SECTION V

Neurology
Neurocysticercosis (NCC) is the commonest helminthic infestation of central nervous system. This disease is caused by larval form of Taenia solium. Although the disease has a worldwide distribution, there is little infestation in developed countries, whereas endemic Taenia solium taeniasis persists in most developing countries and a number of large series of cases have been reported from Mexico, South America, Spain and India. Cerebral cysticercosis was first reported from India by Surgeon Armstrong in 1888, during post mortem examination of a lunatic from Madras Asylum. NCC accounted for 0.05% of all neurological admissions over 20 years in Madras (1970) and consisted of 4% of all paediatric admissions at AIIMS, New Delhi. A few series of cases and sporadic cases have been reported from various parts of India. NCC is the main cause of late onset of epilepsy (20 to 50% in different series) specially in developing countries Latin America, Asia & Africa.

Aetiology
Cysticercosis is infestation with larval stage (cysticercosis) of Taenia solium, the pork tapeworm. In the life cycle of this cestode, human usually is the definitive host and harbours the adult tapeworm whereas pig is the intermediate host harbouring larval form or cysts.

The adult T. solium inhabits the human small intestine. Gravid proglottides containing fertile eggs detach from distal end of the worm and are excreted with human faeces. The pig ingests stool contaminated with eggs and when invasive oncospheres in eggs are liberated by action of gastric acid they penetrate bowel wall, enter blood stream and carried to skeletal muscle and other tissues where they develop into larval cysts. Ingestion of undercooked pork containing those cysts results in evagination of larval in small intestine of human being attachment of scolices to intestinal mucosa and development into adult worm.

By accidental ingestion of taenia eggs (through consumption of vegetables contaminated with human excreta containing egg of T. Solium), man may also act as intermediate host leading to development of extra intestinal encysted, larval forms of T. Solium which is known as cysticercosis. This may be noted in this juncture that internal autoinfection by regurgitation of proglottides into stomach has also been suggested as a possible mode of development of human cysticercosis.
Pathogenesis and Pathology

Cysticercosis are liquid filled vesicles containing a vascular wall and scolex. The speciality of cysticercosis lies in the fact that although encysted larval can develop in any extra intestinal tissue, central nervous system (CNS) is involved in 60-96% of patients giving rise to NCC. This occurs possibly because the immune response in CNS is limited.

Cysticercosis in brain are usually small and tend to lodge in cerebral cortex. They may be located in subarachnoid space, in basal cisterns or sylvian fissure. Basal cysticercosis may undergo disproportionate growth resembling a bunch of grapes.

Viable vesicular cysticercosis elicit little in ammatory change because of active immune evasion mechanism. The initiation of symptoms is equated with immunological attack from the host resulting in a process of degeneration that end with death of parasite and calcification viable cysts may coexist with degenerating cysts of calcified element. Cerebral forms of NCC are most frequent and intramedullary forms are rare. Only 31 cases of intramedullary cysticercosis are recorded in literature. When it involves spinal cord, it grows more often in subarachnoid space. The neurological signs of intramedullary forms are due to in ammatory reaction, mass effect of cysts & cord degeneration due to vasculitis.

Clinical Feature

The clinical profile is variable and depends upon number of cysts, their precise location in brain in ammatory reaction evoked by them or obstruction of ventricular system. NCC may present as seizure disorder raised intracranial tension, chronic meningitis, dementia etc. However, convulsive seizure is the most common presentation. The incidence of seizure has been reported from 22-98% in different series. On the other hand, the reported incidence of NCC among epileptics varies in different series depending on age group of patients and the region of study.

Other common focal signs include pyramidal tract signs, sensory deficits, and involuntary movements. These manifestations follow a subacute or chronic course, Isolated cranial nerve palsy has also been reported.

Due to hydrocephalus caused by secondary arachnoidites or ventricular cysts, NCC may present with features of increased intracranial tension. An encephalitis picture may result from overwhelming in ammation around many cysts and in contrast. Some patients may tolerate hundreds of intraparenchymal cysts with only minor symptoms. Involvement of skeletal muscles result in pseudohypertrophic change of muscles.

Laboratory Diagnosis

The wide spectrum of clinical manifestation vis-à-vis paucity of specific, clinical signs has made it almost impossible to diagnose NCC on clinical grounds alone. In endemic areas, late onset seizure in otherwise healthy individuals, raises the possibility of NCC. However radioimaging and immunological tests are mandatory in context of the fact that histological confirmation by stereotactic biopsy is not universally available and not practicable.

A. Imaging

Computerized Tomography (CT) Scan and Magnetic Resonance Imaging (MRI) of brain have been utilized as very helpful diagnostic tool. Findings in CT or MRI depend on involution of cysts. Viable cysticerci appear as rounded cystic lesions on CT, hypointense on MRI without enhancement, whereas degenerating parasites are seen as focal enhancing lesions with surrounding edema and calcification as hypertense dot or nodule.

CT and MRI findings in subarachnoid involvement include hydrocephalus, abnormal meningeal
enhancement and subarachnoid cyst. However it is to be remembered that multiple ring like or nodular enhancing lesions pose a diagnostic challenge as because a number of other conditions (e.g. tuberculoma, neoplasm, etc.) can produce similar picture.15

B. Immunological Tests
Serodiagnosis was introduced by Weinberg in 1909. Precipitation gel diffusion test and Indirect Haemagglutination (IHA) were further developed.16,17 Other tests include complement fixation test (CFT), immuno-uoresent anti body test, immuno-electrophoresis, ELISA, Dot immunobinding assay, radio immune assay western blot & Enzyme linked Immuno Electro Transfer Blot (EITB).
EITB is the best available serological test for NCC. Tsang et al.18 reported 98% sensitivity and 100% specificity when serum was examined. But CSF antibody detection is far superior and direct evidence of CNS disease than demonstrating serum antibodies. Rosas et al,19 have demonstrated superiously IgM antibodies in CSF indicating recent infestation.
Lastly we should not forget that though majority of NCC cases produces antibodies in response to antigenic stimulation, there exists a section of proved NCC who are non immune responders.
A set of diagnostic criteria based on Neuro imaging studies, serological tests and clinical presentation has been prepared by Del Brutto et al.20

Treatment
Seizure secondary to parenchymal NCC can be controlled with anticonvulsants. However the optimum length of anticonvulsant therapy has not been determined.
Antiparasitic agents destroy viable cysts. Currently albendazole is the drug of choice although single day praziquantel has demonstrated similar activity. The effectiveness of both have been established in various studies.21, 22, 23 Temporary adverse reactions in the form of aggravation of headache and deterioration of sensorium, recurrent convulsion have been reported.
Some forms of NCC should not be treated with antiparasitic agents. In patients with cysticercosis encephalitis, these drugs may result in worsening cerebral oedema and fatal hemiation. The main stay of therapy in such cases is high doses of cortico steroids to reduce in ammatory response. In patients with both hydro ocephalus and parenchymatous cysts, antiparasitic drugs should be started after placement of ventricular shunt. Antiparasitic drugs need be used with caution in patients with giant subarachnoid cysticerci.
Although most cysts disappear after antiparasitic treatment, it’s uncertain whether this is associated with better control of seizures. On the other hand treatment with simple anti convulsant may be sufficient in patient with Parenchymal NCC and seizure24, 25

Conclusion
The importance of improvement of sanitary facilities and veterinary inspection can not be over emphasized in context of control of cysticercosis & NCC. But recent study from Mexico 26 suggest parasite – host immune relationship is more important.
New tools for control are Oxfendazole, an effective single dose therapy for porcine cysticercosis and the candidate porcine vaccine which is under trial.27

References