



CASE REPORT & LITERATURE REVIEW

The Third Ventricle Colloid Cyst Spontaneous Rupture Phenomenon: A Rare Clinical Observation and World Literature Review

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Abstract

Background: Colloid cysts of the third ventricle are rare congenital benign lesions that can cause acute occlusion of CSF pathways with the risk of a fatal outcome. Up to 6% of colloid cysts are asymptomatic, incidental findings. Colloid cysts tend to slowly increase in size over a long period of time. Management of patients with colloid cysts of the third ventricle depends on clinical and radiological findings. Spontaneous shrinkage (rupture) of the colloidal cyst is an extremely rare phenomenon, described only in 8 cases worldwide.

Methods: This article presents the clinical observation of a 28-year-old woman with a symptomatic colloid cyst of the third ventricle, which decreased in size by almost 3 times after having had mild traumatic brain injury, with subsequent regression of occlusive hydrocephalic symptoms.

Results: During the follow-up period (9 years), there is an extremely slow increase in size of the colloid cyst and a slight increase in the Evans index without the appearance of any clinical symptoms.

Conclusions: Although the cyst is currently asymptomatic, considering its dynamics, this patient may require surgical treatment in future. Spontaneous colloid cyst regression phenomenon cannot be considered as an alternative to surgical treatment.

Keywords

Colloid cyst, Third ventricle, Natural history, Spontaneous rupture, Rare phenomenon

Abbreviations and Acronyms

CC: Colloid Cyst; WHO: World Health Organization; CSF: Cerebrospinal Fluid; MRI: Magnetic Resonance Imaging; CT: Computer Tomography; TBI: Traumatic Brain Injury; EI: The Evans Index

Introduction

Third ventricular colloid cysts are congenital, non-neoplastic cystic formations typically located in the anterior-superior part of the third ventricle under the fornix columns near the interventricular foramina (of Monro). Colloid cysts of the third ventricle are 0.5-2% of all primary brain lesions and 15-20% of intraventricular lesions [1]. Histological and immunohistochemical studies have established that colloid cysts originate from ectopic elements of the entoderm, which migrate to the velum interpositum during development of the central nervous system [2].

Clinical manifestations debut is possible in all age groups; however it can be found more often in the young and middle age groups (according to WHO data) - 18-59 years-old with approximately an equal sex distribution [3]. There is data on family cases of colloid cysts in the next of kin; it is assumed that is the most likely autosomal dominant mode of inheritance [4,5].

The clinical picture may vary depending on the location, size of the cyst, and degree of CSF obstruction. The symptoms of obstructive hydrocephalus are dominant - due to occlusion of the interventricular foramina (of Monroe), manifested by headaches of varying degrees of intensity and duration, sometimes accompanied by nausea and/or vomiting. Progressive chronic hydrocephalus and direct impact of CC on fornix structures (in the case of large and gigantic cyst sizes) result in memory disorders of varying severity.



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Episodes of acute persistent CSF pathways occlusion are pathognomonic, manifested by a very sudden onset severe headache (thunderclap headache) with or without subsequent short-term (up to a minute) without convulsive loss of consciousness - "drop attack"; or severe weakness in the legs with a fall ("sinking") of the patient without loss of consciousness. In a third of cases, they are the initial manifestations of the disease [3,6-8]. These conditions can develop both against the background of slowly, undulating progressing hydrocephalus, and without it, and are the most dangerous as they can cause sudden death of the patient due to irreversible occlusion of the interventricular foramina (Monroe) by a colloid cyst.

The risk of such events can reach up to 10% according to the world literature [9].

In about 6% of cases, a colloid cyst may be an asymptomatic, "accidental finding" on patient neuroimaging or autopsy [3].

Colloid cysts are revealed as single-chamber, rounded, well-demarcated formations in the interventricular foramina (Monroe) area or septum pellucidum cavity on neuroimaging (MRI, CT) which vary in size from several millimeters up to 8 cm in diameter [10] (most cases are 10-15 mm).

