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Neurocysticercosis (NCC) in Children: A Review

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Abstract

Neurocysticercosis (NCC) is the commonest parasitic infestation of the brain caused by Taenia solium larvae and a preventable cause of epilepsy worldwide. NCC may be parenchymal or extra-parenchymal, and its clinical presentation may vary based on the location, number, and host immune-response to the cysts. Seizures are more frequently seen in children, and single colloidal parenchymal cyst is the common radiologic finding in pediatric NCC. The diagnosis is generally by neuroimaging, a scolex within the cyst being pathognomic; however, serology may help when the neuroimaging is inconclusive. Initial treatment generally focuses on symptomatic management by antiepileptics and corticosteroids. Albendazole is the cysticidal drug of choice in most cases; praziquental is added for multiple lesions. The outcome depends on the type, location, number of lesions, seizure recurrences and radiologic resolution of the lesions. Single lesions have a better prognosis. In endemic areas, NCC should be a differential in any child presenting with recent-onset seizures, headache, vomiting, or focal motor deficits, in the absence of another known neurological condition. Due to the potentially eradicable nature of cysticercosis, preventive measures need to be encouraged.

Key words: Children; Cysticercus; Epilepsy; Neurocysticercosis; Parasitic disease; Ring enhancing lesion; Taenia solium; Tapeworm.

Introduction

Taenia solium (pork tapeworm) causes two different human infections– i) taeniasis, a generally mild intestinal infection by adult tapeworm, and ii) cysticercosis, an infection caused by the larvae with potentially serious consequences.[1] Neurocysticercosis (NCC), the commonest parasitic infestation of the brain,is the most frequently preventable cause of epilepsy worldwide. It accounts for 30% of epilepsy in endemic countries, and up-to 70% in certain communities.[2] Approximately 2.56 to 8.30 million people worldwide are asymptomatic/ symptomatic for NCC.[2] Cysticercosis has also been recognized by the World Health Organization as an endemic, parasitic 'Neglected Tropical Disease' in South East Asia.[3] In 2011, human NCC-associated active epilepsy produced an estimated annual median loss of 12.03 billion rupees and 1.73 DALYs (Disability-adjusted life year) per thousand persons per year. Thus, human NCC contributes to significant health and economic burden in India.[4]

Methodology/Search Strategy used for writing the review:

The Pub - Med database was used to conduct a literature search using word combinations

of controlled vocabulary (MeSH terms): 'neurocysticercosis' and 'children'. These controlled vocabulary terms were combined using 'AND' as the Boolean connector. The results were limited to studies available in English language. The age limit was set from birth to 18 years of age. With these search parameters, we obtained 536 results. On limiting the search to the period from 2005 to 2020, there were 299 articles. Other important and relevant articles from Google Scholar as well as those hand searched from other sites and resources were also included. From these search results, and a couple of book chapters, 39 articles were finally shortlisted to include citations relevant to our review (we excluded the articles whose results were already incorporated in the published consensus statements/ guidelines and other reviews) with an emphasis on review articles and landmark articles.

Epidemiology

The prevalence of NCC is higher in communities with close contact with pigs, poor sanitary conditions, and where raw or undercooked pork is consumed. It occurs globally; being endemic in Latin America, Sub-Saharan Africa, India and East Asia.[5] With rapid globalization and international travel, the incidence of NCC is rising in the developed, non-endemic countries such as the USA, UK and Australia.[6]

In India, NCC is prevalent in all the regions/ states; but the distribution varies with geography, religious traditions, food preferences, education, hygiene, etc. Fewer cases have been reported from Kerala (due to high literacy and better hygiene) and from Jammu & Kashmir (due to prohibition of pork consumption by certain religious traditions).[7] Cysticercosis is highly prevalent in the northern States of Bihar, Orissa, Uttar Pradesh and Punjab, with a point prevalence of 4.5 per 1,000 population in the rural Northwest India.[8,9] An alarming increase in cases has been observed from Orissa, Madhya Pradesh, Maharashtra, Manipur, Chandigarh, West Bengal, Karnataka, and Tamil Nadu.[8]

