

Hypothalamic dysfunction from a third ventricular arachnoid cyst presenting as central precocious puberty: Endoscopic management and clinical outcome

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Abstract

Arachnoid cysts of the third ventricle are rare benign cerebrospinal fluid lesions that may become symptomatic through obstruction of cerebrospinal fluid pathways and compression of hypothalamic-pituitary structures. Central precocious puberty represents an uncommon mode of presentation. We report the case of a 5-year-old boy referred for progressive precocious puberty associated with accelerated growth and pubertal development. Endocrinological evaluation confirmed central precocious puberty, while brain magnetic resonance imaging revealed a large third ventricular arachnoid cyst associated with severe obstructive hydrocephalus. The patient underwent successful neuroendoscopic ventriculocystocisternostomy with an uneventful postoperative course. This case highlights the importance of considering intracranial lesions in the evaluation of central precocious puberty and supports neuroendoscopic ventriculocystocisternostomy as a safe and effective minimally invasive treatment for symptomatic third ventricular arachnoid cysts.

Keywords: Third ventricular arachnoid cyst; Central precocious puberty; Obstructive hydrocephalus; Neuroendoscopy; Ventriculocystocisternostomy; Pediatric neurosurgery.

1. Introduction

Arachnoid cysts are congenital benign lesions formed by accumulation of CSF within arachnoid membrane forming a fluid filled sac. It accounts for 1% of intracranial lesions of which 9% of them occur within the suprasellar region [1]. It is estimated that about 1-2% of the general population are diagnosed with arachnoid cyst and often times found incidentally with male predominance of about 2-4 times the females [2].

Most of the arachnoid cysts are found within the middle fossa then followed by posterior fossa [3]. Our focus is on the suprasellar arachnoid cysts, the primary presentation of our patient presented.

Overall, in most cases arachnoid cysts are asymptomatic. Some arachnoid cysts become symptomatic during the early childhood [4]. Patient with arachnoid cysts may present with signs of increased intracranial pressure, seizures, sudden deterioration either due to rupture of the cyst or bleeding within the cyst and sometimes may form a focal skull protrusion.

As for the suprasellar arachnoid cyst, patient may present with obstructive hydrocephalus from compression of the third ventricle, endocrine dysfunction which occurs in about 60% particularly precocious puberty, head bobbing as well as visual impairment [5].

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2. Case Report

2.1. Clinical Presentation

A 5-year-old boy with no significant past medical history was referred for evaluation of precocious puberty. Several months prior to admission, his parents noticed progressive staturo-ponderal acceleration associated with pubic hair development, enlargement of the testes and penis, facial acne, and voice deepening. Clinical examination revealed a conscious and stable child with marked growth acceleration (27 kg, 128 cm) and macrocephaly (head circumference: 54 cm). Pubertal assessment showed penile enlargement measuring 8 cm, bilateral testicular hypertrophy, and pubic hair development corresponding to Tanner stage G3P3A1. Neurological examination showed no focal deficits. Endocrine investigations revealed a TSH level of 1.26 uIU/ml, an 8 a.m. cortisol level of 10.4 µg/dl, FSH of 1.1 IU/L, basal LH of 0.59 IU/L, and testosterone level of 1.93 ng/ml. The LHRH stimulation test demonstrated a peak LH of 9.57 IU/L and peak FSH of 2.28 IU/L, consistent with central precocious puberty. Brain MRI demonstrated triventricular hydrocephalus associated with a third ventricular lesion consistent with an arachnoid cyst.

