**Taenia solium** cysticercosis in India: A vетero-medical update

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**ABSTRACT**

Cysticercosis is an infection caused in pigs and man by the metacestode of *Taenia solium*, the pork tapeworm of man. It is a major food-borne zoonosis, which is an economic constraint to the pig industry as well as a serious medical problem. Neurocysticercosis (NCC) is regarded as the most frequent parasitosis of the central nervous system (CNS) and the most common cause of epilepsy in India. The commonest lesions are presented as granuloma. Unhealthy pig-rearing practices, lack of sanitation, inadequate meat inspection, rural-to-urban migration and other factors have contributed to the endemic status of the disease in India. At the same time, increasing number of detections consequent to improved serological and imaging techniques have aided definitive diagnosis, increasingly incriminating NCC as the cause of considerable morbidity and mortality with resultant socio-economic burden. While there are sporadic reports on pigs, largely based on prevalence at postmortem or serological, considerable epidemiological, diagnostic and clinical data has come up on human side in recent years. In the spectra of clinical manifestations, ocular disease and disseminated cysticercosis are being reported with increasing frequency. Detection and anthelmintic treatment of human taeniasis is the main component of a control strategy. Porcine cysticercosis should also receive due attention, and development of an onchospher antigen-based vaccine is a promising line. The problem calls for improved collaboration between medical and veterinary services, community involvement and greater awareness of public health workers.

**Key words**: Cysticercosis, Taeniasis, *Taenia solium*, Pigs, Zoonosis

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associated with pig slaughter in Bengaluru (Selvam et al. 2005) and 18.6% in a rural pig-farming community in Uttar Pradesh (Prasad et al. 2007). Reports from rural Goa (Vora et al. 2008) found 9.7% prevalence and from Asom (Borkataki et al. 2009) 8.93 to 16.00% in 3 districts of the state. Prevalence of porcine cysticercosis in India varied between 4.24 and 20.8% in studies from different parts of the country (Kumar and Gaur 1994). Infection appeared more common in areas where backyard pig-keeping was practiced. The data also varied according to the diagnostic test employed. In general, postmortem or meat inspection found much lesser positivity (D’Souza and Hafeez 1998, 1999b; Sarma et al. 2000, Avapal et al. 2003, Sharma et al. 2004, Gaurat and Gatne 2005) than detection of C. cellulosae antibodies in pig serum (Sreenivasamurthy et al. 1999, Selvam et al. 2004, Sharma et al. 2005, Dhanalakshmi et al. 2006). A focus of high porcine (and human) infection in rural north India (Mohanlalganj, Lucknow, Uttar Pradesh) was reported (Prasad et al. 2002) wherein 26% of slaughtered pigs had cysticercosis and 40% of them had cysts in the brain. The distribution of cysticercosis in different muscles viz. neck, shoulder, masseter, thigh, heart, tongue and diaphragm (Kumar and Gaur 1994, Avapal et al. 2003, Dhanalakshmi et al. 2005) has been reported from time to time. Other tissues / organs involved were brain and eye. In heavily infected pigs, cysticerci are also found in the liver, lungs and intercostal muscles (Kumar et al. 1991). An unusual case of asymptomatic mediastinal cysticercosis due to Taenia solium was also reported in a dog (Banga et al. 2005).

