ADC, FA and T2-relaxation values in Cerebral White Matter of Hypoxic-Ischemic Neonates

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Introduction

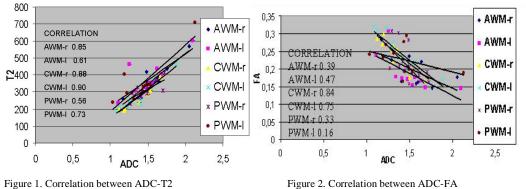
Neonates who suffer from perinatal hypoxic-ischemia (HI) may experience cerebral white matter (WM) injury, most vulnerable are the watershed areas in the anterior, central and occipital WM. This injury is not always detectable on conventional MRI. DTI and calculated T2 give more detailed information about tissue structure. In adults, ADC is decreased, FA is increased and T2 is not changed in the first hours to days after the insult. Thereafter, ADC and T2 increase to above normal and FA decreases to below normal values when tissue damage persists. These changes also occur in newborns, although the exact time-scale is not well known and may differ from adults. Conflicting results exists about the relation between ADC, FA and T2, in experimental and human stroke studies. A correlation between ADC and T2, as well as a correlation between T2 and FA are reported (1,2). We investigated the relation between ADC, FA and T2 in cerebral WM in 11 newborns with perinatal HI. We hypothesized that a correlation exists between ADC, FA and T2, as a result of the major changes of water in the intra- and extracellular compartment in the first 2 weeks after a HI insult.

Methods

Included were 11 term newborns with neonatal encephalopathy caused by perinatal HI, without typical stroke lesions on MRI. Excluded were perinatal infections, neuro-metabolic diseases and major congenital malformations. Perinatal HI was diagnosed when clinical symptoms of neonatal encephalopathy were present with 2 or more of the following risk factors: abnormal fetal heart rate pattern; umbilical artery pH <7.10; meconium stained fluid; Apgar score 5 minutes <7.

MRI was performed between day 5 and 17 after birth. Sedation with chloralhydrate was used in all patients. MRI protocol: Philips Gyroscan 1.0 Tesla, included T1, T2, IR-T1 and DTI images in the axial plane. T2 measurements with a double echo TSE sequence: TE1/TE2/TR = 12.6/120/4381 ms; slice thickness 4mm; slice gap 0.4mm. DTI with a single-shot EPI sequence and Pulsed Field Gradients in 6 directions; slice thickness 4mm; slice gap 0.4mm; voxel size 1.2x1.2x4mm; b-values 0, 400 and 800s/mm². The ADC, FA and T2 were calculated bilaterally in the anterior (AWM), central (CWM) and occipital white matter (PWM) at the level of the Centrum Semiovale. The region of interest (ROI) was hand drawn and was in the same range for the corresponding contralateral measurement (Fig3). ADC, FA and T2 calculations were done in Mathematica® software.





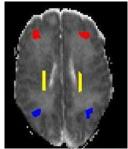


Figure 3. ROI selection

Mean values for ADC, T2 and FA in WM regions are shown in Table. In most patients,			
mean-ADC and m-FA are in the range of published normal values for term neonates			
(ADC 1.5 and FA 0.2) (3); mean T2 is significant higher than published normal values (217-			
228 msec) (4), but with a large sd. Comparison of normal values is hampered by the different			
techniques which are used in the studies. A significant time-dependant influence was only			
detected for T2 in the PWM-left ($r^2=0.63$; p<0.0001).			

	ADC 10 ⁻⁹ m ² /s (sd %))	T2 msec (sd %)	FA (sd%)
AWM	1.52 (16)	367 (26)	0.19 (23)
CWM	1.35 (14)	266 (26)	0.24 (19)
PWM	1.45 (15)	330 (29)	0.22 (21)

A strong correlation was observed between ADC and T2 (Fig1) in all WM regions

 $(r^2=0.56-0.90; p-values < 0.003)$. A correlation between ADC and FA (Fig2) was observed in 5/6 WM regions $(r^2=0.33-0.84; p-values < 0.04)$. T2 and FA showed a strong correlation with an inversed relationship only in the CWM $(r^2 > 0.62; p-values < 0.0001)$.

Discussion

This study shows that in most patients who suffered from hypoxic-ischemia, T2 in WM is increased compared to normal values. Also, a strong correlation between T2 and ADC exists. The T2 and ADC characteristics can be explained by an increased total amount of water in WM. The normal to high ADC values suggest that the increased amount of water in WM is particularly in the extracellular compartment and not intracellular. The etiology of this phenomenon remains unclear (vasogenic edema, membrane disruption), although the inversed relation between ADC and FA suggests that in cases with a high ADC and a low FA, membrane disruption might be the cause. In CWM, ADC and T2 are lower and FA is higher than in AWM and PWM. This can be the result of the more myelinated CWM and of the less vulnerability of the CWM for HI.

References

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