APPARENT DIFFUSION COEFFICIENT (ADC) AND MAGNETIZATION TRANSFER RATIO (MTR) IN PEDIATRIC HYPOXIC-ISCHEMIC BRAIN INJURY

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SUMMARY

Background and purpose: a review of the literature reveals the increasing interest in using Diffusion magnetic resonance imaging, with diffusion weighted images (DWI) and ADC (Apparent Diffusion Coefficient) quantitation, in pediatric hypoxic-ischemic brain injury. However, ADC and MTR (Magnetization Transfer Ratio) as quantitative tools have not been investigated together in these pathological conditions in young pediatric patients. The aim of this study was to apply a quantitative method by using ADC and MTR calculation in order to propose a reproducible quantitation of brain parenchymal lesions.

Methods: we conducted a prospective study including all children presenting with suspected cerebral hypoxic-ischemic injury. 15 children were included, among them 10 males and 5 females aged from 36 weeks of gestation to 17 months with a median age of 10.5 months. All MR examinations were performed at 1.5 Tesla unit including conventional MR (T1, T2 and Inversion-recovery sequences) and DWI with ADC map. ADC and MTR ROI (region of interest) measurements were made, in the frontal subcortical and periventricular white matter (WM) as well as in the gray matter (GM=basal ganglia), and in focal lesions.

Results: ADC and MTR values were abnormal in focal lesions and in diffuse injury with no evidence of lesion on conventional MRI and DWI. We observed a strong inverse correlation between these ADC and MTR (R=0.66 in WM; R=0.61 in GM).

Conclusion: ADC and MTR calculation may be helpful as a reproductive method to quantify the lesions and detect diffuse lesions in hypoxic-ischemic pediatric brain injury.

Key words: Diffusion weighted imaging, apparent diffusion coefficient, magnetization transfer, hypoxic-ischemic encephalopathy, magnetic resonance imaging.

INTRODUCTION

Diffusion weighted imaging (DWI) and ADC (Apparent Diffusion Coefficient) have been evalu-ated in the pediatric age group by several recent studies, especially in hypoxic-ischemic brain damage [3, 8, 16, 17, 24-26, 29, 37, 47, 48], and periventricular leukomalacia in neonates [6, 22]. The results of these studies tend to confirm the hypothesis that diffusion is superior to conventional Magnetic Resonance Imaging (cMRI) to detect brain injury.

Magnetization transfer imaging (MTI) has been shown to be sensitive for the detection of white matter abnormalities. The investigations about MTI in children reported in the literature mostly concern the detection of non visible white matter abnormalities in the early stage of Multiple Sclerosis (MS) and its prognosis [10, 13, 46]. MT has also been used in the evaluation of brain maturation [36, 45] and in Tuberous sclerosis [19, 23]. ADC and MTR were evaluated by Hanyu et al in adult stroke [21]. Nevertheless, there is no report of the use of both techniques as a quantitative tool in the context of hypoxic-ischemia brain injury in the pediatric population. Therefore, we elected to conduct a prospective study in order to add to DWI a quantitative approach of brain parenchymal lesions by using ADC and MT Ratio (MTR) calculation.

MATERIALS AND METHODS

Population

From November 2001 to April 2002, we systematically added Diffusion weighted imaging and MT to each MR examination performed in any patient in the pediatric age. The inclusion criteria were all patients presenting with suspected hypoxic-ischemic brain injury. The patients with brain metabolic disease, malformation or tumour were excluded. Initially, 26 patients were examined. Among them, 11 were secondarily excluded because of incomplete exams (sedation failure) or presence of too many movement artefacts on images. Finally, 15 patients were included in this study with 10 males and 5 females, aged from 36 weeks of gestation to 17 months with a median age of 10,5 months. Clinical information concerning birth (prematurity, Apgar score, weight and blood examination) and reason for imaging were recorded for each patient.

Imaging

MR examinations were performed using a 1.5 Tesla Gyroscan Intera (Philips system, Best, The Netherlands). Conventional MRI included T1-weighted (TR/TE: 420/12; slice thickness: 4mm; matrix: 179×256; FOV: 200mm), T2-weighted (TR/TE: 3500/110; slice thickness: 4mm; matrix: 234×512; FOV: 220mm), and Inversion-recovery sequences (FLAIR-T2w; TR/TE/TI: 8600/140/2100; slice thickness: 4mm; matrix: 203×256; FOV: 180mm and or IR-T1w; TR/TE/TI: 3600/23/400; slice thickness: 4mm; matrix: 295×512; FOV: 270mm). Acquisition time was about 35 minutes. Patients were sedated if necessary and monitored while within the magnet. The presence of a paediatrician inside the magnet room was not required. All patients were imaged within ten days after clinical onset.

