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RESEARCH ARTICLE

A REVIEW ON NEUROCYSTICERCOSIS- PARENCHYMAL VERSUS EXTRAPARENCHYMAL AND PEDIATRIC VERSUS ADULT

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ABSTRACT

NCC is a common parasitic infection of childhood. Its presentation is variable both in children and adults. Cysticercosis most commonly infects central nervous system. It can present as both parenchymal and extraparenchymal infection. It can be completely asymptomatic or can present with seizures, headache or vomiting. It might present with complications like hydrocephalus, cerebral edema and raised intracranial pressure in patients. Neuroimaging modalities like CT or MRI are mainstay of diagnosis. Serological tests are not routinely used. Albendazole is an anti cysticercal drug used for its cure although its usage and duration of its use is widely debatable. Surgical interventions like ventriculoperitoneal shunt and excision of cysts are used for treatment of complications.

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INTRODUCTION

Neurocysticercosis is a common parasitic infection of both children and adults. It is more prevalent in developing countries but because of recent globalization and increased travelling it is now a days seen in developed countries as well. It usually infects brain but other organs may also be affected. It is a treatable disease but sometimes due to unusual presentation and complications it might lead to morbidity and mortality. Human cysticercosis is caused by larval stage (cysticercus) of pork tapeworm **(**Taenia solium). It is a common parasitic central nervous system infection of human (Blanton 2007; Bhattacharjee *et al*., 2011). Humans are definite hosts. Human cysticercosis is acquired through ingestion of undercooked pork and contaminated food and water and through autoinfection by eggs refluxing from intestine to stomach. Individuals infected with adult worm may infect themselves with eggs by feco-oral route (Blanton 2007). Eggs of taenia hatch in human intestine and release larvae that penetrate intestinal mucosa and migrate throughout body to produce human cysticercosis. These cysts have predilection for areas with rich blood supply. These mature cysts are found in CNS, skeletal muscle, subcutaneous tissue, and eyes though they may be seen in any human tissue. Life cycle of parasite is completed when pigs ingest parasitic eggs passed in human faeces while grazing on contaminated soil. These eggs hatch in intestine into larvae that invade mucosa to reach various tissues where they mature into cysticerci.

There are 4 stages of cyst in human brain –

1) **Vesicular stage**- In it, the cyst is filled with clear fluid with a thin semitransparent wall and an eccentric opaque 4–5 mm scolex. These cysts are viable and produce scarce inflammatory changes and are usually asymptomatic.
2) **Colloid stage-** The host immune system overcomes the protective mechanisms of cyst, it degenerates and elicits an inflammatory response thus the larva undergoes hyaline degeneration and the cyst fluid is replaced with gelatinous material giving rise to colloidal stage.
3) **Granular nodular stage-** In due course of time cyst contracts, its walls are replaced by focal lymphoid nodules and necrosis, and the scolex is transformed into coarse mineralized granules forming the granular nodular stage**.**
4) **Nodular calcified stage**- In this stage collagenous structures and calcification develops in place of granulation tissue (Escobar 1983).

NCC patients have varied range of presentation from asymptomatic cases to cases with severe neurological problems. The clinical manifestations in NCC depends on

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location, number and viability of cysts and host immune response. The clinical presentation also differ across countries like in India most cases present with single degenerating lesion while in Latin America and China cases present with few viable lesions (Singh 1997).

