Original Research Article

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Surgical Intervention in Neurocysticercosis: An Institutional Experience and Review of Literature.

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Abstract

Objectives: Neurocysticercosis (NCC) is currently considered the most common parasitic disease of the CNS, affecting thousands of individuals living in developing countries and in industrialised nations with a high index of immigration of people from endemic areas. It is one of the most common causes of acquired epilepsy in the developing world especially in India. In this study we present our experience in the surgical management of NCC, complications arising following of the procedures and the lessons learnt at a tertiary care centre in Southern India. *Methods*: In this study,the records of 69 cases of NCC were prospectively and retrospectively analysed. Eleven patients underwent surgical intervention due to the complications arising out of the infection. Patients were followed up for a period of one year and the short term outcome of the interventions were evaluated. *Results*: Six (54.5%) patients presenting with hydrocephalus were treated with VP shunt, two (18.2%) patients presented as fourth ventricular SOL and underwent posterior fossa craniotomy and excision, while three (27.3%) had supratentorial lesions who underwent craniotomy and excision. One patient developed pseudomeningocele with CSF leak another developed wound infection which improved on conservative management. *Conclusion*: Cysticercosis is considered as a "biological marker" of social and economic development. It is a global health problem and developing countries, like India, bear most of the disease burden. In the present study the short term prognosis was good.HoweverNCC being a chronic disease these patients require prolonged follow up to understand the long term complications and their impact on the overall outcome and survival of the patient.

Keywords: Neurocysticercosis; Hydrocephalus; Fourth Ventricular SOL.

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Introduction

Cysticercosis is a zoonotic disease caused by infection with the larval stage of the parasite Taenia solium (T. solium), commonly known as pork tape worm. In humans, the parasite has a propensity to infest the central nervous system (CNS), where it produces a spectrum of clinical disorder called neurocysticercosis (NCC). Cysticercosis is currently considered the most common parasitic disease of the CNS, affecting thousands of individuals living in developing countries and in industrialised nations with a high index of immigration of people from endemic areas.[1] The disease is endemic in the subcontinents of Central and South America, Eastern Europe and Asia and poses as a major health problem in these regions. The International League Against Epilepsy (ILAE), designates NCC as the most common cause of acquired epilepsy in the developing world, where prevalence rates of active epilepsy are twice those of developed countries.[2] In this series we present our experience with the surgical management of NCC patients treated at the Institute of Neurosurgery, Madras Medical College, over a period of one year and a review of literature in the context of surgical management and outcome.

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Life cycle and Pathogenesis:

T. Solium has a two host life cycle (Fig.1), humans behave as definitive hosts by lodging the adult worms and pigs acts as intermediate hosts. However, either by regurgitation of eggs, ingestion

of food contaminated with cysticercal eggs or by orofecal contamination, human becomes accidental intermediate hosts and develop cysticercosis. The oncospheres reaches the CNS, via the bloodstream and gets lodged in the capillaries of the brain parenchyma. The embryo actively crosses the capillary wall and reaches the interstitial space where it evolves to the cystic form, transforming into C. cellulosae. Ventricular infestation is attributed to active passage of oncospheres through the capillaries of the choroid plexus, immediately followed by their transformation into cysticerci. The cysticercus is carried by the CSF from the ventricles to the subarachnoid space depending on its size. Controversy exists regarding the direct arrival of the oncospheres in the subarachnoid space or the ventricles through meningeal or subpial vessels.[3] C. cellulosae begins to grow in the CNS, usually reaching 4 to 20 mm in widest diameter in the brain parenchyma. In the ventricles or in the subarachnoid space the cysticerci usually reach a larger size and take the form of C. racemosus, characterized by an irregular membrane, absent scolex, and sometimes clustered in multiple, interconnected vesicles, resembling a raceme.

