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Taenia solium Cysticercosis: Present Scenario: A Review

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ABSTRACT

Cysticercosis is one of the most important neglected parasitic diseases caused by the larval stage of the tapeworm *Taenia solium*. It is the most common cause of epilepsy in the areas with improper pig husbandry practices and poor sanitary conditions. In pig the larvae lodges in the muscle and subcutaneous tissue and seldom manifests any clinical sign. In human beings the larval stage resides in the CNS thus leading to the condition Neurocysticercosis. The disease can be diagnosed by using various neuroimaging techniques and immunodiagnostic methods. Available therapeutic options include cysticidal drugs, steroids, anti-epileptic drugs *etc*. The disease can be controlled by anti-parasitic treatment of the pig and humans residing in endemic areas, vaccinating the pigs, creating awareness and other measures.

Key words: Cysticercosis, Cysticidal drugs, Epilepsy, Neurocysticercosis, Neuroimaging, *Taenia solium*, Vaccination.

Taenia solium cysticercosis is regarded as an important neglected, public health concern especially in the areas with poor sanitation and improper animal husbandry practices (Secka *et al*., 2010 and WHO, 2014). It is a food-borne parasitic disease with a global perspective (Robertson *et al*., 2013 and WHO, 2010). Pigs act as the intermediate host whereas human beings can act as both definitive and intermediate host. Pigs reared under free range management system is considered as one of the major risk factor of cysticercosis as it allows pigs to have free access to human excreta particularly in the areas where latrines are not utilised, absent or improperly constructed (Ngowi *et al*., 2004 and Sikasunge *et al*., 2007). Activities of human have an impact on every stage of the parasitic life cycle. Human beings bearing the pork tapeworm in their intestinal lumen are accountable for contamination of the environment with *T. solium* eggs by open defecation (Sarti and Rajshekhar, 2003), thus transmitting *T. solium* infections in humans and pigs cycle. Pigs and humans acquire cysticercosis by consumption of feed/food and water contaminated with the faeces of human carrier laden with eggs or gravid proglottids of *Taenia solium*. In case of humans, larvae commonly reside in the skeletal muscles, subcutaneous tissues, eyes and brain leading to the condition neurocysticercosis (NCC). In pigs, presence of cysticerci in its various predilection sites *viz*. masseter muscle, skeletal muscle, heart, diaphragm *etc*. makes the pork unsafe for human consumption and causes vivid economic losses as a result of loss of market value. As per the global economic impact studies (Carabin *et al*. 2006; Praet *et al*., 2010), pigs with *T. solium* infection accounts for 4.7%-26.9% of total expenses of pig farming, which result into a loss of €10 million and US$18.6 to US$ 34.2 million annually respectively. However, this assessment only provides an indication rather than giving an exact determination of economic loss (Bulaya *et al*., 2015). Even though economic losses caused by porcine cysticercosis may be extensive due to condemnation of infected pig carcasses (Yoder *et al*., 1994; Ogunremi and Benjamin, 2010). This cannot be considered as the major problem of the rural communities where animals brought for human consumption are not slaughtered in a slaughter house or not inspected for presence of any diseases. Therefore in these communities, effect of *Taenia solium* infections is considered as a public health crisis.

Here, epidemiology, risk factors, pathogenesis, clinical manifestation, methods of diagnosis and therapeutic processes and control measures of *Taenia solium* cysticercosis is reviewed.

Epidemiology

As per WHO, globally 50 million people are infected with cysticercosis with highest cases recorded from Africa, Mexico and South and Central America (Fig 1). In this countries nearly 50 thousand people dies of NCC (Roman *et al*., 2000). NCC affects 4% of the population (Sarti *et al.,* 1992). According to Sarti *et al*. (1992), it has been found that it affects 4% of the population in endemic areas where

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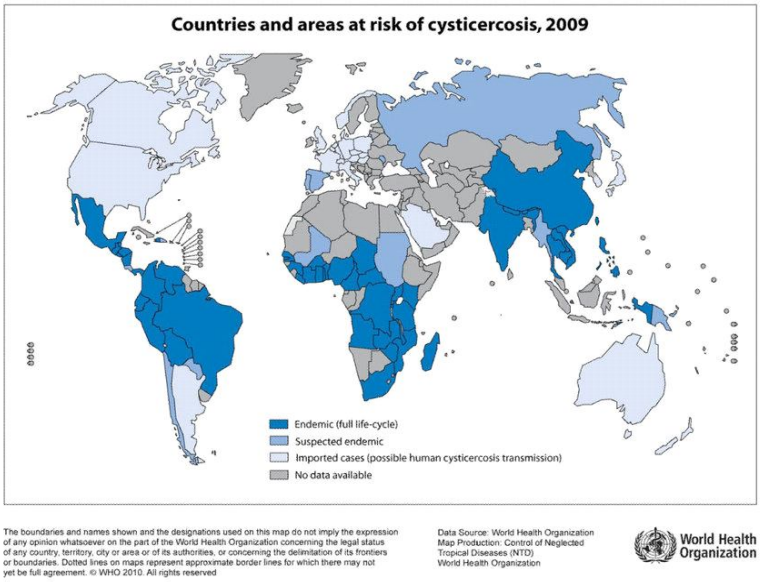