Parenchymal NCC

In children cysticerci usually lodge in brain parenchyma. In brain parenchyma, parietal lobe is most commonly involved because of tendency to involve middle cerebral artery and its branches (Roos *et al*., 2005). Clinical manifestations of parenchymal and extraparenchymal NCC are different. Parenchymal NCC present most commonly with seizures. In NCC seizures usually occur suddenly in an otherwise healthy individual. Seizures are both generalized (Basu *et al*., 2007; Gauchen *et al*., 2011; Prasad *et al*., 2012; Bhattacharjee *et al*., 2013) and focal (Singhi *et al*., 2000; Singhi 2011; Singhi *et al*., 2004; Morales *et al*., 2000; Talukdar *et al*., 2002). Prevalence of seizures vary from 70–90% in various case reports. In a study done in India on 500 children, seizures were reported in 94.8% cases (Singhi *et al*., 2000). Seizure is a common presentation during transitional stages of the cyst. Seizures are usually present in degenerating cysts due to inflammatory responses elicited by these cysts but some of the patients with degenerating cysts can be completely asymptomatic. Presence of seizures is also related to some geographic and racial distribution. In Asian countries, seizures are usually associated with degenerating cysts (Singh 1997; Singhi *et al*., 2000) while in Latin American countries, seizures are associated more with vesicular cyst (Carpio *et al*., 2008; Garcia *et al*., 2004). These patients can also present with status epilepticus and it has been reported in 1.7–32% of children (Singhi *et al*., 2000; Morales *et al*., 2000; Talukdar *et al*., 2002; Baranwal *et al*., 1998). Various neurological deficits have been documented in 4–6% of children depending on the location of the cysts. Transient hemiparesis and monoparesis can occur. Ptosis can also occur with cysts in the midbrain (Singhi *et al*., 2008). Persistent headache is other presenting complaint in NCC and is common in children with calcified cyst. Headache and vomiting were observed in 63% cases in a study (Bhattacharjee *et al*., 2013) and in 33% cases in another study (Singhi *et al*., 2000). In one study, 44% incidences of headache because of raised intracranial tension have been reported (Kalra *et al*., 1992).

Extraparenchymal NCC

Extraparenchymal NCC is commonly seen in adults and is uncommon in children. Intraventricular cysts may remain asymptomatic for years but may become symptomatic if they obstruct the CSF flow causing hydrocephalus and increased ICP. The lateral ventricles and the fourth ventricle are the most common locations. Intraventricular disease is associated with significant morbidity and mortality (Bandres *et al*., 1992; McCormick 1985; Richards *et al*., 1985; Vasconcelos *et al*., 1987). Intraventricular cysts usually presents with raised intracranial pressure and hydrocephalous while subarachnoid cysts presents with arachnoiditis and chronic meningitis. Hydrocephalous can result either because of direct obstruction of cerebrospinal fluid (CSF) pathways by intraventricular cysts or secondary to inflammatory obstruction. Extraparenchymal NCC is seen more in Latin American countries as compared to Indian subcontinent. This is due to a complex interaction between host, parasite and environmental factors (Fleury *et al*., 2010).

Other locations

Cysticercus has a remarkable tendency to lodge in eyes, muscles, and CNS (Sotelo *et al*., 1985). This is because of the high content of glucose or glycogen and rich vascular network in these organs (Escobar 1983). Cysts in and around spinal cord occur in 1–5% cases of adult NCC. These result in various spinal dysfunction. Cysts present in eyes and in extraocular muscles cause visual deficits, limitation of eye movements and other eye symptoms. There are certain unusual presentations of NCC like behavioural changes, neurocognitive deficits and dystonias.

Adult versus Pediatric NCC

The manifestations of NCC varies according to age. Most cases of childhood NCC present with mild to moderate symptomatology and single lesion. Extraparenchymal NCC is common in adults as compared to children (Bhattacharjee *et al*., 2013; Singhi *et al*., 2004; Saenz *et al*., 2006; Antoniuk *et al*., 2006). In children, extraparenchymal NCC carries a poor prognosis (Singhi *et al*., 2004). According to a study done by Saenz *et al*., (Saenz *et al*., 2006) seizures were more common in children (80.4% versus 56.1%), while intracranial hypertension and headaches were more common in adults (27.2% versus 15.2% and 35.1% versus 21.7% respectively). In this study, cerebrospinal fluid inflammation was greater in adults than in children. Single colloidal parenchymal cysts are common in children while in adults multiple viable parasites in the basal subarachnoidal cisterns or in the ventricles are present (Saenz *et al*., 2006). Higher frequency of generalized seizures (60%) were reported in adults in a study (Del Brutto *et al*., 1992). It may be because of higher number of adult cases with multiple lesions. Children usually present with a single brief seizure lasting less than 5 minutes but some children may have repeated seizures. Clustering of seizures is also common in adults as compared to children. Acute encephalitic presentation is common in young children and adolescents, especially females, with numerous cysts and diffuse cerebral oedema, and severe acute raised intracranial pressure. Children are also known to have unusual presentations like behavioural changes, neurocognitive deficits and dystonia.

