

Cystic Lesions of Brain Anatomical Surgical and Radiological Study

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1. Introduction

1.1. Material and Methods

We retrospectively analysed MRI and CT Scan Reports from Patients from Private clinics of Srinagar, Kashmir, India between 2013-2016. A detailed recording was made of the presenting features, neurological signs, investigations, type of surgery, recurrence of hydatid disease and other therapeutic modalities.

1.2. Results

Multiple (three) cases of intracranial hydatids were detected during the period 2013-2016. Hemiparesis, Papilloedema and Seizures were present in two cases. In three patients, radiological investigations included CT scan. In one case, both CT and MR scans were done. One patient had solitary cyst in parietal lobe. There were no Hepatic hydatid cysts.

2. Discussion

Echinococcosis, or hydatidosis, is the most frequent cause of liver cysts in the world. These cysts are relatively rare in brain. The most common form is due to *Echinococcus granulosus*, although occasionally *E. multilocularis* is the infective agent. The adult *E. granulosus* is a tapeworm that resides in the jejunum of dogs. Eggs are passed in the stool and ingested by cows, sheep, moose, caribou, or humans. Embryos pass through the intestinal mucosa into the portal circulation and are filtered by the liver and occasionally by the lungs. Of the serologic tests, the indirect hemagglutination test and the Cassoni skin test have approximately an 85% sensitivity. The complement fixation test has approximately a 70% sensitivity. Calcification of the cystic wall is present in over half the patients. Treatment is primarily surgical.

Hydatid disease is caused by the infestation of the larvae of *echinococcus*. The definite hosts of *echinococcus* are various carnivores, the common being the dog. All mammals (more often being sheep and cattle) are intermittent hosts. Humans get infected through the faeco-oral route by ingestion of food or milk contaminated by dog faeces containing ova of the parasite or by direct contact with dogs. The eggs lose their enveloping

layer in the stomach, releasing the embryos. The embryos pass through the wall of the gut into the portal system and are carried to the liver where most larvae get entrapped and encysted. Some may reach the lungs and occasionally, some may pass through the capillary filter of the liver and lungs and get entry into the systemic circulation. These may even reach the brain. In India, the hydatid disease is more commonly seen in Andhra Pradesh, Tamil Nadu and in Punjab. We have analysed three cases of the hydatid cysts and discussed their mode of presentation, radiological features and outcome.

Intracranial hydatid disease is a disease of rare occurrence, with reported incidence of less than 1% of all cases with hydatid disease [1]. Hydatid disease is endemic in the middle east, Mediterranean countries, South America, North Africa and Australia [2]. Cerebral hydatid disease is more common in paediatric population [3]. Intracranial hydatid cysts are more frequently located in the supratentorial compartment. The other less common sites reported are skull [4], cavernous sinus [5], eye ball [6], pons [7], skull, extradural [8], cerebellum and ventricles [9]. Solitary hydatid cyst in the lateral ventricle, as seen in one case in the group of patients being reported, is relatively rare site for intracranial hydatid cyst. The cerebral hydatid cysts are slow growing and present late when they increase in size and become large.

Intracranial hydatid cysts are commonly solitary. Multiple intracranial cysts are rare. Intracranial hydatid cyst may conveniently be classified as primary or secondary. The primary cysts are formed as a result of direct infestation of the larvae in the brain without demonstrable involvement of other organs. In primary multiple cysts, each cyst has a separate pericyst with brood capsule scolices and these originate from multiple larvae affecting brain after crossing the gastrointestinal tract, liver, lungs and right side of heart without affecting them. The primary cysts are fertile as they contain scolices and brood capsules, hence rupture of primary cyst can result in recurrence. The secondary multiple cysts result from spontaneous, traumatic or surgical rupture of the primary intracranial hydatid cyst and they lack brood cap-

sule and scolices. The secondary intracranial hydatid cysts are therefore, infertile and the resultant risk of recurrence after their rupture is negligible.

