

# Pediatric colloid cysts of the third ventricle: management considerations

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## Abstract

**Purpose** Pediatric colloid cysts (CC) have a congenital origin, and yet, there are very few studies focussing exclusively on their occurrence in the pediatric population. Pediatric CC has been associated with more aggressive clinical and radiological patterns than their adult counterparts. In this study, undertaken on children with anterior third ventricular CC, excised using the interhemispheric transcalsal approach, the characteristic clinicoradiological features and management options are studied.

**Methods** Five pediatric patients (aged 16 years or less; mean age 13.8 years; mean duration of symptoms:7.6 months) out of 38 patients with CC operated between 1995 to 2009 were included. The clinical manifestations included those of raised intracranial pressure ( $n=4$ ); exacerbation of occipital headache on reading ( $n=1$ ); secondary optic atrophy ( $n=3$ ); and, drop attacks ( $n=1$ ). On computed tomography scan, the cyst was hyperdense, enhancing in two patients and not enhancing in three patients. All had bilateral lateral ventricular dilatation with periventricular lucency. On magnetic resonance imaging ( $n=3$ ), the cyst was T1 hypointense and T2 isointense in one, hyperintense on both T1 and T2 with a

hypointense capsule and nonenhancing on contrast in one (with a giant colloid cyst), and T1 hyperintense and T2 hypointense in one patient. An interhemispheric, transcalsal trajectory combined with transforminal approach ( $n=3$ ); combined transforminal and subchoroidal approaches ( $n=1$ ); and, interformiceal approach ( $n=1$ ) were used.

**Results** Total excision was performed in four patients. In one patient, a small part of capsule was left attached to thalamostriate vein. Symptoms of raised intracranial pressure showed improvement in all the patients with resolution of hydrocephalus. There was no tumor recurrence at follow-up.

**Conclusions** Pediatric colloid cysts are rarer than their adult counterparts due to their late detection only after manifestations of raised intracranial pressure, visual or cognitive dysfunction or drop attacks occur. Their radiological appearance varies depending upon the amount of mucoid content, cholesterol, proteins, and water content. The fast development of clinical manifestations in children may be related to rapid enlargement of cyst due to higher water content within them. The transcalsal approach is the “gold standard” of surgery and usually ensures gratifying and lasting results.

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## Introduction

Colloid cysts, benign neoplasms located in the anterior third ventricles, are usually detected following bilateral foramina of Monro obstruction [5, 17]. This may lead to clinical manifestations of raised intracranial pressure, sudden drop attacks due to intermittent cerebrospinal fluid pathway obstruction, or amnesia due to forniceal compres-

sion [2, 9, 11, 16, 27, 31, 57]. In 1858, they were first described in an autopsy study [60]. Dandy performed the first successful resection in 1921 [13]. Despite their benign histological features, they may be associated with sudden death in children due to development of acute hydrocephalus and brain herniation [9, 48, 57]. Pediatric colloid cysts have been associated with more aggressive clinical and radiological patterns than their adult counterparts [4, 40, 43]. Microsurgical resection, usually utilizing the transcallosal approach, has become the “gold standard” in the management of third ventricular colloid cysts [7, 19, 26]. In this study, undertaken on pediatric patients (aged 16 years or less) with anterior third ventricular colloid cysts and operated using the interhemispheric transcallosal approach, the characteristic clinicoradiological features, unusual radiological presentation, and management options are studied and pertinent literature reviewed.

### Material and methods

Five pediatric patients (aged 16 years or less) with a third ventricular colloid cyst (out of a total of 38 patients) operated between 1995 to 2009 were included in this study. Their mean age was 13.8 years. There were three male and two female patients. Four patients presented with manifestations of raised intracranial pressure such as severe headache and vomiting with or without diplopia. Of these one patient became drowsy but arousable prior to his surgery. The patient who did not have manifestations of raised intracranial pressure still had exacerbation of his occipital headache on reading with increase in lacrimation. There was progressive diminution of vision with secondary optic atrophy in three patients. One patient had a history of drop attacks with sudden fall on the ground without loss of

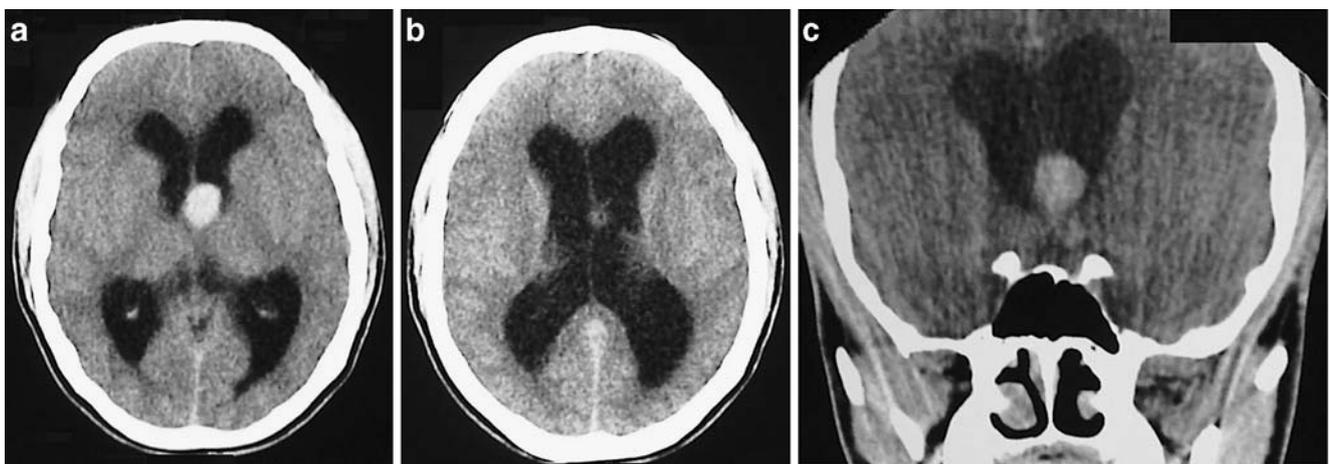
consciousness with spontaneous recovery. One of the patients also had an unexplained left facial numbness, decreased facial sensations in V1, and V2 distribution with decreased corneal reflex that did not improve following surgery. The mean duration of symptoms was 7.6 months.

