ΜΗ ΑΛΚΟΟΛΙΚΗ ΕΓΚΕΦΑΛΟΠΑΘΕΙΑ WERNICKE: ΜΙΑ ΑΝΑΣΤΡΕΨΙΜΗ ΔΙΑΤΑΡΑΧΗ ΠΟΥ ΔΕΝ ΠΡΕΠΕΙ ΝΑ ΔΙΑΦΥΓΕΙ ΤΗΣ ΔΙΑΓΝΩΣΗΣ

Βασιλική Ζούβελου[1], Γεώργιος Βελονάκης[2], Ειρήνη Πάντου[2]

[1] 1η Νευρολογική Κλινική, Αιγινήτειο Νοσοκομείο, Εθνικό και Καποδιστριακό Πανεπιστήμιο Αθηνών
[2] Μονάδα Έρευνας Ακτινολογίας, 1ο Ακτινολογικό Τμήμα, Εθνικό και Καποδιστριακό Πανεπιστήμιο Αθηνών

NONALCOHOLIC WERNICKE ENCEPHALOPATHY: A REVERSIBLE DISORDER YOU SHOULD NOT MISS

Vasiliki Zouvelou[1], Georgios Velonakis[2], Eirini Pantou[2]

[1] 1st Neurology Department, Eginition Hospital, National and Kapodistrian University of Athens, Greece
[2] Research Unit of Radiology, 2nd Department of Radiology, Medical School, National and Kapodistrian University of Athens, Greece

Key words: Wernicke, encephalopathy, nonalcoholic, MRI

A 72-year-old man was admitted with a two-day history of inability to stand, diplopia with horizontal nystagmus and apathy. His personal history included gastric surgery and vomiting for 5 days before the onset of symptoms. Alcohol intake was not reported. Nonalcoholic Wernicke encephalopathy (WE) was suspected and parenteral thiamine was immediately administrated.

Brain MRI was performed on a 3Tesla scanner at admission and two weeks after thiamine administration (Figure). MRI revealed T2-fluid-attenuated inversion recovery (FLAIR) bilateral and symmetrical hyperintensities in medial thalami (arrows A, arrow C), mammillary bodies (arrowheads B, empty arrow C), tectal plate and the periaqueductal area (empty arrow B, arrowhead C) and vermian hyperintensity (curved arrow C). No atrophy was found. The imaging findings were typical of WE [1]. Two days after treatment initiation, there was a dramatic recovery of ocular dysfunction while two weeks later the patient could stand without support and was mentally improved. Follow up MRI revealed complete lesions resolution except for partial resolution of the abnormalities involving the medial thalami (D).

WE is caused by thiamine deficiency. Although the most common causative factor is alcoholism, WE can occur in other nutritional deficiency states (e.g., gastric surgery, hyperemesis gravidarum, malignancy). Prompt diagnosis and timely administration of thiamine results in clinical recovery and radiological resolution of brain lesions [2].

References

[1] Sinha S, Kataria A, Kolla BP, Thusius N, Koukianova LL. Wernicke Encephalopathy-Clinical Pearls. Mayo Clin Proc 2019; 94: 1065-1072 doi: 10.1016/j.mayocp.2019.02.018

[2] Ha ND, Weon YC, Jang JC, Kang BS, Choi SH. Spectrum of MR imaging findings in Wernicke encephalopathy: are atypical areas of involvement only present in nonalcoholic patients? AJNR Am J Neuroradiol 2012; 33:1398-402 doi: 10.3174/ajnr. A2979

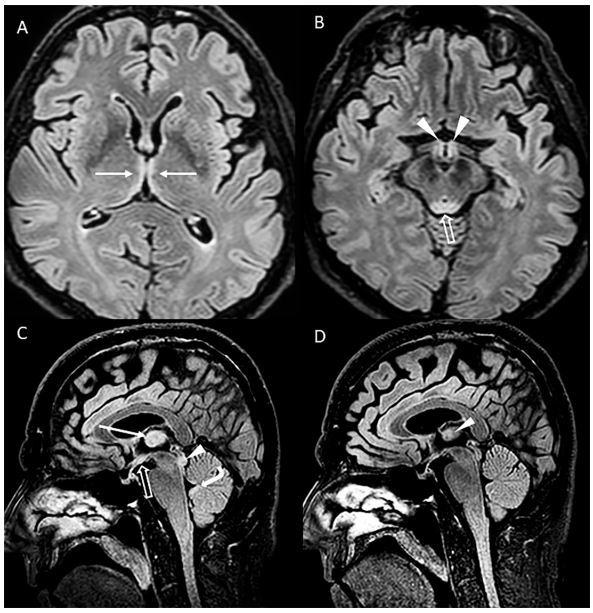

Figure:
Brain MRI in Wernicke Encephalopathy