In humans, in any particular geographic area, economic, socio-cultural and religious factors strongly affect the prevalence of cysticercosis (Singhal and Ladiwala 1995). According to WHO (2003) report, the whole of India is marked among areas of high prevalence or endemicity for cysticercosis. However, there do not seem to be any reports from largely-vegetarian and pig-free states like Rajasthan, Gujarat and Madhya Pradesh (Prasad et al. 2008). Also the predominantly Muslim Kashmir valley is pig-free but endemic for T. saginata, the beef tapeworm (Lateef et al. 2008). According to Kuruvilla et al. (2001), NCC is rather uncommon in Kerala, possibly due to better socio-economic status, high literacy rate and improved sanitation in that state. However, the premise of Singh et al. (2002) that human cysticercosis is more prevalent in the northern states than the south is debatable in the light of reported seroprevalence of 15.9% (17.7% in the rural population) in Vellore (Prabhakaran et al. 2008), 22.4% in rural Goa (Vora et al. 2008) and an endemic situation among epileptic patients in Puducherry and neighbouring districts of Tamil Nadu (Parija and Raman 2011). On a national basis, antibodies range in 17.4 to 29.2% of sera in patients of epilepsy and intracraninal space-occupying lesions (Khurana et al. 2006). A genetic predisposition to cysticercosis was suggested (Jain et al. 1999) to explain high seropositivity and clustering in families of children with single small enhancing lesions (SELs). Singh et al. (2000) found that 2.7% of household contacts of children with solitary cysticercus granulomas (SCGs) were positive for cysterceral antibodies by enzyme immunotransferase blot (EITB) test. An increasing trend in clinically suspected neurocysticercosis in children was noted (Singh et al. 2000, Grover et al. 2002, Kumar et al. 2006). Interestingly, there has been a solitary instance of neurocysticercosis in a chimpanzee at Lucknow Zoo (Shukla 2003). After the advent of newer imaging techniques, NCC is the most frequently observed parasitosis of the CNS (Malla 2000, Narula and Bawa 2003, Parija et al. 2005). It has been found to be the cause of epilepsy in up to 50% of Indian patients presented with partial seizures (Gulati et al. 1994, Sawhney et al. 1996, Rajeshkhat et al. 2003, Kumar et al. 2006, Prasad et al. 2008). Parija and Gireesh (2009) found 5% seropositivity in patients with AIDS but opined that they are nevertheless a high risk group.

**Clinicopathology**

T. solium cysticercosis in pigs is usually a symptomless infection except when the cysticerci get lodged in the eye and brain (Kumar and Gaur 1994). The parasite may lodge in any body tissue but shows a predilection for the two named above. According to Chawla et al. (2004a) the course of infection and histopathology is similar to that of humans. As such, neuro-cysticercosis (NCC) in pigs is regarded as a valid entity with wide variation (10% to 60%) in prevalence (Singh and Parihar 1999, Prasad et al. 2006). Prasad et al. (2006) reported clinical signs like excessive salivation, excessive blinking and lachrymation, and subconjunctival nodule in swine with cysticercosis. Singh and Parihar (1999) described the histopathology of NCC in Indian pigs. Most cysts were found loosely attached to the parenchyma, which detached easily on manipulation. Brain showed slight peripheral congestion. Viability of porcine NCC can be predicted with proton magnetic resonance spectroscopy wherein creatine appears to be a marker (Chawla et al. 2004b). Further, metabolite pattern in the cysticerci is not influenced by surrounding tissue location (Chawla et al. 2004c). Gross and histopathological examination of brains of free-roaming pigs in Bareilly (Uttar Pradesh) revealed NCC in 3% (Prakash et al. 2007).