Fig 1: Countries and areas at risk of cysticercosis.

hygiene, habits-food and sometimes religious trends can determine the incidence and prevalence of the disease (Savioli, 2010). Systematic reviews have showed seizure/ epilepsy as the most frequent clinical manifestation among patients diagnosed for NCC (Carabin *et al*., 2011) and in endemic countries, the prevalence of NCC among individuals having seizure is found to be 29.0% (Ndimubanzi *et al*., 2010). In 2010, *Taenia solium* infection was associated with 2.8 million disability adjusted life year losses (WHO, 2015). The overall burden of NCC across the globe has been roughly calculated to be more than 2 million per annum (Torgerson and Macpherson, 2011). Cysticercosis is highly prevalent in Sub Saharan Africa, Asia and Latin America countries (Phiri *et al*., 2003; Zoli *et al.,* 2003; WHO/FAO/ OIE, 2005). In China, NCC was estimated to affect up to 7 million people (Coyle *et al*., 2012). In Tanzania, cysticercosis is common among the pig producing areas (Ngowi *et al.,* 2004). The prevalence rate in Northern highway and Southern highway of the country was 0.3- 17.4% and 5.5% respectively (Boa *et al.,* 2001; Boa *et al.,* 2006). Cameroon has been recorded to be endemic for both porcine and human cysticercosis (Assana *et al*., 2010; Nguekam *et al.,* 2003; Zoli *et al*., 2003). In China, the burden of cysticercosis was estimated to be 3-6 milion and 11-29 million in Latin America (WHO, 2013). Globally, 80% of population associated with epilepsy live in countries having poor resources where majority of the individuals are affected by neurocysticercosis (NCC), cysticercosis of CNS, with the prevalence rate of 5 to 50% in epileptic patients (Bruno *et al*., 2013; Moyano *et al*., 2014; Rottbecket *et al*., 2013). It is regarded to be a pivotal aetiology of seizure/epilepsy in areas endemic for *T. solium* (Del Brutto and Garcia, 2012; Moyano *et al*., 2014). In Madagascar, the seroprevalence of *T. Solium* infection in human and pig ranges from 7-21% and 7-48% respectively (Michelet *et al*., 2010). Cysticercosis is also present in other islands OF Indian Ocean, particularly in La Réunion (Michault *et al*., 1990). In India, NCC is most common parasitic disease responsible for 47% of epilptic cases in South India (Chowdary *et al.,* 2004). In North India, NCC is the major cause of focal seizures in children (Singhi and Singhi, 1997). Khurana *et al*., (2006) recorded 17.3% seroprevalence of cysticercosis from a North Indian city with 8% presenting seizure. In Burundi, the prevalence of NCC among patient with epilepsy was 11.7% and normal individuals were 2.8% (Newell *et al*., 1997). In a nation abattoir survey of Eastern Cape Province (ECP) conducted in 1937, the prevalence and incidence of cysticercosis was recorded to be 25% and 10% respectively (WHO, 2003).

Risk factors

There are several factors that contribute to the prevalence of cysticercosis in many developing countries. It is a disease of poor socioeconomic background. The disease is mainly associated with poverty, system of pig management, rearing of pigs and persons harbouring pork tapeworm in their intestinal lumen, cultural factors (Boa *et al*., 2001; Juyal *et al*., 2008; Willingham III *et al*., 2010). It is mostly present is rural areas with inadequate sanitation having no facilities for meat inspection (Phiri *et al*., 2003; Assana *et al*., 2012). The spread of the disease in human is facilitated by multitude of factors which include use of untreated or partially treated human waste in agriculture, improper food handling, lack of knowledge concerning the risk of infection while visiting endemic areas and consumption of raw or uncooked meat *etc*. (Swastika *et al*., 2016). Activities of human affect every stages of parasitic life cycle. Human beings bearing the pork tapeworm in their intestinal lumen are responsible for environmental contamination (Sarti and Rajshekhar, 2003). Feed type, source of water supply, lack of deworming of pigs, purchase of replacement pigs, source of income of

the farmers and pigs managing on uncooked swill are some of the risk factor associated with cysticercosis in pig (Samuel *et al*., 2018). Pig husbandry is one of the important factors associated with porcine particularly in areas where latrines are absent or poorly constructed (Pouedet *et al*., 2002; Ngowi *et al*., 2004; Sikasunge *et al*., 2007).

