Neurocysticercosis

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ABSTRACT. Neurocysticercosis (NCC) is one of the common parasitic central nervous system (CNS) infections. Improperly cooked pork and eggs of the tapeworm *Taenia solium*, entering the body through the feco-oral route, are the common sources of its infection. Affected person may remain asymptomatic for long periods and can present with a variety of neurological manifestations, including focal neurological deficits and generalized seizures. Neuroimaging along with serological test can aid in its diagnosis. Treatment of NCC varies from case to case and must always be individualized based on the patients’ condition. Common therapeutic strategies include surgery and treatment with drugs, such as antiparasitic agents (albendazole) and corticosteroids (hydrocortisone), apart from other agents which are based on the patient presentation. Proper prevention strategy has to be followed to control the spread of infection within and among the individuals. We herewith present a case of NCC in a tertiary care hospital of Hyderabad, India.

Introduction

Neurocysticercosis (NCC) is a very common outcome of helminthic infection of the nervous system and a leading cause of acquired epilepsy worldwide. It is a preventable parasitic infection caused by pork tapeworm *Taenia solium*. Humans are infected after consuming undercooked food, particularly pork, or water contaminated with tapeworm eggs, or through poor hygienic practices. Teniasis is the intestinal infection of adult tapeworm in the humans and is an initial phase of infection. If left untreated, the parasite can survive inside the body for many years, leading to more hazardous conditions, such as cysticercosis which develops when the larvae of *T. solium* invade inside the body and develop in the muscles, skin, and eyes. If the larvae invade the central nervous system (CNS), the infection leads to NCC.¹

NCC is considered as the most frequent preventable cause of epilepsy in the developing world and a growing public health concern. It presents with symptoms such as headache, blindness, seizures, meningitis, and dementia.²
Life Cycle of Neurocysticercosis

The life cycle of *T. solium* (adult tapeworm) involves two hosts: humans and pigs. Humans are the definitive hosts and acquire intestinal infection (teniasis) from pigs, the intermediate hosts, by the ingestion of undercooked pork infected with live cysticerci (encysted larvae). Humans acquire cysticercosis via consumption of food or water contaminated with *T. solium* eggs or by autoinfection. NCC results when the larval stages lodge in the brain (Figure 1).

Epidemiology

NCC is one of the leading health problems in India, Latin America, and Southeast Asian countries. As a result of increased travel and migration of people from endemic areas, NCC has become an important emerging infection in the industrialized world. In most of the developing countries, 10% of acute neurological cases are patients with NCC. Epilepsy and raised intracranial pressure are common clinical forms of NCC. It accounts for 50% of patients presenting with partial seizures in some parts of India.

Diagnosis

Clinical manifestation of NCC is wide in range, but the two most common signs and symptoms are seizures and increased intracranial pressure. Diagnosis of NCC includes initial evaluation followed by laboratory interpretation. Initial evaluation includes careful history and physical examination. Studies recommend serologic testing with enzyme-linked immunotransfer blot as a confirmatory test in patients with suspected NCC. Neuro-imaging studies such as magnetic resonance imaging (MRI) and contrast computed tomography (CT) scan are recommended for classifying patients with newly diagnosed NCC.

Challenges in Management

Management of NCC has been debated around several issues: (1) Effectiveness of the antiparasitic drugs in killing the cysts and in improving the clinical outcome (defined as fewer seizures, in patients with intra-parenchymal NCC); (2) choice of the optimal cysticidal drug; (3) use of steroids; and (4) optimal duration of antiepileptic drug therapy. Unders-

Figure 1. Life cycle of neurocysticercosis.
tandably, the benefits of antiparasitic regimens are more evident in patients with multiple viable cysts and less evident in patients with degenerating lesions.

**Treatment**

In patients presenting with untreated diffuse cerebral edema or hydrocephalus, studies recommend the management of elevated intracranial pressure alone and not antiparasitic treatment; hydrocephalus usually requires a surgical approach. In the absence of raised intracranial pressure, it is recommended to use antiparasitic drug in all patients. For patients presenting with 1–2 viable parenchymal cysticerci, a monotherapy of albendazole for 10–14 days is recommended at a regimen of 15 mg/kg in two divided doses along with food, with a maximum dose of 1200 mg/day. Albendazole at 15 mg/kg/day for 10–14 days is recommended for patients with viable parenchymal cysticerci >2.

**Case Report**

Informed consent was obtained from the patient before publishing the case.

A 23-year-old male patient with no history of psychiatric illness was brought to a tertiary care hospital with the chief complaints of headache and vomiting for 10 days. At presentation, the patient was conscious and cooperative with normal speech. The patient has no relevant comorbidity. On physical examination, it was revealed that the patient was conscious, with right-sided facial palsy and blood pressure of 110/80 mm Hg. Cardiac and respiratory examinations were normal. Neurological examination revealed a power of 5/5 in all limbs. His abdomen was soft with normal bowel sounds. Initial laboratory investigations showed serum glutamate oxalo-acetate of 55 U/L, serum glutamate pyruvate of 21 U/L, direct bilirubin of 0.2 mg/dL, total bilirubin of 0.6 mg/dL, alkaline phosphatase of 47 U/L, total protein of 6.4 g/dL, and albumin of 4.2 g/dL. Other investigations revealed blood urea of 23 mg/dL, serum creatinine of 0.7 mg/dL, serum uric acid of 4.5 mg/dL, sodium of 139 mmol/L, potassium of 3.9 mmol/L, and chloride of 104 mmol/L. MRI, done on the 1st day, showed signs of NCC evidenced by multiple well-defined thin-walled T2 hyper-intense areas, with eccentric hypotensive specks; few showing perifocal edema in the bilateral cerebellar and cerebral hemispheres, numerous small parenchymal calcifications; and few showing mild perlesional edema; the largest lesion measured 9 mm, suggestive of calcified granulomas. Multiple, small well-defined cystic lesions were noted involving both cerebral hemispheres, the largest measuring 15 mm, and few of the cystic lesions showed small specks of calcium within them (scolex). Three small cystic lesions were noted in the right cerebellar hemispheres, the largest measuring 7 mm. A mucosal polyp was noted in the right maxillary sinus.