MRI characteristics differ depending on the structure, density and hydration state of the colloidal substance. In 66%, colloid cysts have a homogeneous structure of varying degrees of density, of which: 52% is a viscous colloid (high signal on T1, low on T2); 10% - dense masses (pronounced hyperintense to the adjacent brain on T1 and hypointense on T2); 4% - watery contents (isointense or hypointense on T1 and high signal on T2). 34% of cysts have a heterogeneous structure: The dense component is predominantly located in the lower-posterior or central part of the cyst (black on T2), and the more liquid colloidal content is in the upper-anterior or peripheral part of the cyst (bright on T2), which creates the effect of a "black hole" on MRI. Colloid cysts do not accumulate a contrast agent, only occasionally showing a thin rim enhancement due to adjacent contrasting and stretched septal veins [3].

The pathogenetic treatment of symptomatic colloid cysts of the third ventricle is their surgical (microsurgical, endoscopic) removal [3,7,11].

Asymptomatic small (up to 10 mm) CCs, which are incidental findings and do not cause any CSF obstruction confirmed by clinical and neuroimaging data can be observed and managed conservatively according to some authors [12,13].

Spontaneous colloid cyst reduction/rupture is extremely rare to happen and has been described in several cases [5,14-20]. This clinical observation is the ninth evidenced case of the third ventricular colloid cyst spontaneous reduction in more than 100 years of

observation.

Clinical Presentation

A 28-year-old woman, in June 2012, developed severe paroxysmal headaches up to several times a day. At the peak of the headaches, the patient experienced weakness in the legs ("the legs became numb"). Analgesics had no effect. A brain MRI (June 25, 2012) revealed a rounded lesion at the interventricular foramina (Monroe) and mild dilation of lateral ventricles. The MRI characteristics were suggestive of a colloid cyst of the third ventricle (Figure 1A).

On 18.07.2012 this woman applied for consultation to Burdenko Neurosurgical Center where she was offered surgical treatment - microsurgical removal of the colloid cyst of the third ventricle. Hospitalization date was set for September 19th, 2012.

On August 18th, 2012 the patient was involved in a car accident (side-impact), hit her head on the steering wheel and lost consciousness for a short period of time.

After this mild traumatic brain injury her headaches regressed. Repeated MRI dated September 6, 2012 showed a reduction of the colloid cyst in size (only the dense part of the colloid remained) as well as the narrowing of the lateral ventricles (Figure 1B). Surgical treatment was cancelled as clinical symptoms disappeared and the radiological picture improved. The patient was suggested performing MRI in dynamics. Ophthalmologist and neuropsychologist follow-up examinations showed no changes in the patient's condition after 13 months of observation. On MRI scan, there is practically no negative dynamics either in the size of the ventricles or in the size of the dense part of the colloid (Figure 1C). Control MRI scans (in 3 years and 9 years) showed some increase in cyst size, mainly due to fluid cyst fraction, as well as mild lateral ventricular dilation, while no clinical symptoms of CC were found in the patient during the follow-up period (Figure 1D and Figure 1E). This patient abstains from planned surgical treatment and is being observed.

Discussion

Spontaneous reduction/rupture of a colloid cyst is extremely rare. Only 8 cases of spontaneous colloid cyst resolution/rupture have been described throughout world literature. In the vast majority of cases the mechanism of this phenomenon is unclear (Table 1).

Macroscopically, a colloid cyst is a well-defined, rounded cystic formation with a capsule of varying thickness (from very thin to quite dense "leathery" - which is more typical for CCs with xanthogranulomatous metamorphosis).

The inner layer of the cyst capsule is represented by pseudostratified cylindrical ciliated epithelium producing mucin [2,21], and therefore CCs can increase

