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# NEUROCYSTICERCOSIS WITH LEFT FRONTAL TUBERCULOMA - A FATAL CASE REPORT

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## Abstract

Neurocysticercosis (NCC) is a neurologic infection cause by the larval stage of the tapeworm Taenia solium is the most common cause of acquired epilepsy, when the central nervous system (CNS) gets involved. Tuberculosis is an infectious bacterial disease caused by mycobacterium tuberculosis, which most commonly effects lungs. When this bacteria effects the central nervous system then it may leads to most serious form of tubercular infection, termed as "Intracranial Tuberculoma". Recently near about 2.5 million people worldwide are carriers of adult tapeworm with prevalence of 3-6%. Clinical presentation of NCC vary from asymptomatic to life threatening symptoms of seizures, headache, visual disturbances, nausea, abdominal cramps and diarrhoea. The available diagnostic tools include CT, MRI and serology. We present a case of a 30 years old female patient with chief complaints of seizures of GTCS type with 5 episodes, each episode lasting around 5 minutes and gap between each episode is 10 minutes, unrolling of eyeballs, froth discharge, no incontinence of urine was present.

**Keywords:** Neurocysticercosis, Tuberculomas, Seizures, CT, MRI, AKT4.

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## INTRODUCTION

Here we present a rare case of co-existing infection of Neurocysticercosis (NCC) and left frontal tuberculoma. Although neurocysticercosis is not new to the developing countries but co-infection of tuberculoma in patients with neurocysticercosis is a rare occurrence. Neurocysticercosis(NCC) is a neurologic infection cause by the larval stage of the tapeworm Taenia solium, which in the developing countries like India forms the most common cause of acquired epilepsy, when thecentral nervous system(CNS) gets involved [1–3]. Now it has also been a problem in industrialized countries because of the immigrants oftapeworm carriersfrom the endemic area of this disease [4, 5]. Taenia solium is a two-host parasite making human and swine as natural definitive and intermediate host respectively. In human NCC is the result of accidental ingestion of eggs of Taenia solium usually due to contamination of food by people with taeniasis, such as partially cooked pork and uncooked vegetables. Human accidently ingest the Taenia egss containing infective oncospheres, and the parasite become established in the tissues almost anywhere in the body as larval cysts and reach their maturity in about 3 months [6,7]. Clinical presentation of NCC vary with the locations of the lesions, the number of parasites, and the host's immune response[8], from asymptomatic to life threatening symptoms of seizures (70%),headache, visual disturbances, nausea, abdominal cramps and diarrhoea. While dysarthria, extra-ocular movement palsy, hemi paresis, hyperreflexia and hyporeflexia are rare.Seizures due to cysticercosis usually occur when the dying cyst incites aninflame-matory reaction. The available diagnostic tools include CT, MRI and serology.The current assay of choice is the electroimmunotransferblot (ETIB) using partially purifiedantigenic extracts[9,10]. Recently near about 2.5 million people worldwide are carrier of adult tapeworm, and many more are infected with cysticerci, and are thought to be more prevalent in endemic areas of central and south America, eastern Europe, Africa and some regions of Asia with a prevalence of 3-6% [3,11,12,13]. Tuberculosis is an infectious bacterial disease caused by mycobacterium tuberculosis, which most commonly effects lungs. When this bacteria effects the central nervous system then it may leads to most serious form of tubercular infection, termed as “Intracranial Tuberculoma” (ITC) Intracranial tuberculomas are space-occupying masses of granulomatous tissue that result from hematogenousspread from a distant focus of tuberculous infection. Only 1% of patients with develop an intracranial tuberculoma. In developing countries tuberculomas

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still constitute 30% of intracranial space-occupying lesion which is as little as 0.1 -0.2%In the developed countries [6].

**CASE REPORT:**

A 30 years old female patient was admitted in general medicine ward on 23rd June with chief complaints of seizures of GTCS type with 5 episodes, each episode lasting around 5 minutes and gap between each episode is 10 minutes, unrolling of eyeballs, froth discharge, no incontinence of urine was present. On examination it was found that the patients blood pressure (B.P) and pulse rate were fluctuating which can be revealed from the table given below. The patient was normotensive as well as non-diabetic and non-alcoholic. But she was past smoker and hyperthermic since last more than 2 months.

**Table 1: Physical Examination**

| PARAMETERS | DAY 1 | DAY 2 | DAY 3 | DAY 4 | DAY 5 | DAY 6 | DAY 7 |
|---|---|---|---|---|---|---|---|
| B.P.(mm Hg) | 90/50 | 140/90 | 130/90 | 110/70 | 120/80 | 120/80 | 120/90 |
| Pulse | 128 | 101 | 100 | 78 | 80 | 80 | 70 |

**Lab investigations**

A part from the above mentioned physical examinations the patient underwent routine test as well as definitive tests.

