A Case Report on Early Diagnosis of Brain Tuberculoma in an Adolescent

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ABSTRACT

The design of the work is to describe brain tuberculoma in an adolescent. Tuberculoma is defined as the unfamiliar incidence of tuberculosis in divergent parts of the body. Fifty years of Tuberculosis Control projects have been abortive in declining the prevalence of the infection in major parts of the world even after using potentially potent drugs however TB persists to kill all age groups (1). Tuberculoma (TC) is defined as the unfamiliar incidence of tuberculosis in divergent parts of the body (2). TB can exist as disseminated form with connivance of CNS existence is very frequent. TCs may evolve in the course of treatment or can be present at diagnosis (3). The most familiar site of TC has been reported to beat the grey-white matter junction and the periventricular region. They can also be even begin in the epidural, subdural and subarachnoid spaces and the brain stem (4). Generally, the development of intracranial TC is from the hematogenous seeding of the tubercle bacilli to the leptomeninges and in brain parenchyma, resulting in origination of tubercles which enlarges and coalesce and are encircled by a fibrous capsule (5). As a repercussion of possibility of concomitant infections and liable microbes, it is very much essential to crucial all the feasible affiliated sources of the infectious diseases (5).

1. Introduction

Fifty years of Tuberculosis (TB) Control projects have been abortive in declining the prevalence of the infection in major parts of the world even after using potentially potent drugs however TB persists to kill all age groups (6). Tuberculoma (TC) is defined as the unfamiliar incidence of tuberculosis in divergent parts of the body (2). TB can exist as disseminated form with connivance of CNS existence is very frequent. TCs may evolve in the course of treatment or can be present at diagnosis (3). The most familiar site of TC has been reported to beat the grey-white matter junction and the periventricular region. They can also be even begin in the epidural, subdural and subarachnoid spaces and the brain stem (4). Generally, the development of intracranial TC is from the hematogenous seeding of the tubercle bacilli to the leptomeninges and in brain parenchyma, resulting in origination of tubercles which enlarges and coalesce and are encircled by a fibrous capsule (5). As a repercussion of possibility of concomitant infections and liable microbes, it is very much essential to crucial all the feasible affiliated sources of the infectious diseases (5).

2. Case Presentation

A boy of 14 years old was brought to the Clinic, presented with history of seizures associated with altered consciousness, unrolling of eyes, frothing from angle of mouth, tongue bite, vomiting and post ictal confusion, weakness and blurred vision of left eye. He had three indistinguishable incidents of seizures. The child had been given vaccination as per schedule. The child had often grumbled of diplopia, headache during the episodes of epilepsy. Family history was positive where there was a history of seizures for his father’s younger brother. His family did not have any symptoms of TB and he was studying in a high school with low level of sociocultural state of south India (Telangana). On examination patient was conscious and afebrile. Provisional diagnosis was Epilepsy associated with TC/ NCC? His motor and sensory systems were found to be normal. Treatment given was Inj. Levipil 500mg/IV/BID, Inj. Pantop 40mg/IV/OD, Tab.Clobazam 5mg/PO/OD/HS, Tab. Folvite 5mg/PO/OD, Tab. MVT/PO/OD, Inj. Eptoin 100mg/IV/TID, Inj. Decadron 1cc/IV/BID, Tab. Albendazole

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400mg/PO/OD and on next day CAT 1 ATT was started. All possible physical and paraclinical investigations were done such as Sputum / skin clip for acid fast bacilli remained negative. MRI – well defined round lesion measuring 12.7x 11.7mm noted in the right high frontal lobe which shows T1 isointensity, T2 intermediate signal intensity T2 flair hypo intensity, shows no blooming on GRE. Perilesional edema noted. DWI shows central dot restricted diffusion, on contrast peripheral rim enhancement noted. No midline shift noted. Final impression states granulomatous lesion likely TC/NCC. CT brain shows well defined round irregular isohyperdense lesion with surrounding disproportionate edema noted in patient right frontal lobe and well defined hypodensity of HU 420 noted in right frontal lobe s/o calcified granuloma. After contrast a thick irregular peripheral rim enhancement of lesion noted with no enhance in surrounding edema. No midline shift noted. Impression states that granulomatous disease (NCC/TC). Liver function test revealed decreased unconjugated bilirubin levels. Mantoux test report was negative. Radiography of chest was found normal. Finally, patient was diagnosed as TC. By adhering to the treatment provided there was noticeable symptomatic relief was seen. He was discharged on the 11th day and child was instructed to follow up for every month and ATT is for 9 months CAT 1 (2A) was instructed. He was on ATT and Discharge medication given was Tab. Albendazole 400mg PO/OD, Tab. Pyridoxine 200mg /PO/OD, Tab.MVT PO/OD.

3. Discussion

Tuberculosis is still being the leading significant source of death among various infection brain TCs may be unaccompanied numerous (or) of the little infiltrating variation. Children with TCs have a miscellaneous presentation the disease manifestations may be influenced based upon the size and location in the brain. Although they commonly present as intracranial space occupying lesion with features of raised intracranial tension, seizures and localizing neurological signs, they may at times come with prolonged low grade fever with or without vague behavioral disturbances or may remain silent, or deteriorate rapidly due to sudden, massive edema around a small silent lesion. In the present case patient has episodes of seizures, Dizziness, Headache, blurred vision of left eye, Diplopia which was quite similar to the study done by Dubey et al where headache and blurred vision is seen since 6 months. Complete blood picture was found to be normal (WBC: 6200/mm³, hemoglobin: 12mg/dl) except lymphocytes where increased count is seen which was juxtapose to the study done by Mansoureh et al. Serum electrolytes were found to be normal which was identical to the work done by Mansoureh et al. MRI-well defined round lesion noted in the right high frontal lobe which shows T1 isointensity, T2 flair hypo intensity, shows no blooming on GRE which was opposite to the study done by Parth et al they reported that tuberculomas were observed. DWI shows central dot restricted diffusion this is like the work done by Parth et al. CT brain shows well defined round irregular isohyperdense lesion with surrounding disproportionate edema noted in patient right frontal lobe s/o calcified granuloma where it is contradictory to the study done by Maryam et al reported that multiple ring enhancing lesions were seen. This patient has been diagnosed early because of a high index of clinical suspicion and early CT examination. However, precautions and chances of recurrence should be explained.

4. Conclusion

In conclusion, the present case has been recovering by using anti-tubercular treatment and the patient is under follow-up. Early diagnosis makes the disease less worsen and it makes patient to respond well to treatment provided. The regulation of tuberculoma recommends regular follow-up and a series of MRI of the brain. Hence tuberculosis should be regarded as an important differential diagnosis of CNS diseases by the physicians because it may lead to life threatening condition if neglected, especially in developing countries.

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Conflict of Interest

The author(s) confirm that this article content has no conflict of interest.

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