Case Report

Neuroendoscopic Management of Lateral Ventricular Neurocysticercosis Presenting as Brun’s Syndrome

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Background: Brun’s syndrome is a phenomenon characterized by sudden onset of severe headache, vomiting associated to a vestibular syndrome triggered by an abrupt movement of the head. Case Presentation: We present a case of a 12-year-old female patient with headache, vertigo, and vomiting; magnetic resonance imaging (MRI) was suggestive of a cystic intraventricular mass in the frontal horn of the left lateral ventricle. The patient underwent endoscopic exploration for the excision of cyst with complete postoperative recovery and histopathology suggestive of intraventricular neurocysticercosis. Discussion: Brun’s syndrome is caused by a mobile deformable intraventricular mass leading to an episodic obstructive hydrocephalus resulting from an intermittent or positional CSF obstruction with elevation of intracranial pressure due to the ball valve mechanism. Treatment is mainly surgical, preferably by the neuroendoscopic technique as it has an advantage of performing septostomies and third ventriculostomies in addition to cyst removal, making this procedure practical for most cases of ventricular cysticercosis even in emergencies.

Keywords: Brun’s syndrome, intraventricular SOL, neurocysticercosis, neuroendoscopy

INTRODUCTION

Neurocysticercosis (NCC) is one of the common parasitic helminthic infections of the central nervous system caused by the larval stage of taenia solium.[1] It has been seen in world literature that NCC is uncommon in childhood, which can be attributed to the prolonged incubation period of the disease and which can range from several months to 30 years (average 4.8 years).[2] The prognosis for intraventricular neurocysticercosis (IVNCC) is worse than that for the intraparenchymal form of the disease, making treatment specially important.[3]

The IVNCC can present with various features. However in some patients, the increasing intracranial pressure due to obstruction of the aqueduct is intermittent, producing relapsing/ remitting symptoms. This infrequent and life-threatening phenomenon is called Brun’s syndrome.[4]

We report a case of a 12-year-old female child who presented with Brun’s syndrome due to intermittent pressure on the foramen of Monro caused by a cystic lesion in the ventricular system managed endoscopically with complete postoperative recovery and histopathology suggestive of NCC.

Case Report

A 12-year-old female presented with complaints of headache since two weeks, which worsened on forward bending of the head associated with abrupt episodes of vertigo and vomiting. There was no history of fever, seizures, or visual symptoms. Fundoscopy was suggestive of early papilloedema. Perimetry showed...
generalized scotomatous points. Gadolinium-enhanced MRI brain showed a large cystic intraventricular mass in the frontal horn of the left lateral ventricle measuring 4(CC)*5(AP) * 3.2(T) cms. The lesion was hypointense on T1 and hyperintense on T2 and FLAIR. No contrast enhancement was seen in the lesion. A tiny solid nodule of 7*6 mm was seen in the periphery of the lesion along the posterior aspect. The lesion caused displacement of the septum pellucidum to the right, and dilatation of both lateral ventricles with a mild periventricular ooze was seen [Figure 1]. Differential diagnosis on MRI comprised ependymal cyst/ arachnoid cyst/ choroid plexus cyst/ colloid cyst. The patient underwent endoscopic exploration with excision of the cyst. Intraoperatively, a large cyst was seen in the lateral ventricle with a gritty surface with a frond-like movement within the ventricle. On aspirating the cyst, clear CSF-like fluid was found, which, on analysis, did not show any growth. The cyst was removed in total and sent for evaluation [Figure 2]. After this, the third ventricle was inspected and any remnants of the cyst in the third ventricle were ruled out. The ventricle was well irrigated with Ringer lactate solution. Histopathology was suggestive of neurocysticercosis. The postoperative period was uneventful, and the patient was discharged with albendazole for 1 month with an improvement in postoperative perimetry. The postoperative CT brain showed complete excision of the cyst, with septum pellucidum attaining its normal midline position [Figure 3].

**DISCUSSION**

It has been seen in world literature that NCC is uncommon in childhood. This low incidence in children is attributable to the long incubation period of the disease, which ranges from several months to 30 years (average 4.8 years). [2]

NCC is divided into parenchymal and extraparenchymal forms; the latter includes intraventricular, subarachnoid, and occasional spinal forms.[5] Patients younger than

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**Figure 1:** Preoperative MRI. (A) T1 image hypointense lesion with a tiny solid nodule causing displacement of the septum pellucidum to the right. (B) T2 hyperintense lesion. (C and D) No contrast enhancement of lesion

**Figure 2:** Intraoperative. (A) Large cyst with gritty surface and normal ependyma of the ventricle in the background. (B) Final portion of cyst being removed. (C) Normal ventricular anatomy postexcision of the cyst. (D) Cyst held with an endoscopic grasper

**Figure 3:** Postoperative CT. (A and B) Axial and coronal scans showing complete excision of the cyst, with septum pellucidum attaining its normal midline position
17 years of age constitute 0.8–27.5% of diagnosed cases of NCC, with the intraventricular form being even rare.