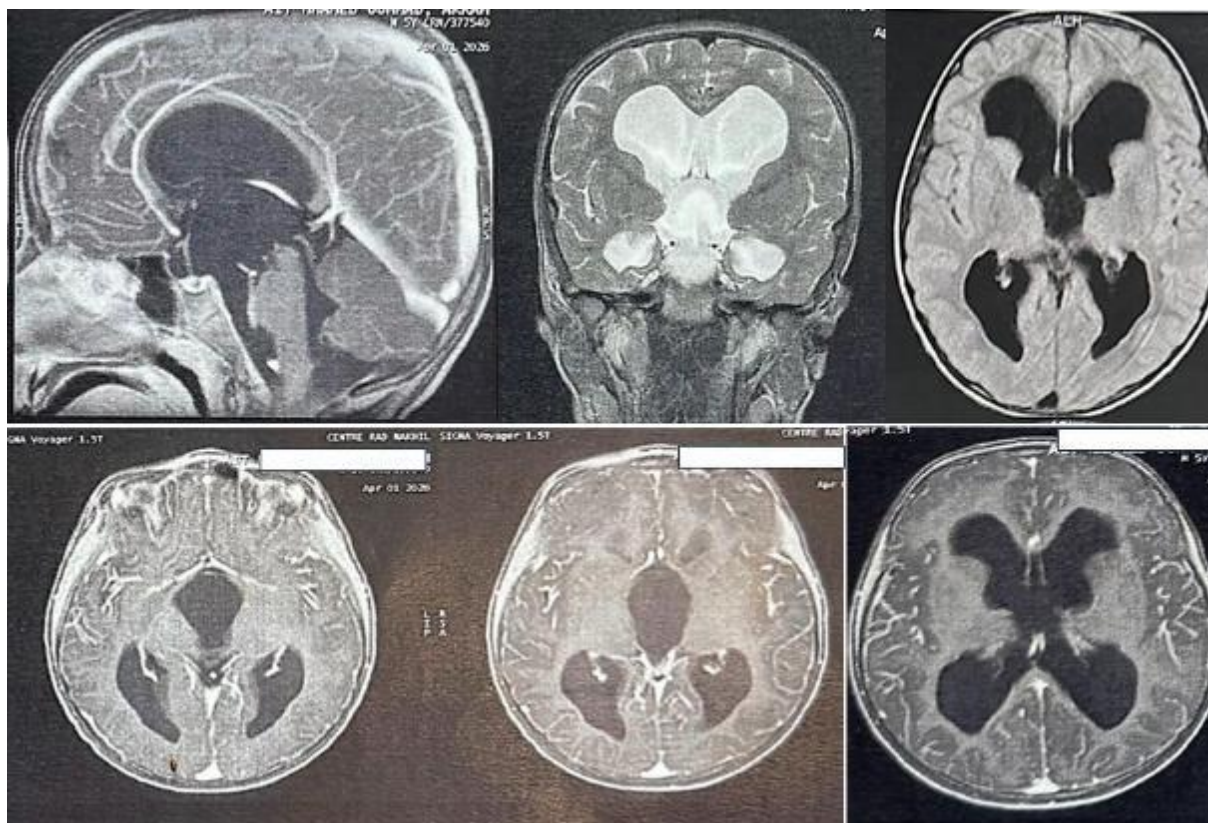


Figure 1 Preoperative brain MRI in axial, sagittal, and coronal planes demonstrating a large third ventricular arachnoid cyst causing marked dilatation of the third ventricle and severe obstructive hydrocephalus

The patient underwent endoscopic ventriculocystocisternostomy under general anesthesia through a right frontal precoronal approach. Endoscopic exploration of the lateral ventricle identified the septal vein medially, the thalamostriate vein laterally, and a translucent cyst membrane obstructing the foramen of Monro. Following bipolar coagulation, the cyst wall was fenestrated and the opening enlarged using a Fogarty balloon catheter, allowing entry into the cyst cavity. The prepontine cistern was subsequently reached, where a thick Lilliequist membrane was identified, opened bilaterally using endoscopic stomy forceps, and further enlarged with a Fogarty balloon catheter. A free pulsatile cerebrospinal fluid flow confirmed adequate ventriculocystocisternostomy (figure 2-3-4-5-6)



Figure 2 Endoscopic view of the lateral ventricle showing the anatomical landmarks of the foramen of Monro. The septal vein is identified medially and the thalamostriate vein laterally. A thin translucent arachnoid cyst membrane is seen protruding through and obstructing the foramen of Monro



Figure 3 Endoscopic coagulation of the arachnoid cyst membrane prior to cyst fenestration. monopolar coagulation was used to devascularize and shrink the cyst wall before opening.



Figure 4 Endoscopic opening of the arachnoid cyst wall followed by enlargement of the fenestration using a Fogarty balloon catheter.