The radiological investigations revealed bilateral lateral ventricular dilatation with periventricular lucency due to bilateral foramen of Monro obstruction. On computed tomography (CT) scan, the five patients had a hyperdense rounded mass in the anterior third ventricular region that was enhancing in two patients and not enhancing in three patients. On magnetic resonance (MR) imaging done in three patients, the mass was hypointense on T1 and isointense on T2 weighted images in one patient, hyperintense on both T1 and T2 with a hypointense capsule and nonenhancing on contrast in a patient (with a giant colloid cyst reaching up to the corpus callosum), and hyperintense on T1 and hypointense on T2 in one patient.

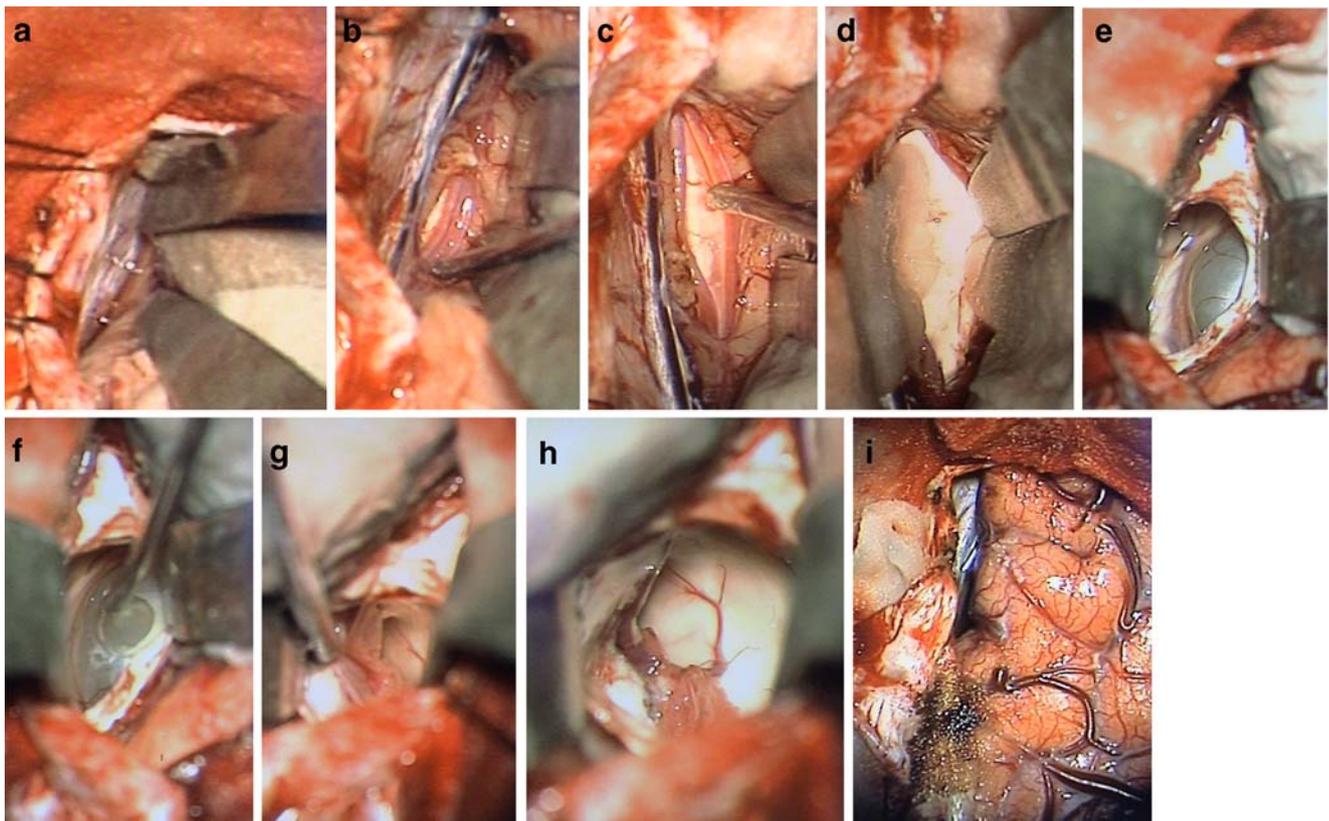
A right frontal parasagittal craniotomy and interhemispheric, transcallosal approach was adopted in all the patients [7]. The access from the lateral ventricle to the third ventricle was gained using the transforminal approach in three patients; and, combining the transforminal and subchoroidal approaches in one patient [53] (Figs. 1a, b, 2a-i, 3a, b, and 4). In one of the patients with a giant colloid cyst that was stretching the body of the corpus callosum and also bilateral fornices, it was possible to directly approach the giant cyst in the third ventricle between the fornices by using the interforniceal approach [6] (Fig. 5a, b).