Males and females are equally affected. Infection possibly occurs earlier in life (5–15 years of age) with presentation predominantly in young adulthood.[10] Within children, the prevalence is higher in older age groups.[11] Besides prevalence, the clinical presentation also varies in children. Seizures are more frequent in children, while intracranial hypertension and headaches are commoner in adults. Single colloidal parenchymal cysts are common radiologic findings in pediatric NCC while multiple viable parasites in the basal subarachnoidal cisterns/ventricles are frequent in adults. Cerebrospinal fluid (CSF) inflammation is also greater in adults as compared to children.[12]

Etiopathogenesis

T. solium completes its life cycle in two hosts: humans (definite hosts) and pigs (intermediate hosts). Humans acquire the intestinal infection (taeniasis) through the ingestion of larval cysts (cysticerci) in undercooked/infected pork. These cysts mature into adult tapeworms and shed eggs in feces, infective to pigs. Following human ingestion of T. solium eggs (shed in the stool of a human tapeworm carrier, through close contact with a carrier or autoinfection), tissue cysticerci develop at one/more sites (brain, muscle, subcutaneous tissues) over three to eight weeks.[13,14] Eating undercooked pork with cysticerci results in tapeworm infection (taeniasis), not human cysticercosis.[14]

Presentation of NCC depends on the site of cyst invasion, parasite stage and host immune-response. There are four stages of parenchymal larval cyst development. The 'vesicular cyst' (viable) has an eccentric scolex and minimal enhancement (lack of host immune-response) on neuroimaging. Death of a scolex (subsequent to cysticidal therapy/ immune-response) may result into cyst rupture, releasing fluid into surrounding tissues. This produces a strong immune reaction and perilesional edema noted as contrast computed tomographic (CT) scan/magnetic resonance imaging (MRI) enhancement (Figure 1).

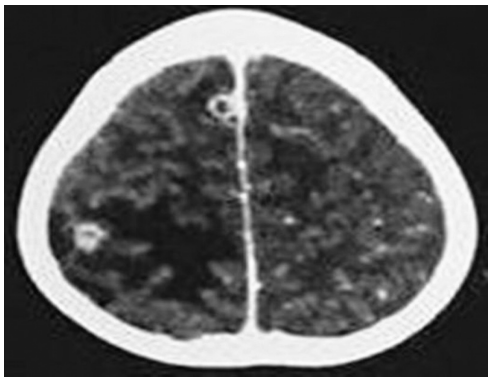

Fig. 1: Contrast CT scan of brain showing well defined peripherally enhancing lesion in the right high parietal (near the falx) and right high frontal region. There is hyperdense scolex within and perilesional edema, suggestive of cerebral neurocysticercosis/NCC (colloid-vesicular stage).

The transparent vesicular fluid in colloid cyst is replaced by a viscous and turbid fluid (easily identifiable on MRI). It further degenerates into a 'nodular cyst', with some contrast enhancement (due to perilesional edema). Finally, it 'calcifies'

and is detected as punctuate calcification on CT scan.[15] In most Indian patients, it presents as a solitary cysticercus granuloma (SCG).[16]

Clinical Manifestations

The clinical manifestations may be parenchymal/ extra-parenchymal or both. Most children present after five years of age, commonest presentation being seizures. In an Indian series, NCC accounted for 50% of pediatric hospital admissions for partial seizures.[17] Most seizures (single/recurrent) are partial, of a short duration and may undergo secondary generalization.[17] Less common presentations include hydrocephalus, diffuse cerebral edema, encephalitis, symptoms of space-occupying lesions, altered vision, focal neurologic signs, and meningitis.[5,14] Cysticercal encephalitis (rare) may occur spontaneously or post-antiparasitic therapy after simultaneous degeneration of multiple cysts. The massive immune response causes diffuse brain edema which may manifest with seizures, headache, acute raised intracranial pressure (ICP), papilledema and altered sensorium.[14] Thus, encephalitis due to NCC should be a differential of acute encephalitis especially in endemic countries.[18] In disseminated or miliary NCC, multiple cysts may occur in varying developmental stages in various locations, including extracranial cysts. It runs a malignant course with poor prognosis.[15,18] Many patients are asymptomatic and incidentally detected on imaging.[14]

