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Radio-Diagnosis

A CYST ON THE MOVE: REVERSIBLE OBSTRUCTIVE HYDROCEPHALUS FROM A MIGRATING THIRD VENTRICULAR NEUROCYSTICERCOSIS CYST

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ABSTRACT Intraventricular neurocysticercosis is an uncommon form of the disease that can cause acute obstructive hydrocephalus, and a mobile cyst may act as a ball valve, producing abrupt and fluctuating neurological deterioration. We report a 25-year-old male who presented with headache and altered sensorium and required admission and intubation. MRI of the brain showed a well-defined, thin-walled cystic lesion in the third ventricle near its opening into the aqueduct of Sylvius, with an eccentric scolex (T1/FLAIR hyperintense, T2 hypointense) and no diffusion restriction. There was upstream dilatation of the lateral and third ventricles with trans-ependymal edema. The patient's sensorium recovered spontaneously within 24 hours. Repeat MRI done about 20 hours after the first study showed dislodgement of the cyst into the fourth ventricle with resolution of hydrocephalus, although trans-ependymal edema persisted. Tiny subependymal focus in the left lateral ventricle showed blooming, consistent with calcified granulomas. Recognition of the scolex, the cyst position relative to the aqueduct and the associated ventricular dimensions on MRI allows a confident diagnosis, explains the fluctuating clinical course, and flags the risk of recurrent obstruction. Serial imaging and early planning of definitive treatment are essential.

KEYWORDS : Neurocysticercosis; Intraventricular Cyst; Third Ventricle; Obstructive Hydrocephalus; Bruns Syndrome; Scolex; Magnetic Resonance Imaging

INTRODUCTION

Neurocysticercosis (NCC), caused by the larval stage of *Taenia solium*, is the most common parasitic infection of the central nervous system and a leading cause of acquired epilepsy in endemic regions such as India [1,2]. Cysts are usually located in the brain parenchyma or subarachnoid space. Intraventricular involvement is seen in roughly 10 to 20% of cases and carries a disproportionate risk of morbidity, because a cyst within the CSF pathway can obstruct flow at the foramen of Monro, the aqueduct or the fourth ventricular outlets [3,4].

Intraventricular cysts are often free-floating and mobile. A cyst that shifts position with posture or head movement can intermittently block and release CSF flow, producing sudden headache, vomiting, vertigo and loss of consciousness that may resolve as abruptly as it began. This ball-valve phenomenon is classically described with fourth ventricular lesions as Bruns syndrome [4,5]. Because the cyst wall is thin and the fluid is isointense or nearly isointense to CSF on conventional sequences, ventricular cysts are easily missed, and the diagnosis often depends on identifying the eccentric scolex or on indirect signs such as a localized ventricular contour abnormality and unexplained obstructive hydrocephalus [3,6].

Herein, we present a 25-year-old male with an intraventricular NCC cyst at the third ventricle–aqueduct junction that caused life-threatening hydrocephalus and then migrated into the fourth ventricle within 24 hours, with documented MRI evidence of the cyst in both positions and of the associated reversal of hydrocephalus.

Case Presentation

We report a case of a 25-year-old male patient who presented with headache followed by altered sensorium with an episode of seizure at the time of admission, there was no history of fever. He was admitted to the intensive care unit and was intubated for airway protection in view of the depressed sensorium.

A contrast-enhanced MRI of the brain was performed on 11th October at 7:20 pm. It revealed a well-defined, thin-walled cystic lesion measuring ~10 × 10.8 × 10.3 mm in the third ventricle near its opening into the aqueduct of Sylvius. An eccentric focus within the cyst was T1 and FLAIR hyperintense, T2 hypointense (Figure 1).

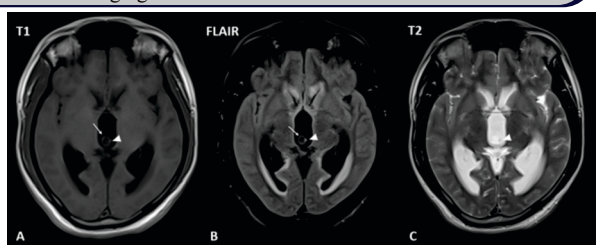


Figure 1: MRI brain (11th October, 7:20 pm) axial T1(A) and FLAIR (B) sequences show a well-defined thin-walled cystic lesion in the third ventricle near the aqueduct of Sylvius (white arrow) with an eccentric scolex (arrowhead) that is T1/FLAIR hyperintense and T2 (C) hypointense.

