CURRENT CONCEPTS IN NEUROCYSTICERCOSIS

Lt Col PC SANCHETEE

MJAFI 1998; 54 : 1-2

KEYWORDS : Cysticercosis, *Taenia solium*, Magnetic resonance imaging

Cysticercosis (NCC) is one of the common parasitic infection of central nervous system (CNS) in many parts of the world including India. The common locations are (i) basilar or meningeal cysts, (ii) solitary or multiple parenchymal cysts, (iii) ventricular localisation, and (iv) spinal cord cyst. Mixtures of these basic patterns may occur, and asymptomatic infections are common. Adult parasite is host specific and man is the only known definitive host for *Taenia solium* [1]. Human infestation occurs following ingestion of measly pork containing cysticercus larvae. Adult worm lives in the intestine for several years and is largely asymptomatic. Pigs, dogs, monkeys, camels, deer, sheep, cats and human are some of the known intermediate host for the embryo of the parasite. Cyst remain viable in the intermediate host for many years (approximately 5-10 years in human) [2]. Further development is possible only if these tissues are ingested by definitive host. Human, if acts as an intermediate host, usually signifies dead end in the life cycle of the parasite as cannibalism is rarely practised now.

Pork eating is relevant to development of adult worm in the intestine and is not responsible for development of cysticercosis in an individual. However, taeniasis is essential for propagation of the life cycle of the parasite and incidence of cysticercosis is high in the area where pork eating and taeniasis are highly prevalent. Cysticercosis is related to ingestion of ova passed in the faeces of an individual who is infested with *T. solium*. Human is infected with *T. solium* by one of the two ways,

(a) Anal-oral contamination which is related to i) external autoinfection due to poor personal hygiene, ii) contamination of food, water and beverages due to promiscuous defecation, unsafe water supply, improper washing of vegetables (particularly salads), iii) use of *T. solium* proglottids by witch doctors, and iv) aberrant sexual practices

(b) Retrograde movement of ova in the intestine (internal autoinfection) due either to spontaneous reversed peristalsis or to use of taenicial (but not ovicidal) drugs e.g. niclosamide.

Development of cysticercus cellulose is not yet well established. Whether it passes through all the stages or skip on some of them and also duration of each stage is not certain and uniform. It is possible that they live between 2-5 yrs. It has been observed that brain cysts as seen in Indian patients are often small, multiple and with a distinct scolex. In contrast, cysts in patients from endemic areas (like Mexico and Brazil) are usually large, fewer in number, has indistinct or no scolex and there is poor inflammatory response in surrounding tissues. Whether it is related to the immunological tolerance of the patients in endemic areas or to the genetic variation in cysts following continuous immunological assault, is not certain. Recemose cysticercus is an aberrant form of the parasite which is seen at the basal cisterns of brain. These are thin walled irregular grape like clusters of amorphous cyst with no living parasite or scolex in it.

The host-parasite relationship in NCC is a complex immunological affair [3,4]. Participation of immune responses appears to be decisive in clinical outcome, diagnosis, treatment and prognosis of the disease. Antigens are mainly localised at the external surface of the tegument (glycocalyx) as suggested by high amino acid and glucose uptake at this site [4]. Vaccines, as well as diagnostic antigens, have been developed from live or irradiated embryos, viable or irradiated eggs, secretion or excretion of cysticerci, whole cysticerci, cysticerci fluid and cysticerci wall. Vaccines developed from adult worm have not been found to be of much value. Incidence of porcine cysticercosis have shown reduction from 5.4 to zero percent one year after intramuscular immunisation [5]. It is possible to induce immunity in human also but results till now are not very encouraging. Young and viable cyst incite

little or no inflammatory reactions. Dying or degenerating cysts results in chronic granulomatous inflammatory infiltration in the cyst and surrounding tissues with neutrophils, eosinophils, lymphocytes, plasma cells and at times giant cells. There may be liquefaction in the centre of cyst with pus formation (granulomatous abscess). In addition there is a variable degree of oedema and vasculitis in the surrounding tissues. Fibrous capsule may form around the cyst with minimal gliosis. Finally there is complete resolution, fibrosis, gliosis or calcification.