### Results

Total excision was performed in four patients. In one patient in whom both the transforminal and subchoroidal



**Fig. 1** a and b Contrast enhanced axial CT scan and c Coronal CT image showing a hyperdense enhancing colloid cyst causing bilateral foramen of Monro obstruction with associated hydrocephalus and periventricular lucency



**Fig. 2** **a** The patient was positioned supine with head end elevated 60° and slightly flexed. By gentle extension of the head position relative to the torso, it was possible during surgery to change the line of sight in the ventricular system from an anterior to posterior direction using the small interhemispheric exposure. An intraoperative image showing that after the frontal parasagittal craniotomy and dural reflection towards the superior sagittal sinus, the frontal lobe is gently retracted laterally exposing the interhemispheric fissure and falx cerebri. **b** Both the anterior cerebral arteries are exposed traversing over the corpus callosum. **c** The two anterior cerebral arteries are retracted towards their respective sides exposing the corpus callosum. **d** The corpus callosum is incised to gain access to the lateral ventricle. **e** The left lateral ventricle is exposed showing the colloid cyst at the foramen of Monro stretching the fornix. The site of the lateral

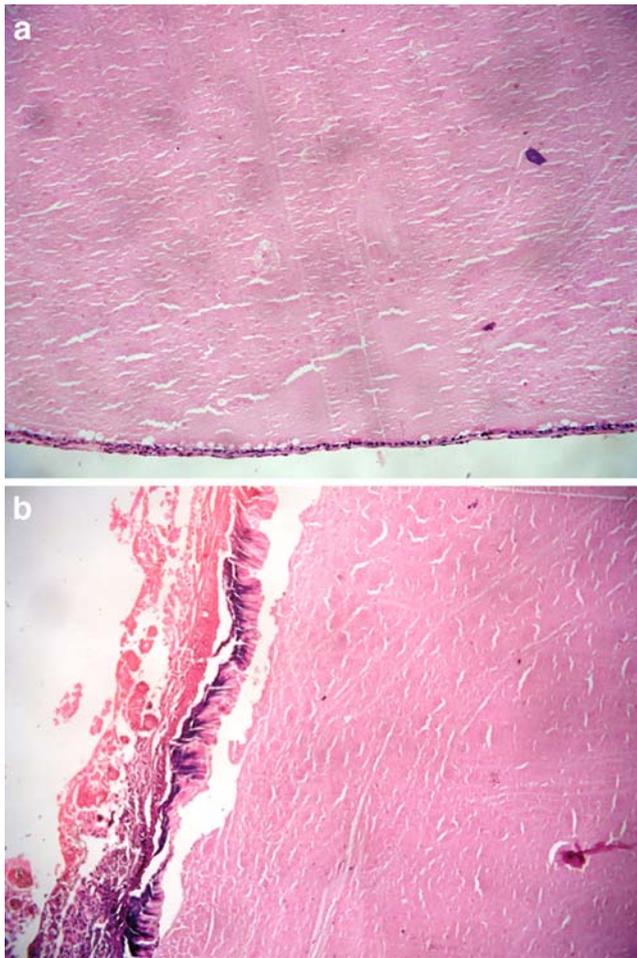
ventricular opening may be determined by observing the relative positions of the choroid plexus and the thalamostriate vein. **f** The cyst is fenestrated revealing the mucoid material. **g** The entire cyst wall is excised showing the floor of third ventricle. **h** After cyst capsule removal, the thalamostriate vein and the choroid plexus are seen at the foramen of Monro. **i** The surface of the well-relaxed brain after excising the colloid cyst. It is possible to gently lift up the cortical vein from the brain surface by dissecting within its arachnoid plane. This facilitates a few additional millimeters of interhemispheric exposure without coagulating the vein. Two consecutive cortical veins should never be coagulated. In case a cortical vein needs coagulation, the retractor should not be placed over the brain surface being drained by the vein to avoid venous infarction

approach was used to access to the third ventricle from the lateral ventricle, a small part of the remaining capsule was left attached to the thalamostriate vein. The symptoms of raised intracranial pressure showed improvement in all the patients. One patient, however, had persistence of sixth nerve palsy and the other, unilateral facial hypoesthesia. In two patients, there was persistent subgaleal cerebrospinal fluid collection that required repeated lumbar punctures for 3 days and acetazolamide (250 mg tds) administration. Hydrocephalus resolved completely following surgery in all the patients and there was no recurrence of tumor at a follow-up of 1.5 to 3 months. The histopathology of the lesion showed the cyst wall with mildly anisomorphic cuboidal to ciliated columnar lining epithelium and enclosing an amorphous eosinophilic material (Fig. 3a, b)

## Discussion

### Incidence

Colloid cysts represent 0.5-1% of intracranial neoplasms and 55% of third ventricular lesions [5, 16, 17]. Its incidence in children is rarer [4, 40, 43]. In a study from Bangalore, India of 50 patients having a third ventricular colloid cyst, 18 (28%) patients were below 18 years [43]. A study from Saudi Arabia reported 16.3% patients below 18 years of age [4]. In our study, five (13.16%) patients were below 16 years of age out of a total of 38 patients seen during the same time frame. Our incidence of pediatric colloid cysts is lesser when compared to the international literature mainly due to the fact that we confined ourselves



