DIFFUSION-WEIGHTED IMAGING IN A CASE OF WERNICKE ENCEPHALOPATHY

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SUMMARY

In a chronic alcoholic patient with progressive confusion, which was consistent with the clinical diagnosis of Wernicke encephalopathy, T2-weighted, FLAIR and diffusion weighted (DWI) MR imaging depicted brain abnormalities located in both medial thalamic nuclei. Apparent Diffusion Coefficient (ADC) measurements in these regions showed unexpected normal values, referring to Wernicke pathological findings and DWI data. DWI may be helpful to diagnose early basal nuclei abnormalities, but may fail to compute ADC values in these locations.

Key words: MRI, diffusion-weighted imaging, Wernicke encephalopathy, basal ganglia.

INTRODUCTION

Wernicke encephalopathy is an unfrequent brain disease, caused by nutritional thiamine deficiency and occurring mainly in chronic alcoholics [8], even if some cases of associated cachectic patients (parenteral nutrition [5], gastroplasty) and children [15] have been reported. This encephalopathy has often been decribed in conventional T2-weighted or FLAIR MR imaging [2, 13, 14, 16], with typical findings such as hyperintensities in the mamillary bodies, surrounding the third ventricle and the aqueduct, and in the thalamic medial nuclei. But very little is actually known about its findings in Diffusion-Weighted Imaging (DWI) apart from one Japanese case report [17]. We describe a patient with Wernicke encephalopathy imaged with DWI 15 days after the neurological onset of symptoms.

CASE REPORT

A 36-year-old chronic alcoholic woman was admitted to the hospital for a 15 days progressive confusion. On admission, this patient was dehydrated and febrile (38°C). Blood pressure was 110/70 mm Hg with regular pulse of 95/min. Blood results showed hyponatremia (129 mmol/L), hypokalemia (2.3 mmol/L) and metabolic alkalosis (pH 7.63, PCO₂ 35 mm Hg, HCO₃ 39 mol/L, PaO₂ 69 mm Hg), with increased gamma Glutamyl Transferase to 5 Normal. She was admitted to the intensive care unit, and a CT scan performed few hours after the admission was normal. The clinical background was complicated with a Glasgow Score 3
coma two days after the admission, which rapidly came back to normal after treatment. The diagnosis of Wernicke encephalopathy was then established on clinical data. Three days after admission, the patient underwent a MR examination on a 1.5 T MR equipment (Siemens Magnetom Vision Plus). Four sequences were performed: 1) Axial Spin Echo T1-weighted: 20 slices, 5 mm thickness, 3 mm spacing, 512 × 144 matrix, 25 cm Field Of View (FOV), 450/12 [TR/TE], 70˚ flip angle, 63.6 kHz bandwidth; 2) Coronal Fast Spin Echo T2-weighted: 16 slices, 3 mm thickness, 3 mm spacing, 512 × 192 matrix, 25 cm FOV, 800/35 [TR/TE], 20˚ flip angle, 63.6 kHz bandwidth; 3) Axial Fast Fluid Attenuated Inversion Recovery (FLAIR): 20 slices, 5 mm thickness, 3 mm spacing, 512 × 182 matrix, 24 cm FOV, 9500/105/2340 [TR/TE/T1], 180˚ flip angle, 63.6 kHz bandwidth; 4) DWI: multislice single shot spin echo diffusion echo planar imaging with a pair of gradient centered around the 180˚ pulse, 5 mm thickness axial slices, 3.5 mm spacing, 128 × 64 matrix, 28 cm FOV, 312/151 [TR/TE], 63.6 kHz bandwidth. Four sets of 10 slices were acquired with two values of b (b = 0 and b = 825 s/mm²), with diffusion gradients applied simultaneously in 3 orthogonal spatial directions. The DWI raw images were transferred to an independent workstation, where a dedicated software allowed DWI averaged isotropic images generation and the quantification of the diffusion changes. Calculation of the Apparent Diffusion Coefficient (ADC) performed on a pixel-by-pixel basis created ADC maps. Small circular ROIs of 44 mm² (9 pixels) were centered on areas with abnormal signal on DWI, T2-weighted and/or FLAIR images to calculate mean ADC values. According to the extent of the lesions, two set of six ROIs were positioned in the brain areas with MR signal changes. Two set of five other ROIs were positioned symmetrically in the adjacent areas which appeared normal on both DWI and FLAIR sequences. Overall, 12 measurements were obtained in abnormal and 11 in normal appearing brain regions. ADC measurements were then averaged to obtain references ADC values.