Etiopathogenesis

The life cycle of the parasite *Taenia solium* involves two hosts: intermediate and definitive. Human beings are the lone definitive hosts which harbours the adult stage of the parasite, whereas pigs and humans both can habour the larval stage of the parasite (cysticercus) thus serving as the intermediate hosts of the pork tapeworm. In human, adult *T. solium* resides in the small intestine and attached themselves to the intestinal wall with the help of hooks and suckers. Gravid proglottids containing the fertile eggs isolate itself from the posterior end of the parasite and are released into the environment along with human faeces. In areas where pigs are reared under free range management system and the prevailing sanitary conditions are poor, pigs come in contact with human faeces containing *T. solium* eggs and ingest them. Inside the intestinal lumen of the pig, the eggs dissolves its coat and release oncospheres that penetrates the intestinal wall and reaches the blood vessels and make its way into the circulatory system, from where they invades tissues and reaches various predilection sites and develop cysticerus. After consumption of insufficiently cooked infected pork by the humans, cysticerci reach the small intestine and there due to the activities of digestive enzymes, evagination of scolex occurs and they attach themselves to the wall of the intestine. Following this, the proglottids multiply and soon mature to adulthood and lead to taeniasis (Delbrutto *et al*., 1998: Del Brutto and Oscar 2012). Humans after consuming *Taenia solium* eggs can also act as its intermediate hosts. In this situation, it leads to human cysticercosis. Humans acquire cysticercosis after consumption of food and water contaminated with the tapeworm eggs or by the auto-infection in individuals carrying the adult pork tapeworm in their intestinal lumen (Fig 2).

Cysticerci mainly consist of vesicular wall and scolex (William, 2008: Del Brutto and Oscar, H. 2012). After entering the CNS, cysticerci remain in vesicular (viable) stage which consists of transparent membrane, vesicular fluid, and invaginated scolex. Cysticerci either remain viable for several years or, undergo degeneration due to defence mechanism of the host's immune system and are subjected to calcifications. In the colloidal stage, which is the initial stage of involution of cysticerci, turbidity of vesicular fluid occurs and the scolex is marked by hyaline degeneration. This is followed by thickening of cyst wall and mineralizationof the scolex; this stage is called granular stage. At this stage, cysticercus remains no longer viable and the remnants of the parasite look like a mineralized nodule (calcified stage)(Escobar and Weidenheim, 2002; Del Brutto and Oscar 2012).

Vesicular cysticerci induce a slight inflammatory reaction that is pronounced in the surrounding tissue. In contrary, colloidal cysticerci are enclosed by collagen capsule and are marked by mononuclear inflammatory reaction containing parasite itself. The parenchyma of the brain is marked by various inflammatory changes which include astrocytic gliosis, microglial proliferation, edema, neuronal degenerative changes and perivascular cuffing of lymphocytes. As parasites encounters the granular and calcified stages, the edema disappears whereas the astrocytic changes in the suroounding tissues become more pronounced, epithelioid cells come and fuses with each other and lead to the formation of multinucleated giant cells (Pittella, 1997; Del Brutto and Oscar 2012). Cysticerci