The cysticerci present a natural evolutionary process that culminates with their degeneration within a period of approximately 2 to 5 years.[3] This process starts after the transformation of the oncosphere into the cysticercus (vesicular stage), inflammatory reaction is less intense during this phase. This is followed by progressive degenerative changes of the cyst characterized by thickening of the membrane and replacement of the clear fluid with a whitish gel (colloidal stage). The cyst wall continues to become thicker and the gel undergoes calcium deposition (granular stage), and finally the cyst becomes completely calcified, being significantly reduced than the original size (calcified nodular stage). The inflammatory reaction surrounding parenchymal cysticerci is mainly composed of lymphocytes, plasma cells and eosinophils, and is usually associated with some degree of oedema and reactive gliosis, and varies according to the stage of development of the cysticerci, being more marked when the cyst starts to degenerate, and progressively declines until the cyst becomes calcified. The intensity of the inflammatory reaction depends on the parasite–host interaction in the nervous system.[3,4] The inflammatory reaction triggered by cysts in direct contact with CSF of the ventricles and subarachnoid space may extend to other sites and provoke extensive areas of inflammation leading to cysticercotic encephalitis, more frequently seen in younger age group and women.[5]

The disease is widely prevalent in India, but the exact disease burden is not known due to the lack of major epidemiological studies. There are wide differences in the frequency and reporting of cysticercosis in India which can be attributed to the diverse geography, ethnicity, sociocultural practise and religious beliefs.[6] Cysticercosis appears to be more prevalent in the northern States Bihar, Uttar Pradesh through Punjab.[6]

Material and Methods:

The records of sixty-nine patients with NCC treated at the Institute of Neurosurgery, Madras Medical College, between, January 2018 and December 2019 were retrospectively and /or prospectively analysed. Fifty-eight patients were managed conservatively and eleven patients underwent surgical intervention. The indications for surgery were - to control of raised ICP, hydrocephalus, to remove cysts causing local compression and refractory to medical management, and rarely, to obtain histopathological diagnosis. Patients were followed up for a period of 6 months to 1 year.

A diagnosis of NCC was based on the revised diagnostic criteria established by Del Brutto et al. seizures were classified according to the ILAE classification. All patients under went MRI and CT for neuroimaging and images were interpreted by neuroradiologists and neurosurgeons.

Medical management for patients showing active cysts consisted of albendazole 15 mg/kg/day for 2 weeks along with corticosteroids to reduce the inflammatory reaction. Repeat albendazole therapy or an additional course of praziquantel 100 mg/kg/day for 2 weeks may be required in non-responders. Seizures were managed with appropriate antiepileptics.

Statistical analysis: Descriptive data were evaluated using proportions or means (±standarddeviation). Statistical software SPSS14.0 was used for statistical analysis.

Results

Sixty-nine patients of NCC were identified during the study period. Of the 69 cases, 45 (65.2%) were male and 24 (34.7%) were female. Mean patient age was 36.3 ± 12.06 years (range:20–55 years). Mean age in males 45.5 ± 5.8 (range: 40–55 years) and in females 25.4 ± 6.7 (range: 20–35 years). Fifty seven patients (82.6%) had multiple lesions and 12 (17.3%) had solitary lesion. According to the recent

version of the diagnostic criteria developed by Del Brutto et al.[7] 59.4% (41/69),28.9% (20/69) and 11.5% (8/69) fell into the absolute, definitive and probable criteria respectively (Fig. 2).

Headache (68%) was the most common presentation in this series. Seizures (46.3%) were the second most common presentation in this series. Most patients had general tonic-clonic seizures (53.1%) followed by partial seizures with secondary generalisation (34.3%) and simple partial seizures (12.5%). Other frequent presentations were nausea and vomiting (28.9%), dizziness and vertigo (26%), papilledema (7.2%), gait abnormalities (5.7%), hemiparesis (2.8%), mental confusion (2.8%) and visual disturbances (2.8%) (Fig. 3).

In the present series, 56% had active parenchymal cysts, 38% had calcified cysts and 6% had intraventricular cysts based on the neuroimaging criteria of Zhao et al. The frequency of cysts at various stages as seen on MRI and CT Brainis shown in (Fig. 4).