The cysts reach the ventricular system through the choroid plexus and are more frequently found in the fourth ventricle, which is likely due to the gravitational forces that favor migration from supratentorial ventricles, or they may directly enter through the choroid plexus.\[^6\]

The natural history depends on the location of intraventricular cysts. The ones attached to the ventricular wall involute and eventually resolve, whereas the cysts that are not attached may migrate and block the cerebrospinal flow causing obstructive hydrocephalus.\[^6\] IVNCC can present with clinical features of hydrocephalus, which include headache, nausea, vomiting, altered sensorium, and papilledema, or they may present infrequently with features of Brun’s syndrome. The mechanism of hydrocephalus is either ventricular obstruction or arachnoiditis.\[^6\,^7\]

Brun’s syndrome, which was first described in 1902, is a phenomenon characterized by sudden onset of severe headaches and vomiting associated to a vestibular syndrome triggered by an abrupt movement of the head. The presumptive cause is a mobile deformable intraventricular mass leading to an episodic obstructive hydrocephalus and resulting from an intermittent or positional CSF obstruction with elevation of intracranial pressure due to the ball valve mechanism.\[^6\]

According to literature, the diagnosis of NCC includes systematic evaluation of clinical features, neuroimaging, and serology.\[^3\] However, the role of serology is limited because of low sensitivity and specificity. The identification of a scolex in a cystic lesion is a pathognomonic radiological finding. Scolices appear as rounded or elongated bright nodules within the cyst cavity.\[^9\] Noncontrast CT scanning is sensitive for calcified and parenchymal lesions, but it is insensitive for extraparenchymal disease. Thus, MRI is a preferred diagnostic test for extraparenchymal NCC as the MRI signal properties of the cystic fluid or the scolex differ and inflammation is marked with hyperintensity signals, allowing for a reliable diagnosis.\[^5\,^3\] Nonvisualization of scolices coupled with the absence of pericystic gadolinium enhancement on MRI posed a problem in the radiological diagnosis of NCC in our case. Hence, a radiological differential diagnosis of ependymal cyst and colloid cyst was considered.

Therapeutic measures include antiparasitic drugs, surgery, and symptomatic medications. As inflammation is common in most forms of NCC, corticosteroids represent the primary form of therapy for meningitis, cysterceral encephalitis, and angitis.\[^9\] Praziquantel and albendazole have been successfully used to treat NCC; they destroy 60% to 80% of parenchymal brain cysticerci after one course. The most accepted regimens of cysticial drugs are albendazole, 15 mg/kg per day for 28 days, and praziquantel, 50 mg/kg per day for 2 weeks.\[^10\] Medical management alone is not recommended because of the limited effectiveness of albendazole and praziquentel. The principles of surgery include managing hydrocephalus and removal of the cyst. Neuroendoscopy is preferred over microsurgery and ventriculoperitoneal shunt. Even in the emergent case of Brun’s syndrome, neuroendoscopy has been proven both diagnostic and curative, as endoscopic third ventriculostomy (ETV) or septum pellucidotomy can be done to prevent delayed hydrocephalus, in addition to cyst removal.\[^1\,^5\] Under endoscopic visualization, cysticerci appear as a “full moon,” which is considered pathognomonic for IVNCC.\[^1\]

According to literature, intraoperative cyst rupture causing postoperative ventriculitis is rare. In our case, cyst rupture occurred intraoperatively; however, the patient did not have postoperative ventriculitis.\[^11\]

**Conclusion**

Although neurocysticercosis is a common neurological infection, intraventricular NCC presenting as Brun’s syndrome is very rare. The presentation is accompanied by features of raised ICP, and early diagnosis and timely intervention can prevent mortality. Treatment is mainly surgical, preferably by the neuroendoscopic technique. The ease of performing septostomies and third ventriculostomies, in addition to cyst removal, makes this procedure practical for most cases of ventricular cysticercosis, even in emergencies.

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**Conflicts of interest**

There are no conflicts of interest.

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