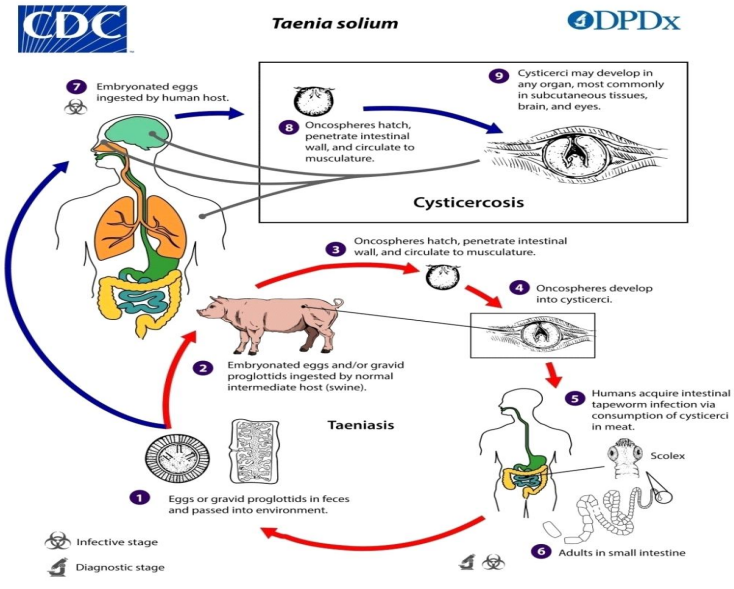

Fig 2: Life cycle of *Taenia solium.*

that develops in the meninges induces an inflammatory reaction in the sub-arachnoid space which is accompanied by the formation of an exudate which is made up of collagen fibres, lymphocytes, multinucleated giant cells, eosinophils, and hyalinised parasitic membranes which causes an atypical thickening of the leptomeninges. This inflammatory reaction may spread and elicit damage to the optic chiasm and cranial nerves, as well as to the small penetrating arteries that arises from the Willis circle. The later may lead to cerebral infraction by blocking the lumen of the vessel (Del Brutto *et al*., 2008 Del Brutto and Oscar 2012). Thickened parasitic membranes and leptomeninges lead to the blocking of Luschka and Magendie foramina which causes obstructive hydrocephalus. Ventricular cysticerci when get attached to ventricular wall or choroid plexus may induces an inflammatory changes. The damaged ependymal lining when projected toward the ventricular cavities causes obstruction of CSF transit, especially when protrusion site is at or near to foramina of Monro or cerebral aqueduct (Pittella, 1997; Del Brutto and Oscar, H. 2012).

Some antigens of the cysticercus elicit an immunological reaction leading to the production of specific antibodies that lay the foundation for immunological diagnosis of cysticercosis (Flisser *et al*., 2002 Del Brutto and Oscar, H. 2012). At the same time, patient with neurocysticercosis shows cellular immune dysfunction due to accumulation of CD8 T-lymphocytes, altered proliferation of lymphocytes and atypical cytokines concentration. It has been presumed that the reduced cellular immunity may be the reason for the association of NCC with the conditions consequent of immune compromised states, along with the glioma formation (Del Brutto *et al*., 1997; Del Brutto and Oscar, H. 2012); in conditions as such, it has been presumed that the proliferation of the glial cells surrounding the parasites, combined with the reduction in the cellular immunity may inhibit immunological scrutiny against cancer, causing malignant transformation of astrocytes.

Clinical manifestations

In pigs, cysticerci generally get lodge in the muscles and subcutaneous fat (Fig 3). Although some pigs may present high infection, cysticercosis seldom manifest any clinical symptoms. Since, pigs are slaughtered at 7 month of age; cyst fails to attain the degenerative stage than manifests clinical symptoms in man.

The clinical manifestation of NCC in human is determined by number and location of the cyst within the Central nervous system (Fig 4). The most frequent clinical manifestation of patient with NCC is seizure. In nearly 70% of the patient, seizure is only the single clinical symptoms of the disease (Del Brutto *et al*. 1992; Del Brutto and Oscar, H. 2012). In third world countries, NCC is considered as the leading cause of acquired epilepsy and also to blame for increased prevalence rate of epilepsy (Del Brutto *et al*., 2005; Medina *et al*., 2005; Montano *et al*., 2005). Seizures are most frequent clinical symptoms manifested in patients with

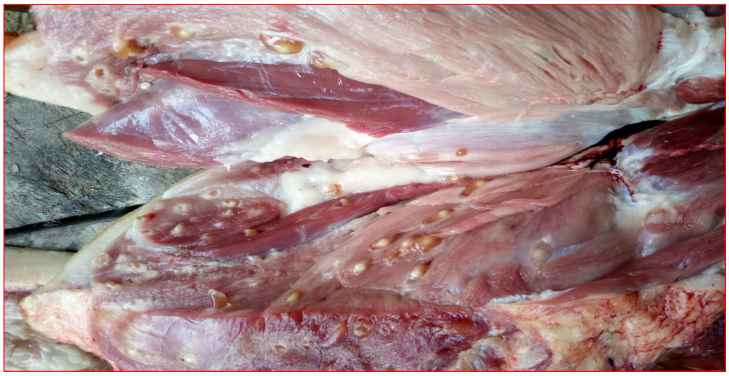

Fig 3: *Cysticercus cellulosae* clinged to the skeletal muscle of pig carcass.

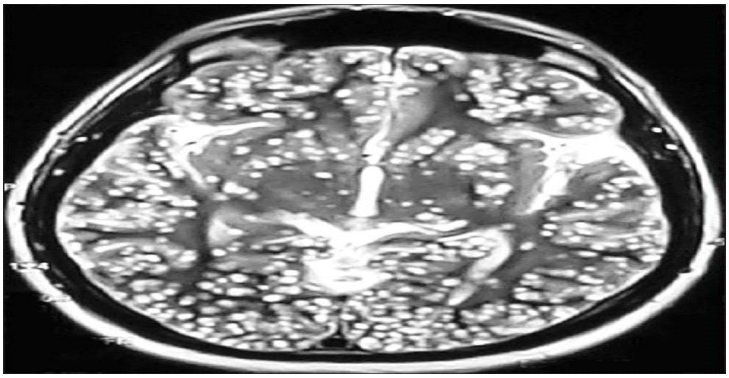

Fig 4: Tapeworm infestation in brain (Neurocysticercosis).

parenchymal NCC as compared with the patient with sub-arachnoid or ventricular cysticerci (Garcia and Del Brutto, 2005; Del Brutto and Oscar, H. 2012). Generation of epilepsy among patients with calcified cysticerci is still a matter of debate (Nash *et al*., 2004). Though calcified cyst have been regarded as an inert lesions, latest data depicts that calcified cysticerci are neither pathologically inert lesions nor clinically inactive, as they may elicit recurrent seizures due to the exposure of trapped antigen of parasite in the calcium matrix to the host immune system in the process of calcification remodelling (Nash *et al*., 2008; Del Brutto and Oscar 2012).