There was upstream dilatation of both lateral ventricles and the third ventricle with trans-ependymal edema. The frontal horns measured ~13.2 mm on the right and ~13.3 mm on the left, and the third ventricle measured ~16 mm (Figure 2).

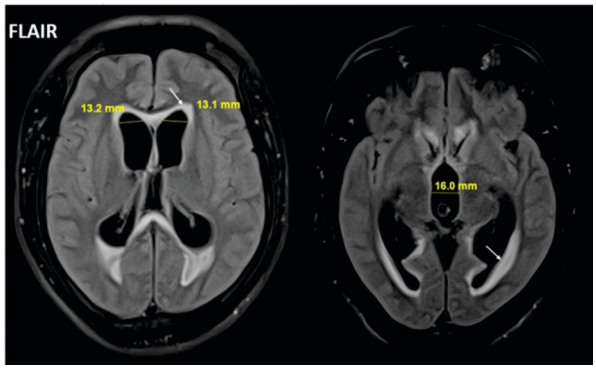


Figure 2: Initial MRI axial FLAIR shows upstream dilatation of both lateral ventricles, the frontal horns measure ~13.2 mm on the right and ~13.3 mm on the left, and the third ventricle measures ~16 mm with periventricular transependymal edema (white arrow).

Within 24 hours of presentation, the patient's sensorium improved spontaneously and a repeat screening MRI was performed on 12th October at 3:15 pm, about 20 hours after the first study, showed that the lesion had been dislodged from the third ventricle into the fourth ventricle. The hydrocephalus had resolved, with the frontal horns now measuring ~4.5 mm on the right and ~5.2 mm on the left, and the third ventricle ~8.2 mm. However trans-ependymal edema was still present (Figure 3).

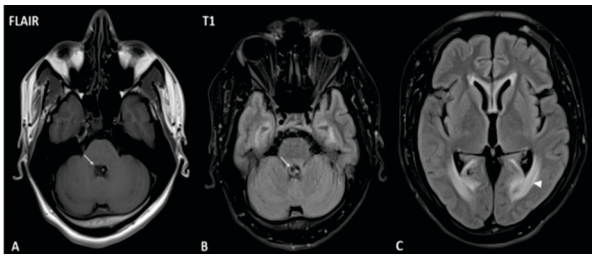


Figure 3: Repeat MRI (12th October, 3:15 pm) axial FLAIR (A) and T1 (B) sequences show the cyst now lying in the fourth ventricle (white arrow), with decompression of the lateral and third ventricles (C). Transependymal edema persists (White arrowhead).

A tiny T1 and T2 hypointense nodule measuring ~2.5 × 3 mm was seen in the subependymal region of the left lateral ventricle. It showed blooming with negative signal on filtered phase images and no post-contrast enhancement, and was considered a calcified granuloma (Figure 4). Both cerebral hemispheres, the cerebellum and the brainstem were normal in signal intensity. The sellar and parasellar regions were normal, no extra-axial collection was seen, major vascular flow voids were maintained, and there was no abnormal parenchymal or leptomeningeal enhancement. Polypoidal mucosal thickening was noted in the right maxillary sinus.

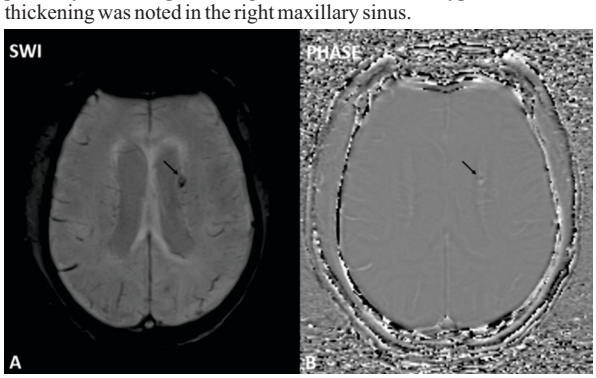


Figure 4: SWI (A) and filtered phase (B) images show a tiny subependymal focus of blooming in the left lateral ventricle with negative phase signal, consistent with a calcified granuloma (black arrow).

The radiological differential diagnosis of a thin-walled cystic lesion in the third ventricle includes colloid cyst, ependymal or neuroglial cyst, arachnoid cyst, epidermoid cyst, cystic neoplasm and other parasitic cysts such as hydatid cyst. The eccentric scolex, absence of diffusion restriction, mobility of the lesion between the two studies and coexisting subependymal calcified granulomas favoured intraventricular neurocysticercosis. The patient was further managed conservatively with oral steroids and anti-parasitic drugs and was suggested follow up.