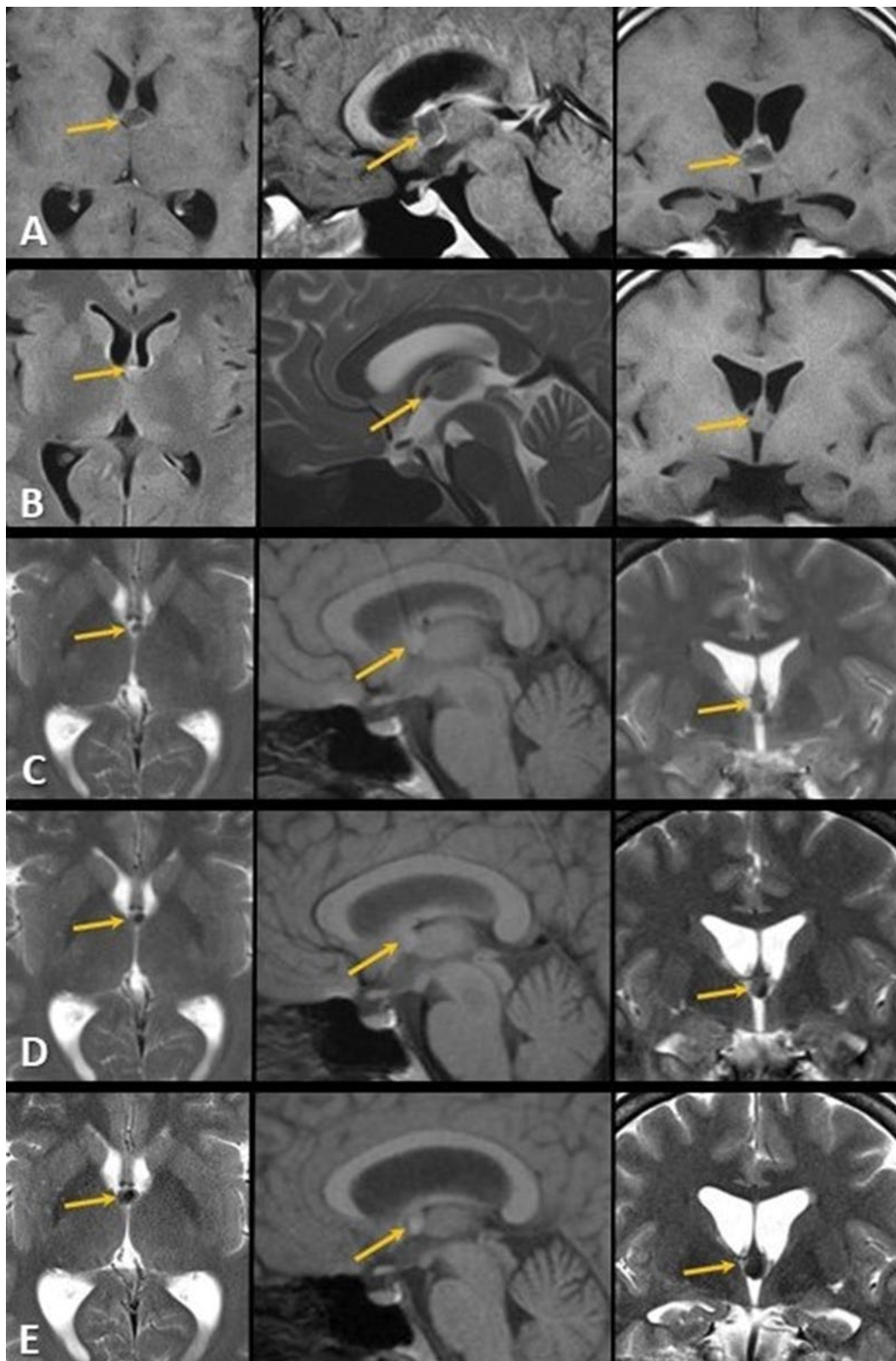


Figure 1: Chronology of colloid cyst III ventricle changes on T1, T1 + C, T2 MRI. (A) Primary MRI T1 + C, 2012. The yellow arrow shows a two-component (dense component in the lower part, more liquid on top) colloid cyst of ventricle III with contrast accumulation in the form of a thin rim around the cyst (contrasting of the septal veins adjacent to the cyst), MAX size 14 mm; moderate ventricular dilation (EI = 0.28); (B) Control MRI T1, T2 after TBI, 2012. Dense colloid cyst component (yellow arrow), MAX size 5 mm; mild ventricular asymmetry (D ≥ S) (EI = 0.24); (C) Follow-up MRI T1, T2, 2013. Dense colloidal cyst component with a small accumulation of colloidal masses (yellow arrow), MAX size 6 mm; IE = 0.25; (D) Follow-up MRI T1, T2 2015. Dense colloidal cyst component with increasing accumulation of colloidal masses (yellow arrow), MAX size 7 mm; IE = 0.26; (E) Follow-up MRI T1, T2 2021. Dense colloid cyst component with increasing accumulation of colloidal masses (yellow arrow), MAX size 8 mm; EI = 0.27.