Focal neurological signs depend on the size, number and location of the parasites in CNS of the patients with neurocysticercosis. Although the chief clinical manifestation includes the pyramidal tract signs, but language disturbances, sensory deficits, parkinsonian rigidity, involuntary movements and dysfunction of brainstem, have been observed in several patients. These signs become apparent after sub-acute or chronic phase when the cyst attain the size of a brain tumour and are more common in patients with large subarachnoid cysts that put pressure on the brain parenchyma (Fleury *et al*. 2011; Del Brutto and Oscar, H. 2012). In 3% of the patient, Stroke syndromes have been recorded, which may be due to cerebral infarctions in the posterior limb of internal capsule, brain stem, or corona radiata (Del Brutto, 2011). In few patients intracranial hypertension have been described with or without seizures or focal neurological signs. The most common etiology associated with intracranial hypertension is hydrocephalus, which may occur due to cysticercotic arachnoiditis, granular ependymitis, or ventricular cysts (Fleury *et al.*, 2011; Del Brutto and Oscar, H. 2012). This syndrome is also present in patients with cysticercotic encephalitis, a serious form of NCC that occurs due to the presence of a large number of *Taenia solium* cysticerci in the brain parenchyma inducing a severe response of the host's immune system. This state is characterized by unconsciousness, visual disturbance, seizures papilledema, vomiting and headache and is more common among children and young women (Rangel *et al*., 1987).

Few patients with NCC may manifest some psychiatric syndrome such as poor performance, dementia, etc (Forlenza *et al*., 1997). Before the existence of CT scan, many patients with NCC presenting psychiatric symptoms were admitted to mental hospitals for many years until the exact diagnosis was made at autopsy (Nieto *et al*., 1982). Cysts when present in the sellar region manifest ophthalmologic and endocrinologic disturbances (Del Brutto *et al*., 1998; Del Brutto and Oscar 2012). Cysts when occurs in the spinal cord parenchyma causes motor and sensory deficits, which differs depending on the severity of the lesion (Alsina *et al*., 2002). Cysticerci of intraocular subretinal region are associated with diminished vision, vitritis, uveitis and endophthalmitis (Madiubba *et al*., 2007). A large number of cysticerci in the striated muscles may cause generalized weakness accompanied by progressive muscle enlargement (Wadia *et al*., 1988; Del Brutto and Oscar 2012).

Ophthalmic cysticercosis

Ophthalmic cysticercosis though less frequent but may occur in many patients (1-3% of the cases). It is commonly present in the vitreous humour or in sub-retinal space. Visual disturbance vary depending on amount of retinal tissue damaged and presence of chronic uveitis (Cardenas *et al*., 1992). Cyst may also occur in anterior chamber, conjunctiva and extraocular muscle. Some cyst may be develops in the retro-ocular space damaging the optic nerve or causing proptosis (Wadia *et al.,* 1988; Chandra *et al*., 2000). Visual loss also results from pappiloedema, chiasm compression or hydrocephalus.

Extraneural cysticercosis

Taenia solium cysticerci may also occur outside the Central nervous system but is rarely associated with any clinical symptoms. Extraneural cysticerci generally occur in arms or chest and are presented as small, movable and painless nodule which gradually becomes inflamed and disappeared (Dixon and Smithers, 1934).

Diagnosis

Several epidemiological studies have used various diagnostic techniques such as tongue inspection (Secka *et al*., 2010; Sikasunge *et al*., 2008), Enzyme-Linked Immunosorbent Assay (Praet *et al*., 2010; Sikasunge *et al*., 2008), latex agglutination, immunoblot technique (Goussanou *et al*., 2014) for diagnosis of porcine cysticercosis. Necropsy examination can be used to quantify the intensity of infection in *T. solium* affected pigs (Flisser *et al*., 2004). Specificity of lingual and carcass examination is nearly 100%, but sensitivity may differ on the basis of degree of infection (Dorny *et al*., 2004). However, on correlation between EITB and necropsy, (Sciutto *et al*., 1998) recorded that a large number of pig which were negative at necropsy were recorded to be EITB positive. The prevalence of porcine cysticercosis is very often underestimated owing to its poor efficiency of visual meat inspection, which has been indicated by many serological tests (Gomes *et al*., 2007). In contrary, its prevalence can too be overestimated through misdiagnosis of other morphological alterations in affected muscles, because the meat safety system is only based on the conventional post-mortem inspection carried out in slaughter houses. Hence, molecular diagnostics is considered in order to validate macroscopic diagnosis of ambiguous lesions as these tests are extremely specific and sensitive (Chiesa *et al*., 2010). Molecular detection of *Taenia solium* infection can be carried out with PCR. Ramahefarisoa *et al.* (2010) evaluated the performance of PCR test targeting mitochondrial cytochrome oxidase gene for the ante-mortem diagnosis of porcine cysticercosis. Sreedevi *et al*. (2012) conducted a molecular study by using PCR to confirm the meat inspection results of the pig carcasses. TBR and Cox1