In humans, clinical spectra of the disease depend upon localization of the cyst and lesions produced. NCC due to the lodging of the cyst in CNS is the most common form with inflammatory changes in the parenchyma around the cyst. Acute symptomatic seizures are the most common manifestation of human NCC, the other symptoms observed being headache, meningitis, focal neurological deficits and psychological disorders (Chandramukhi and Nayak 1990, Mohanty et al. 1997, Sawhney et al. 1998, David and Mathai, 2000, Varma and Gaur 2002). Almost a third of NCC cases are associated with headache and vomiting (Barnwal et al. 2006).
1998, Singhi et al. 2000, Talukdar et al. 2002). Tuberculosis of CNS mimics NCC (Garg et al. 2000a). In extraparenchymal NCC, the most common clinical sign is hydrocephalus (Dass et al. 2010). It is believed that the immunologic process elicited by the release of the dying parasite’s antigen is responsible for clinical manifestations of NCC. When the cysticercus dies, the bladder wall collapses to form a small cyst granuloma (SCG) or most commonly a single enhancing CT lesion (Rajeshkhar 1991, Chandy et al. 1991, Singhal et al. 1997, Garg and Nag 1998a, Malla 2000) or a calcified dot which represents the scolex (Rajeshkhar and Chandy 1996). Clinical manifestations of NCC are pleomorphic (Rahalkar et al. 2000, Varma and Gaur 2002, Patel et al. 2006) and cover a wide spectrum, but radiological abnormality of single small enhancing lesions, are the commonest in Indian NCC patients (Garg and Nag 1998a, Garg et al. 2000b, Mohanty et al. 2008). Reports were received on psychiatric illness (Agarwal et al. 2001), intramedullary spinal (Ahmed and Sharma 2007), extramedullary spinal (Kasliwal et al. 2008), giant intraparenchymal NCC (Umredkar et al. 2009) and hydrocephalus with bilateral optic atrophy (Dass et al. 2010), Garg and Kar (2002) reported HIV and NCC co-infection with unusual racemose/giant cysts. Next to CNS, ocular cysticercosis is commonly reported and is predominantly intra-ocular (Atul et al. 1995, Kaliaperumal et al. 2005). Other reports have dealt with extracranial acquired brown syndrome (Pandey et al. 2001), unifocal subconjunctival cyst (Kumar et al. 2007), decrease in extraocular CS (Madigubba et al. 2007), conjunctival / subconjunctival (Mehdi et al. 2007), and fibrinous anterior uveitis with secondary glaucoma (Mahendradas et al. 2007). Visual loss is a serious consequence, which can result either from direct ocular involvement or secondary to CNS involvement. Various types of disseminated cysticercosis involving an infant (Dhingra and Mishra 2008), heart (Bhalla et al. 2008a), brain, subcutaneous tissues, skeletal muscle, right orbit and thyroid gland (Bhalla et al. 2008b), right orbit, thyroid gland with pulmonary and cardiac involvement (Jain et al. 2010) have been recorded. Uncommon presentations included skeletal muscles (Shankar et al. 1994), superficial muscles and subcutaneous tissue (Couto et al. 1995), eosinophilia with pleural effusion (Salari et al. 2001), maxillo-facial (Sidhu et al. 2002), nodular subcutaneous with a single nodule on tongue (Bassi et al. 2007). Mahajan et al. (2007) reported a case with co-existence of salivary gland cysticercosis with squamous cell carcinoma. An interesting rare observation related to carpal tunnel syndrome (Sharma et al. 2010) caused by compression of the median nerve within the carpal tunnel. Suggestive MRI was supplemented with histological diagnosis consistent with cysticercosis.

**Diagnosis**

The absence of specific clinical symptoms in cysticercosis forced investigators to search for other approaches to diagnosis (Kumar and Gaur 1994). Since the quality of antigen largely determines the specificity and sensitivity of an immunodiagnostic test, efforts were initially directed at fractionation and characterization of antigens. Progress in this direction has resulted in trials of various sero-diagnostic tests by various workers for porcine cysticercosis. Shinde et al. (1991) found indirect haemagglutination (IHA) test with scolex antigen (SA) as 84% effective in detecting cysticercosis in naturally infected pigs, while counter-current immunoelectrophoresis (CIEP) was regarded as the test of choice by some others (Deka and Gaur 1993, D’Souza and Hafeez 1999). According to D’Souza and Hafeez (1999a) serodiagnosis is much more reliable as a certain percentage of infected pigs is usually missed in routine meat inspection. Further, enzyme-linked immunosorbent assay (ELISA) with antigen-B (a purified antigen) detected a higher percentage of infected pigs than CIEP. On the other hand, ELISA using whole cyst antigen (protein A) gave reduced specificity and sensitivity (D’Souza and Hafeez 1999b). Sreenivasamurthy et al. (1999) found that EITB was 97.5% sensitive and 100% specific in comparison to 90 and 50%, respectively, for ELISA. In this study, 18 polypeptides were obtained by SDS-PAGE and among them 68, 43 and 29 kDa components were specifically recognized by confirmed cysticercosis. D’Souza and Hafeez (1999c) compared the serum antibody detection by ELISA using 3 different antigens, viz. whole cyst (WC), scolex (SA) and excretory-secretory (ES) and found ES antigen to be the best. Prasanna et al. (2001) reported that latex agglutination test (LAT) was as efficient as ELISA when antigen B was used. Serodiagnosis of cysticercosis in naturally infected pigs by IHA (Selvam et al. 2004) was also reported with a sensitivity of 85.71 and 80.95%, respectively, with WC and antigen B (partially purified). The test also detected 19.75 and 15.43% of the pigs, which were not detected during meat inspection, respectively, with WC and B antigens. Protein A ELISA was reported (Dhanalakshmi et al. 2006) to give sensitivity and specificity of 76 and 73% with WC and 80 and 83% with SA, respectively.