Surgical Treatment:

In our series, 11 (15.9%) patients underwent surgical intervention during the study period. Nine of the operated patients had multiple lesions and two had single lesion in neuroimaging studies. Surgery was performed to control raised ICP in 9 patients, and local brain compression 2 patients. In the operated patients increased ICP was caused by hydrocephalus in 8 (72.7%), patients and intracranial space-occupying lesion (tumoral form) in 1(9%). Based on the pathophysiological mechanisms of intracranial hypertension detected on conventional CT scanning and/or MR imaging, different surgical approaches were indicated. Of the three patients harbouring tumoral lesions, two underwent right frontal craniotomy and excision and one patient underwent right parietal craniotomy and excision. Six (54.5%) patients presenting with hydrocephalus were treated with VP shunt, two (18.1%) patients presented as fourth ventricular SOL and underwent Posterior fossa craniotomy (PFC)and excision (Fig. 5).

Procedure related complications:

One who underwent PFC developed pseudomeningocele with CSF leak which was managed conservatively and patient improved. One patient undergoing right frontal craniotomy developed wound infection which improved on conservative management. One patient developed shunt dysfunction which was revised at a later date (Fig. 6-12).

Follow up:

The patients were followed up for a period of 6 months to one year. There was no mortality in the present series. Patients without post of neurological deficits fared better in terms of functional outcome. Two patients who had hemiparesis preoperatively showed improvement in power by 1 MRC grades in the follow up period. None presented with new onset seizure however all of them were on regular antiepileptics in the follow up period.

Table 1: showing the comparison of clinical features among various studies.

Clinical features	Present study	Colli et al27	Rangel-Castilla et al
Seizure	46.3%	22.5%	16%
Headache	68%	74.4%	94%
Nausea/vomiting	28.9%	46.9%	14%
Papilledema	7.2%	45%	-
Gait abnormality	5.7%	25.6%	13%
Dizziness/ Vertigo	26%	21.9%	10%
Hemiparesis	2.8%	8.1%	16%
Mental confusion	2.8%	11.3%	9%
Visual disturbance	4.3%	15.6%	32%

Table 2: Showing the proportion of surgical procedures in various studies**.**

Procedure performed	Present study	Colli et al[28]	Rangel-Castilla et al[12]
VP shunt	54.5%	37.5%	51.6%
Supratentorial Craniotomy	27.2%	15%	29.%
Posterior fossa craniotomy	18..1%	17.5%	-
Endoscopic	-	3.2%	12.9
Others	-	26.7%	

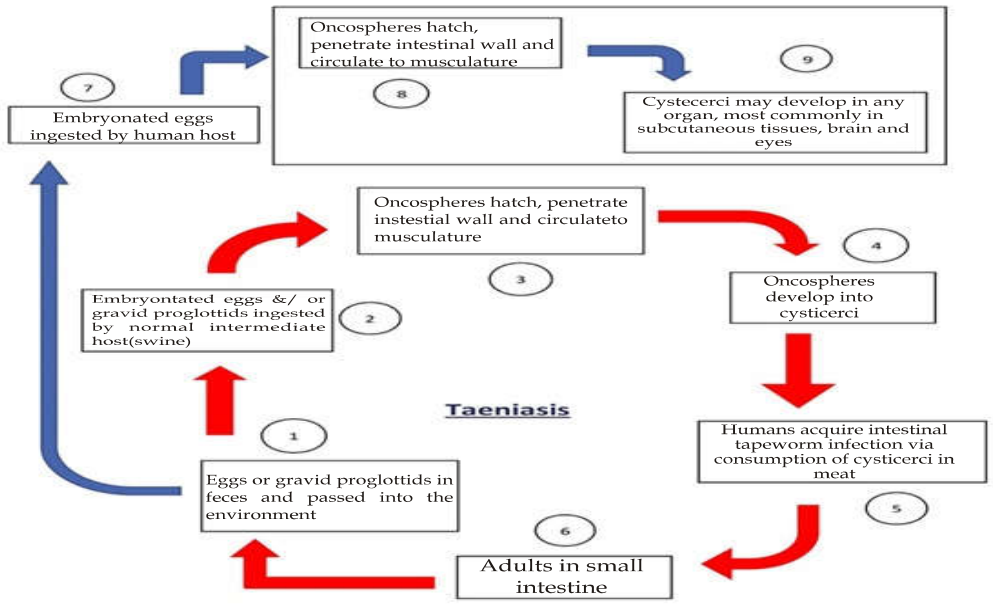

Fig 1: Life cycle of T.Solium.