primers were used targeting rRNA and cytochrome oxidase subunit 1 gene of the parasite *Taenia solium* respectively. Lokhande *et al*. (2016) carried out PCR to validate the results of meat inspection by targerting cytochrome oxidase gene. With the emergence of new neuroimaging technique, accuracy of diagnosing NCC in humans has drastically changed. CT scan and MRI provide the necessary information not only on the number and location of lesions, but also onstage of involution of cysticerci (Garcia and Del Brutto, 2003; Del Brutto and Oscar, H. 2012). In CT scan and MRI, vesicular cysticerci look like small and rounded cysts which are well demarcated from the brain parenchyma surrounding it. In such cases, neither oedema and nor contrast enhancements are seen. Interior of these lesions, an eccentric hyperdense nodule indicates the scolex, which give them a pathognomonic "hole-with-dot" appearance. Colloidal and granular cysticerci seem like an ill-defined lesions encircled by oedema; after administration of contrast medium, they represent a ring or a nodular pattern of enhancement. This kind of pattern is often referred to as "cysticercus granuloma"(Singh *et al*., 2010; Del Brutto and Oscar, H. 2012). Persons with cysticercotic encephalitis show a specific neuroimaging pattern such as diffuse brain edema, ventricular system collapse without midline shift, and multiple small ring-like or nodular enhancing lesions spread within the brain parenchyma (Rangel *et al*., 1987; Del Brutto and Oscar, H. 2012). On CT scan, calcified cysticerci look like a small hyperdense nodules without perilesional oedema or abnormal enhancement after administration of contrast medium.

The major neuroimaging finding among patients with subarachnoid neurocysticercosis is hydrocephalus due to the inflammatory blocking of foramina of Luschka and Magendie. The basal fibrous arachnoiditis that is related to hydrocephalus appears as focal or diffuse areas of abnormal enhancement of leptomeninges. Cystic lesions within CSF cisterns act as mass occupying lesion by giving a multilobulated appearance and displacing the structures present nearby (Fleury *et al*., 2011; Del Brutto and Oscar, H. 2012). MRA, a noninvasive imaging technique is helpful in diagnosing patients with subarachnoid neurocysticercosis, by detecting segmental narrowing or blocking of intracranial arteries (Del Brutto, 2008). In CT scan, cyst on ventricles seems like hypodense lesions which alter the ventricular system leading to the development of asymmetric hydrocephalus. As ventricular cysticerci and CSF are isodense, they can only be differentiated by dint of distortion of the shape of the cavities of the ventricles (Madrazo *et al*. 1983). In contrary, MRI can readily visualize ventricular cysts due to difference in the signal properties of the scolex/or cystic fluid from that of CSF (Do Amaral *et al*., 2005; Del Brutto and Oscar, H. 2012). "Ventricular migration sign" which occur due to mobility of the cyst within the cavities of the ventricles in response to the head movement, can be used as a basis for diagnosing ventricular cysticercosis. In MRI, Intramedullary cysticerci produce characteristic rounded lesions which have an eccentric hyperintense nodule indicating the scolex (Garcia *et al*., 2003; Del Brutto and Oscar, H. 2012). After administration of contrast medium, cyst periphery is marked by abnormal enhancement. There is enlargement of the spinal cord, and if the scolex cannot be detected then difficulty may arise to infer neurocysticercosis from spinal cord tumour. Leptomeningeal cysts can move within the space of the spinal subarachnoid and may rotate during the examination in response to the movements of the patient in the examination table.