There were bithalamic hyperintense T2-weighted, FLAIR and DWI, hypointense T1-weighted lesions which were mainly located in the medial nuclei (figure 1). ADC measurements in these nuclei shown normal or slight increased mean ADC values (right medial nucleus: 1.077 × 10⁻³ ± SD 0.064 × 10⁻³ mm²/s, left medial nucleus: 1.077 × 10⁻³ ± SD 0.058 × 10⁻³ mm²/s), compared with that of normal adjacent DWI appearing thalami (right thalamus: 0.945 ± SD 0.069 × 10⁻³ mm²/s, left thalamus: 0.960 ± SD 0.031 × 10⁻³ mm²/s). These areas of FLAIR hyperintensities which appeared normal on DWI, bilaterally in the insular cortex, and slightly surrounding the aqueduct and the mamillary bodies. No other focal areas of signal abnormality were found.

Hydroelectrolytic and thiamine deficiency corrections have partially drawn the confusion out, and the patient was transferred to a clinical unit 8 days after the admission. She escaped few days after without any imaging nor clinical follow up.

DISCUSSION

Wernicke encephalopathy is caused by thiamine deficiency [7], and mainly occurs in chronic alcoholics. The classic triad consists of ophthalmoplegia, ataxia and confusion, although these findings are not invariably present [8]. There is a characteristic MR distribution, with both gray and white matter involvement [13]. The periventricular regions, the medial thalamic nuclei, massa intermedia, third ventricular floor, and mamillary bodies are most frequently affected [3], and the periaqueductal region, midbrain reticular formation and tectal plate commonly involved.

T2-weighted and FLAIR images may reveal hyperintensities [2] in these locations, with Gadolinium contrast enhancement on T1-weighted imaging [4, 9, 12], sometimes surrounding the third ventricle and the aqueduct. Atypical locations may be found such as caudate nuclei, and focal cortical involvement [5]. These areas of abnormality may resolve in few days with ad integrum normalization of imaging findings [6].

Indeed, we observed selective T2-weighted and FLAIR hyperintensities on the both medial thalamic nuclei, which also appeared hyperintense on DWI. These DWI hyperintensities might be associated with the DWI T2 shine through [10]. Moreover, the ADC findings in the medial thalamic nuclei are quite unexpected. Pathologic findings include intracellular edema, demyelination, petechial hemorrhage, and astrocytic microglial proliferation [18]. Edema and gliosis are known to increase ADC values due to vasogenic oedema [11]. In that case, the medial thalamic nuclei ADC values are in the normal range compared with that of DWI normal appearing thalami. We have hypothesized that early correction of thiamine deficiency and hydrolelectrolytic troubles might have stopped the necrosis process.

Although, hyperintensities bilaterally seen on insular cortices are quite unusual in Wernicke encephalopathy [2, 13, 17], and might be correlated with local cortical anoxia (coma) [1]. The diagnosis was established on clinical basis. Unfortunately no clinical nor imaging follow up was available, the patient escaped few days after her transfer into the clinical unit. In this case, follow up MRI would have been interesting to correlate DWI and ADC values with the clinical outcome, especially in case of chronic atrophy or transient brain abnormalities [13], and to
see the expected normalization of areas of FLAIR hyperintensities.

Lastly, our method of ADC calculation has some pitfalls: we used relatively small ROIs in order to avoid partial volume effect on ADC values; however, ADC values calculated on a pixel-by-pixel basis were averaged over ROIs of 44 mm². This would mask local ADC changes since ROIs containing the same amount of pixels of low and high ADC values would result in normal averaged ADC. In addition,
we used ADC in the adjacent normal appearing brain matter as a reference for normal ADC values. Consequently, ADC values measured in gray and white matter were pooled to obtain normal ADC values. Ideally, ADC in abnormal gray or white matter should be compared to that of normal gray or white matter, respectively [5]. Unfortunately, when positioning a ROI in these small areas, it is often impossible to distinguish between gray or white matter.

**CONCLUSION**

DWI may be helpful to establish early the diagnosis and precise the extent of Wernicke encephalopathy in case of chronic alcoholic progressive confusion. It may fail to compute useful ADC values, and to differentiate from other basal ganglia diseases without clinical follow up data.

**REFERENCES**


