CASE REPORTS

JAPANESE ENCEPHALITIS AND NEUROCYSTICERCOSIS CO-INFECTION

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Abstract

We report an unusual case of Japanese encephalitis with co-infection of neurocysticercosis in a twelve year old child from a low socio-economic back ground. Child presented altered sensorium and dystonia and was managed aggressively but was left behind with neurological sequelae. The presence of neurocysticercosis infection in this case appears to adversely affect the presentation and outcome.

Keywords: Japanese encephalitis, Neurocysticercosis, Co-Infection.

Introduction

Japanese encephalitis (JE) is the most common human endemic encephalitis. (1) Clinically, the disease manifests as fever, headache, and altered sensorium with or without focal neurologic symptoms. On CT and MR imaging, JE lesions are most commonly seen in the thalami. Other areas involved are the substantia nigra, basal ganglia, cerebral cortex, hippocampus, pons, midbrain, medulla, cerebellum and white matter. (1) Neurocysticercosis (NCC) is the most common parasitic infection of the central nervous system caused by the larval stage of Taenia solium (2). Neuroimaging by CT or MR imaging helps in diagnosis. Co-infection of JE and NCC is a rare phenomena. Though both diseases have common epidemiologic and socio-demographic factors like pig rearing, poor socio-economic status, unhygienic conditions and malnutrition, more than a casual association of the two conditions has been suggested. (3) The present case highlights that the presence of NCC as co-infection serves as poor prognostic marker in outcome in JE.

Case Report

A 12-year old male child from a low socio-economic background was admitted with complaints of fever, altered sensorium and generalized abnormal body movements for fifteen days. On examination he had fever, altered sensorium with increased tone in both upper and lower limbs, exaggerated deep tendon reflexes and bilateral extensor plantar reflex. Dystonic posturing with non-intentional coarse tremors were present. Hemogram, clinical biochemistry and blood cultures were non-contributory but ELISA for IgM antibody against JE was 11.2 IV (positive > 6.1 IV). Cerebrospinal fluid (CSF) examination was near normal with mild pleocytosis. CSF JE and NCC ELISA was not done. There was a history of one episode of generalized seizure about a month ago for which a CT brain was that revealed generalized brain edema and ring enhancing lesion with surrounding edema in left high parietal area suggestive of NCC. (Fig. 1) Cysticercosis IgG and IgM in serum were positive. He was given phenytoin and albendazole for about three weeks.

MRI brain after admission in the current episode revealed altered signal intensity in bilateral thalami and middle cerebral peduncle appearing hypointense on T1 and hyperintense on T2/FLAIR images, which was consistent with the radiological picture of JE (Fig. 2). A focal tiny cystic lesion with eccentric dot in left occipital lobe in grey-white junction with minimal perifocal edema was seen. The child was treated symptomatically and the seizures and dystonia responded but neurological sequelae persisted in the form of tone abnormality and altered sensorium.

Figure 1: CT brain showing generalized brain edema and ring enhancing lesion with perilesional edema in left high parietal area.

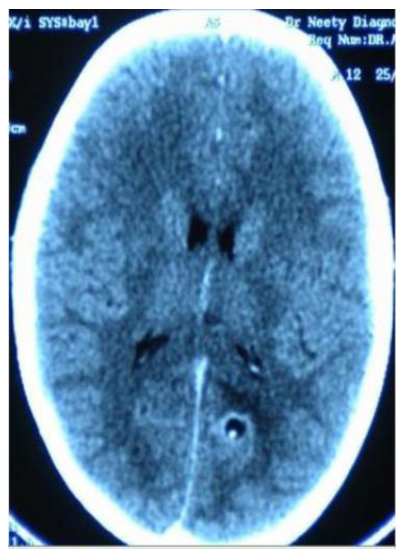
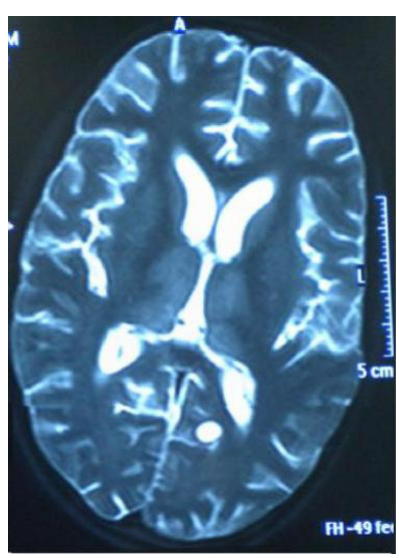


Figure 2: MRI brain showing hyperintense lesion in bilateral thalamus and middle cerebral peduncle with focal cystic lesion with eccentric dot in left occipital lobe.



Discussion

The observation that NCC and JE occur together was reported first by Hsu in 1940. (4) A high incidence of intestinal parasitic infections has been reported in areas endemic to viral encephalitis and has been postulated that it facilitates entry or reactivates latent neurotropic viruses by altering the blood brain barrier or host immune response. (5) The effect caused by the parasite on host immune system in dual infections due to metabolic products secreted by the larvae as well as the immune response to irrelevant host-like antigens normally present on the surface of the larvae and parasitic infections are known to induce generalized immune-suppressions particularly cell-mediated immuno-suppression, which may predispose the host to a fulminant JE infection. (6,7) A significant association of intestinal helminthic infections and JE in humans has been proved. (6-8) A few autopsy studies have shown that nearly a third of the brains of those who died as a result of JE also harbored NCC. (6,8) Desai et al in a prospective study to ascertain the frequency of effect coexistent NCC and JE in patients who survive encephalitis found that the frequency was notably higher (37.42%) than the prevalence of NCC in the general population (4%). (8) An imaging study by Handique et al also shows higher frequency of coexistence of NCC and JE (19.3%). (4) The reason behind the higher incidence of NCC among patients with JE has been theorized to be multiple common factors in the etiopathogenesis of the two pathogens, the most common being the rearing of pigs. Pigs are efficient amplifiers of the JE virus in its natural cycle and are the intermediate host of Taenia solium. Other factors shared by the two illnesses include a relatively higher incidence of intestinal parasitic infections, poor socioeconomic status, and unhygienic conditions as well as malnutrition. These factors are known to predispose a patient to diseases by neurotropic viruses.

The characteristic MR imaging findings in cases of JE without NCC have been described as symmetrical bilateral lesions in the thalamus and the basal ganglia. (9) In patients with co-infections, lesions of JE tend to be asymmetrical (4,10) and a greater number of JE lesions are seen on the side with the most NCC with edema. (4,8) Our patient had symmetrical lesions, however there was significant lateralization of JE lesions to the side bearing the most NCC.

Children having NCC with JE have more severe encephalitis as evidenced by deep coma, high frequency of refractory seizures, and higher mortality and morbidity rates.

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