Quantitative analysis

Conventional MRI, Diffusion-weighted and ADC map images (two sets) were reviewed by two experienced pediatric neuroradiologists, separately, in order to detect and interpret signal abnormalities. Consequently, the patients were divided in two different groups: the control group, including the patients with no signal abnormality on any of the images in agreement with both neuroradiologists and, the patients group including the others (signal abnormality found on one or both images sets by at least one of the neuroradiologists).

Quantitative analysis (post-processing)

ADC measurements were directly obtained by drawing ROI (region of interest) on ADC maps. ROIs were manually drawn with an electronic cursor (implemented in the Philips gyroscan system, Release 8) in the frontal subcortical and periventricular white matter and in the caudate nucleus, putamen and thalami for the gray matter, bilaterally (figure 1). MTR measurements were made in the same ROIs (location of the MT pulse, using a gradient echo 3D off-resonance acquisition (TR/TE: 37/4,4; slice thickness: 4mm; matrix 256×256; FOV: 230mm). The MT effect was calculated by using the MT ratio: MTR (%) = [1-Ms/Mo]×100 where Mo and Ms are the intensities of signal respectively before and after MT pulse (11). Acquisition time was about 6 minutes.

Total acquisition was about 35 minutes. Patients were sedated if necessary and monitored while within the magnet. The presence of a paediatrician inside the magnet room was not required. All patients were imaged within ten days after clinical onset.

Fig. 1. – Axial T2 weighted image in a control case showing the locations of the ROI (region of interest) drawings where ADC and MTR were systematically calculated: subcortical and periventricular for the white matter and, caudate nucleus, putamen and thalamus for the gray matter.

Fig. 1. – Image pondérée T2 dans le plan axial chez un cas du groupe contrôle, démontrant la localisation des régions d’intérêt (ROI) où le CDA et le RTA ont été systématiquement calculés : zones sous-corticales et pérventriculaires pour la substance blanche, caudales, putaminales et thalamiques pour la substance grise.
**TABLE I.** – The control group with cases and indications for imaging, conventional and Diffusion imaging outcome (NSA = no signal anomaly).

<table>
<thead>
<tr>
<th>Cases</th>
<th>Sex</th>
<th>Age (months)</th>
<th>Birthweight (grams)</th>
<th>Appgar score (1/5/10 minutes)</th>
<th>Reasons for imaging</th>
<th>cMRI</th>
<th>DWI + ADC map</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>(40 weeks)*</td>
<td>1400</td>
<td>7/8/8</td>
<td>Prematurity</td>
<td>NSA</td>
<td>NSA</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>2,75</td>
<td>2170</td>
<td>9/9/10</td>
<td>Deep faintness</td>
<td>NSA</td>
<td>NSA</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>5,50</td>
<td>2620</td>
<td>–</td>
<td>Developmental delay</td>
<td>NSA</td>
<td>NSA</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>13,00</td>
<td>–</td>
<td>–</td>
<td>Seizures</td>
<td>NSA</td>
<td>NSA</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>17,00</td>
<td>–</td>
<td>–</td>
<td>Seizures</td>
<td>NSA</td>
<td>NSA</td>
</tr>
</tbody>
</table>

(* = gestational age.

**TABLE II.** – The patients group with indications for imaging, conventional MR and Diffusion weighted imaging outcome of each patient. Signal abnormalities on cMRI often corresponded to hyperintense T2/FLAIR and hypointense T1 lesions; depending on the case, signal abnormalities on DWI corresponded to hyperintense or hypointense lesions, respectively hypo- and hyperintense on ADC map.

