Colloid Cyst of the Third Ventricle: A Case Report

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ABSTRACT
Colloid cyst of the third ventricle (CC) represents approximately 1% of intracranial tumours and 20% of intraventricular tumours. CC usually occurs between 20 and 50 years of age. During the first decade of life, it is diagnosed very rarely (1–2%). It can be most commonly found in the anterior part of the third ventricle at the foramen of Monro (1). It is often visualised during the computed tomography (CT) examination as a hyperdense focal lesion, it has variable change of the signal during magnetic resonance imaging (MRI) (2). CC has a benign character, however, a strategic position which may lead to acute hydrocephalus, intracranial hypertension syndrome, consciousness disorder, and even sudden death. This peracute hydrocephalus is an indication to an acute neurosurgical procedure (3).

KEYWORDS
colloid cyst; intracranial hypertension; malignant brain edema; external ventricular drainage

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CASE REPORT

A 30-year-old, previously healthy man developed headache with a maximum in the frontal region 3 weeks ago. The pain had a tension character, and the patient did not report any provoking factor. The headaches significantly deteriorated after one week, he still woke up at night due to pain, was unsafe when walking, eventually preventing him from walking even to the toilet. He started to be drowsy and was transferred to neurology at night in this condition. He vomited repeatedly at the outpatient clinic. His psychomotor rate was slower upon admission, he cooperated actively; his pupils were equal, with preserved photoreaction, abdominal reflexes were low, without irritation pyramidal symptoms on the lower limbs, no nuchal rigidity, Lasegue was negative, standing position very unstable, BP 205/105, HR 56/min, regular. CT examination of the brain and CT angiography of carotids and vertebral arteries reported a hyperdense spherical focal lesion of 18 × 26 mm in the midline in area of the 3rd ventricle, with dilatation of lateral ventricles to 22 mm, small hypodense rims at the occipital corners with the infiltration of cerebrospinal fluid. There was no dilatation of the 3rd and 4th ventricles. Supratentorial subarachnoid cisterns were faded, basal cisterns were preserved (Figure 1). Angiography of cerebral arteries was normal. He was admitted to the neurological ICU, the anti-oedematous treatment (mannitol, NaCl 10% 20 ml) was initiated immediately, and a neurosurgeon was consulted. Laboratory levels revealed no clear deviations. Ophthalmological examination was acutely performed, revealing fundus congestion with haemorrhages. However, generalized epileptic paroxysm manifested at the same time with the subsequent development of coma. Vomiting of the gastric content occurred again. Brief external cardiac massage was performed within the resuscitation care, the patient was subsequently intubated and artificial pulmonary ventilation was initiated. He was subsequently transferred to the surgical theatre to undergo acute external ventricular drainage. A follow-up brain CT scan was additionally performed during transfer. At arrival to the surgical theatre, his pupils were not round bilaterally; opening pressure of the introduced drainage was 30 cm of the water column, fluid was clear. After introduction of ventricular drainage, the patient was handed over to the care of the anaesthesiology-resuscitation department.