In humans, serological diagnostic tests employed were essentially identical to those used in pigs. As such, various investigators used a battery of highly sensitive techniques, with cysticercol antigens, initially crude ones with low specificity, and later on fractionated ones for detection of antibodies (or antiserum to detect antigens) in serum and other body fluids such as cerebro-spinal fluid (Katti and Chandramukhi 1991, Mahajan et al. 1995). Most commonly used ones are immuno-assays, viz. ELISA (Malla et al. 1992, Kaur et al. 1996, Rajeshkhar and Oommen 1997, Bedi et al. 2001, Mandal et al. 2006, Kumar et al. 2006, Prabhakaran et al. 2007, Sahu et al. 2009) and EITB (Rajeshkhar et al. 1991, Rajeshkhar and Oommen 1997, Singh et al. 1999, Prabhakaran et al. 2004, Mandal et al. 2006, Atluri et al. 2009, Parija and Gireesh 2009). Other variants of immuno-
assays employed included dot-ELISA (Biswas et al. 2004), dot-blot (Mandal et al. 2006, 2008) and MTT lymphocyte proliferation assay (Verma et al. 2010). Varying degrees of sensitivity and specificity were recorded by the various investigators listed above. The antigens used were also variable, such as lentil-lectin affinity purified glycoproteins (Prabhakaran et al. 2004, 2007), crude soluble extract (CSE) antigen (Mandal et al. 2006), scolex and membrane antigens (Shukla et al. 2008), lower molecular mass antigens (Mandal et al. 2008, Atluri et al. 2008), CSE and excretory secretory (ES) antigens (Atluri et al. 2008, 2009). The latter study suggested highest sensitivity with ES antigens. Shukla et al. (2008) showed that antigens prepared from C. fasicolaris could be used for immunodiagnostic procedure of NCC with 94% sensitivity. Lymphocyte transformation test was proposed (Prasad et al. 2008) as another alternative with higher sensitivity and specificity. Das et al. (2002) had suggested that antigen B detection in CSF samples may be a useful adjunct to clinical suspicion and radiological observations. Specific antibody detection can also be done in serum, urine and saliva samples (Parija et al. 2004, Sunita et al. 2007). Needle aspiration cytology was considered a satisfactory alternative to open biopsy (Arora et al. 1994, Khurana and Jain 1999) while others (Bhalla et al. 2008a, 2008b) used incision biopsy to find cysts in subcutaneous swellings and in muscles. Neuro-imaging modalities such as CT scan and high resolution MRI have greatly improved the accuracy in the diagnosis of NCC. Up to 50% of all patients presented with seizures were diagnosed with SCG (Misra et al. 1994). Sonographies have since been used increasingly in the diagnosis of cysticercosis (Garg and Kar 2002, Sharda et al. 2002, Asrani and Morani 2004, Dass et al. 2010, Singal et al. 2010).

Immunity

Immunized pigs have comparatively less severe pathological changes than the non-immunized pigs (Kumar and Gaur 1994). Specific IgG anti-cysticercus antibodies were produced in experimentally infected pigs (Kaur et al. 1995b). Their initially low levels, increased as infection progressed, predominantly IgG1 isotype. Sreenivasamurthy et al. (1999) reported diagnostically important antibody response towards polypeptide masses of 68, 43 and 29 kDa for the diagnosis of procine cysticercosis. In studies on piglets infected with T.solium eggs, kinetics of cellular responses indicated significant increase in CD4 + T cells at 60 days post infection (Grewal et al. 2000a). On the other hand, intense humoral response was triggered early viz. 10–30 days post-infection and persisted up to 90 days.