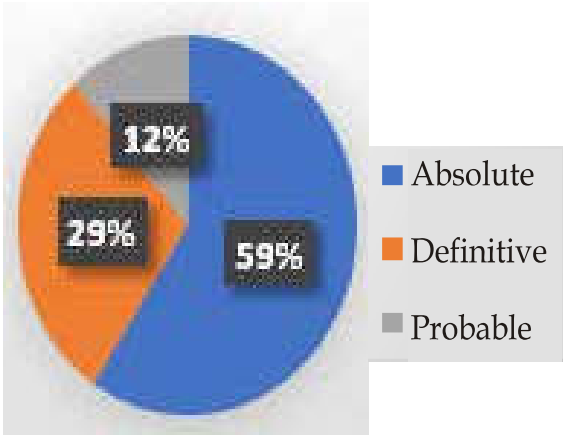

Fig. 2: Distribution of the patients according to Del Brutto et al diagnostic criteria

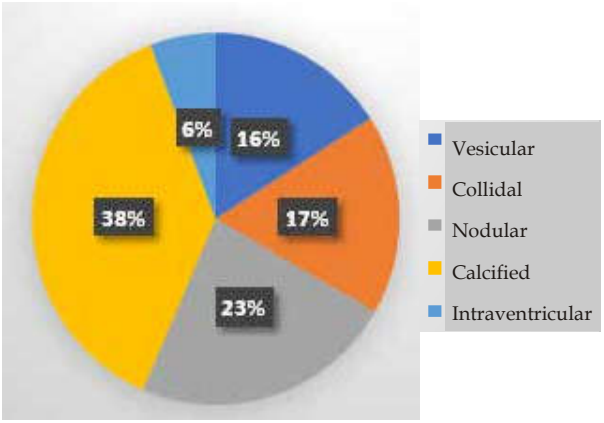

Fig. 4: Proportion of cysts at various stages of development as seen on neuroimaging.

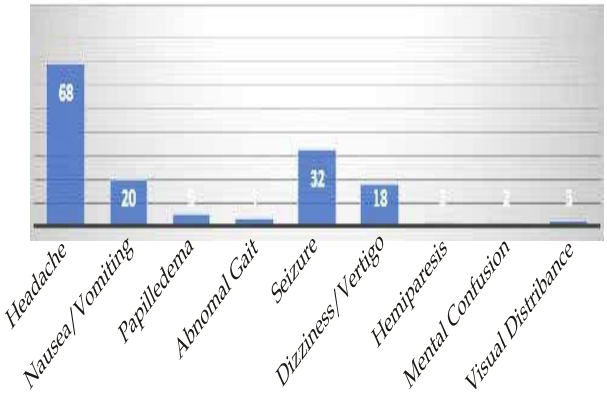

Fig. 3: Showing the frequency of clinical features in the study group.

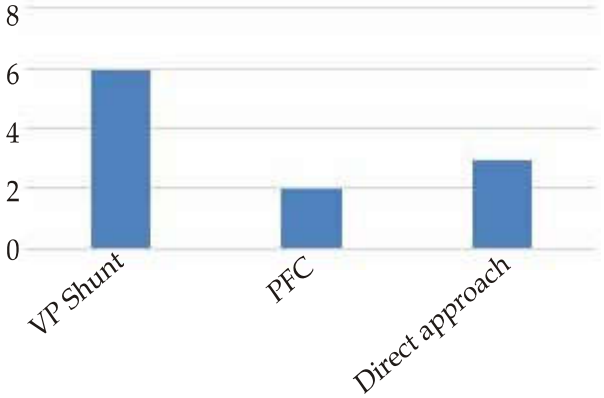

Fig. 5: Showing the various surgical procedures. (Direct approach- two right frontal craniotomy and one right parietal craniotomy)