He was managed with injection dexamethasone 8 mg intravenous (IV) BD, albendazole 400 mg BD along with other medications such as injection pantoprazole 40 mg OD and injection ondansetron 8 mg BD. On day 2, seizures were reported in the patient, which was managed with the injection levipil 500 mg IV BD. On day 4, headache had subsided and he was discharged on day 5 with the following medications: tablet albendazole 400 mg BD, tablet pantoprazole 40 mg OD, tablet naxdom 250 mg (SOS), and tablet levipil 500 mg BD.

**Discussion**

NCC is the most common cause of preventable epilepsy and is reported to be caused by cysticercus cellulosae, the larval form of *T. solium* commonly known as tapeworm. Events of human cysticercus occur via either endogenous route, i.e., autoinfection in the tapeworm carriers, or through exogenous route, i.e., by ingesting eggs of *T. solium* after fecoral contamination. It is an uncommon manifestation of a common infection. Generalized promulgation of the larvae can result in the involvement of almost any organ in the body. The hallmark of NCC is its heterogeneity, whereas clinical manifestation depends on the
number, localization, and evolutionary stage of the parasites as well as the intensity of inflammation. Patients with NCC may be asymptomatic or may present with whole variety of symptoms. The main presentation of NCC includes dementia, headache, epilepsy, and increased intracranial pressure or focal neurological signs based on the localization and amount of cysts. Diagnosis is usually based on neuroimaging abnormalities, which involve CT scan or MRI in which differential diagnosis of cysticerci cerebral lesion includes tubercle, abscess, metastasis, and glioblastoma. Serology can be beneficial, and the results can vary depending on the activity of cyst and the number of lesions; the major drawback is that negative result will not rule out NCC. Postmortem studies have shown that 80% of the cases with NCC remain asymptomatic.

Therapeutic intervention should be individualized, particularly for patients with mixed form of NCC. Since extraparenchymal NCC involves poorer prognosis, there was a consensus toward more aggressive management.

Management is cysticidal, surgical, i.e., removal of cysts and ventriculoperitoneal shunt, and symptomatic, i.e., antiepileptics and steroids. Albendazole and/or praziquanuel are commonly used, but their role is controversial. NCC is a life-threatening complication and those with active cysts remain at the risk of serious complications. Besides, treatment/therapy can result in the cysticidal syndrome, characterized by features of raised intracranial tension, which may occur in 50% of the cases. Efficiency of treatment should be monitored by repeating CT scan. This case presented with symptoms of headache associated with vomiting for the past 10 days with no history of seizures, and later developed it, and was diagnosed with the help of MRI, CT scan, and serology, and treatment provided was levetriacetam, albendazole, domperidone, and naproxen.

Future Prospects

To improve the knowledge of NCC, it is important to develop the validated criteria for diagnosis, since the current available diagnostic criteria have not been validated so far. Newer insights into the immune mechanisms underlying symptomatic human cysticercosis and helminth-induced immunosuppression are being obtained through recent studies. Toll-like receptor-4 and soluble intercellular adhesion molecule 1 K469E polymorphisms have been suggested to predispose to symptomatic infection. The role of genetic susceptibility to NCC is also being evaluated and preliminary work has reported positive association of HLA-DRBII-13 with SSECTL(s). Prospective cohort studies about epileptogenicity of NCC are needed to assess the association of different evolutionary phases of the parasite, in the development of seizures and epilepsy.

The understanding of these immune and genetic mechanisms will help develop newer drugs such as tamoxifen and newer drug delivery systems, such as lactic acid–conjugated solid lipid nanoparticles bearing albendazole and prednisolone, for effective management of NCC. Results of preclinical animal studies on the pharmacokinetics, safety, and toxicology of oxefonazole for humans have been encouraging. Development of effective antiparasitic drugs for the treatment of swine cysticercosis is also a potential area of research, especially combination therapy.

Conclusion

NCC is an important acquired cause of epilepsy and other neurological manifestations, especially in endemic areas. Because of its pleomorphic presentation, NCC should be considered in the differential diagnosis of a number of neurological conditions. Accurate diagnosis with proper symptomatic assessment, followed by a well-designed therapeutic intervention by the healthcare provider, contributes significantly to the morbidity and mortality of the NCC. Treatment with cysticidal therapy leads to reduction in seizure frequency and a faster resolution of lesions. A single antiepileptic drug, usually carbamazepine or phenytoin, is sufficient to control seizures due
to single-lesion NCC. Prevention of NCC is important and is feasible. Public sanitation and hygiene awareness are of utmost importance to the prevention of this disease. Development of newer cysticidal drugs and drug delivery systems for both human and swine population are the potential areas of research. Well-structured prevention strategy also plays an important role in controlling the incidence of NCC.

**Conflict of interest:** None declared.

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