Table 1: Summary of cases with colloid cyst spontaneous rupture.

Case/year [ref. No.]	Gender/age	Presentation	Size (mm)/CC contents/MRI characteristics	Ventricular size	CC dynamics/Follow-up	Ventricular size dynamics	Symptoms Dynamics	Mechanism of CC reduction
Motoyama Y, et al. 2002 [17]	♂/83	Progressive hydrocephalus	large (not reported)/homogeneous/- T1, ↓T2	+++	Rupture of the CC with preservation of the capsule /10 days	No	Regression followed by hydrocephalus after 6 months (refusal of CSF shunting surgery)	Unknown
Annamalai G, et al. 2008 [14]	♂/35	Asymptomatic finding (due to trauma)	5/homogeneous/↑T1, ↓T2	++ (D > S)	Complete resorption/15 months	No	No	Unknown
Gbejuade H, et al. 2011 [15]	♂/65	F	8/homogeneous/↓T2	N	Complete resorption/19 months	No	Regression	Unknown
Duncan G, et al. 2014 [16]	♀/22	Asymptomatic finding (due to migraine)	3/homogeneous/↑T1	N	Increase of colloidal component up to 6 mm (↓T1) with subsequent decrease to 3 mm (↑T1)/50 months	No	No	Unknown
Bakhtevari MH, et al. 2015 [18]	♂/69	Progressive hydrocephalus; meningism	50 /homogeneous/ - T1, ↑T2	+++	Rupture of CC with development of chemical meningitis; endoscopic removal of CC /42 months	++ (slightly less than before surgery)	Regression	Unknown
Peeters S, et al. 2016 [20]	♀/46	Asymptomatic finding (due to dysmenorrhea)	0.675 cm ³ /homogeneous/↑T1, ↓T2	N	Colloid content increase to 2.852 cm ³ (↓T1, ↑T2) with subsequent decrease to 0.07-cm ³ (↑T1)/36 months	No	Headaches with amnesic and mental disorders (during size increase of CC) with subsequent regression	Unknown
Turel M, et al. 2017 [19]	♀/45	Asymptomatic finding (due to nonspecific headaches, vertigo)	11 /heterogeneous/↑T1 with ↓T1 center; ↑T2 with ↓T2 center	N	Complete resorption/24 months	Slightly narrowed	No	Unknown
Agrawal D, et al. 2020 [5]	♂/21	Asymptomatic finding (familial form)	6 (CT)	N	Increase of colloidal component up to 8 mm (↑T2) with subsequent decrease to 4 mm (↑T2)/38 months	Slightly expanded with a subsequent narrowing	No	Unknown
Present case	♀/28	Headaches, occlusive seizures	14 /heterogeneous /↑T1 with - T1 center; ↑T2 with ↓T2 center	++	Rupture of the CC with preservation of the dense component/108 months	Narrowed	Regression	Hydrodynamic impact

“-” – isointense MRI signal; ↑ - hyperintense MRI signal; ↓ - hyperintense MRI signal; “+++” - pronounced dilatation; “++” - mild dilatation; N - normal size or slight dilatation

in size (especially in young patients) over a long period of time (years), and develop from "asymptomatic findings" to potentially life-threatening conditions.

Pollock, et al. study showed that 8% of patients with initially asymptomatic colloid cysts developed symptoms within 10 years of observation [12].

A cause of a sharp increase of cyst size with the rapid development of the clinic, in most cases leading to a lethal outcome, is hemorrhage into the colloid cyst, which is a very rare phenomenon [9,22]. Gradual increase in cyst size followed by its reduction (due to the rupture) has been described in Duncan G. and Peeters S' studies [16,20].

Pollock BE identified 4 factors which most provoke the appearance of symptoms in patients with CC: younger age, cyst size > 10 mm, ventricular dilation and increased signal on T2-weighted MRI, which points to its more liquid content [12].