<table>
<thead>
<tr>
<th>Cases</th>
<th>Sex</th>
<th>Age (months)</th>
<th>Birthweight (grams)</th>
<th>Appgar score (1/5/10 minutes)</th>
<th>Reasons for imaging</th>
<th>cMRI</th>
<th>DWI and ADC map</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>F</td>
<td>(36 weeks)*</td>
<td>1450</td>
<td>9/9/9</td>
<td>Congenital infection</td>
<td>uncertain frontal SCWM anomalies</td>
<td>confirm frontal SCWM anomalies</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>(38 weeks)*</td>
<td>1300</td>
<td>7/9/9</td>
<td>Acute respiratory distress</td>
<td>bilateral PVWM anomalies</td>
<td>bilateral PVWM anomalies</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>0,20</td>
<td>3880</td>
<td>1/5/5</td>
<td>Fetal hypoxia</td>
<td>basal ganglia anomalies</td>
<td>basal ganglia anomalies</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>0,50</td>
<td>1885</td>
<td>8/9/9</td>
<td>Birth asphyxia</td>
<td>N</td>
<td>frontal SCWM and PVWM anomalies</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>0,50</td>
<td>3325</td>
<td>3/8/8</td>
<td>Fetal hypoxia</td>
<td>residual subependymal hemorrhage</td>
<td>subependymal anomalies</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>1,50</td>
<td>2500</td>
<td>9/9/9</td>
<td>“Shaken baby syndrom”</td>
<td>SDH, SAH, left temporal hematoma bitemporal cortical contusions extensive and ill-limited WM lesions</td>
<td>left temporal anomaly bitemporal and distal cortical lesions well delineated parenchymal extensive lesions</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>1,50</td>
<td>3100</td>
<td>–</td>
<td>Cyanotic cardiac malformation</td>
<td>ill-limited cortical subcortical parietal lesion basal ganglia anomalies</td>
<td>well delineated cortical subcortical parietal lesion basal ganglia anomalies</td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>2,00</td>
<td>2820</td>
<td>–</td>
<td>Trauma</td>
<td>left SDH and parietal contusions</td>
<td>subcortical left parietal anomaly</td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>3,00</td>
<td>–</td>
<td>–</td>
<td>Infection (meningitis)</td>
<td>no evidence of parenchyma lesion</td>
<td>left basal ganglia lesion</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>5,33</td>
<td>–</td>
<td>–</td>
<td>Peripheral hypertension</td>
<td>frontal and parietal WM anomalies</td>
<td>more precise parietal WM anomalies</td>
</tr>
</tbody>
</table>

(*) = gestational age. SCWM= subcortical white matter; PVWM= periventricular white matter; SDH= subdural hematoma; SAH= subarachnoid hemorrhage.

ROI size varied between 10.5 to 35mm² with same size in contralateral matching areas. To ensure that the measurements were valid, a ROI was systematically placed in the lateral ventricular CSF. When a focal lesion was detected during qualitative analysis, ADC and MTR were calculated inside and at the periphery of the lesion and also in the contralateral normal appearing area.

We decided to use the ADC and MTR median values obtained in our control group and the values measured in normal appearing brain parenchyma contralateral to the lesion, as normal reference with age correlation.

Statistical analysis

The “Mann and Whitney” non parametric test was used to evaluate the difference of ADC and MTR values between the control and the patients group. The p value under 0.05 was considered significant. The ADC-MTR correlation was evaluated by the Spearman test.

RESULTS

After the qualitative analysis, the children arrangement in control versus patients group is summarized in table I and II. In the tables, all ages are given with respect to the normal gestation term. The reasons of suspected hypoxic-ischemic brain injury or risk condition recorded are: prematurity (1), foetal hypoxia (2), birth asphyxia (1), seizures (2), deep faintness (1), cyanotic cardiac malformation (1), acute respiratory distress (1), trauma (1 fall and 1 shaken baby syndrome), infections (1 congenital infection and 1 meningitis), and abnormal neurological examination (2), the latter including, peripheral hypertonia and developmental delay.

Five children were categorized normal in the control group and, 10 were abnormal and categorized in the patients group. Among these 10 children, the focal parenchymal lesions were more obvious and better delineated in 6 cases (8, 11-15; see table II). In four cases (6, 7 9, 12 and 14), some lesions were uncertain or missed on T2WI but were clearly demonstrated on DWI, confirmed by the ADC map. Some cases are illustrated on figures 2, 3 and 4.