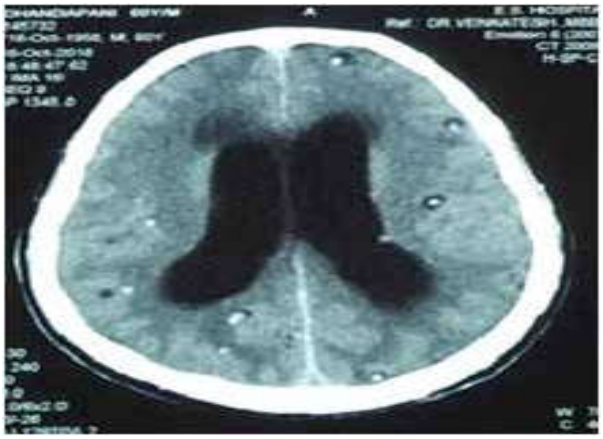

Fig. 6: CT Brain showing multiple calcified NCC with hydrocephalus

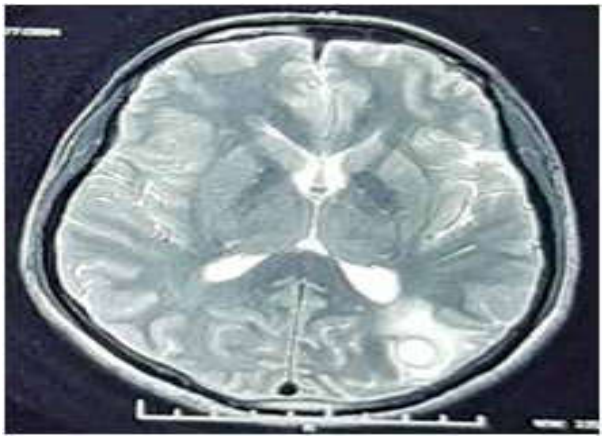

Fig. 8**:** T2WI MRI Brain showing an active cyst with perilesional edema in left parietal region.

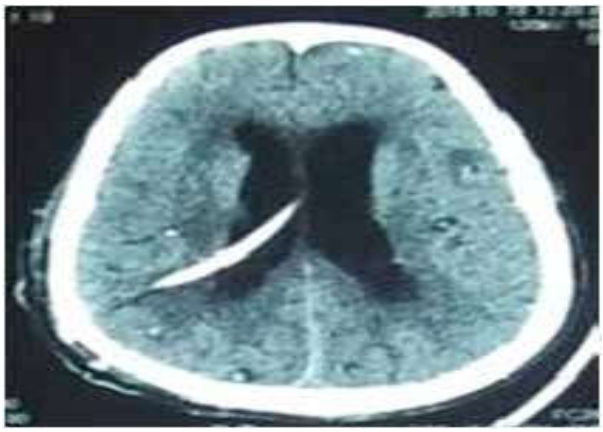

Fig. 7: Post op CT Brain after right VP shunt insertion

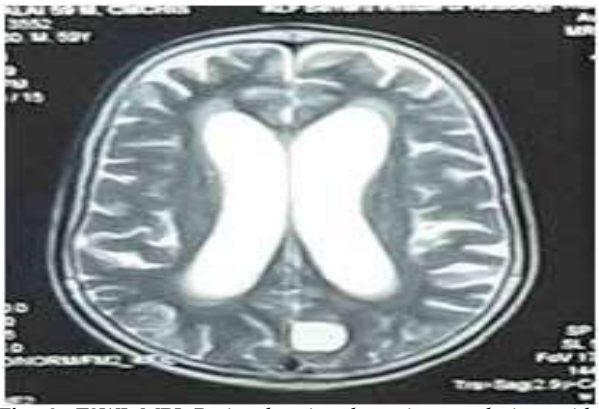

Fig. 9: T2WI MRI Brain showing hyperintense lesion with central scolex with hydrocephalus