**Fig. 3** **a** and **b** Photomicrograph showing section from the cyst wall with mildly anisomorphic cuboidal to ciliated columnar lining epithelium and enclosing an amorphous eosinophilic material (Haematoxylin and Eosin X 100 Fig. 3a), X 200 (Fig. 3b)

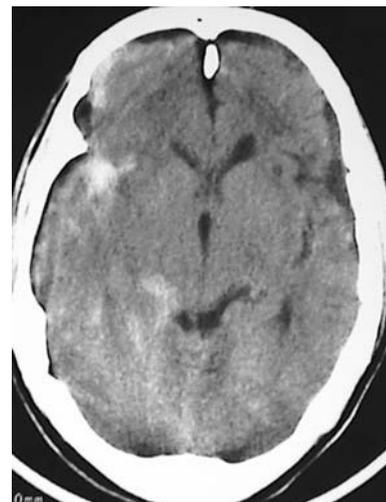
to the patients in the age group of 16 years and younger whereas the reported series have included patients up to the age of 18 years [40, 43].

#### Location and development

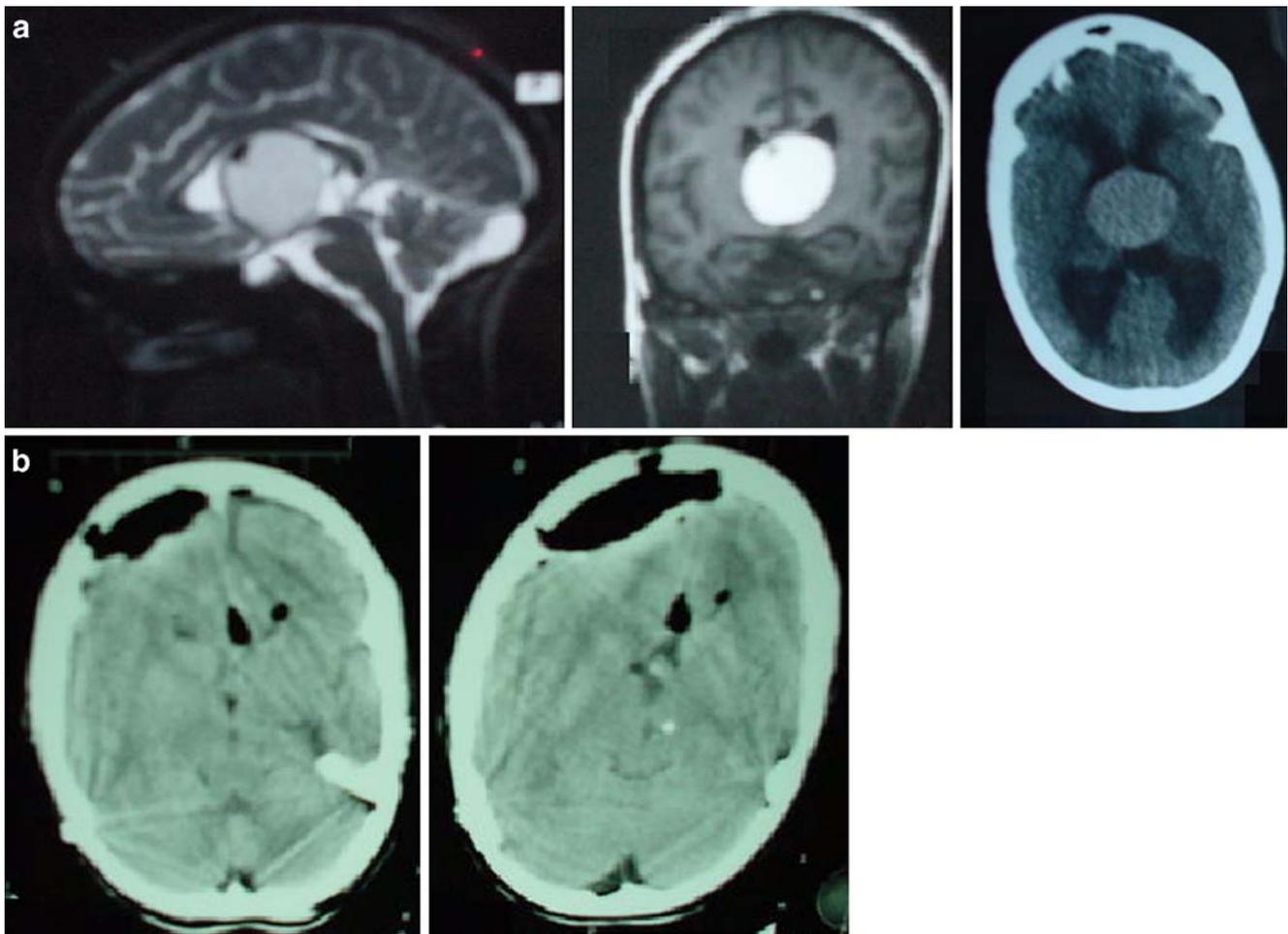
All our pediatric patients had colloid cyst at the conventional location in the anterosuperior aspect of the third ventricle. Even when small in size, their strategic location blocks both foramen of Monro leading to bilateral lateral ventricular dilatation. Occasionally, however, they may be situated a little posterior to the foramen of Monro in the body of the third ventricle maintaining the patency of the foramen and the cerebrospinal fluid pathway. A search of the international literature revealed quite a ubiquitous distribution of colloid cysts in several unusual locations such as the optic chiasma, sellar area, cerebral convexity, lateral ventricle, septum pellucidum, posterior third ventricular region, fourth ventricle, brain stem, subarachnoid space, and even the spine [24].