DISCUSSION

Intraventricular NCC arises when cysticerci reach the ventricular system through the choroid plexus, and the cysts may then remain attached to the ependyma or float freely within the CSF [3,4]. Cysts may be found in the lateral, third or fourth ventricles, and the fourth ventricle and the aqueduct region are commonly involved [4,5]. Free-floating cysts can migrate with changes in head position, CSF pulsation and ventricular pressure, which explains the intermittent and sometimes dramatic clinical course. In the present case, a cyst lodged at the third ventricle–aqueduct junction produced marked hydrocephalus and coma severe enough to require intubation, and a spontaneous shift of the cyst through the aqueduct into the fourth ventricle relieved the obstruction within 24 hours. The clinical recovery parallels the imaging change, with the frontal horns falling from about 13 mm to under 6 mm and the third ventricle from 16 mm to about 8 mm, which supports a mechanical, positional cause for the deterioration [4,5].

CT has limited sensitivity for ventricular cysts, as the cyst fluid is isodense to CSF and the scolex may not be visible [3,6]. MRI is the modality of choice. Cyst fluid is usually isointense to CSF on T1 and T2, but the scolex appears as an eccentric mural nodule that is T1 and FLAIR hyperintense relative to CSF and may enhance, giving the characteristic "hole with dot" appearance. FLAIR and heavily T2-weighted three-dimensional steady-state sequences (CISS/FIESTA) help to delineate the thin cyst wall, and susceptibility-weighted imaging helps to detect calcified granulomas [3,6]. Demonstration of a scolex within a cystic lesion is an absolute diagnostic criterion for NCC in the revised criteria of Del Brutto and colleagues [2]. In the present case, the scolex was identified on the initial study, which allowed a specific diagnosis rather than a presumptive one.

Trans-ependymal edema indicates raised intraventricular pressure with transudation of CSF into the periventricular white matter. Its persistence on the second study, despite normalisation of ventricular size, is not unexpected, since interstitial fluid takes longer to clear than the ventricles take to decompress. Ependymal inflammation related to the parasite may also contribute [3]. The coexisting tiny subependymal foci in the left lateral ventricle are in keeping with calcified granulomas of previous infection. On filtered phase images, calcification and haemorrhage typically show opposite phase polarity, which helped to favour calcification for the first focus; the second focus remained indeterminate between microbleed and calcified granuloma [6]. The presence of calcified lesions indicates earlier disease activity, and the intraventricular cyst may represent a separate or a more recent focus.

The main clinical lesson is that spontaneous recovery does not mean the problem has resolved. A mobile cyst may re-obstruct the aqueduct or the fourth ventricular outlets, and the fourth ventricular position may produce recurrent episodes of the type described by Bruns [4,5]. Endoscopic removal is widely regarded as the treatment of choice for symptomatic intraventricular cysts, as it removes the obstructing lesion and avoids the need for permanent CSF shunting in many patients [7,8]. Albendazole with corticosteroids can be effective in selected patients, but treating a cyst within the CSF pathway medically carries the risk of an inflammatory reaction, cyst swelling and acute re-obstruction, so it needs close monitoring and a plan for urgent CSF diversion [1,7]. CSF diversion may be required if the hydrocephalus recurs or if ependymitis or arachnoiditis develops.

Radiologists play a central role in this setting. The diagnosis may be suspected when obstructive hydrocephalus is unexplained or when ventricular dimensions differ between studies, and the cyst should be actively looked for in every ventricle, including the fourth ventricle and its outlets, when the cause of hydrocephalus is not evident. Comparing serial studies is crucial, since the cyst position, and thus the site of obstruction, can change within hours.

CONCLUSION

Intraventricular neurocysticercosis can present as sudden, life-threatening obstructive hydrocephalus that resolves spontaneously when the cyst moves. In this 25-year-old male, MRI documented a scolex-bearing cyst at the third ventricle–aqueduct junction with hydrocephalus and transependymal edema, and about 20 hours later showed the cyst in the fourth ventricle with resolution of hydrocephalus. Radiologists should look carefully for thin-walled cysts and an eccentric scolex, recognise the ball-valve mechanism and the risk of re-obstruction, and recommend prompt repeat imaging with early neurosurgical involvement for definitive management.

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