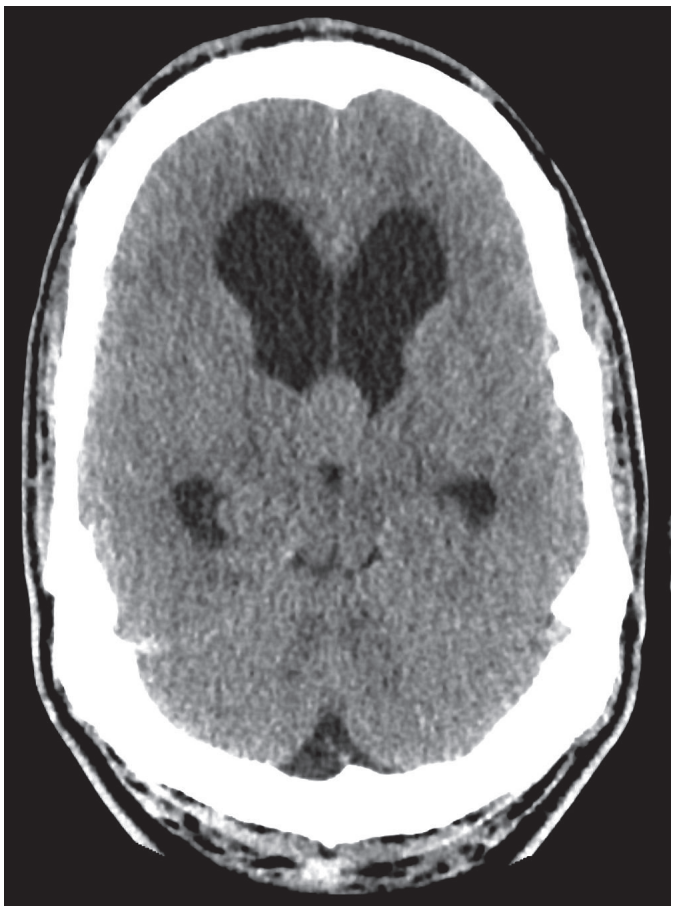

Fig. 1 Initial CT examination.

Dilatation of lateral ventricles and faded subarachnoid space associated with slightly hyperdense spherical expansion of the 3rd ventricle.

Tab 1 Course of patient with colloidal cyst nad peracute syndrom of intracranial hypertension.

9. 12. 2022	Headache, worsening
16. 12. 22 02:35	Neurological examination because of increasing headache, then weakness, sedation, at emergency he vomitted
04:10	Head CT and then CT angiography
04:20	Admission to neurological ICU – antiedematous medication
04:30	Neurosurgical examination – plan of EVD, before surgery – CT control, ophthalmological examination and continuing in antiedematous therapy
04:45	Ophthalmological examination
05:03	Epileptic paroxysm with subsequent short resuscitation, then intubation and arteficial ventilation
06:38	Head CT control – extincted cysterns, increased hydrocephalus
07:00	Mydriatic pupils
07:10	Beginning of surgery – CSF, leaking with increased pressure (30 mm H2O)
07:30	Induced EVD, end of surgery
07:35	Transfer to Anesthesiology, anisocoria – right 5mm and left 3 mm
09:07	Head MRI, induced EVD, diffuse brain edema

Brain MRI was performed (Figure 2). Figure 3 short, transient clinical improvement (for several hours) was followed by a period of hypertension that was difficult to control, with unilateral mydriasis on the right, due to which further acute CT scan was performed (Figures 4, 5). Faded differentiation between the grey and white matter was obvious here, with insignificant haemorrhage into the ventricular system, and a virtually collapsed ventricular system. During the following several hours, bilateral mydriasis developed, followed by brainstem areflexia within 24 hours from the initial examination. CT angiography revealed no filling of cerebral vessels except for a questionable M1 segment on the left. The patient was handed over to the transplant programme.

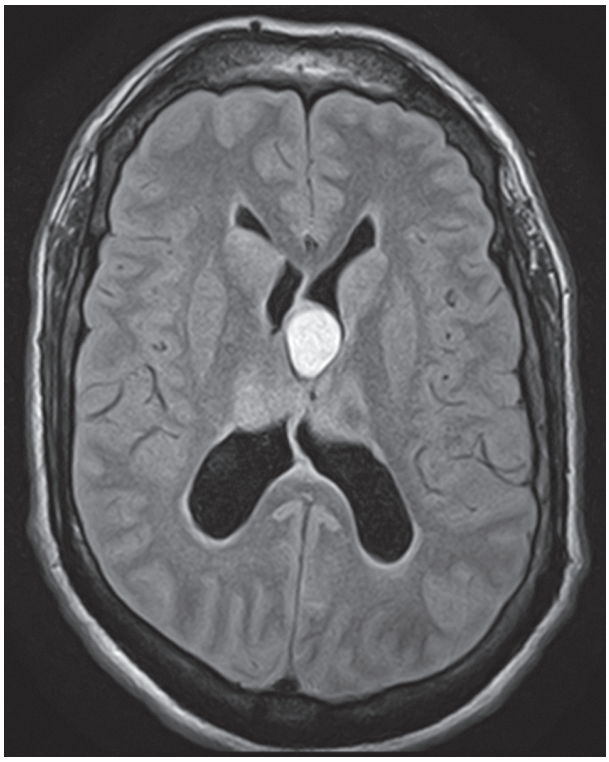

Fig. 2 MRI examination – FLAIR sequence.