Immunodiagnostic methods are useful in detecting cysticercosis in absence of neuroimaging technique (Garcia *et al*., 2012; Ito *et al*., 2006; Del Brutto and Oscar, H. 2012). Enzyme linked immunoelectrotransfer blot (ETIB) assay is a reliable immunodiagnostic tool for detecting specific antibody against antigen of *Taenia solium* (Tsang *et al*., 1989). The major drawback of the test is that in 50% of the person with solitary cerebral cyst or those with calcified cyst it gives false negative result (Singh *et al*., 2010). Second drawback is that it gives positive test result for the individuals harbouring the adult stage of the parasite in absence of the larval stage (Garcia *et al*., 2005; Del Brutto and Oscar, H. 2012). Thus it becomes difficult to infer whether the test is positive for taeniasis or cysticercosis. Monoclonal antibodies that detect the circulatory antigen cannot be used to screen neurocysticercosis as it has poor sensitivity, although, it can be use to check the response of cysticidal therapy (Mahanty and Garcia, 2010). The result of stool examination for *T. solium* eggs differ among patients and depend on severity of infection (García and Del Brutto, 1999; Gilman *et al*., 2000; Del Brutto and Oscar, H. 2012). Detecting coproantigen by ELISA and PCR can be used for screening *T. solium* carriers in endemic areas (Mahanty and Garcia, 2010).

In spite of these developments in neuroimaging and immunodiagnostic method, diagnosis of NCC still remains a problem in some patients. Clinical symptoms are not characteristic of the disease, findings of MRI and CT scan are not pathognomic, serological test often show cross reactivity. The intervention of clinical, radiological, immunological, and epidemiological data is required to diagnose patients with suspected for neurocysticercosis (Del Brtto *et al*., 2001). This includes four criteria-absolute, major, minor, and epidemiological (Fig 5). Unambiguous diagnosis of NCC, is given by absolute criteria, major criteria advocate diagnosis though cannot solely gives confirmation of the disease, minor criteria are recurrent but are not specific, and epidemiologic criteria are incidental facts supporting the diagnosis of NCC. Analysis of the criteria gives two diagnoses -definitive and probable-depending on the chance of presence of NCC in patients.

Treatment

A single method of treatment may not necessarily cure every patient with NCC. Rational therapy depends on cysts viability, response of human immune system against the parasite, and number and location of lesions (Garcia *et al*., 2002;

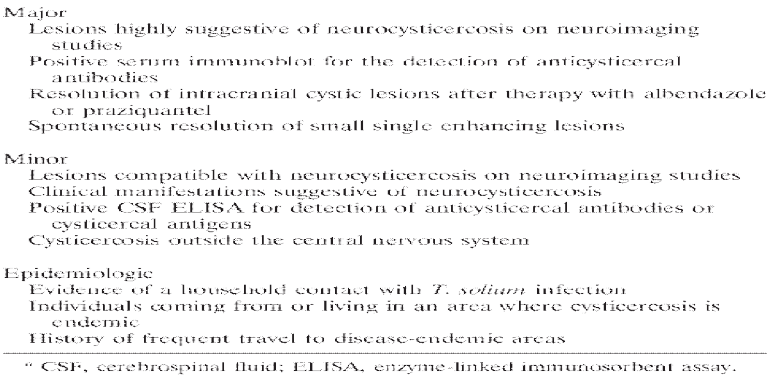

Fig 5: Diagnostic criteria for Neurocysticercosis.

Del Brutto and Oscar 2012). Therapy usually needs blending of both symptomatic and cysticidal drugs. Surgery may not be necessary in few patients. Utilisations of two potent cysticidal drugs (praziquantel and albendazole) have drastically changed the faith of many patients with neurocysticercosis (Sotelo and Diaz-Olavarrieta, 2010; Del Brutto and Oscar, H. 2012). The initially the dose of praziquantel for treatment of NCC was fixed at 50mg/kg/day (given every 8 hours) for 15 days (Sotelo *et al*., 1984). If we treat cysticerci with higher concentrations of the drug which is maintained for 6 hours -3 individual doses @ 25 to 30mg/kg at two-hour intervals, might destroy the parasites. Initial results were encouraging (Del Brutto *et al*. 1999), in single cyst single day course of praziquantel works better whereas in case of multiple cysts 15 days course of praziquantal should be given (Pretell *et al.,* 2001; Del Brutto and Oscar, H. 2012). Albendazole, another anthelmentic drug with cysticidal effect was primarily given at doses of 15mg/kg/day for a period of one month (Escobedo *et al.,* 1987). Later, it was found that duration of therapy could be reduced to a week without affecting the efficacy of the drug (Garcia *et al*., 1997), and in case of solitary cyst the duration of the therapy could be reduced to even three days (Bustos *et al*., 2006; Del Brutto and Oscar, H. 2012). Albendazole have better efficacy than praziquantal (Bustos *et al*., 1990; Takayanagui and Jardim E, 1992). Albendazole can kill both subarachnoid and ventricular cysts (Del Brutto, 1997). In patients with large subarachnoid cysts, prolonged treatment or higher dose of albendazole (up to 30mg/kg/day) may be required (Fleury *et al*., 2011; Góngora-Rivera *et al*., 2006; Proaño *et al*., 2001; Del Brutto and Oscar, H. 2012).