Diagnosis

NCC should be suspected in all cases who have ring enhancing brain CT lesion of less than 2 cm size presenting with seizure with no neurodeficit and no evidences of systemic diseases (White *et al*., 2008). A close differential diagnosis of NCC is tuberculoma. NCC and tuberculoma can be differentiated by neuroimaging as lesion in tuberculomas are more than 20 mm diameter, wall thickness more than 2 mm, presence of midline shift, lack of white dot like scolices and lack of spontaneous disappearance of tuberculoma (White *et al*., 2008; Kalra 2009 IAP; Kalra 2009 Ghai). In addition the absence of permanent focal neurodeficit strongly supports the diagnosis of neurocysticercosis. The definition of subarachnoid giant cysts in the literature is not precise. Some authors have described it as a surgically removable cyst approximately 50 mm in diameter and 60 ml in volume (Joubert *et al*.,1990; Castellanos *et al*., 2000; Berman *et al*.,1981). Other authors disagree with the sole criteria of size to define these cysts. According to few authors, intracranial hypertension and severe compression and displacement of adjacent brain structures as documented by imaging should also be taken into consideration.

Neuroimaging

MRI and CT are the imaging modalities commonly used to diagnose NCC. In a study (Hector *et al*., 1989) MR was compared with CT for diagnosis of NCC and it was observed that MR was four times more sensitive in the detection of the cysts (85% vs 21 %). This is because MR could detect cysts in the brainstem, in a subependymal location, in the cerebellum, in the subarachnoid space, and inside the ventricles. Bone and other artifacts on CT usually do not allow the identification of cysts in these areas. On MRI, migration of IVNCC is also reported although rare. In a study (Thomas *et al*., 2005) 10 year old boy presenting with headache and vomiting had MRI findings of marked hydrocephalus with cystic lesion isointense to CSF, with a thin wall and a small nodule (scolex) in the left temporal horn. Contrast images performed 20 minutes later revealed migration of the cyst to the occipital horn. MRI also has limitation in detecting NCC. Intraventricular neurocysticercosis is often missed on routine magnetic resonance imaging (MRI) of brain owing to similar signal intensity of cyst in cerebrospinal fluid (CSF) spaces and absence of contrast enhancement of cyst wall. Recently fast imaging employing steady state acquisition (FIESTA) and susceptibility weighted angiography (SWAN) sequences have been shown to better delineate the neurocysticercosis in the CSF spaces. FIESTA also called 3D constructive interference in steady state (CISS) is a heavily T2-weighted (T2W) high-resolution sequence, which increases the conspicuity of lesions in cisterns, sulci, and intraventricular spaces. It is the best noninvasive sequence to show subarachnoid and intraventricular neurocysticercus (Govindappa *et al*., 2000). There is recommendation for routine use of these sequences in patients with suspected neurocysticercosis (Hingwala *et al*., 2011). Many cases of intraventricular obstructive hydrocephalus have been diagnosed using 3D CISS that were misdiagnosed on conventional sequences (Dincer *et al*., 2009). SWAN or susceptibility-weighted imaging (SWI) is another advanced modality of investigation. Study has showed increased visibility of scolex in SWI thus it is recommended to use FIESTA and SWI sequences in addition to conventional MRI sequence in evaluation of patients with unexplained hydrocephalus specifically from endemic areas of neurocysticercosis (Verma *et al*., 2012).

Serologic tests

In certain studies sensitivity and specificity of the presently used commercial ELISA in serum was reported to be 85% and 94% respectively (Sahu *et al*., 2009; Biswas *et al*., 2004). Sensitivity of a test detecting antibodies for the parasite is also related to the number of lesions present in brain. In patients with multiple larvae there is release of more antigens resulting in a stronger antibody response in the infected host (Zea Vera *et al*., 2013). In a comparative study of enzyme-linked immunosorbent assay (ELISA) and enzyme linked immunoelectrotransfer blot (EITB) done in children, it was found that both were more sensitive in cases with multiple brain lesions (100%) than in those with a single lesion (87%) (Mandal *et al*., 2006). The antigen detecting ELISA has better sensitivity using CSF samples as compared to serum samples. However, ELISA has less sensitivity as compared with EITB for both serum and CSF samples, for both intraparenchymal and extraparenchymal NCC (Rodriguez *et al*., 2009). Till now there is no ideal serologic test for single-lesion NCC. A positive serodiagnostic test supports the diagnosis of NCC, however, a negative test does not exclude NCC. In endemic areas individuals may be seropositive without having NCC.