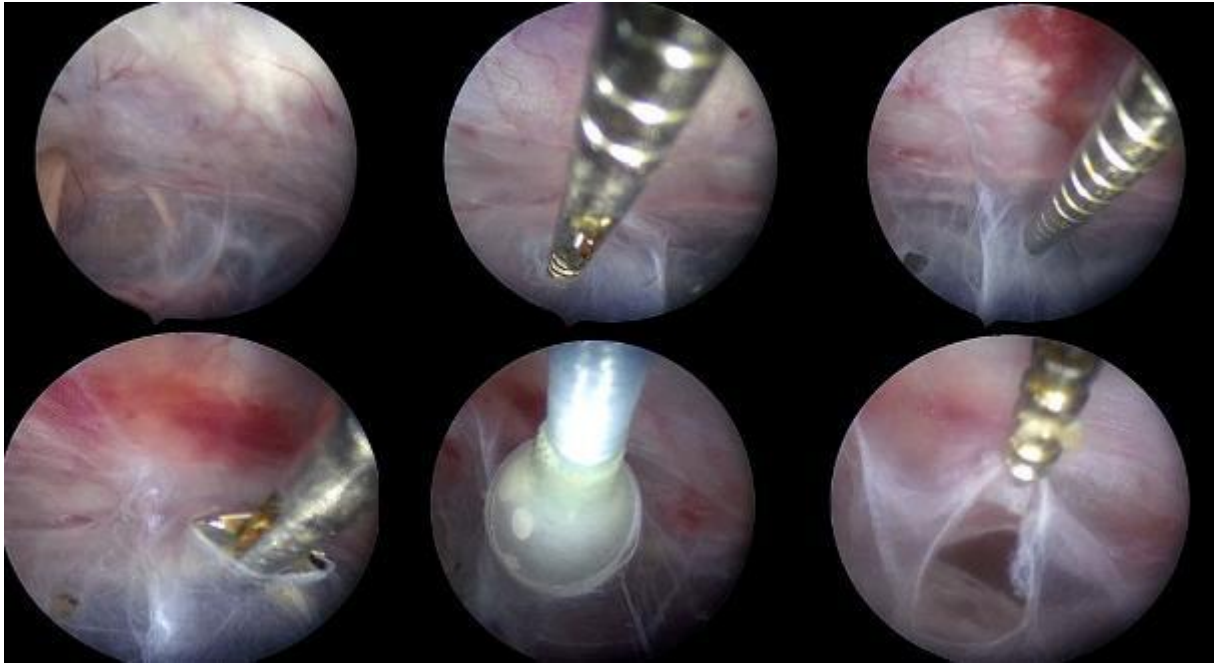


Figure 5 Endoscopic visualization of the basal cistern following traversal of the cyst cavity. The thick Lilliequist membrane was identified, fenestrated bilaterally using stomy forceps, and progressively enlarged with a balloon catheter. A pulsatile cerebrospinal fluid flow through the fenestration confirmed adequate ventriculocystocisternostomy



Figure 6 Endoscopic view showing the basilar artery, bilateral PCAs, PComAs, dorsum sellae, and pituitary stalk through a large and patent stoma, confirming adequate communication with the basal cisterns

The postoperative course was uneventful. The patient remained neurologically stable and was discharged 48 hours after surgery. He was subsequently referred to the Pediatric Endocrinology Department for further management and follow-up

3. Discussion

Giant suprasellar arachnoid cysts (GSAC) are extraparenchymal collections of cerebrospinal fluid (CSF) accounting for between 5% and 12 % of all intracranial arachnoid cysts [6]. When they grow significantly, they can extend into the third ventricle, causing marked ventricular distension and critical structural compression.

These cysts are predominantly congenital and result from an abnormality of the Lilliequist membrane or from dilation of the interpedal cistern [7]. Their expansion is often attributed to a one-way valve mechanism (slit valve), whereby CSF enters the cyst via arterial pulsations but cannot escape, or to active secretion by the cyst wall. This slit valve mechanism has been observed in some cases of congenital suprasellar arachnoid cysts. In these cases, a small opening in the arachnoid wall of the cyst was present directly above the basilar artery, forming a one-way slit valve for the flow of CSF [8].

Many arachnoid cysts are clinically silent and are identified only incidentally [9]. Expansion of suprasellar cysts arachnoid cysts (SACs) is one of the major causes for the progression of clinical symptoms and signs.

Suprasellar arachnoid cysts may present with endocrinopathies months to years prior to development of visual symptoms. Other presenting symptoms of arachnoid cysts include headache, seizures, visual loss, hydrocephalus or focal neurological deficits corresponding to cyst location [10]. SACs can lead to hormonal disturbances as they are located near the hypothalamic-pituitary area [11]. The most common reported endocrine manifestation of SACs is precocious puberty which is suggested to be related to the disruption of posterior hypothalamus inhibitory signals on the gonadotrophs.

Suprasellar arachnoid cysts are benign, probably congenital, cerebrospinal fluid collections with poorly understood pathophysiology. Several theories have been proposed to explain the development of suprasellar arachnoid cysts, including the slit-valve and osmotic gradient mechanism or transudation from remnants of choroid plexus tissue or ectopic glial cells [6]. Nowadays, the slit-valve mechanism is widely accepted. However, these primary cysts are hypothesized to result from unexplained membrane splitting [12]. Secondary cysts may occur due to trauma, surgery, or hemorrhage. Primary cysts begin as an abnormality in the Lilliequist membrane or interpeduncular cisterna. It then enlarges and displaces the floor of the third ventricle, mammillary bodies, optic nerves, and pituitary stalk [7].