**Routine Test**

Differential counts revealed increased Eosinophils count 9% (0-3% normal range) which increasing the chance of provisional diagnosis, while the other parameters were found to be normal. Decreased serum calcium and magnesium level with value7.0mg/dl(8.5-10.2 mg/dl normal range)1.6 (1.8-2.6 mg/dl) respectively was found along with increased inorganic phosphate level 8.2 (2.4 -4.1 mg/dl normal range). hrough complete blood picture (normal), WBC count-15000 cells/cumm(4000-11000 cells/cumm), Electrolyte test revealed increased chloride levels-112 mEq/l (95-105 mEq/l). LFT and thyroid profile was found to be normal.

**Difinitive Tests**

CT and MR imaging were done.MRI revealed a thick and hypointense wall with marked perilesional edema.

The final impression was a small inflammatory granuloma in the frontal lobe.

Ventricular cysts was found to be appearing on CT images as cystic lesions.

Histological examination of cerebrospinal fluid revealed scolex of pork tapeworm surrounded along with inflammatory granulomatous infiltrates.

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As a part of differential diagnosis the patient also underwentMonteux test whose result was found to be positive (induration-20mm).

**Final Diagnosis**

On the basis of above mentioned investigation the interpretation of final diagnosis was made as a co-existing infection of NCC along with intracranial tuberculous granuloma.

**Treatment**

When we talk about the treatment, it is found that the patient was prescribed rational treatment. As the patient was diagnosed for co-infection with NCC and ITG, he was prescribed Albendazole(400 mg) as cysticidal agent along with four first line antitubercular drug AKT-4. The prescribed antitubercular AKT-4 kit is composed of Pyrazinamide(750mg), Isoniazid(300mg), Rifampicin(450mg), and Ethambutol(800mg). Injection Phenytoin (100mg/PO/BD) and controlled release Tab. Carbamazepine(200 mg/PO/BD) was also prescribed to manage seizures induced by NCC. Rifampacin should be taken before food. The patient was also advised to go for physiotherapy to regain the strength in facial and limb muscles.

## DISCUSSION

The present case about a co-infection of Neurocysticercosis (NCC) and intracranial tuberculoma where the tuberculos granuloma may be the manifestation of NCC. NCC is a specific type of common neurologic infection caused by the larval stage of the tapeworm Taenia solium is the most common cause of acquired epilepsy [3,14,15,16]. Signs and symptoms of NCC are pleomorphic which range from asymptomatic to life threateningand depending upon the number, location, growth, stage of degeneration and inflammation, host factors and parasite genotype [15,17,18]. The most common clinical features of NCC, occurring in about 50-80% of the patients include seizures or multiple seizures and sometimes headache [15]. In this present case, the patient presented with a history of multiple seizures, which can be attributed to NCC, as the patient was not having any past history of seizures. NCC acquired epilepsy occurs due to parasitic inflammation leading to a granuloma formation. Although the main diagnostic tool of neurocysticercosis is the neurological imaging, the diagnosis of neurocysticercosis depends upon different combined findings which include epidemiology, signs & symptoms, serology, and available pathology [19]. In endemic areas like India, the suspected patients presenting as lesions in CT scans can be a suggestive of NCC. The treatment is started with anticonvulsants and a twelve days course of albendazole along with dexamethazone.

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Cerebral tuberculomas are the result of haematogenous spread of Mycobacterium Tuberculosis [20].

Anti tubercular treatment with the four drugs (HRZE) is important for curing the disease [21].

The activity of the enhancing lesions is not related to stopping the treatment. The treatment is continued till all the lesions disappear. Tuberculomas are 1% of CNS tuberculosis and is the least presentation of the disease. There are also reports of developing fresh lesions during the therapy [22]. Poymerase chain reaction test (PCR) also helps in documenting the diagnosis [23]. Intracranial giant tuberculomas can mimic in appearance like malignant pathology [24].

**Table 2: Different between Neurocysticercosis and CNS Tuberculomas Lesions**

| NEUROCYSTICERCOSIS | TUBERCULOMA |
|---|---|
| Lesions are <20mm and may be single or multiple | Often multiple lesions >20mm because of conglomeration. |
| Meningitis feature is not there. | Meningitis is usually associated. |
| Present at grey-white matter junction. | Most common in posteriror fossa. |
| Other involvements like muscles, eyes or subcutaneous tissues. | Spread is mostly secondary to infection somewhere else. |
| T2W shows hyper sensitivity with hypointense scolex in it. No midline shift and ring enhancement is there depending upon the staging. | Hypointensity seen in T2W and midline shift may be present. |
| MR spectroscopy shows multiple amino acid peaks | MR spectroscopy shows lipid peaks. |

## CONCLUSION

Tuberculomas and neurocysticercosis lesions may be similar and resemble, number of lesions and symptoms. There is also difference in the MR spectroscopy findings, the former shows lipid peaks and the latter shows amino acid peaks. History of seizures is most commonly seen in tuberculomas. Timely management can avoid the complications.

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