Utilisation of cysticidal drug in some cases in questionable. Earlier it was considered that cysticidal drugs can cause destruction of the cysts but have failed to alter the clinical course of the disease (Salinas *et al*., 1999; Del Brutto and Oscar, H. 2012). However, latest studies have demonstrated that cysticidal drugs also cause clinical improvement in many patients. In a placebo-controlled trial, it was found that albendazole was effective in the treatment of viable parenchymal brain cysticerci (Garcia *et al*., 2004; Del Brutto and Oscar, H. 2012). Other controlled trials demonstrated that patients who have received treatment for colloidal brain cysts have better prognosis than who received any treatment (Gogia *et al*., 2003; Baranwal *et al*., 1998; Kalra *et al*., 2003; Del Brutto and Oscar, H. 2012). Results of meta-analysis have showed that cysticidal therapy can better resolute the vesicular and colloidal cysticeci and can reduce the occurrence of seizure associated with it.

Despite above mentioned advantages, cysticidal drugs should not be used in to treatment of some form of NCC as they may aggravate the condition by increasing intracranial pressure (García *et al*., 2002; Del Brutto and Oscar, H. 2012). Cysticidal therapy in patients with parenchymal brain cyst and hydrocephalus is recommended only after placement of ventricular shunt. Precaution should be taken while treating patient with giant subarachnoid cyst with cysticidal drug as they induce inflammatory reaction due to the destruction of parasite which may block leptomeningeal vessels adjacent to the cyst. Albendazole though causes destruction of ventricular cyst but may produce hydrocephalus due to inflammatory responses, if cysticeri are present in the 4th ventricle or close to foramina of Monro. Cysticidal drugs should not be given to patients with calcified cyst as they already contain the dead parasite (García *et al*., 2002; Del Brutto and Oscar, H. 2012).

Seizure in patients with NCC can be controlled with antiepileptic drugs (Del Brutto *et al.,* 1992; Garcia *et al*., 2004; Del Brutto and Oscar 2012). Even after receiving proper treatment, in 50% of the patient it was seen that seizure had relapsed after withdrawal of antiepileptic drugs (Del Brutto *et al*., 1994). Factors related with recurrence of seizure are calcification of cyst and existence of multiple brain cysts. In few patients with colloidal cyst, calcification of cyst may occur after therapy, which may be associated with seizure reoccurrence (Singh *et al*., 2010; Del Brutto and Oscar 2012). Neuro-endoscopy can be used to remove cysts which can be easily reached. Ventricular cysts which do not adhere to ependyma can be removed through neuro-endoscopy. Surgery is necessary for intra-ventricular cysts, hydrocephalus, large cisternal cysts, large parenchymal cysts. Intra-ventricular cyst can be removed by endoscopic surgery. Hydrocephalus can be cured by using ventriculo-

peritoneal shunt but the limitation it that it may lead to obstruction in few patients and require multiple revisions (Rajshekhar, 2010).

Control

For effective conrtol of cysticercosis, eradication programme should consider all the factors responsible for occurrence of the disease such as human carrier, infected pigs, environmental contamination with *T. solium* eggs etc (Keilbach *et al*., 1989; Pawlowski, 2006). All efforts should be directed to interrupt the life cycle of the parasite. Vaccinating pigs against *T. solium* metacestode infection is an effective way of controlling *T. solium* infection in humans (Lightowlers, 2010).Vaccine potential of many recombinant vaccines has been investigated for utilisation in pigs including cloned antigen of *T. Solium* oncosphere and S3P vaccine (Sciutto *et al*., 2008). Maximum protection was recorded in oncospheral antigen. Nearly complete protective immune responses has been recorded in pigs when vaccinated by using three kinds of cloned *T. solium* oncospheral antigen namely TSOL18 (Gauci *et al.,* 1998). TSOL45-1A(Gauci and Lightowlers, 2001) and TSOL16 (Gauci and Lightowlers, 2003) under experimental conditions .The highest protection was induced by TSOL18 antigen.TSOL18 induce protective immune response both by antibody and cell mediated destruction of the parasite (Kyngdon *et al*., 2006). Owing to the small size of TSOL18 antigen (112 amino acid), it is feasible to produce whole antigen at a purity and cost suitable to use it as a vaccine directly. Cysvax, a recombinant vaccine developed by Indian Immunological Limited, Hyderabad has been recently licensed in India for the prevention of porcine cysticercosis (WHO, 2017). Transmission of the disease can also be blocked by deworming the pigs with benzimidazole, oxfenazole at the dose rate of 30mg/kg (Gonzalez *et al*., 1996; Gonzalez *et al*., 1997; Gonzalez *et al*., 1998). In addition to it, more epidemiological survey should be carried out to ascertain the global burden of the disease and to influence the community at all levels to participate in parasite control interventions (Anonymous, 2003; Ngowi *et al*., 2004).

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