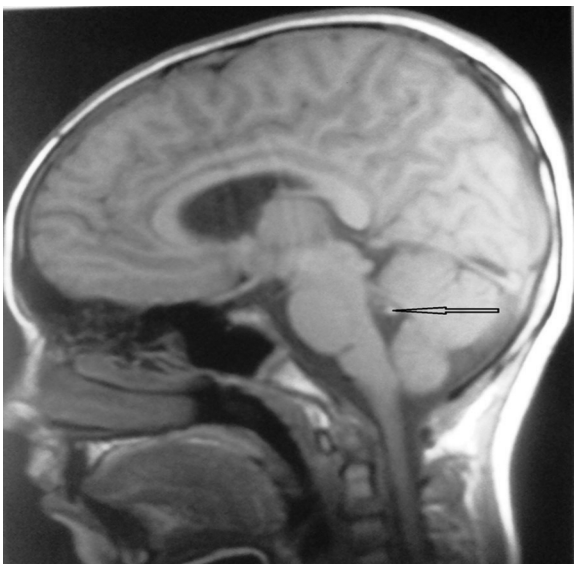

Fig. 2: MRI brain (sagittal view), note the intraventricular cystic lesion (Neurocysticercosis- NCC) in the fourth ventricle marked with an arrow.

Extra-parenchymal neurocysticercosis (ventricles, subarachnoid space, spine, and/ or eye), is rare in children and may coexist with intra-parenchymal lesions.[14,19] Ventricular and subarachnoid NCC may manifest as basilar arachnoiditis, obstructive hydrocephalus, or chronic meningitis (Figure 2).

Some subarachnoid cysts enlarge without scolices, become racemose, and act as a space-occupying lesion.[13] Spinal cysticercosis cysts may be leptomeningeal or within the cord, presenting with radicular pain, paresthesia, cord compression, paraplegia or sphincter disturbances.[13]

Cysticercosis cysts may lodge anywhere in the eyes, may be asymptomatic or produce visual deficits, sudden blindness, limitation of eye movements, etc.[13] Degenerating cysticerci may produce chorioretinitis, retinal detachment, or vasculitis due to inflammation, necessitating ophthalmologic evaluation in all NCC patients (to rule out ocular cysticercosis) prior to anti-parasitic therapy. Extra-neural cysticercosis may involve muscle or subcutaneous tissue; and are generally asymptomatic. Intramuscular cysts often undergo calcification, detected radiographically as 'cigar-shaped calcifications'.[14]

Diagnosis

NCC should always be suspected in a child with seizures or hydrocephalus,with relevant neuroimaging findings (cystic lesions, enhancing lesions, and/or calcifications) and h/o residence in endemic area.[5,14] The revised diagnostic criteria for neurocysticercos is by Brutto et al. has four categories- *absolute* (histological demonstration of cysticerci, scolex on neuroimaging, and direct visualization of subretinal parasites by fundoscopy), *major* (highly suggestive lesions on neuroimaging, positive serum enzyme-linked immunoelectrotransfer blot (EITB) for antibodies, resolution of intracranial cystic lesions after cysticidal therapy, and spontaneous resolution of single enhancing lesions); *minor* (lesions compatible with NCC on neuroimaging, suggestive clinical manifestations, positive CSF Enzyme-linked immunosorbent assay [ELISA] for cysticercal antibodies/antigens, and extra-neural cysticercosis); and *epidemiological* criteria.[20] While interpretation of these criteria permits two degrees of diagnostic certainty-definitive or probable diagnosis, it cannot differentiate from a tuberculoma, an important differential in endemic countries, like India.[20,21] Cost limitations for serological tests make it difficult to assess all criteria. Thus, usefulness of these criteria in routine practice may be limited.[21]