With regards to the quantitative aspects, the ADC and MTR values in white and gray matter in the control group are presented on (Table III). There was a significant difference between these values and the values recorded in the patients group (p<0.05). The ADC and MTR values were abnormal in all focal lesions on diffusion (Table IV). The ADC value in focal lesions was lowered compared to the symmetric contralateral normal appearing area. Conversely, the MTR values were elevated in the same ROIs, except in case 14. In diffuse injuries, ADC and MTR values were compared to their respective median values obtained from the control group. ADC was increased in white matter damage and decreased in gray matter involvement. MTR values vary conversely except in case 8 where both were lowered. The Spearman test (R) showed a strong inverse correlation between ADC and MTR (figure 5) among the patients group (R= −0.67) as well as among the control group (R= −0.42).

DISCUSSION

Hypoxia or ischemia may damage brain by causing focal lesions or diffuse injury. In our study, the change on DWI in focal lesions was a hypersignal (hyposignal on ADC map) with a decrease of ADC value. Diffusion technique is based on random water motion [27, 28]. Several studies in adult, neonates...
Fig. 3. – Case 12: Cyanotic cardiac malformation; a1) on T2WI the recent left parietal infarction is evident but not well delineated (double arrow); b1) and c1) Diffusion and ADC map show clearly the margins of the focal infarct (double arrow) and a small focal lesion in the left frontal subcortical area (single arrow), not evident on T2 (in the lesion ADC=50.92±9.54 and MTR=44.8; normal contra lateral respectively 154.44±7.26 and 40.47); a2), b2) and c2) diffusion and ADC map reveal another peripheral focal cortical infarct (arrow) undetectable on T2.

Fig. 3. – Cas 12 : Malformation cardiaque cyanogène ; a1) sur l’image en pondération T2, l’infarctus pariétal gauche récent est évident mais ne présente pas des limites nettes (double flèche) ; b1) et c1) la Diffusion et la cartographie CDA montrent avec plus de précision les limites de l’infarctus (double flèche) ainsi qu’un petit infarcissement focal sous-cortical frontal gauche (flèche unique), mal individualisée sur le T2 (dans la lésion CDA=50.92 ± 9.54 et RTA = 44.8 ; en zone contro-latérale d’apparence saine respectivement 154.44 ± 7.26 et 40.47) ; a2), b2) et c2) la diffusion et la cartographie CDA révèlent des infarcissements focaux périphériques (flèche) indétectables sur l’image T2.

Fig. 4. – Case 14: Purulent meningitis; a) on T2WI it is very difficult to detect any signal abnormality in the basal ganglia area, even if a subtle hyposignal on left thalami could be suspected (arrow); b) the anomaly is more evident on DWI (arrows) and on c) ADC map (arrows) (left thalamic ADC=72.15±8.93 and MTR=44.09; in normal contralateral respectively 98.21±8.42 and 55.45).