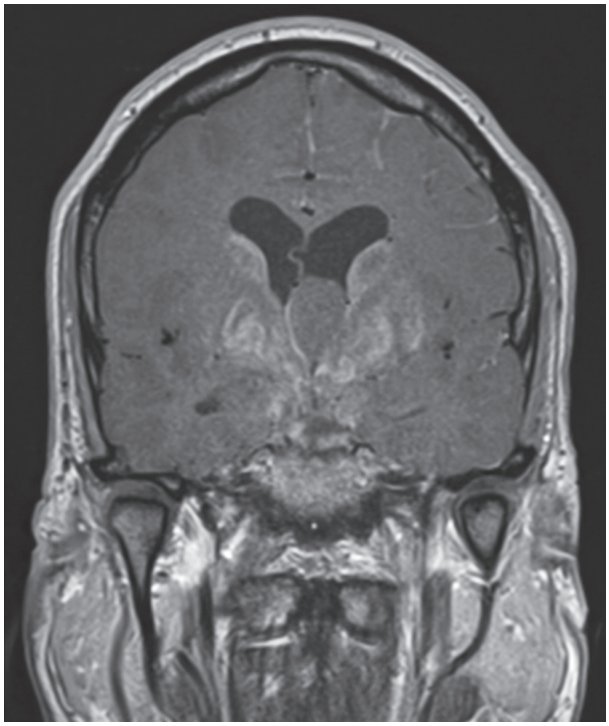

Fig. 3 MRI examination: contrast-enhanced T1-weighted images in the coronary plane.

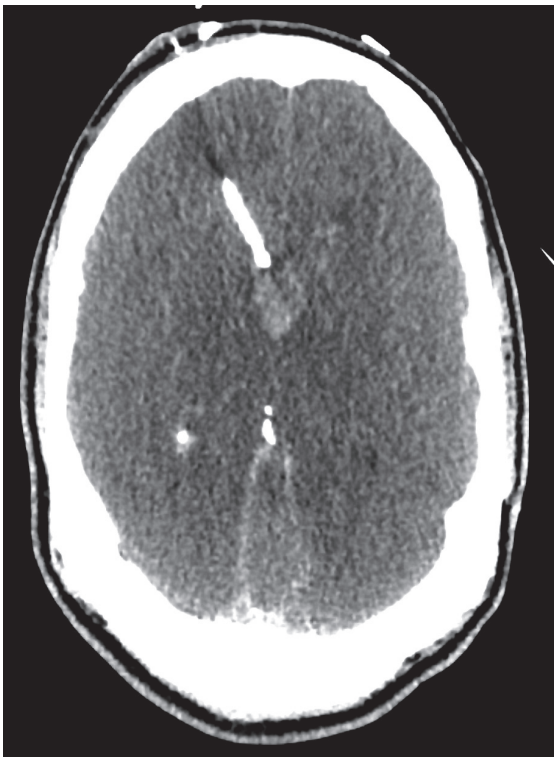

Fig. 4 Follow-up CT examination after 34 hours.

