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°1 C). Blood pressure was 110/70 mm Hg with regular pulse of 95/mm. 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 Japanese case report [17]. We describe a patient with Wernicke encephalopathy imaged with DWI 15 days after the neurological onset of symptoms.
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 ophtalmoplegia, 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 mammillary 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 thalamus. We have hypothesized that early correction of thiamine deficiency and hydroelectrolytic 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 mm$^2$. 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 extend 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