Fig. 4. – Cas 14 : Méningite purulente ; a) sur l’image pondérée T2, il est difficile d’objectiver une anomalie de signal au niveau des noyaux de la base, même si un discret hyposignal peut être suspecté au niveau du thalamus gauche (flèches) ; b) l’anomalie de signal est plus évidente sur l’image pondérée en diffusion (flèches) et sur c) la cartographie ADC (flèches) (Thalamus gauche CDA = 72.15 ± 8.93 et RTA = 44.09 ; dans les régions contralatérales d’apparence normale, respectivement 98.21 ± 8.42 et 55.45).
and infants have established that some signal changes are visible early on DWI in acute hypoxic-ischemic brain lesions [7, 11, 42, 48]. As demonstrated in animals, this is due to the restriction of extracellular water motion by cell swelling resulting from cytotoxic oedema [9, 18, 38, 40]. The ADC decrease in ischemia remains incompletely understood, but could partly be explained by cytotoxic edema. The results of some recent studies support the hypothesis that Diffusion is efficient in pediatric brain as in adults [31, 41, 42, 48]. However, we know that newborn and infant brain differs from adult about the global water content and that brain maturation and myelination will influence free water distribution [2, 15, 33, 43]. Then, normal ADC is age-related and the same for its pathological variations [5, 30, 34, 44]. Several authors concluded that ADC calculation can provide an objective measurement of hypoxic-ischemic brain injury [41, 48]. Time course study of diffusion signal abnormalities and ADC values inside lesions revealed a hypersignal on DWI and low ADC at early stages of ischemic insult and increase of diffusion at late stages[1, 32, 39]. Our findings corroborate that DWI is more sensitive than eMRI in detecting focal brain hypoxic-ischemic injury and lesions can be quantified by measuring ADC values. This quantification could be optimised by adding another tool like MTR. In the particular case of shaken babies, the interest of DWI has been well demonstrated [3, 4]. ADC and MTR measurements can provide a comparative database for further examination, so that the outcome could be predicted more accurately. In our series, the same correlation was found also in patients with normal preterm infants revealed a hypersignal on DWI and low ADC at early stages of ischemic insult and increase of diffusion at late stages. Our findings corroborate that DWI is more sensitive than eMRI in detecting focal brain hypoxic-ischemic injury and lesions can be quantified by measuring ADC values. This quantification could be optimised by adding another tool like MTR. In the particular case of shaken babies, the interest of DWI has been well demonstrated. ADC and MTR measurements can provide a comparative database for further examination, so that the outcome could be predicted more accurately. In our series, the same correlation was found also in patients with normal preterm infants revealed a hypersignal on DWI and low ADC at early stages of ischemic insult and increase of diffusion at late stages. Our findings corroborate that DWI is more sensitive than eMRI in detecting focal brain hypoxic-ischemic injury and lesions can be quantified by measuring ADC values. This quantification could be optimised by adding another tool like MTR. In the particular case of shaken babies, the interest of DWI has been well demonstrated.
appearing brain parenchyma (Table III). One hypothesis to understand this inverse correlation is that, as mentioned above, the exchange rate will be increased by the limitation of water motion. Nevertheless, further experimental studies could provide more details for a better understanding. Our results support that MTR is applicable as an additional tool to quantify brain lesions in pediatric hypoxic-ischemic injury.

The diagnosis of diffuse injury remains difficult and should take into account the age and brain maturation. The calculation of ADC and MTR values may be helpful in detecting diffuse injury. In our study, we found increased ADC values in injured white matter areas. Similar findings have been reported by Counsell et al. [8]. This can be illustrated by case 9 (figure 6 and Table V). In that case, the DWI abnormalities needed to be confirmed by quantitative analysis. Only the marked difference between ADC and MTR values, compared to the median values in the control group, allowed the conclusion that there was diffuse injury of the frontal white matter. Nevertheless, we cannot definitely exclude that these quantitative variations may simply be due to brain maturation. Thus, brain damage could be quantified in a coherent way by using these two different parameters: ADC and MTR.

A comment could be made on cases 8 and 14 when considering the MTR values in the lesion and in normal appearing contralateral parenchyma or median values in control group (Table IV). As statistically forecasted, MTR should increase when diffusion is restricted. Paradoxically, in these cases, MTR decreases as well as ADC in basal ganglia (case 8) and in the left thalamus (case 14). We did not find any explanation to explain these observations and do not know if cellular density, higher in grey matter compared to white matter, could play a role in this phenomenon.

Our preliminary results should be confirmed by evaluation of larger patient populations. Our study presented some limitations due to the heterogeneous population sample and small number of patients. The multishot diffusion technique is more prone to