The patients with intracranial hydatid cysts usually present with focal neurological deficit and features of raised intracranial pressure; the latter may be due to the large size or due to interference with pathway of CSF flow.

MR and CT scans characteristically show hydatid cyst as a spherical, well defined, non-enhancing cystic lesion without peripheral oedema [10,11]. The fluid density is generally equal to that of CSF on both CT and MR scan. A fine rim of peripheral enhancement with perilesional oedema may be seen in the presence of active inflammation. MR scan may show a low-density cyst wall and relations with surrounding structures are better delineated than on CT scan. Other features can be presence of daughter cysts (as seen in our case), detached membranes, parenchymal distortion, hydrocephalous and increased intracranial pressure. Growth rate of brain hydatid has been reported between 1.5-10 cm/year. On MR I, cyst is hypointense on T1 weighted, hyperintense on T2 weighted images with cyst wall being hypointense on T1/T2 weighted images. Presence of surrounding oedema and rim enhancement indicates complicated cyst. The most common location for intracranial hydatid cysts is the hemispheric parenchyma, particularly in the perfusion territory of the middle cerebral artery. Hydatid cysts are usually spherical, solitary, and unilocular. The cysts may contain translucent fluid and may also contain daughter cysts with an appearance resembling small white grapes. Protoscolices within cysts form a granular deposit known as hydatid sand. The best diagnostic clue of a hydatid cyst is a single, large, thin-walled, spherical, nonenhancing CSF-attenuation cyst in the parietal region of the brain. The two visible imaging components are the cyst and the pericyst. The pericyst is a peripheral capsule of the cyst. While MR imaging is more sensitive in demonstrating the pericyst, CT is more sensitive in depicting cyst calcification.

Differential diagnosis is that of a Neoplasm Primary or metastatic (primary often known) or Abscess, arachnoid cyst, epidermoid cyst, and neurocysticercosis. The treatment of hydatid cyst is surgical and the aim of surgery is to excise the cyst in without rupture to prevent recurrence and anaphylactic reaction.

3. Cysticercosis

is caused by the larva of *Taenia solium*. Infection is acquired by ingesting the tapeworm eggs. This clinical picture can resemble ICSOL and should be differentiated from brain abscess, a granuloma such as tuberculoma, or a brain tumor. Gravid proglottids have 5-10 uterine branches. Life cycle: Humans ingest undercooked pork containing cysticerci. Larvae attach to gut wall and develop into adult worms with gravid proglottids. Terminal proglottids detach, pass in feces, and are eaten by pigs. In the gut, oncosphere (hexacanth) embryos hatch, burrow into blood vessels, and migrate to skeletal muscles, where they develop into cysticerci. If humans *Taenia solium* eggs in food contaminated with human feces, the oncospheres burrow into blood ves-

sels and disseminate to organs (e.g., brain, eyes), where they encyst to form cysticerci. Laboratory Diagnosis of *Taenia solium* is gravid proglottids visible in stool. Eggs seen less frequently. Usually the Lesions in the Brain are Cystic and the cyst wall is usually thin and well differentiated from Parenchyma without Perilesional Edema.

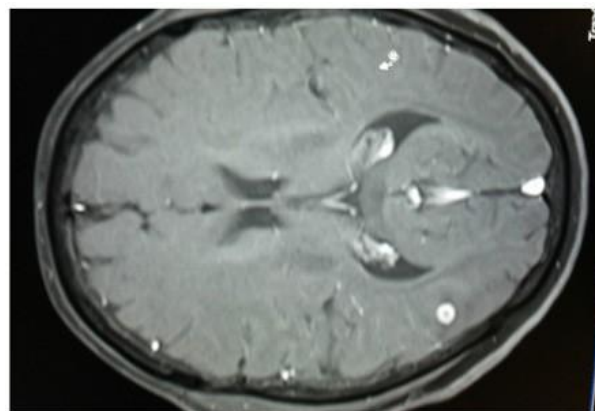


Figure 1: Neurocysticercosis.