Radiological modalities: While CT is beneficial for calcification, parenchymal and ocular lesions, MRI is useful for smaller lesions, degenerating changes, edema around calcifications, scolioses within calcifications, and extra-parenchymal lesions.[14] Intra-parenchymal NCC may manifest as non-enhancing hypodense cystic lesions, contrast enhancing lesions (Figure 1) and calcifications. The presence of scolex within cyst is pathognomic for cysticercosis. Less common manifestations include involvement of the brainstem, cerebellum, or basal ganglia, mass effect, diffuse cerebral edema, cerebral infarction, giant cysts (>20 mm), and multiple cysts (>50).[14] Multiple cysts may appear with typical 'starry-sky' appearance'.[22]

NCC detection may be enhanced by additional sequences like susceptibility-weighted images (calcified lesions), fluid-attenuated inversion-recovery (FLAIR) and diffusion weighted images (visualization of scolex), proton MR spectroscopy (differentiation from tuberculoma), magnetization transfer images and ratio (detect perilesional gliosis) and three-dimensional constructive interference in steady state (3DCISS) for differentiating cyst from CSF.[23] Radiographic manifestations of extraparenchymal NCC include intraventricular (Figure 2)/subarachnoid cysts, leptomeningeal enhancement and hydrocephalus.[14] Serological tests are useful in those where neuroimaging is not diagnostic.

Antigen Detection Tests: Available tests include crude to semi-purified/purified antigen preparations in serum, urine, saliva and even tears.[22] The antigen ELISA detects circulating larval antigens in the serum, thereby effectively detecting active disease. Though mostly positive in subarachnoid disease, it has poor sensitivity (72 to 86%) and is negative in calcified and single parenchymal lesions. However, it can help monitor the effects of therapy as levels drop following successful therapy.[24]

Antibody Detection Tests: These are useful in the absence of scolex on neuroimaging. Lentil lectin purified glycoproteins-based EITB assay is best for sero-diagnosis (100% specific and 98% sensitive for active, multiple and extra-parenchymal lesions, sensitivity in serum > CSF). ELISA has better sensitivity with CSF. They are frequently negative in single/calcified lesions and cannot differentiate between active disease, past disease and asymptomatic seropositivity. Tests may be false positive in extra-neural cysticercosis, other cestodes/helminths and up-to 1 year after antiparasitic therapy.[24]

Other tests: Real-time polymerase chain reaction assay may be useful for subarachnoid neurocysticercosis (sensitivity 97%, specificity 100%).[25] Though promising as diagnostic measures they are not commercially available. Non-specific findings include peripheral eosinophilia and cerebrospinal fluid pleocytosis (mostly mononuclear cells), increased protein, and low glucose levels in cysticercal meningitis. Brain biopsy is the last option, to differentiate NCC from an abscess/malignancy, when neuroimaging and serology are not confirmatory.[14]

Differential Diagnosis

If the scolex is not visualized, tuberculoma is an important differential diagnosis, particularly in resource-limited/endemic countries. Features suggestive of a tuberculoma are - size of 20 mm on CT, location at the base of the brain/posterior fossa (NCC lesions are often seen at the gray–white matter junction), lobulated irregular shape, marked edema causing midline shift, presence of raised intracranial pressure, progressive focal neurological deficits, presence of a lactate peak, and choline/creatine ratio >1 on MR spectroscopy.[26] Other conditions mimicking single/ multiple ring/ nodular enhancing lesions include-pyogenic brain abscess, mycotic granuloma, primary or metastatic brain tumor, toxoplasmosis, and septic emboli. Differentials for cystic brain lesions are cystic echinococcosis and coenurosis. CT parenchymal calcifications are also observed in metabolic disorders, vascular malformations, HIV infection, intracranial neoplasms, and congenital anomalies.[14]