<table>
<thead>
<tr>
<th>Cases</th>
<th>Controls</th>
<th>Focal lesions (DWI and ADC map)</th>
<th>Diffuse injury (DWI and ADC map)</th>
<th>ADC (10-5 mm²/sec) (mADC)</th>
<th>∆ADC (%)</th>
<th>MTR (%) (mMTR)</th>
<th>∆MTR (%)</th>
</tr>
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<tr>
<td>(1-5)</td>
<td></td>
<td></td>
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<tr>
<td>6</td>
<td></td>
<td>frontal SCWM</td>
<td>165.58±8.13</td>
<td>49.30 up</td>
<td>31.14</td>
<td>37.58 down</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td></td>
<td>frontal PVWM</td>
<td>186.96±14.82</td>
<td>68.58 up</td>
<td>37.78</td>
<td>24.27 down</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td></td>
<td>bilateral basal ganglia anomalies</td>
<td>92.31±8.22</td>
<td>16.76 down</td>
<td>46.72</td>
<td>6.35 down</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td></td>
<td>frontal SCWM fronto PVWM</td>
<td>131.93±18.84 160.93±21.77</td>
<td>18.96 up 45.11 up</td>
<td>38.97</td>
<td>21.88 down</td>
<td>18.01 down</td>
</tr>
<tr>
<td>10</td>
<td></td>
<td>subependymal anomalies</td>
<td>133.91±23.44</td>
<td>13.29 down</td>
<td>42.83</td>
<td>12.41 up</td>
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<td>11</td>
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<td>left temporal injury</td>
<td>72.21±8.48</td>
<td>35.68 down</td>
<td>46.70</td>
<td>3.18 up</td>
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<td></td>
<td></td>
<td>diffuse bilateral basal ganglia injury</td>
<td>80.06±6.19</td>
<td>27.80 down</td>
<td>50.79</td>
<td>1.80 up</td>
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<tr>
<td>12</td>
<td></td>
<td>cortical subcortical parietal lesion</td>
<td>50.92±9.54</td>
<td>67.02 down</td>
<td>44.80</td>
<td>10.70 up</td>
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<tr>
<td>13</td>
<td></td>
<td>left parietal anomalies</td>
<td>92.94±9.98</td>
<td>10.15 down</td>
<td>53.29</td>
<td>17.56 up</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td></td>
<td>left basal ganglia lesion</td>
<td>72.15±8.93</td>
<td>26.53 down</td>
<td>44.09</td>
<td>20.48 down</td>
<td></td>
</tr>
<tr>
<td>15</td>
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<td>parietal WM anomalies</td>
<td>118.00±12.21</td>
<td>6.40 up</td>
<td>47.06</td>
<td>5.67 down</td>
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</tr>
</tbody>
</table>

Table IV. – Quantification of focal and diffuse brain injuries by ADC and MTR calculation; ∆ = percentage variation of the pathologic value (Vp) related to the normal reference value (Vr); Vp is obtained in the center of the lesions; Vr is obtained in the contralateral normal appearing matching area for focal lesions and corresponds to the median value (mADC and mMTR) for diffuse involvement; [∆ = (1 - Vp/Vr) × 100].

Tableau IV. – Quantification des lésions cérébrales focales et diffuses par calcul du CDA et du RTA, avec les pourcentages de variation des valeurs pathologiques intralésionales par rapport aux valeurs de référence normales obtenues en zone contralatérale d’apparence saine (pour les atteintes focales) ou correspondant aux valeurs médiane obtenues dans le groupe contrôle (pour les atteintes diffuses).
movement artefacts. The single shot technique with shorter acquisition time could resolve this problem. Otherwise, ADC and MTR values considered normal in patients with no abnormal signal on cMRI and DWI are somewhat arbitrary. We should include more normal cases to get a reliable normal database.

CONCLUSION

Our preliminary results indicate that coupling ADC and MTR measurements may be useful, as a reproducible method, to confirm focal lesions and to detect diffuse brain injury with a strong inverse correlation between both values.
substance blanche frontale périventriculaire est suspectée sur la diffusion (hypersignal sur la carte CDA) (figure 6) ; la comparaison des valeurs de CDA et de RTA avec les valeurs moyennes respectives obtenues dans le groupe contrôle (classe 1) montrent une différence significative (p<0,00016 pour le CDA ; p<0,00021 pour le RTA). La concordance entre la Diffusion, la cartographie CDA et les valeurs anormales de CDA et de MTR est en faveur d’une atteinte bilatérale de la substance blanche frontale.

TABLEAU V. – Dans le cas 9, l’IRM conventionnelle ne démontre pas d’anomalie de signal. Un discret hyposignal de la substance blanche frontale périventriculaire est apparent sur la diffusion (hypersignal sur la cartographie CDA) (figure 6) ; la comparaison des valeurs de CDA et de RTA avec les valeurs moyennes respectives obtenues dans le groupe contrôle (classe 1) montrent une différence significative (p<0,00016 pour le CDA ; p<0,00021 pour le RTA). La concordance entre la Diffusion, la cartographie CDA et les valeurs anormales de CDA et de MTR est en faveur d’une atteinte bilatérale de la substance blanche frontale.

<table>
<thead>
<tr>
<th>FPVWM</th>
<th>ADC (10-5 mm²/sec)</th>
<th>MTR (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>right</td>
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<tr>
<td>Case 9</td>
<td>174.48±16.75</td>
<td>180.93±21.77</td>
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<tr>
<td>Control</td>
<td>mADC p&lt;0.00016</td>
<td>110.90±30.75</td>
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**RÉFÉRENCES**

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