Before the advent of CT scan, radiology was of little value in parenchymatous cysticercosis. The live cysts appear as low density circumscribed lesions with or without high attenuation eccentric scolex. Granulomatous stage of the cyst is characterised by contrast enhancing disc or ring lesions. CT scan is of value in confirming intracranial calcifications.

MRI allows demonstration of live cysticerci and better visualisation of early changes in the parasites as a whole and scolex in particular [6]. The adult, viable metacestode larva is noncystic and may not be visible on MRI. With the formation of bladder proper, the typical changes of cyst (i.e. iso- or hypointense on T1W1 and hyperintense on T2W1) appear. Scolex, which is situated eccentrically at one end of the cyst, appears as hyperintense on T1W1 and hypointense on T2W1. With these findings one can make a definite diagnosis of NCC even if the lesion is single. Over a period of several weeks the larvae age and degenerate with some degree of inflammation or even formation of granulomas [7]. These lesions appear as hyperintense core, hypointense rim and surrounding hyperintense oedema on T2W1. However, rim and core appear as iso- or hypointense on T1W1. The specificity of these findings for diagnosis of NCC is not high and similar findings may be seen in tuberculosis, pyogenic infection and fungal infection. Multiplicity of lesions and presence of extra-CNS lesions at this stage are helpful in marking a diagnosis of NCC. Calcified lesions were undoubtedly visualised better with CT scan as compared to MRI.

Therapy for neurocysticercosis must be individualised according to the activity of the disease and location of cysticerci. NCC due to parenchymal cysts carries a good prognosis regardless of therapy. Spontaneous resolution of cysts have been noted in few of the cases. Cysticidal drugs (praziquantel, albendazole)

which can penetrate blood brain barrier have greatly improved the prognosis [8-10]. Varied degrees of responses that were observed could be due to differential sensitivity of cysts and maturation stage at the time of treatment. For ventricular disease, most cases can be managed with shunting procedures either alone or together with the administration of antiparasitic agents without extirpation of the cysts. Subarachnoid disease in addition may require surgical extirpation of the cysts. For taeniasis massive single-dose treatment with praziquantel is recommended. Persistent efforts to prevent contamination of pork have eradicated the infection in industrialised countries and diminished seroprevalence. Cysticercosis is a major public health problem in developing countries and application of adequate control measures are needed to contain its menace.

REFERENCES

1. Lee DL. Classification and anatomy of parasite. In : Braude AI, editor. Medical microbiology and infectious diseases. London : WB Saunders Company 1981; 162-83.
2. Mc Greevy PB, Nelson GS. Larval cestodes infections In : Strickland GT, editor. Hunter's Tropical Medicine. Philadelphia : WB Saunders Company 1991; 843-59.
3. Flisser TIE, Perez-Montfort R, Larrable C. The immunology of human and animal cysticercosis : the reviews. *Bull WHO* 1979; 57 : 839-56.
4. Shankar SK, Suryanarayana V, Vasantha S, Ravi V, Ravikumar BV. Biology of neurocysticercosis parasite related factors modulating host response. *Medical Journal Armed Forces India* 1994; 50 : 79-88.
5. Molinari JL, Soto R, Tato P, *et al.* Immunization against porcine cysticercosis in an endemic area in Mexico : a field and laboratory study. *Am J Trop Med Hyg* 1993; 49 : 502-12.
6. Jena A, Sanchettee PC, Gupta RK, Khushu S, Chandra R, Lakshmi N. Cysticercosis of brain shown by magnetic resonance imaging. *Clin Radiol* 1988; 39 : 542-6.
7. Jena A, Sanchettee PC, Tripathi R, Jain RK, Gupta AK, Sapra ML. MR observations on the effects of praziquantel in neurocysticercosis. *Magnetic Resonance Imaging* 1992; 10 : 77-80.
8. Cruz M, Cruz I, Horton J. Albendazole versus praziquantel in the treatment of cerebral cysticercosis: clinical evaluation. *Trans R Soc Trop Med Hyg* 1991; 85 : 244-47.
9. Sanchettee PC, Venkataraman S, Dhamija RM, Roy AK. Albendazole therapy for neurocysticercosis. *J Assoc Phys India* 1994; 42 : 116-7.
10. Sanchettee PC, Dhamija RM, Roy AK, Venkataraman S, Krishnan NR. Praziquantel therapy in neurocysticercosis. *Neurology India* 1990; 38 : 139-46.