Case report

DIFFUSION WEIGHTED IMAGING OF CEREBELLAR LESIONS IN WERNICKE’S ENCEPHALOPATHY

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SUMMARY

We report unusual findings on MR imaging in a 62-year-old woman with Wernicke’s encephalopathy (WE). Initial fluid-attenuated inversion recovery (FLAIR) and diffusion weighted MR imaging (DW-MRI) showed hyperintense lesions in the cerebellum and medial thalami, with a decreased apparent diffusion coefficient (ADC) in the cerebellum (reduced by 45%). After thiamine supplementation, the T2 and diffusion hyperintensities disappeared. However, clinical examination at three months showed persistent cerebellar impairment. The importance of the ADC values should be further investigated.

Key words: diffusion weighted magnetic resonance imaging, Wernicke’s encephalopathy, apparent diffusion coefficient, cerebellum.

INTRODUCTION

Wernicke’s encephalopathy (WE) from thiamine deficiency has been well characterized on MR imaging [6]. Few reports have focused on the prognostic value of DW-MRI in WE [1, 2, 4, 7, 15]. Here we report unusual cerebellar low ADC and T2 abnormalities in WE that reversed on serial MRI after treatment. However, the radiological recovery was not associated with clinical improvement.

Case report

A 62 year-old alcoholic woman presented with sudden-onset gait ataxia and dysarthria. She reported gait disturbances and disorientation over a few years and drinking 1.5 liters of wine daily with a severely restricted diet. On examination, she was mildly confused, dysarthric, with truncal ataxia and bilateral cerebellar tremor. The upper and lower limbs were symmetrically hypotonic and weak, and deep tendon reflexes were absent in the lower limbs. There was no nystagmus or gaze palsy. She had severe truncal ataxia and could not stand without assistance. Cerebrospinal fluid was normal and the electroencephalogram had fast rhythms without paroxysms. MRI examination (1.5T GE system) with axial T2 FLAIR (2.532/80/1) and diffusion-weighted images (EPI; 6.600/160/1; field of view 275mm; matrix 128x128, b values = 0 and 1.000s/mm$^2$ on the three spatial axes) revealed hyperintense lesions of the medial thalami, vermis and paravermian superior cerebellum (figure 1a, 1b, 1c and figure 2a). Relative ADC values were obtained from corresponding regions of interest (ROIs) from affected and healthy areas. Mean ADC values of 3x3 pixels of the ROI in the cerebellum were low (520 to 540x10^{-6}mm$^2$/s) compared to healthy cerebellum (880x10^{-6}mm$^2$/s) (figure 2c). Mean ADC values of the thalamus lesions were likely meaningless because of their small size and close proximity to the third ventricle. T1-weighted slices before and after contrast administration (TR/TE/NEX 720/14/2) showed a non-enhancing hypointensity of the cerebellar lesions. Mild cerebellar atrophy was present.

The patient received high-dose IV thiamine (1000mg per day). Follow-up MRI (DW-MRI and FLAIR) 15 days after treatment showed complete resolution of the cerebellar and thalamic lesions (figure 1b, 1d and figure 2b) and normalization of ADC values (figure 2d). The confusion disappeared but cerebellar symptoms persisted at three months.

DISCUSSION

In the present case, there were MRI lesions in the vermis and paravermian superior cerebellum (figures 1a, 1b, 1c and figure 2a), in addition to the classic lesions in the paramedian thalami associated
FIG. 1. – Initial DWI and FLAIR images revealed hyperintense lesions of the medial thalami vermis and paravermian superior cerebellum (a, c). Follow-up MRI performed 15 days after thiamine therapy showed lesion reversibility (b, d).

FIG. 1. – L’IRM initiale en séquences de diffusion et FLAIR montre des anomalies hyperintenses du thalamus médian et de la région para-vermienne supérieure du cervelet. (a, c). L’IRM réalisée 15 jours après la supplémentation en thiamine montre la réversibilité des anomalies (b, d).

FIG. 2. – Initial DW-RMI image shows hyperintense cerebellar lesions (a). Mean ADC value of affected cerebellum was low (520 to 540 x 10⁻⁶ mm²/s) (arrow) in comparison to healthy cerebellum (880 x 10⁻⁶ mm²/s) before thiamine therapy (c). After treatment, DWI hyperintense lesions disappeared and ADC value was normalized (b, d).

FIG. 2. – L’IRM de diffusion initiale montre une anomalie hyperintense du cervelet. (a). L’ADC moyen de cette anomalie est bas (520 to 540 x 10⁻⁶ mm²/s) (flèche) par comparaison avec le cervelet sain (880 x 10⁻⁶ mm²/s), avant traitement par thiamine (c). Après traitement, cet hypersignal en diffusion a disparu et l’ADC s’est normalisé (b, d).
with WE [1]. Cerebellar lesions on MRI are rare but have been previously reported [1, 14, 17]. Pathological studies show a higher prevalence of cerebellar lesions than on imaging studies, and some have suggested cerebellar involvement in more than half of WE cases [20]. Serial DW-MRIs in WE have shown different temporal patterns of cerebellar lesions, with some showing complete resolution [1] or partial reduction [14] of the lesions over time. Therefore, it is difficult to correlate clinical outcome with lesion severity.

The importance of ADC values in the diagnosis of WE is unclear, since decreased, normal or increased ADC values have been reported in WE [2, 4, 6, 8, 16]. Decreased ADC values in cerebellar lesions could reflect cytotoxic edema with irreversible lesions induced by thiamine deficiency [2, 4], suggesting a poor prognostic value. However, case reports of DW-MRI in WE do not confirm this hypothesis. These disparate patterns could be explained by different time windows of MRI examination. On the other hand, the pathophysiological process involved in WE may be similar to venous infarcts, where ADC values are variable and not correlated with the final outcome [5, 9, 13].

The regression of the FLAIR lesions was unexpected since the cerebellar symptoms persisted. This apparent “mismatch” between clinical and radiological outcomes has been previously reported [7]. It is possible that the reversibility of FLAIR lesions masks patchy neuronal loss. Consistent with this hypothesis, MR spectroscopy reported persistent increased lactate peaks and decreased N-acetylaspargate/creatinine ratios after thiamine treatment [14, 18].

CONCLUSION

Acute restricted cerebellar DWI lesions are possible in the setting of WE, however the importance of the ADC values should be further investigated.

RÉFÉRENCES