Its developmental origin is well-established and therefore, its relatively lower incidence in children as compared to the adult patients is rather surprising [4, 40, 43]. Perhaps, the extremely slow growth potential leads to its late detection only when it blocks the foramen of Monro leading to obstructive hydrocephalus and manifestations of raised intracranial pressure; or, when it becomes large enough to exert mass effect on the surrounding structures such as the fornix leading to memory loss [24]. Its origin is believed to be from the diencephalic vesicle or from the persistence of the embryonic paraphysis. Paraphyseal rudiments normally disappear by total degeneration at 3.5 months of age. Colloid cysts are proposed to be arising from the nondegenerated part of these rudiments [21]. Shuangshoti and Ibrahim, on the other hand, suggested that colloid cyst is a derivative of the neuroepithelium [29, 54] while Tsuchida et al., Mackenzie et al., and Ho and Garcia proposed that it is an endodermal derivative [21, 28, 41]. Therefore, until now, its exact origin is a matter of controversy. The presence of intrasellar colloid cysts supports the theory that not only paraphysis but also the neuroepithelium takes part in its formation.

Colloid cysts usually have a sporadic occurrence but there are a few instances where familial occurrences have been seen with first degree relatives having the same lesion [3, 47, 51]. In consonance with its congenital origin, there may be other associated congenital abnormalities like frontal encephalocele, agenesis of corpus callosum, lipoma, craniopharyngioma, neurofibromatosis, and capillary hemangioma of the choroid plexus [24]. One should be particularly aware of the coexistence of an associated aqueductal stenosis while dealing with anterior third ventricular colloid cysts. In the latter case, despite success-



**Fig. 4** Postoperative image showing complete excision of the colloid cyst



**Fig. 5** **a** T2 weighted sagittal and T1 weighted coronal MR image showing a giant colloid cyst stretching the corpus callosum. The lesion is hyperintense on both T1 and T2 images with a hypointense rim of capsule. It was nonenhancing on contrast. The axial CT image showing the hyperdense colloid cyst causing bilateral foramen of Monro obstruction, the characteristic splaying of the posterior end of

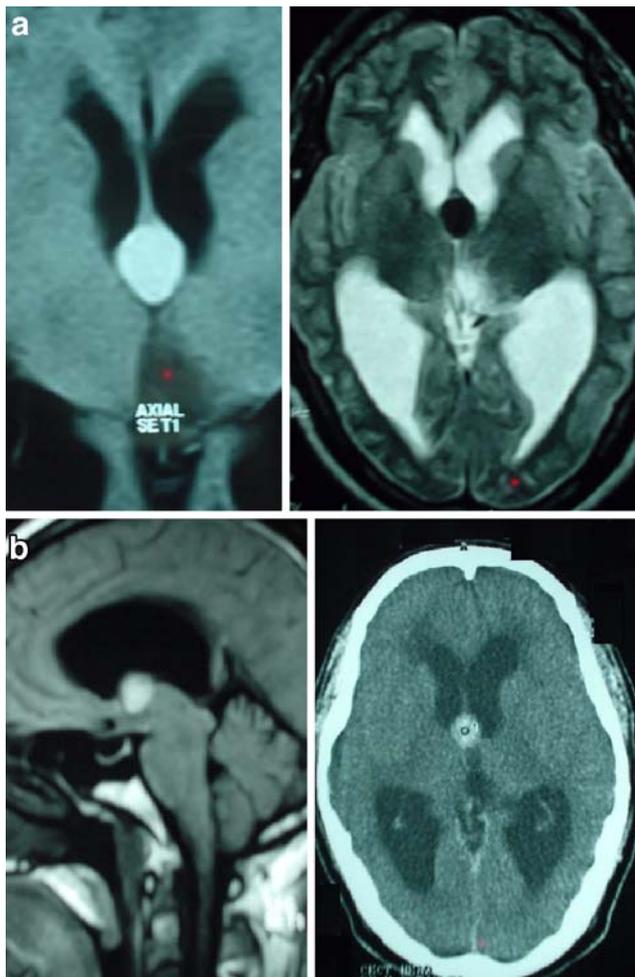
the frontal horns, gross hydrocephalus with periventricular lucency, and obliteration of the sulci and gyri. **b** The axial CT scan showing the postoperative image after excision of the colloid cyst by the interhemispheric transcallosal interforaminal approach. The ventricles are collapsed. There is air along the interhemispheric fissure, in the lateral ventricle, and in the extradural space below the bone flap

ful removal of the colloid cyst, obstructive hydrocephalus may persist mandating a cerebrospinal fluid diversion procedure.