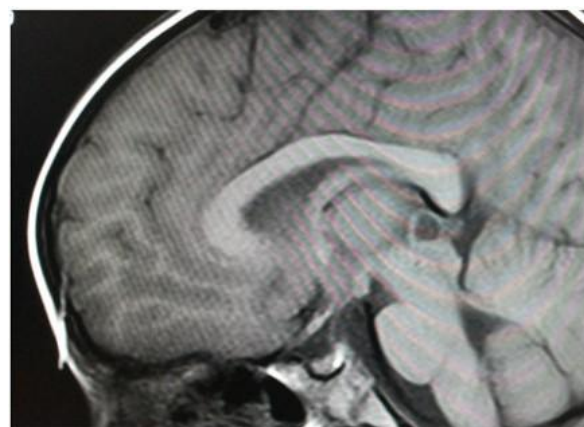


Figure 2: Pineal Cyst in the Region of Pineal Gland.

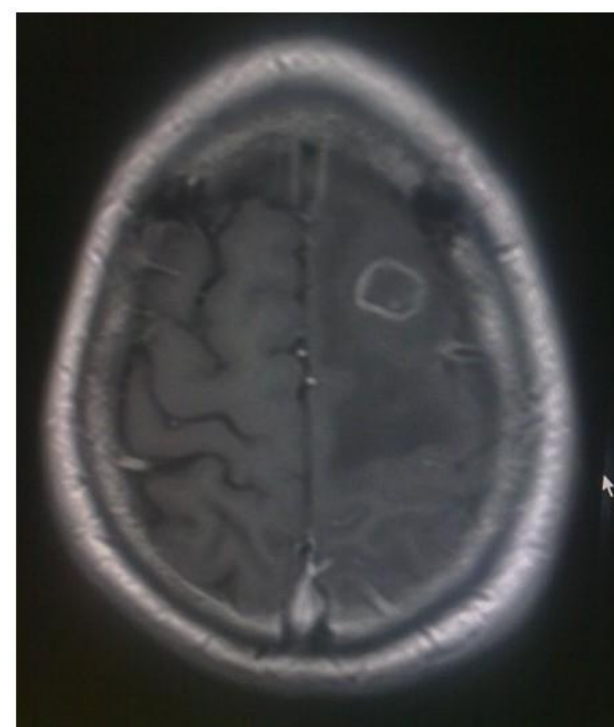


Figure 3: Tubercular Cyst in the Brain.

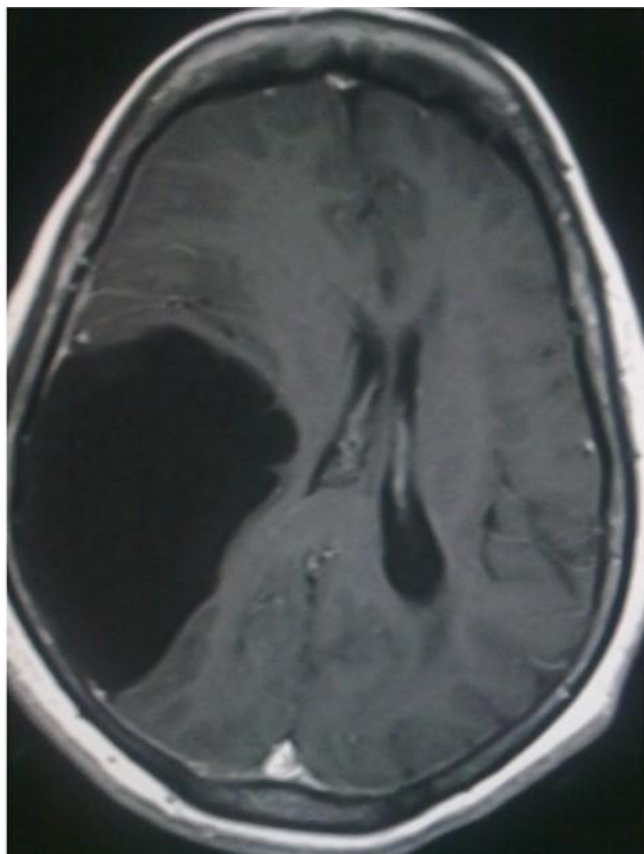


Figure 4: Porencephalic Cyst.

