivo (58 - 91 · 10-5 mm2/s).

On a larger scale, we compared the mean ex vivo ADC at a certain time (6 h p.m.) with our in vivo control group. In the cerebrum there was a significant difference between ex vivo ADC (24.8 ± 5.0) · 10-5 mm2/s and in vivo ADC (75.1 ± 4.1) · 10-5 mm2/s ($p < 0.001$). In the thalamus and cerebellum, the difference between ex vivo and in vivo ADCs was significant, too ((36.4 ± 5.7 versus 69.9 ± 7.6) · 10-5 mm2/s, $p < 0.001$ and (46.9 ± 11.2 versus 72.3 ± 3.8) · 10-5 mm2/s, $p = 0.045$, respectively).

Conclusion

It could be shown that ADC values of in vivo and ex vivo human brain differed significantly. Furthermore there is a characteristic postmortal time pattern of ADC which is similar to that after stroke. The course of ADC after death might be explained by the same cellular phenomena. Especially in the thalamus there is a strong decrease of ADC (55 %) from 2 to 19 h post death.

With the knowledge of the ex vivo data, DWI may be added to the MRI methods for a virtual autopsy (virtopsy). E. g., temperature effects will cause deviations from the obtained ex vivo data and may give hints to the storage conditions of a corpse after a crime.

To improve our understanding of the course of water diffusion, the time immediately after death as well as the reproducibility has to be examined.

Key words: Forensic Medicine, MRI, DWI