#### Clinical presentation

In our study, the mean age of presentation was 13.8 years which is nearly the same as that reported in other studies of pediatric colloid cysts (where the mean age was reported to be 13 to 14 years) [4, 40, 43]. In contrast, medical literature indicates that 80% of patients with colloid cysts are between 30 and 60 years of age [25]. The male predominance in our pediatric patients (male: female ratio = 3:2) has also been noted in other series (male: female ratio = 2:1 and 4:3, respectively) [4, 40, 43]. The mean duration of symptoms in our study was fairly short, that is 7.6 months,

considering that a colloid cyst is a benign developmental tumor with a low growth potential. An even shorter mean duration of symptoms (5.3 months) has been reported in the literature. A likely explanation for the rapid development of the clinical manifestations in children having a colloid cyst when compared to the adult population is the high water content in the cyst in children that is responsible for its rapid enlargement. Once cerebrospinal fluid pathway gets obstructed, manifestations of raised intracranial pressure rapidly progress. In children, it is also rare to pick up early clinical manifestations. The underlying pathology, therefore, becomes evident only when the child already has a fully developed hydrocephalus. The late detection, therefore, leads to a rapid progression of the clinical picture in pediatric patients. The most common clinical presentation of pediatric colloid cysts include the nonspecific syndrome



**Fig. 6** **a** The T1 coronal MR image showing the uniformly hyperintense colloid cyst situated at the foramen of Monro. The T2 weighted axial MR image showing its uniformly hypointense nature. **b** In another patient, the T1 weighted axial MR image showing the hyperintense colloid cyst. The axial CT image showing the hyperdense colloid cyst with periventricular lucency

of increased intracranial pressure without localizing sign and progressive or fluctuating dementia with or without headache, associated at times with raised intracranial pressure [2, 9, 11, 16, 27, 57]. Four of our patient presented with symptoms of raised intra cranial pressure and decreased vision with secondary optic atrophy; one of them also had dementia [2, 27]. Occasionally paroxysmal attacks with complete recovery in-between may also be observed. The latter presentation that was seen in one patient in our series is due to sudden obstruction of the foramen of Monro by the pedunculated lesion leading to the rapid development of hydrocephalus. It is also proposed that loss of tone and drop attacks are due to the pressure of the rapidly expanding lateral ventricular walls on the white matter tracts of the centrum semiovale that are responsible for the lower limb function, in close vicinity to the ventricular walls. The momentary brain stem distortion and disruption

of the physiological function of the reticular activating system may be responsible for the transient loss of consciousness. The spontaneous recovery of manifestations is brought about by the relief of cerebrospinal pathway obstruction once the pedunculated tumor again moves away from the foramen of Monro [9, 48, 57]. Some of the other unusual presentations reported in literature include symptoms due to hemorrhage within the cyst or rupture of the cyst due to vigorous head movements, cerebrospinal fluid rhinorrhea, hypopituitarism, diabetes insipidus, aseptic meningitis, quadriplegia, and spasmodic torticollis [24].

### Radiology

The computed tomographic images in all our five patients showed a hyperdense tumor. It was enhancing in two patients and not enhancing in three others. A few studies have suggested that the density pattern of a colloid cyst on CT scan depends upon the relative concentrations of the cholesterol and protein in the cyst contents. The more the amount of cholesterol content, the more the hyperdensity is evident on CT scan [16, 33, 42]. The enhancement seen in two patients was possibly due to the enhancement of the thick cyst wall. The mucoid content of the cyst does not enhance. In the case of scanty contents in a small cyst with thickened walls, the whole cyst appears to be enhancing. The T1 and T2 weighted MR images in our patients showed a variable intensity pattern (Figs. 1a, b, 5a, and 6a, b). The mass was hypointense on T1 and isointense on T2 weighted images in one patient, hyperintense on both T1 and T2 with a hypointense capsule, and nonenhancing in contrast in a patient (with a giant colloid cyst reaching up to the corpus callosum) [24, 34], and hyperintense on T1 and hypointense on T2 in one patient. Again, the variability in the protein, cholesterol, and water contents of the cyst may be responsible for the ensuing MR picture [16, 24, 33, 42, 52]. The predominance of cholesterol content in the cyst gives the colloid cyst its characteristic MR appearance of hyperintense on T1 and hypointense on T2 weighted image. Some of the colloid cysts appear homogenous in intensity while others are more heterogenous in character occasionally with intracystic fluid levels. Colloid cysts have been known to change their intensity characteristics over time without any interventional procedure being performed on them [33]. The principal constituent of a colloid cyst is mucin (an intracellular proteinaceous carbohydrate) which has not been found to undergo a serial change with time. The change in intensity characteristics of colloid cysts, therefore, may be due to acute hemorrhagic or xanthogranulomatous (chronic inflammation typically involving giant cells, mediated by foamy macrophages and large number of fibrin laden macrophages that occasionally replace the entire epithelial lining) changes [23, 45] and also shrinkage

or enlargement of cyst due to osmotic fluxes from the ventricular cerebrospinal fluid (the latter resulting in a lower protein to water ratio). Pollock et al. reported a trend towards hyperintensity on T2 weighted MR images in children due to the presence of larger water content [49, 50]. It is believed that cysts that are hyperintense on T2 weighted MR images tend to grow rapidly. Aspiration of these cysts is easier due to their larger water content. Similarly, hyperdense cysts are difficult to aspirate when compared to iso-/hypodense cysts [16]. Hyperintensity on both T1 and T2 weighted images may be attributed to hemorrhagic fluid within a colloid cyst [33].