Identification of antigenic fractions of C.cellulosae by western blotting indicated that lower molecular mass (LMM) of 20 kDa was most immunoreactive for ELISA and EITB assays (Kaur et al. 1995a). Shankar et al. (1995) identified the glycocalyx as the most significant site of C.cellulosae relevant to human NCC. That humoral response to NCC is very heterogeneous was evidenced from the fact that patient antibodies may react to 1–8 antigens on immunoelectrophoresis and up to 30 antigens on EITB (Pardini and Vaz 2001). By modulating the host immunity, co-existing NCC might influence the poor outcome of certain human carriers and viral infections like JE according to a study from NIMHANS, Bengaluru (Desai et al. 1997). Grewal et al. (2000b) suggested that CMI responses especially T cells and cytokines play a role in human cysticercosis. The granulomatous inflammatory response developing around the cysticerci in both human and swine is an expression of immune response (Prasad et al. 2008). Immune response in symptomatic human neurocysticercosis responsible for severe neuropathology including the formation of granulomas is thought to be of the Th1 phenotype predominantly (white et al. 1997). Toll-like receptors (TLRs), particularly TLR 4 polymorphisms, are significantly associated with symptomatic neurocysticercosis (Varma et al. 2010).

Treatment and control

In experimental cysticercosis in pigs, treatment with albendazole at 15 mg kg⁻¹ body weight daily for 30 days from day 0 or 15 days post infection resulted in 100% cure rates (Kaur et al. 1995b). In human infections, benefit of anthelmintic therapy especially with albendazole was controversial. Padma et al. (1994) did not find any efficacy of the drug in multiple or single ring lesions in epilepsy. On the other hand, Raina et al. (1996) reported that administration of albendazole (25 mg/kg/day along with prednisolone 1 to 1.5 mg/kg/day for 4 weeks) resulted in spontaneous extrusion of cyst from the subconjunctival location. According to Garg (1997), both praziquantel and albendazole have their merits and demerits, and the efficacy of chemotherapy in altering the natural course of the disease is subject to doubts due to the tendency of cysticercus lesions to resolve. Garg and Nag (1998b) described the efficacy of anthelmintic (albendazole) therapy in spinal cysticercosis in several cases with resolution of neurological deficits. The occurrence of side effects of albendazole therapy with headache, nausea, vomiting and recurrence of seizures (Rajeshkhar 1998) indicated the cysticercal nature of the lesion. Praziquantel appears to kill the scolex and protoscolex. The usual dose is 50 mg/kg/day divided into 3 doses for 15 days (Garg 2001). Singh and Sander (2004) questioned the usefulness and safety of anticysticercal treatment in seizures. As such, recommended treatment modalities should include a combination of (i) anticysticercal drug, (ii) corticosteroids to suppress the host’s immune response, (iii) anti-seizure medication, and (iv) surgical intervention where required, by excision (Garg 2001, Kasliwal et al. 2008) or vaso-expression from eye (Kai et al. 2008).

Identification and elimination of human T.solium tapeworm carriers is the only direct way of destroying the
source of infection for both humans and pigs and for this 2 effective anthelmintics are niclosamide and praziquantel (Pawlowski 2008). Anthelmintic resistance of T.saginata in Kashmir was reported against niclosamide (Koul et al. 1999) and both niclosamide and praziquantel (Lateef et al. 2008). Lateef et al. (2008) found nitazoxanide therapy as effective against T.saginata. However, there does not seem to be any such report for T.solium in India.

As per prevailing indications, T solium cysticercosis cases are likely to increase in Asia in proportion to a growing consumption of meat (Rajshekhar et al. 2003). As such, epidemiological data to estimate the magnitude of the problem has to be gathered (Rajshekhar 2004). Girotra et al. (2011) reported that awareness of neurocysticercosis was poor among patients and the public, and educational programmes are needed to fill this lacuna in endemic areas. The urgency for control of taeniasis / cysticercosis was brought to the attention of the global representatives (WHO, 2003) for medical and economic reasons. Suggested measures in endemic areas include improved collaboration between medical and veterinary services, taenicial treatment of high risk group of people (like meat industry workers and pig keepers), extension of diagnostic facilities, improved pig husbandry and proper meat inspection. Another promising approach viz. using onchosphere antigen ( TSOL18) (Lightowlers 1999) for development of vaccine against porcine cysticercosis should be prioritized. Application of this vaccine along with a single treatment with oxfendazole (Lightowlers 2010) was found highly effective in a field trial on pigs naturally infected with cysticercosis.

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