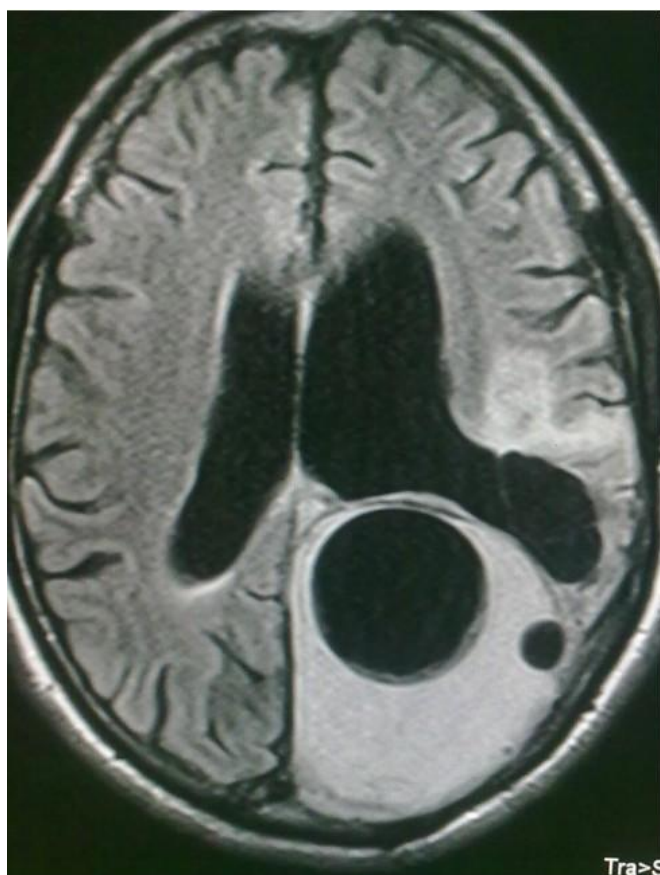


Figure 5: Hydatid Cyst Brain.

3.1. Tubercler Cysts

are caused by *Mycobacterium tuberculosis*. Causes Tuberculosis. Characteristics of *Mycobacterium tuberculosis* are Aerobic,

Acid-fast rods. Grows very slowly which requires that drugs be present for long periods(months). Laboratory Diagnosis of *Mycobacterium tuberculosis* is acid fast rod seen with Ziehl-Neelsen or (Kinyoun) stain. Tuberculosis can affect brain, Spinal Cord, Meninges, Cranial as well as peripheral nerves.

3.2. Pineal cyst

Pineal gland is a small conical body projecting downwards between two superior colliculi.

The pineal body has far long been regarded as a vestigial organ of no importance. Recent investigations have shown that it is an endocrine gland of great importance. It produces hormone that may have an important regulatory influence on many other endocrine organs (including the adenohypophysis, the neurohypophysis, the thyroid, the parathyroid, the adrenal cortex and medulla and the gonads).

Pineal cysts are present in 5% or 10% of normal population.

3.3. Porencephalic Cysts

are seen as cavities or cysts in the cerebral hemisphere. They are formed as a result of destructive brain lesions and can cause neurological and physiological symptoms. There may be associated defective developmental problem. They are common in neonates and younger age group.

3.4. Conclusion

Cysts of the Brain are not that Infrequent. The Cysts of the brain presents clinically as intracranial space occupying lesion and can be seen in any age group.

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