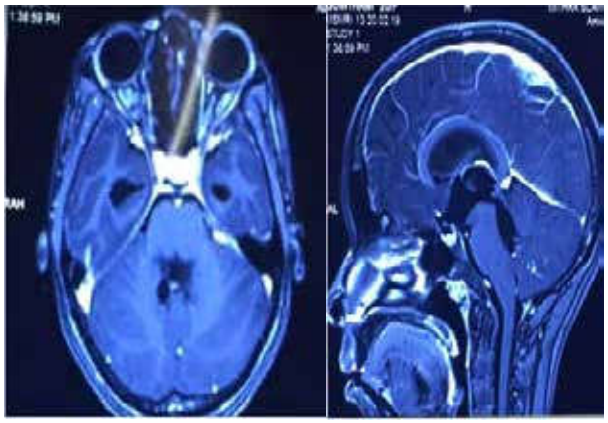

Fig. 10, 11: Axial and Saggital section MRI showing cyst in the 4th ventricle.

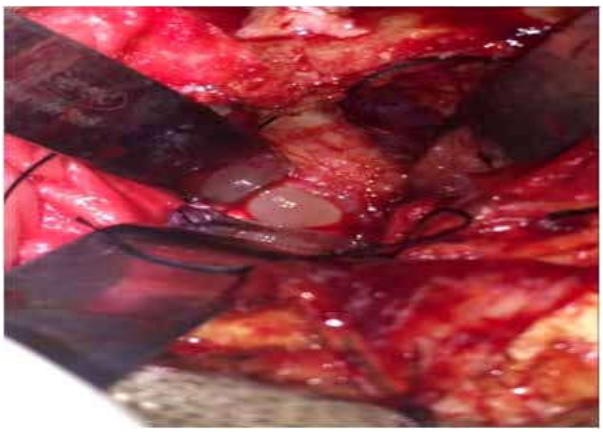

Fig. 12: Posterior fossa craniotomy for excision of a 4th ventricular NCC with the cyst in situ.

Discussion:

Antiparasitic drugs praziquantel and albendazole, along with steroids forms the cornerstone of medical management of active parenchymal and subarachnoid cysts. Albendazole has been shown to reduce the recurrence of seizures in children with a single non–viable lesion, and improve the radiological clearance of lesions in patients with a few viable lesions. Corticosteroids have been shown to reduce the incidence of adverse events during treatment.[9] Residual lesions have significant correlatation with the recurrence of seizures. In children with complete disappearance of lesions following treatmemt, no seizure recurrence was seen even with short periods of antiepileptic drug treatment.[10]

Headache is the most common presentation in the present series, various studies have shown an association between healed NCC and headache, and with migraine in particular. Aura was more likely to be present in migraine patients with NCC than those without, also lateralization of migraine to the side of calcified NCC has been seen in most of the cases.[11]

Surgical removal through a safe approach is the recommended treatment for intraventricular cysts.[12] In patients with parenchymal cysts causing local compression or giant cysts causing intracranial hypertension or significant mass effect, cyst removal is indicated. It is recommended to manage elevated ICP in patients with untreated hydrocephalus or diffuse cerebral edema prior to initiation of antiparasitic therapy.[13]

Indications for surgical treatment of NCC

1) spinal cord compression or of nerve root and cauda equina compression by NCC
2) local compression of the brain and of cranial nerves;
3) intracranial hypertension.

Spinal cysticercosis:

The incidence of spinal cysticercosis varies from 1.2% to 5.8%.They can be intramedullary, extramedullary-intradural, extradural or rarely even within the vertebral body. It has been found that Indian patients who tend to have more intraparenchymal forms of disease cranially with less chance of spinal NCC. Moreover in Indians spinal cysticercosis is often intramedullary than intradural-extramedullary. Every patient with cysticercus anywhere in the nervous system should undergo a thorough craniospinal imaging to identify asymptomatic disease in other parts of the nervous system.[14]

Cisternal Forms Causing Local Compression

Patients with cysticerci in the basal cisterns have lesions that are varied in size and cause symptoms of local compression independent of signs of hydrocephalus or raised ICP. These forms are also known as C. racemose. Optic nerve and the chiasm, the oculomotor nerve, the trigeminal nerve, and the facial nerve are the most commonly involved cranial nerves in these lesions.[15] Parinaud syndrome and focal signs due to compression of the cerebral cortex may be seen due to compression induced by cysts located in the cistern of the quadrigeminal lamina.[16] Surgical excision of cysticerci in the basal cisterns is not an effective procedure due to their multiplicity and adhesion to the cranial nerves, vessels, and brain parenchyma due to arachnoiditis.