Beaumont TL, et al. identified and combined 5 criteria (age < 65 years, headaches, axial diameter of CC \geq 7 mm, hyperintensity in FLAIR and location of CC in the risk zone - 3 zones of the third ventricle were identified in which CC can be located) into a clinical scale called the risk scale for colloid cysts (CCRS) to identify symptomatic lesions and risk stratify. Thus, surgery should be obligate for all patients with CCRS \geq 4 because they are included in the high-risk subgroup. Beaumont TL, et al. also mentioned 2 patients with incidentally identified asymptomatic colloid cysts who showed a reduction in cyst size by more than 30% during their follow-up period (mean time was 5.1 years), details not provided [6]. The relative prognostic value of such scale cannot be overlooked as it does not take into account such factors as cyst mobility and possibility of its sudden (due to hemorrhage) or gradual increase in size.

An important factor in the dynamics of the size of a cyst is the consistency of its contents. Cyst contents can be both homogeneous: Liquid; containing homogeneous colloid masses; either have dense contents or heterogeneous - colloid combined with dense masses [3].

Kachhara, et al. study showed some decrease in CC size and at the same time there was an increase in its density (thickening) following a ventriculoperitoneal shunt. This phenomenon is probably due to the extraction of water from the cyst through its permeable capsule following CSF diversion [23].

Spontaneous regression of CC is probably associated with a rupture of the cyst capsule and resorption of the liquid/colloid component in small cysts according to cases described in the scientific literature. As a result of the rupture of large cysts chemical (aseptic) meningitis was described by Bakhtevvari MH, et al. [18]. We can also assume that a 2-days headache with irradiation to the neck and subsequent regression of the patient's

symptoms in the case described by Peeters S. were meningism signs [20]. Inflammatory changes due to aseptic meningitis can result in the development of communicating hydrocephalus (Motoyama Y, et al.) [17]. All these cases show symptoms regress while factors which led to the capsule rupture and colloid cyst size reduction remain unclear.

It is assumed that in the present case, the cyst capsule (probably thin) rupture and a partial decrease in the volume of the colloid cyst due to the more liquid fraction occurred due to a pressure surge (hydrodynamic shock) in the cyst that occurred at the time of the TBI. Due to unblocking of interventricular foramina and restoration of normal CSF dynamics, the patient showed neurological and radiological symptoms regression. However, the presence of the preserved cyst capsule provides its further enlargement as accumulation of colloid fraction continues due to liquid production by secreting cells of the cyst capsule's inner layer. This fact explains CC recurrence after its stereotactic aspiration [24] or incomplete surgical removal [3,11]. Cyst enlargement rate is 0.02 mm/month in the present case. Despite an extremely slow CC size increase and Evans index increase in dynamics, it is likely that over time this CC will become symptomatic and will require surgical treatment.

Patients' asymptomatic small (up to 1 cm) colloid cysts management is being discussed in the world's literature. In particular, Pollock suggests to monitor the patients' condition in dynamics. O'Neil, et al. (2018) reviewed 132 publications, and 176 cases of asymptomatic colloidal cysts of the third ventricle were selected for detailed analysis. The average size of the cysts was 7.5 cm with significant variation in their diameter from 3 to 18 mm. Ventricular dilation was described in 1/3 of observations. A 5-year risk of clinical progression and increase in size with further surgical removal was 5-15% [25].

Conclusion

Third ventricle colloid cysts spontaneous regression phenomenon is extremely rare. Factors contributing to its development seem to be a thin capsule and liquid contents of the cyst, but its causes in the vast majority of cases is unknown and cannot be considered as an alternative to surgical treatment in symptomatic cysts.

Patients with clinically asymptomatic colloidal cysts ("accidental finding") having radiological signs of ventriculomegaly (Evans index > 0.3) are subjects for surgical treatment - CC removal. Conservative management (MRI monitoring) of patients with asymptomatic colloid cysts not accompanied by ventriculomegaly may be an option in elderly patients (> 60 years). At the same time, patients should be informed about a possible sudden disturbance in CSF circulation and aggravation of symptoms and should

have MRI control in dynamics (once a year, or earlier in case of symptoms aggravation).

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

The patient provided verbal and written consent for this case report.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

MS conceived of the study, data acquisition and analysis and drafted the manuscript. AK participated in the supervision and revision of the manuscript. VT contributed to the writing of the original manuscript. All authors read and approved the final manuscript.

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