Electroencephalogram (EEG)

EEG is not usually done in typical cases of NCC. It is usually done in cases with status epilepticus, with refactory seizures and in whom diagnostic dilemma is present. Study has showed that EEG demonstrated little relation to symptoms and CT lesions in patients with neurocysticercosis (Carpio 2002).

Other tests

Lumbar puncture test is usually done in cases suspected of having NCC meningitis. CSF examination in these patients may show mild elevation of protein, hypoglycorrhachia and pleocytosis which may be lymphocytic, monocytic or polymorphonuclear. CSF is usually normal in cases of parenchymal NCC, thus it is not routinely indicated. Other tests that may support the diagnosis of NCC include biopsy of subcutaneous nodules (if present) to look for the parasite, and blood counts, which may show peripheral eosinophilia in about one third of patients. Tapeworms in stool microscopy is indicative of taeniasis but does not necessarily indicate the presence of NCC. These tests do not carry much significance hence are not routinely done.

Treatment

Albendazole, a member of the benzimidazole group, destroy parenchymal NCC and may destroy some of the intraventricular cysts as well. It is less expensive, better tolerated and has better penetration into subarachnoid space. Administration of steroids with albendazole increases bioavailability of albendazole (Jung *et al*., 1990) while phenytoin or carbamezepine does not affect it. The bioavailability of praziquantel decreases with co-administration of steroids (Vazquez *et al*., 1987), phenytoin and carbamezepine (Bittencourt *et al*., 1992). Albendazole is used at a dose of 15 mg/kg/day (maximum 800 mg) in two or three divided doses in children. Albendazole is a safe drug with transient gastrointestinal symptoms such as nausea, diarrhoea and vomiting which occur rarely but prolonged use may cause an increase in serum aminotransferase, jaundice, headaches, loss of hair, leukopenia and thrombocytopenia. Allergic phenomena like urticaria, rashes and oedema can also rarely occur. Praziquantel is used at a dose of 50 mg/kg/day. Side effects include abdominal discomfort, headache, dizziness and drowsiness. Idiosyncratic side effects include pruritus, fever, rashes and muscle and joint pains. The usual duration of therapy is for a period of 15 days. The use of albendazole in children is a debatable topic. There are supporters favouring no albendazole therapy, 7 days short course therapy and 28 days long course albendazole therapy (Blanton 2007; Singhi *et al*., 2004; Kalra 2009 IAP; Kalra 2009 Ghai; Cuellar *et al*., 1999; Kaur *et al*., 2010).

Albendazole and other anticysticercal drugs are contraindicated for spinal and ocular cysticercosis, as drug induced inflammation may result in irreversible damage to these organs. Use of anticysticercal drugs is debated in degenerating cysts and it is of no use in calcified lesions. Albendazole is more effective than praziquantel (Cruz *et al*., 1991; Sotelo *et al*., 1990; Escobedo *et al*., 1987).

Antiepileptic therapy

Seizures in NCC are usually well controlled with a single antiepileptic drug (AED). The recurrence rate after AED withdrawal is low in cases with single-lesion NCC both in children (Talukdar *et al*., 2002; Baranwal *et al*., 2001) and adults (Goel *et al*., 2010). The usual practice is to give AED for 2 years seizure-free interval but it can be withdrawn after 1 year seizure free interval in cases where lesion has disappeared and EEG has normalized. Prolonged treatment is needed for those with persistent or calcified lesions.

Corticosteroids

Oral corticosteroids are administrated generally few days before and a few days along with anticysticercal therapy to prevent any adverse reactions that may occur due to host inflammatory response. Usually oral prednisolone 1–2 mg/kg is used. In cases with disseminated lesions and extensive cerebral oedema, steroids may be given for prolonged period.

