Cerebral Sparganosis

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Abstract
A 22 years male patient presented with recurrent seizures, CT and MRI diagnosis of tuberculoma was made and the patient was treated. When seizures persisted, a craniotomy was done and the excised mass revealed an abscess with a segment of broad solid non-cavitatory body, wall with no scolex and loose stroma and smooth muscle fibers. A diagnosis of sparganosis cerebral abscess was made. The case is reported in view of the rarity of cerebral sparganosis in India and the need for awareness of the entity in India.

INTRODUCTION

Human sparganosis is caused by infestation of tape worm Sparganum, the migrating plerocercoid (second stage) larva of pseudophyllidean cestodes especially of the genera Diphyllobothrium and Spirometra. The majority of infections in man are probably caused by larvae of Spirometra mansonioides. Infections caused by both the larvae and adult worms have a worldwide distribution1 but most common in China, Japan and Southeast Asia.2,3 Sparganum usually involves subcutaneous tissues and/or muscles of various parts of the body, but involvement of other sites such as orbit, pleural cavity, brain, urinary tract, scrotum, and abdominal viscera has been documented.1,4 Central nervous system (CNS) involvement is distinctly rare. Cerebral and spinal cord involvement is reported.1,4-7 Reports of CNS sparganosis from India are very few.8 We report a case of sparganosis brain abscess.

CASE REPORT
A 22 years male presented with recurrent right focal onset generalized tonic clonic seizures with secondary generalization of two years duration. There was no history of fever, headache, vomiting. On examination, higher intellectual functions were normal. There were mild left cerebellar signs present. There were no neurological deficits. There were no cranial nerve palsies. Ocular fundi were normal. The other systems were normal. CT scan brain showed iso to hyperdense lesion in left frontal lobe. There was ring enhancement with perilesional edema (Fig. 1). MRI on T2W1 showed nodular hyperintense lesion with surrounding hypointensities suggestive of perilesional edema in left frontal lobe. Contrast MRI showed confluent ring enhancement (Fig. 2). With a possible diagnosis of tuberculoma, patient was treated with rifampicin, isoniazid and pyrazinamide for one year. There was no improvement and seizures persisted. A left frontal craniotomy was done and the mass was excised. The mass measured 4 x 3 x 2 cm with central necrotic areas. Multiple sections studied showed an abscess with central necrotic material surrounded by neutrophils, few eosinophils, foamy histiocytes and lymphomononuclear cells. There were no granulomas. Amidst the necrotic material, a broad, solid, non-cavitory irregularly ridged segment of parasite measuring 2 to 3 mm was seen (Fig. 3). The tegument was thick and homogeneously eosinophilic. At the anterior end the worm is flattened and grooved vertically. Scolex was absent. The parenchyma was composed of loose stroma, fluid-filled spaces and a few smooth muscle fibers. There were no calcareous corpuscles or calcification. Serial sections failed to bring out the entire length of the worm. The wall of the abscess showed fibrosis and hyalinization. The adjacent brain parenchyma
showed gliosis. The morphology of the parasite was identified as Sparganosis sp based on the characteristics of body wall, tegument and anterior end. Post-operative contrast CT showed residual lesion with perilesional edema. Patient was treated with albendazole for six weeks. Patient was doing well at six months follow up.

**DISCUSSION**

Neurohelminthiasis due to larval forms of cestodes in India include cysticercosis and echinococcosis. Both these infections are transmitted by contaminated food or water and man is the accidental second intermediate host. Sparganosis is reported very rarely from India. Human sparganosis may result from ingestion of infected cyclops containing the procercoid stage. The adult tapeworms live in the intestines of dogs and cats. The routes of human infestation are by i) ingestion of water contaminated with copepods, ii) ingestion of raw second intermediate hosts, such as snake, frog and triton, iii) topical application of a slice of raw meat to open wounds and mucous membranes, iv) ingestion of the pleocercoid larva through carriers.

The clinical manifestations are non-specific; however a long history of seizures is the most common clinical manifestation as was seen in our case. History of eating raw intermediate hosts, if present, may be helpful. The neuroradiological findings are also not specific. Chang et al reported that the CT characteristics of cerebral sparganosis are as follows: i) unilateral involvement, ii) extensive or multifocal areas of low density along white matter fasicles, with ipsilateral ventricular dilatation and localized cortical atrophy, iii) nodular or irregular enhancement with spotty calcification and iv) change in location of enhancing nodules on sequential scans.

Kim et al described magnetic resonance features as widespread white matter degeneration and cortical atrophy, mixed-signal lesion with irregular dense enhancement of central foci. The authors conclude that MR is the most valuable modality for the early detection of cerebral sparganosis. As India is not an endemic area for sparganosis, the findings on CT and MR were interpreted as those of tuberculoma.

ELISA test performed for sparganum-specific immunoglobulin (IgG) is very sensitive. The serum ELISA test has 88% sensitivity and CSF ELISA test has 93% sensitivity.

Negative results on ELISA test after surgical removal predict good long-term outcome and persistent positive reaction strongly suggests incomplete removal of the worm.

The tissue reaction depends upon whether the worm is live or dead. Brain involvement is seen as a granuloma or abscess. Complete removal of the worm by surgery gives good long term outcome. Drug therapy with praziquantel did not have a favourable outcome. Sequential scanning may indicate whether the worm is alive or dead within the tissue.

India is endemic for cysticercosis and awareness of sparganosis in the differential diagnosis is essential. Awareness on the part of the pathologist also is important. It is necessary to establish ELISA tests for identification.

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**REFERENCES**