### Surgical approaches

We utilized the transcallosal–transventricular approach to excise the lesion [7, 19, 26] (Fig. 2a–i). In four patients in the series, the colloid cyst was positioned at the foramen of Monro and could easily be accessed using the transforaminal approach [7]. In a patient with a giant colloid cyst, the cyst was large enough to cause stretching of the corpus callosum and access to the third ventricle was possible directly using the interforaminal approach between the two stretched fornices [6] (Fig. 5a, b). In one other patient, the pedunculated cyst retracted posterior to the foramen of Monro and was not completely accessible using the transforaminal approach (Table 1). In this patient, the third ventricle was accessed from the lateral ventricle by the subchoroidal approach working between the choroid plexus superiorly and the thalamus inferiorly [53]. We utilized the transcallosal approach in all of our pediatric patients since the approach does not cause any cortical breach (which may precipitate epilepsy) and is the shortest approach to the lateral and third ventricle. By gently decreasing the height of the patient's head, it is possible to change the trajectory of approach from an anterior approach to a posteriorly directed one. Venous infarction due to compromise of cortical bridging veins draining into the superior sagittal sinus was avoided by: a gentle cortical retraction, cerebrospinal fluid drainage from the interhemispheric fissure and the ventricles (the latter after the corpus callosotomy), mobilization of bridging vein within its arachnoidal covering from the cortical surface below, constant irrigation of the veins to retain their elasticity, avoiding coagulation of two contiguous veins to gain a wider corridor, and avoiding retractor placement at the site of drainage of a coagulated vein whenever venous coagulation became unavoidable [16, 20, 30]. When it was impossible to separate the vein that was entering the dura before its entry into the sagittal sinus, a small dural sleeve was left attached to the vein. A larger anteroposterior craniotomy ensured utilization of multiple corridors between the bridging cortical veins. An anterior disconnection syndrome is an extremely rare possibility

while using the transcallosal approach provided the callosotomy is placed anteriorly and kept limited to approximately 2 cm [16, 30]. Once the ventricular cerebrospinal fluid gets drained, the ensuing brain relaxation permits a spontaneous retraction of the callosotomy edges. Hence, a longer callosotomy is not required [7, 30]. One of our patients had a mild memory loss of recent events while the remote memory was preserved. Perhaps the forniceal compression by the pressure of the colloid cyst was responsible for this amnesia which did not recover completely despite the cyst removal [2, 27]. The genu of the internal capsule lies in close proximity to the lateral ventricular wall. While placing the retractors, one has to be careful that the lateral ventricular walls were not retracted forcefully to avoid the danger of contralateral hemiparesis [7]. Finally, hemorrhage from the vascular pedicle of the colloid cyst that may withdraw out of sight from the surgeon while utilizing the transforminal corridor to gain access from the lateral to the third ventricle; from the vascular choroid plexus; or, from injury to the ventricular ependyma should be meticulously avoided. In one of our patients, a small part of the capsule was left attached to the thalamostriate vein. The coagulation of the latter vein may result in venous infarction in the striatal-internal capsular region with consequent hemiparesis and often alteration of sensorium [7]. The surgical approaches have been summarized in Table 2.

The transcortical-transventricular approach has the advantage of avoiding the bridging veins [22, 55]. It was, however, not used in any of our patients as the cortical incision may lead to the development of epilepsy, the range of movement becomes restricted by the margins of the corticectomy, the retraction of the cortical margin may precipitate edema, and the obliquity of approach makes the surgeon's orientation within the ventricular system more difficult as compared to the transcallosal approach. In none of our patients was the cyst sufficiently anteriorly placed in the third ventricle to adopt a subfrontal lamina terminal approach. The latter in any case can only be utilized for decompression of a colloid cyst and not for its complete removal. A rarely utilized approach that may be useful in cases of small cysts without ventricular dilatation is the infratentorial, supracerebellar approach with the patient in the sitting, head flexed position [39]. The approach being extremely tedious, requiring traversing a long distance within the third ventricle from the posterior third ventricular region to the foramen of Monro with its trajectory surrounded by vital neurovascular structures was not considered in our patients. The minimally invasive approaches like stereotactic aspiration, endoscopic removal and endoscopic assisted microsurgical removal are increasingly being used to excise third ventricular colloid cysts. The procedures are facilitated by the lateral ventricular

**Table 1** The spectrum of pediatric patients with colloid cyst

Serial number	Name, age, sex	Clinical history	Neurological examination	Radiological features	Surgery and extent	Postoperative status
1	SK 16/m	Headache vomiting and diplopia: 6 months	Vision bilateral 6/12, fundus papilloedema, right sixth nerve paresis	CT: small hyperdense, enhancing mass obstructing foramen of Monro with bilateral lateral ventricular dilatation	Interhemispheric transcallosal transforaminal approach and total excision	No deficits
2	DS 15/m	Holocranial headache and vomiting: 9 months, one drop attack: 4 months back, progressive diminution of vision: 4 months	Memory impaired, vision right: 6/24, left: finger counting at 1 m. Bilateral secondary optic atrophy	CT: hyperdense nonenhancing cyst obstructing foramen of Monro causing bilateral lateral ventricular dilatation	Interhemispheric transcallosal transforaminal approach and total excision	No deficits. Subgaleal collection
3	AS 16/f (Figs. 1a, b, 2a-i, 3a, b, and 4)	Bifrontal headache: 7 months Left facial numbness: 1 month	Visual acuity bilateral 6/9, Fundus papilloedema, hypoesthesia in left V1 and V2 distribution, corneal reflex diminished	CT-enhancing globular mass at the level of foramen of Monro with dilatation of lateral ventricles. MR: T1 hypointense, T2 isointense lesion	Interhemispheric transcallosal transforaminal approach and total excision	No headache. Left facial hypoesthesia persisting
4	BC 14/m (Fig. 5a, b)	Bilateral ocular pain and lacrimation off and on: 1 year, occipital headache on reading: 1–2 years	Vision bilateral 6/6, no papilloedema	CT: hyperdense nonenhancing colloid cyst causing bilateral foramen of Monro obstruction, splaying of posterior parts of the frontal horns, gross hydrocephalus with periventricular lucency and obliteration of sulci and gyri MR: giant colloid cyst stretching corpus callosum, hyperintense on both T1 and T2 images with a hypointense capsule and non enhancing on contrast	Interhemispheric transcallosal interformiceal approach and total excision	No deficits
5	BS 8/f (Fig. 6a, b)	Headache and vomiting : 4 months, drowsy: 5 days	Preference to sleep but arousable on verbal commands, vision 6/6, fundus bilateral mild disc pallor, mild ataxia	CT: rounded hyperdense nonenhancing mass causing obstruction at foramen of Monro with bilateral lateral ventricular dilatation MR: T1 hyperintense and T2 hypointense, spherical lesion in the anterior part of third ventricle with bilateral lateral ventricular dilatation	Interhemispheric transcallosal transforaminal and subchoroidal approach. Small part of capsule left attached to thalamostriate vein	No deficits