Forms Progressing to Intracranial Hypertension

The incidence of increased ICP in patients with cerebral cysticercosis ranges from 25 to 65.9%.[16,17], In our series 81.8% of the patients presented with features of raised ICP. Patients with raised ICP could be classified in three groups according to the underlying pathophysiological process:

Group 1- hypertension caused by space-occupying cysticerci (tumoral form);

Group 2- hypertension caused by diffuse cerebral edema (pseudotumoral form); and

Group 3- hypertension secondary to hydrocephalus caused by obstruction of CSF circulation.

These mechanisms can be isolated or combined. The relative frequency of Group 1 ranges from 22 to 67%, in Group 2 from 9 to 44%, and in Group 3 from 18 to 41%.[16,17] In our series 27.2% belonged to Group 1, none to Group 2, 81.8% to Group 3.

Because of their particularly high morbidity and mortality rates, patients afflicted with forms of NCC manifesting intracranial hypertension usually require surgical treatment.

Tumoral Form-The response to anticysticercal drugs in these forms especially the ventricular and cisternal giant cysticercitypes remains controversial.[13] There is always the risk of cyst degeneration with induced inflammatory reaction in patients with giant cystecerci and mass effect.[18] In our opinion surgical excision is the best option

for the tumoral form of NCC. Complete resection of giant cysts is usually relatively easy when the cyst is located within the parenchyma or cisterns, as well as when the cysts are in the active phase because they are still loosely adherent to the parenchyma. However degenerating cysts especially those close to the eloquent cortex and blood vessels should be approached with caution, because of inflammatory reaction surrounding these cysts.

Pseudotumoral or Encephalitic Form- This form is characterized by increased ICP secondary to a diffuse inflammatory reaction of the brain parenchyma due to massive infestation with cisticerci,and it is more frequent in children.[16,17] It has been observed in GRE sequences of MRI that the calcified cysts with scolex are also associated with perilesional edema which is probably due to preservation of antigenic material in these calcified cysts.[19] Medical management with osmotic diuretics, steroid, and CSF drainage forms the cornerstone of treatment in these forms.[20] Decompressive craniectomy was recommended in some of these cases to avoid visual loss due to papilledema, but its results were inconsistent.[17]

Obstructive Form- Hydrocephalus is the most frequent mechanism of intracranial hypertension in NCC Mechanism of hydrocephalus

1.Direct obstruction by intraventricular cysts

2. Cysticercal meningitis

3. Ependymitis

Foramen of Magendie and the cerebral aqueduct are the usual sites of cyst related obstruction leading to obstructive hydrocephalus, causing obstructive hydrocephalus. Inflammatory reaction to the cysts can occur at any point in CSF circulation, leading to obstructive or communicating hydrocephalus, but is more common around the fourth ventricle, posterior fossa and the basal cisterns.The cysticercus cysts are smooth walled and slimy and exhibits extreme plastic deformation and mobility. These character of the cysts permits their movements within the narrow ventricular confines.This malleability of the intraventricular NCC cyst can lead to intermittent hydrocephalus due to blockage of the ventricular system caused by the mobile cyst with spontaneous relief of symptoms due to further passage and relief of obstruction of the CSF pathways.[21]

Treatment planning of cysticercosis-related hydrocephalus requires identification of its pathogenesis. Cyst removal can relieve obstruction caused by free cysts.[22] VP shunt is considered the best treatment for patients with obstruction caused by an inflammatory process.[23]

CT scan is not helpful in identification of free intraventricular cysts; CT cisternography can demonstrate cisternal cysts and when there is no obstruction of the ventricular system, it can identify intraventricular cysts. CT ventriculography allows adequate evaluation of the ventricular system.[24] MRI can demonstrate cysticerci and inflammatory reactions in the parenchyma, cisterns, and even ventricles. However, unless there are features of mass effect, absence of the adjacent CSF flow, and obstruction of the ventricular flow, intraventricular cysts can be visualised on MRI only after they start to degenerate.[25,26]

