Case Report

Left Cerebellary Toxoplasmosis with No Immunodeficiency: Case Report

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Abstract

Toxoplasmosis is caused by an infection with an obligate intracellular parasite, Toxoplasma gondii. The patients are usually immunocompromised. It causes a silent infection in healthy individuals. Trimethoprim sulfamethoxazole is the most common drug used for toxoplasmosis.

A fifty year old male applied to our out-patient department with the complaint of right hemiparesis, walking difficulty, ear humming and facial asymmetry on May 2010. Magnetic resonance imaging revealed a left cerebellary mass lesion. He was operated and the pathological findings were consistent with 'toxoplasmosis'; although we could not demonstrate an immunodeficiency in this patient.

Keywords

Cerebellar mass; Toxoplasmosis; Immunocompromised patient

Introduction

Toxoplasma gondii is an obligate, intracellular, opportunistic parasite causing a life-threatening disease 'toxoplasmosis'; especially in acquired immunodeficiency syndrome (HIV) and immunocompromised patients. Cerebral white and grey matter, retina, lung alveolar layer, heart and skeletal muscle are the most common affected areas. Here, we report a case of cerebral toxoplasmosis who did not exhibit immunodeficiency.

Case

A fifty year old male admitted to Uludag University, School of Medicine neurosurgery out-patient department on May 2010 with the complaint of right hemiparesis, walking difficulty, ear humming and facial asymmetry that persisted for two months. The neurological examination revealed ataxia, left cerebellary incapacity and left peripheral facial palsy (House Brackmann Grade III). The laboratory examinations were normal.

Magnetic resonance imaging (MRI) confirmed a peripheral enhancing mass lesion (18×22 mm) representing hyperintense hemorrhagic areas in T1-weighted images; hyperintense perilesional edema in fluid attenuated inversion recovery (FLAIR) images and hyperintense hemosiderin content in T2-weighted images in left middle cerebellar peduncle extending through pons and medulla oblongata (Figure 1A-1C).

Discussion

Toxoplasma gondii infection can be diagnosed indirectly with serological methods and directly by PCR, hybridization, isolation, and histology [2]. Serology is frequently positive, but specificity is low because only one third of the cases show a high titered IgG antibody [3], and only half of the cases show intrathecal antibody production for the agent [4]. Therewithal, the recent studies have denoted low sensitivity for polymerase chain reaction for toxoplasma gondii in plasma and cerebrospinal fluid [5,6] and occasional false-positive results [7]. In addition to all of these handicaps; the neuroradiological findings are also helpful for the diagnosis of toxoplasmic encephalitis. On the other hand, for a diagnosis, suspicion about the multiple lesions on MRI is required primarily.
Toxoplasmosis lesions are most commonly located in the cerebral hemispheric white and subcortical gray matter, such as thalamus and basal ganglia [8,9]. The characteristic finding is the asymmetric ‘target sign’ to which MRI is more sensitive than computed tomography. Typically, toxoplasmic lesions are iso-hypointense on T1-weighted images with rim enhancement and have hyperintense foci on T2-weighted images [10]. If there are multiple intracerebral lesions with mass effect or contrast uptake, some authors assert that the positive predictive value of MRI for toxoplasmosis can reach up to 100%; especially if at least one of these lesions is located in the basal ganglia or in the thalamus [11] but this is not pathognomonic to cerebral toxoplasmosis. Single toxoplasmic lesions are mentioned as ‘infrequently’ in literature [12,13].

Perilesional edema is associated with patient’s ability of inflammatory response formation [14]. The more edema formation means the more inflammatory response which is the sign of a good prognosis. Edema is also correlated with CD+4 quantity [15,16]. The definitive method of diagnosis is brain biopsy and this procedure is applied for the patients who did not respond to 2-4 weeks’ empirical medication.

Conclusion

Our case is among the unique ones in literature of toxoplasma cerebellitis because of the single lesion with rim enhancement on T1, hyperintense hemorrhagic areas on T2 weighted MR images with cerebellar localization in a non-immunocompromised patient. To our knowledge, it is very rare in literature especially as a solitary lesion. They are curable lesions by antibiotherapy and surgery when needed.

References


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