



## ***Taenia solium* cysticercosis in India: A vetero-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 onchosphere 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

*Taenia solium* cysticercosis in pigs and in man is a zoonotic disease of global significance (Flisser *et al.* 2006). In the pig industry, it is responsible for heavy economic losses in terms of rejection and condemnation of infected meat. In humans, cysticercosis, particularly its neurological form is a frequent cause of serious complications, like epileptic seizures, encephalitis and other manifestations. Not less than 20 million people worldwide are infected with *T. solium* cysticerci and conservative estimates place annual deaths from NCC at 50 000 (Pawlowski 2008). Man is the definitive host and harbours the adult tapeworm as essentially symptomless condition. The pig acquires the infection through ingestion of tapeworm eggs / proglottids passed in human stools and the metacestode stage *Cysticercus cellulosae* develops mainly in the muscles of pig. Eating raw or undercooked pork by man completes the cycle of this parasite which is unique in the sense that man can be definitive as well as intermediate host. Neurocysticercosis is acquired by eating food accidentally contaminated with

*Taenia solium* eggs, either through already contaminated food or through transmission from tapeworm carriers in the household (Garcia *et al.* 2003). Studies using mitochondrial and DNA sequences have proved genetic similarity between *T. solium* cysticerci isolated from human brain and from pigs (Juarez *et al.* 2008). Lack of sanitary services, free access of pigs to scavenging, and inadequate meat inspection are the main factors favouring perpetuation of infection (Kumar and Gaur 1994, Flisser *et al.* 2006, Singhi 2011) Owing to the immense public health importance of the disease, much work has been carried out over the past two decades, on various aspects of human cysticercosis which has helped improve early and specific diagnosis, clinico-pathology and immunity. By contrast, porcine cysticercosis has not received due attention. This review attempts to compile information from an Indian perspective from medical as well as veterinary sources.

### *Epidemiology*

The adult of *T. solium* is cosmopolitan, but is more common in Mexico and other latin American countries, Africa and Asia. In India, the prevalence of *T. solium* taeniasis based on stool examination for ova varied between 3.9% in Sikkim (Mitra and Patwari 1998) and a high 16% in persons

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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 intracranial 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 cysticercal antibodies by enzyme immunoassay (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, Rajshekhar *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.*

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 (Rajshekhar 1991, Chandy *et al.* 1991, Singhal *et al.* 1997, Garg and Nag 1998a, Malla 2000) or a calcified dot which represents the scolex (Rajshekhar 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 extraocular 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 cysticercal 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, Rajshekhar 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 (Rajshekhar *et al.* 1991, Rajshekhar 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 lentin-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.fasciolaris* 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 IgG<sub>1</sub> isotype. Sreenivasamurthy *et al.* (1999) reported diagnostically important antibody response towards polypeptide masses of 68, 43 and 29 kDa for the diagnosis of porcine 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<sup>-1</sup> 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 (Rajshekhkar 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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