Hydrocephalus and dilatation of the third ventricle were present in certain patients with GnRH-dependent sexual precocity and was thought to cause damage to the hypothalamus [13]. In our case, we could not confirm whether the etiology of the patient's precocious puberty was the arachnoid cyst or/and hydrocephalus, because both were found on the brain MRI.

Typically, imaging is used to make the diagnosis of SACs as they are well-defined cysts with a thin wall that displace surrounding structures that follow CSF density on CT and CSF signal intensity on MRI [11]. In our patient, the MRI showed arachnoid cyst (AC) with an enlarged lateral ventricle (LV) and third ventricle that compressed the pituitary gland so that our patient was immediately admitted for endoscopic fenestration of the AC the next day.

Various surgical treatment options were adopted for SACs. Before the widespread use of neuroendoscope, microsurgical excision and cystoperitoneal shunting were the principle surgical treatments [14]. There has been a paradigm shift toward neuroendoscopic management of SACs for the last two decades and today neuroendoscopy became the treatment of choice.

The significant potential morbidity of microsurgical fenestration and the current overall favorable opinion for using a shunt-independent approach encouraged neurosurgeons to search for an alternative procedure. The endoscope is an ideal instrument for exploration of fluid-filled cavities, and the close proximity of suprasellar arachnoid cysts to the basal cisterns make such lesions appropriate for endoscopic treatment. Although flexible endoscopes have been used in their management, most neurosurgeons use rigid endoscopes because they have greater light intensity and superior optics that allow better [8].

Within neuroendoscopic fenestration, various techniques have been analyzed to determine the efficacy and rate of both short-term and long-term complications. The fenestration can be performed only at the apical membrane, usually at the level of the foramen of Monro, between the ventricle and the cyst (ventriculocystostomy or VC), or to basilar fenestration toward the prepontine cistern called VCC [15].

The operative technique for endoscopic fenestration of suprasellar arachnoid cysts involves two types: Ventriculocystostomy and ventriculocystocisternostomy. In our case, we performed a ventriculocystocisternostomy, a technique that involves creating a communication between the cyst and the basal cisterns. This approach is considered when the cyst extends into the interpeduncular or prepontine cisterns. Similar to ventriculocystostomy, the patient is placed under general anesthesia and in a supine position. A burr hole is made in the skull, typically in the frontal or parietal region, and a rigid endoscope is inserted. The endoscope is advanced through the ventricles until it reaches the suprasellar region. In ventriculocystocisternostomy, the fenestration is directed toward the basal cisterns, allowing the cyst fluid to drain into this space. This technique can be more challenging due to the anatomical structures in the region, and careful navigation is required to avoid injury to vital structures [16]. The endoscope is guided to the basal cyst membrane, where a cystocisternotomy is carried out anterior to the basilar artery. At this stage, several fenestrations are made in the avascular areas of the membrane using blunt biopsy forceps and scissors, taking care to navigate around the cranial nerves that exit the brainstem. Once the fenestration is complete, the endoscope is removed, and the dura is closed tightly. Both ventriculocystostomy and ventriculocystocisternotomy are minimally invasive procedures that offer an alternative to open surgery for the management of suprasellar arachnoid cysts. The choice of technique is determined by the location and extent of the cyst and of course the surgeon's experience.

Despite the high success rate of endoscopic fenestration in relieving hydrocephalus and mass effect, the evolution of endocrine abnormalities remains less predictable. Several authors have reported stabilization or partial improvement of endocrine dysfunction following cyst decompression, whereas others observed persistence of precocious puberty despite adequate radiological results, suggesting that hypothalamic injury may become irreversible once established. Therefore, long-term follow-up should include both serial neuroimaging and comprehensive endocrinological assessment. Early recognition and treatment of suprasellar arachnoid cysts may help prevent progressive hypothalamic dysfunction and improve overall functional outcome.

4. Conclusion

Suprasellar arachnoid cysts are cerebrospinal fluid dilations that cause obstructive hydrocephalus, ataxia, and other endocrinological disorders. The treatment requires communication of the cyst to the surrounding ventricles or cisterns. The current literature supports the superiority of the ventriculocystocisternostomy (VCC) over other interventions. Our case presentation highlights the neuroendoscopic VCC in a five-year-old patient and provides a step-by-step illustration of a safe technique and the relevant third ventricular anatomy.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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