With the use of microsurgical techniques, proper planning and suitable approach the excision of free ventricular cysts is relatively simple with minimal morbidity and mortality. Surgery is advocated for free intraventricular cysts as these may dislocate within the ventricular system causing acute obstructive hydrocephalus. These cysts tend to increase in size and start to behave as intraventricular tumors, especially in the fourth ventricle, as was seen in two of our patients. Fourth ventricle is accessed by a posterior fossa craniotomy (PFC), and the free cysts can be delivered through the foramen of Magendie toward the cistern magna. In cases where the cysticerci are adherent to the ventricular wall or when the foramen of Magendie is stenotic or obstructed, sectioning the inferior portion of the cerebellar vermis can be done to widen the foramen to allow better visualization of the ventricle. In our series two patients presented as fourth ventricular masses, both were approached by PFC and excision. The mortality rate observed in patients undergoing PFC range from 0 to 60%.[15,27] In our series there was no mortality in patients who underwent PFC and in both case no significant adhesion was seen intraoperatively in both the cases. Outcomes are better in patients with free ventricular cysts, than those with who present with inflammation and adhesion around the intraventricular cyst or cases with aseptic meningitis due to intraoperative rupture of the cyst.[28] Hence it is prudent to identify free ventricular cyst and differentiate between cyst-related obstructions and inflammation, on preoperative imaging for planning an appropriate surgical approach.

When inflammatory reactions are intense, it is suggested to perform VP shunt after the cyst resection, to avoid hydrocephalus developing from CSF circulation blockade due to inflammation within a few weeks or months.[15] In our series none of the patients undergoing PFC developed hydrocephalus during the follow up period.

Stereotactic approaches to cyst resection or drainage,[29] and endoscopic cyst drainage or resection,[30] have been proposed as alternatives to excision of giant parenchymal and ventricular cysts. Evacuation of the cyst by puncturing has also been suggested as, the rupture of a cyst triggersthe degenerative process. Recurrence after puncture and drainage has been attributed to the existence of racemose cyst at the punctured site.[30]

More recently, endoscopic excision has been advocated both for supratentorial and infratentorial intraventricular cysts. In cases of cyst rupture during endoscopic removal intraventricular steroid injectionto prevent an anaphylactic reaction has been suggested.[31] Combined endoscopic removal of the cysts with a third ventriculostomy and/ or a septal pellucidotomy has shown excellent results with shunt avoidance in most of the cases. Endoscopic removal of fourth ventricular cysts has also been described, pre-operative USG or image guided navigation is helpful in these procedures.[32] Overall, endoscopic removal of cysts is a minimally invasive procedure, reduces blood loss and operative time and provides excellent visualisation of the anatomy.

Post-operative complication and Prognostic Factors: Infectious complications were most commonly seen in the postoperstiive period in the present study.

Complication	Present study (%)	Colli et al[27](%)
Bacterial Infection	18%	30.6%
PFC complication (meningitis)	9%	10.2%
VP shunt complication	9.09%	25.2%

Subarachnoid cysts (extraparenchymal cysts), especially at the basal cisterns, causing chronic arachnoiditis, has been identified as poor prognostic factor.[15,28] Patients requiring VP shunt for the treatment of inflammatory obstruction and cysts fared worse than in those who underwent removal of intraventricular cysts.[28] Male sex and age younger than 40 years are found to have a poor prognosis.[28]

Conclusion

Cysticercosis is considered as a "biological marker" of social and economic development. It is a global health problem and developing countries, like India, bear most of the disease burden. Other than measures like health education, public awareness, accessible health care, mass treatment of carriers, sanitary conditions and changes in porcine-rearing practises, development of an effective vaccine against porcine cysticercosis may prove the best potential tool for eradication of the disease. In the present study the short term prognosis following surgery was good. However NCC being a chronic disease these patients require prolonged follow up to understand the long term complications and their impact on the overall outcome and survival of the patient.

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