Management

Antiparasitic therapy is never an emergency; managing acute symptoms such as increased intracranial pressure (via surgical intervention and/ or corticosteroids) and seizures (via antiseizure drug therapy) is a priority.[1]

Antiparasitic therapy: Cyst stage determines whether to treat with an antiparasitic medication. While viable and colloidal (early degenerating/ inflammed) cysts may benefit from antiparasitic treatment, granular/calcified cysts do not.[1] Antiparasitic therapy aids faster resolution of active cysts, decreases seizure risk, and reduces recurrence of hydrocephalus.[27] It is contraindicated in untreated hydrocephalus, high cyst burden (e.g. cysticercal encephalitis) and only calcified lesions.[28] Additionally, children with markedly elevated intracranial pressure and ophthalmic cysticercosis may worsen with cysticidal therapy, and thus

corticosteroids without cysticidal therapy should be considered.[22] Based on the disease burden, albendazole is preferred in single/two cysts, and praziquental is added in presence of more than 2 cysts.[29]

Albendazole is less expensive, better tolerated with fewer side-effects, has better subarachnoid space penetration, improved bioavailability with corticosteroid co-administration and unaffected by phenytoin or carbamazepine.[30] It is commonly administered at 15 mg/kg/d in 2–3 divided doses for 7 days.[5] Longer courses may be required for multiple lesions (28 days) and subarachnoid cysts; patients requiring longer duration (>14 days) should be monitored for hepatotoxicity and leucopenia.[5,29] Praziquantel is used in a dose of 50 mg/kg/d for 15 days, especially in persistent lesions.[22]

Corticosteroids (Anti-inflammatory agent): Severe inflammation due to antiparasitic treatment may cause death/disability, thus favoring adjunctive corticosteroids to minimize seizures, particularly in single enhancing lesions. It also reduces the diffuse cerebral edema (multiple inflamed cysticerci) in cysticercal encephalitis.[28] Routine corticosteroid use in calcified lesions/perilesional edema, remains unjustified due to risk of rebound edema while tapering.[29] Oral prednisolone is given at 2 mg/kg/d after meals, 2–3d before starting albendazole, continued for the duration of the albendazole therapy, with rapid tapering over next few days.[22] Children with raised intracranial pressure, numerous disseminated lesions, cysticercal encephalitis, subarachnoid disease, vasculitis, extensive cerebral edema or intraocular cysticercosis may need intravenous or prolonged courses of corticosteroids; such patients need to be screened for strongyloidiasis, latent tuberculosis, and vitamin D deficiency.[1,22]

Anti-seizure drug therapy: NCC lesion(s) act as a focus for recurrent seizures (focal epilepsy), necessitating antiseizure management even with a single seizure.[29] Antiepileptic selection depends on local availability, cost, interactions and potential side effects; most prefer phenytoin, carbamazepine, or newer therapies like leviteracetam.[22,28] Anticonvulsant therapy is recommended till neuroradiologic resolution and seizures have not occurred for 6 months (for single lesion) or 1 to 2 years (for multiple lesions).[1] Risk of seizure recurrence is high in children with abnormal neuroimaging (persistent or calcified lesion or multiple lesions prior to cysticidal therapy) and abnormal electroencephalograph during antiepileptic withdrawal; thus longer durations of anti-epileptic drugs may be necessary.[31]

Managing elevated intracranial pressure: Diffuse cerebral edema can be managed medically with corticosteroids (dexamethasone 0.2 to 0.4 mg/kg per day) to reduce the inflammation. Elevated intracranial pressure due to obstructive hydrocephalus (intraventricular disease) requires surgical removal of obstructing cysticercus or placement of an external ventricular drain/ shunt, while communicating hydrocephalus (subarachnoid disease) needs cerebrospinal fluid diversion via ventriculo-peritoneal shunt.[28]