Surgery

In intraventricular NCC either a ventriculoperitoneal shunt or a temporary ventriculostomy must be performed if both hydrocephalus and increased ICP are present (Del Brutto *et al*., 1993). Indications for surgical removal of cysts are- obstruction of CSF flow, or if they are complicating shunt functioning. Only medical therapy without any surgical intervention is required in patients with intraventricular cysts without hydrocephalus, or with only slight dilatation of the ventricles. Hydrocephalus sometimes return in patients of intraventricular cysts even after disappearance of cyst. There is liberation of antigenic substances inducing inflammatory response in ventricular system with dying cysts which cause ventriculitis (Madrazo *et al*., 1983). This in turn causes blockage of ventricular system as the cyst capsule attaches to ventricular wall or subarachnoid tissue with strong adhesions and thickeneing. This tissue cannot be removed surgically as it may damage the brain tissue (Madrazo *et al*., 1983). Thus ventriculoperitoneal shunt is required. These ventriculoperitoneal shunt can sometimes get blocked because of obstruction by either a gelatinous material from cyst or high CSF protein level. Patients who have inflammatory cyst may also have active cysts that might not be visible on neuroimaging, thus it is recommended to give antihelminthic drugs in inflammatory cysts as well to accelerate the death of the parasite in the active cysts which have been missed. (Fleury *et al*., 2010; Del Brutto *et al*., 1993; Vazquez *et al*., 1992; Cuetter *et al*., 1992; Rawlings *et al*., 1989; Sotelo *et al*., 1988).

Subarachnoid cysts in the basilar cisterns are known to produce hydrocephalus, raised ICP and meningeal irritation. Subarachnoid cysts are usually associated with parenchymal cysts thus the recommendation is to treat these cysts with anthelmintic drugs and shunting procedures if there is hydrocephalus, and surgical removal of the cysts (Del Brutto *et al*., 1993). Some authors suggest that surgery should be performed as soon as possible after the diagnosis of subarachnoid cysts to minimize the risk of sequelae or death (Castellanos *et al*., 2000; Colli *et al*., 1994; Soto *et al*., 1996), while some authors advocate that surgical removal of cysts should be considered only in patients who have life-threatening intracranial hypertension despite treatment with corticosteroids. Thus surgical procedures and their potential complications can be avoided in most patients with neurocysticercosis with giant subarachnoid cysts which was earlier considered to be an absolute indication for surgery (Proano *et al*., 2001).

Newer modality of endoscopic IVNCC cyst excision is being used these days. It is a safe and effective option and avoids shunt and its related complications in these children (Suri *et al*., 2008). Magnetic resonance imaging is also a guide for treatment of IVNCC. The absence of pericystic Gadolinium enhancement on MR imaging is an indication for excision of lesions and if pericystic enhancement is present, shunt surgery should be performed. Jonathan Stuart *et al*., proposed a treatment algorithm based on patient symptoms, cyst location, and MR imaging Gd enhancement characteristics (Citow *et al*., 2002). Indications of surgery are: cysts that compress the brain and cranial nerves locally, intracranial hypertension or edema refractory to medical treatment, intraventricular NCC with hydrocephalus and raised ICP, spinal NCC with cord or root compression and ocular cysts (Sinha *et al*., 2009)

Follow up

Follow up is required for all the patients on treatment. A CT scan is repeated after 3–6 months to determine whether the lesions have resolved or not. A repeat course of cysticidal therapy is often given in cases with persistent but not calcified lesions.

Prevention

NCC is a preventable disease. Its burden can be reduced by educating masses for proper hygiene practices. Pigs should be treated for cysticercal infection. Many vaccines are under trial for preventing pigs from infection.

Conclusion

NCC is a cause of seizures in pediatric as well as adult population. NCC can be treated by medical therapy alone if addressed on time but sometimes it can land up into complications requiring surgical intervention. Tuberculoma being a close differential of NCC should be ruled out before labeling a patient with NCC. NCC is a preventable disease and best approach to reduce its burden is to prevent it.

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