**Table 2** Surgical approaches for excision of a third ventricular colloid cyst

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A. Microsurgery:
Transcallosal–transventricular approach-
> Transforaminal
> Interformiceal
> Subchoroidal
Transcortical-transventricular approach-
> Transforaminal
> Subchoroidal
Subfrontal approach-
Infratentorial supracerebellar approach-
B. Stereotactic guided microsurgical removal
C. Frameless stereotactic guided microsurgical removal
D. Endoscopic removal of cyst
E. Endoscopic assisted microsurgical removal

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dilatation associated with the foramen of Monro obstruction due to the colloid cyst [1, 8, 10, 12, 14, 15, 18, 25, 32, 35–38, 46, 56, 58]. Stereotactic and endoscopic drainage of cyst contents facilitate removal of its capsule that collapses away from the surrounding structures. These techniques, therefore, attain excision of the cyst utilizing only a small burr hole or craniotomy. However, complete cyst removal with its capsule may occasionally not be possible utilizing these techniques. The operative view through the endoscope is two dimensional as opposed to the three dimensional view obtained via the operative microscope. The field of view through the endoscope is restricted to the small area just distal to it. The anatomical structures in the vicinity of the traversing scope are not visible to the surgeon. Injury to the fornix, hemorrhage from the choroid plexus, or the pedicle of the colloid cyst may, therefore, occur. The frameless stereotactic approach was not preferred as this is equipment intensive and requires a cortical incision.

Small incidental colloid cysts have often been reported. Patients having a small cyst with neither symptoms nor hydrocephalus, when treated conservatively, were asymptomatic at a long follow-up [16]. Pollock and Huston retrospectively reviewed the cases of third ventricular colloid cysts observed during the modern neuroimaging era to determine their natural history. The incidences of symptomatic progression related to the cyst in their study were 0%, 0%, and 8% at 2, 5, and 10 years, respectively. No patient died suddenly during the follow-up interval. Two (6%) of 34 patients in whom follow-up imaging was performed either exhibited cyst growth (one patient) or experienced hydrocephalus (one patient) at a mean of 41 months after diagnosis. This study concluded that patients in whom asymptomatic colloid cysts are diagnosed

can be cared for safely with observation and serial neuroimaging. If a patient becomes symptomatic, the cyst enlarges, or hydrocephalus develops, prompt neurosurgical intervention is necessary to prevent the occurrence of neurological decline from these benign tumors [49, 50]. Maqsood et al., on the other hand, had a slightly differing opinion when focusing on the mortality in patients less than 18 years of age with a colloid cyst. In a review of the studies reported in the literature, they found that most of the patients who died had a background history of headaches, had arrived in altered sensorium, and the majority had associated hydrocephalus. They reported a risk of acute deterioration in symptomatic patients with colloid cyst as high as 34% [43]. The autopsy findings conducted for finding out the cause of sudden death in children often discovered colloid cysts retrospectively to be the incriminating pathology [40, 44]. Thus, it was recommended that small colloid cysts without hydrocephalus in asymptomatic patients may be managed conservatively; however, surgery is advised when follow-up is likely to be irregular [43]

The hydrocephalus resolved in all our patients following excision of the cyst. Therefore, a cerebrospinal fluid diversion was not required. Occasionally, there may be associated aqueductal stenosis or arachnoidal granulation block (either due to congenital malformation or as a result of ventricular hemorrhage during surgery) that may lead to persistence of hydrocephalus despite successful establishment of the cerebrospinal fluid pathway at the level of the foramen of Monro following the colloid cyst excision. This may mandate an additional cerebrospinal fluid diversion procedure. In case a mild intraventricular hemorrhage ensues during cyst excision, an external ventricular drain may be placed for approximately 3 days. This ensures drainage of the hemorrhagic cerebrospinal fluid into a closed drainage bag that prevents its accumulation within the ventricular system; monitoring the extent and duration of hemorrhage by a regular inspection of the drainage bag; the maintenance of a low ventricular cerebrospinal fluid and brain pressure in the immediate postoperative period; and, the feasibility of ventricular irrigation and intraventricular antibiotic instillation. Fortunately, none of our patients required a postoperative closed ventricular drainage.

To conclude, pediatric colloid cysts have unique diagnostic and operative considerations. Although they may be present for a long time, they are usually detected when they start causing rapid or slowly progressive obstruction of the cerebrospinal fluid pathway. The fast development of the clinical manifestations in children having a colloid cyst when compared to the adult population may often be due to the high water content in the cyst in children that is responsible for its rapid enlargement. Surgical excision of these benign lesions, although technically demanding, usually ensures a gratifying result.

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