Surgical management for NCC: This is recommended in extra-parenchymal NCC, intraventricular cysts, hydrocephalus due to racemose cysts/ependymitis, spinal cysticercosis (intramedullary/ extramedullary), large parenchymal colloidal cyst/ subarachnoid racemose cyst causing mass effect, an atypical SCG (to confirm diagnosis) and intractable epilepsy associated with NCC.[32]

Ocular cysticercosis is treated by surgical excision of the cysticerci and not with anti-helmintic drugs (as drugs may exacerbate inflammation).[1] Optimal management in asymptomatic patients with incidental radiological enhancing lesions is unclear and should be individualized based on patient preference. There is no benefit of therapy in an asymptomatic patient with non-viable lesion.[28]

Future prospects include research into role of genetic susceptibility to NCC and helminth-related immune mechanisms.[22] Newer drugs (tamoxifen and oxfendazole) and drug delivery systems (lactic acid-conjugated lipid nanoparticles bearing albendazole and prednisolone) are being considered to optimize drug delivery.[33,34]

Prognosis

The outcome depends on the type, location (parenchymal vs. extra-parenchymal), number of lesions, seizure recurrences and radiologic resolution of lesions.[22,35] Good prognosis and good seizure control is anticipated with single lesions (resolution in >60% cases within 6 months).[22] Seizure recurrences are frequent in multiple and calcified lesions; risk being higher if >5 lesions exist, lesions persist on follow-up neuroimaging and calcified lesions are seen in recent neuroimaging.[35] Therapy with steroids with/without albendazole is significantly associated with better seizure outcome as compared with no cysticidal therapy.[35] Cysticercus encephalitis, racemose and extra-parenchymal NCC have guarded prognosis.[22]

Singhi et al. in their follow up of 500 children with NCC and epilepsy observed cognitive impairment in one-fourth, more often in those from lower socioeconomic status and with multiple-lesion neurocysticercosis. Other problems noted on follow-up include poor scholastic performance, aggressive behavior, conduct problems, poor attention span, anger management issues, poor memory, and chronic headache.[36] NCC causes decline in cognitive function and behaviors in older children, which needs to be identified early for appropriate management and preventing undue parental anxiety.[37]

Recommendations

NCC should be considered as a differential diagnosis in a normally developing child presenting with sudden-onset seizures, headache, vomiting, or focal motor deficits with no suggestion of another underlying neurological disorder.[22] In endemic areas, physicians should be aware of atypical presentations such as communicating hydrocephalus, vasculitis, stroke, dorsal midbrain syndrome, ptosis, amaurosis fugax, dystonia, neurocognitive deficits, involuntary movements like myoclonus/hemiballismus and psychiatric presentations.[26,38,39] Due to the difficulty encountered on purely clinical diagnosis, neuroimaging and serology may be necessary.[26] An ophthalmologic evaluation is mandatory before starting of antiparasitic treatment (to exclude ocular cysticercosis). Seeking Taenia carriers among household contacts (especially in non-endemic areas) will allow detection of the potential source of infection and reduce further disease spread.[1]

Prevention

Due to the potentially eradicable nature of cysticercosis, preventive measures should be encouraged. These include preventing open defecation, improving food-handling practices, ensuring proper meat inspection, and enforcing strict personal and public hygiene. Other measures are- mass administration of anti-helminthic drugs to humans and pigs (in endemic areas), mass vaccination of pigs, improved animal husbandry, and raising public awareness about the disease.[2] Prevention of NCC should be part of health policy in endemic areas.

Conclusion

NCC remains a serious public health issue impacting both adults and children. The disease manifestation may vary, and diagnosis often involves combining imaging, clinical features and serologic studies. Since symptoms are due to host immune-response and not active parasites, initial treatment should focus on symptom management. Despite effective cysticidal therapy, the prognosis often remains uncertain. NCC is often considered as a biologic marker of poverty and underdevelopment. Meticulous case finding, improved diagnosis and management, and public health information campaigns are essential to control and break life cycle of the parasite. Involvement of private and public sectors is needed to intensify control strategies for T. solium and improve management of affected patients.

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