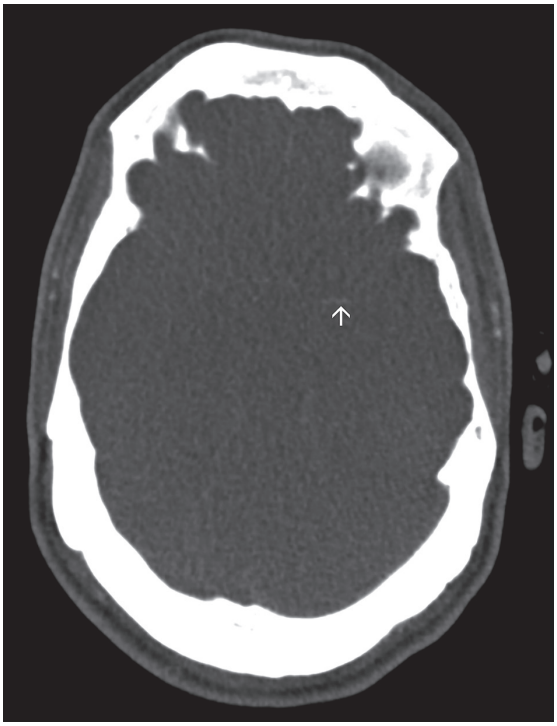

Fig. 5 CT angiography of the carotid circulation.

Dilatation of lateral ventricles with an image of active hydrocephalus associated with a hyperintense lesion of the 3rd ventricle, a typical image of the colloid cyst at the top of the 3rd ventricle.

The lesion of the 3rd ventricle without enhancement after administration of contrast substance. However, pathological enhancement of both thalami and basal ganglia is obvious with diffuse oedema and impaired blood-brain barrier.

Image of diffuse cerebral oedema with faded differentiation between the grey and white cerebral matter. Ventricular drainage on the right in the frontal region.

Only poor MCA filling obvious intracranially on the left (marked with an arrow).

DISCUSSION

Opinions on the origin of CC and especially on the tissue type it comes from have gone through long evolution. In the 1920s, there was discussion about paraphysis, the embryonic tissue from which CC could arise. In the 1950s, ependyma of the third ventricle was considered. Then, there was a theory of a neuroepithelial origin. The epithelium in the choroid plexus and ependyma of the 3rd ventricle share the same origin – the neuroepithelium – and CC most probably forms from this tissue. Current microscopic demonstration of cilia on these epithelial cells and the presence of goblet cells, demonstration of desmosomes points to significant resemblance of the CC structure to respiratory epithelium. Hence, there is considerable similarity with Rathke's cyst (4).

The genetic theory is the result of epidemiological studies where CC has occurred in siblings (especially females) and also in twins (1).

Cardiovascular symptoms occur quite commonly. It is a myocardial contractility disorder (stunned myocardium), associated with pulmonary oedema and heart failure. There was a case report of a 29-year-old woman admitted due to hypertension, tachycardia, and pulmonary oedema. She had significant elevation of cardiac markers in laboratory collections. The transthoracic ECHO examination revealed global hypokinesia and a drop in ejection fraction to 30–35%. There was no diastolic function disorder. Myocardial function also improved after endoscopic CC excision. In pathogenesis of cardiac dysfunction, authors emphasize pressure of growing CC on hypothalamic cardiovascular regulatory centres (3).

The incidence rate of CC is thought to be even lower at ≈0.9 per million. The most common clinical symptom of CC is headache (68–100%); there are typical attacks, lasting for seconds to minutes. However, the duration and intensity of pain is variable depending on the position of the head; worse when sitting upright and improved when lying supine (2). Other symptoms include nausea, vomiting, visual disturbances, and ataxia of the standing position and gait (5). Urinary incontinence, epileptic seizures, falls, sudden death may occur. Although this is a benign process, the risk of sudden neurological deterioration or death caused by colloid cysts is 3% to 35%.

Hydrocephalus (in 99%) is associated with higher probability of death, compared to patients without hydrocephalus. Patients presenting with cyst apoplexy have a high mortality rate (50%), with an average cyst size of 2.5 cm. Mortality is generally higher in women, even with a smaller CC size (2).

Papilledema occurs in up to 57% of patients. Based on a study of 140 patients, authors report that patients without congestion have higher probability of death. However, this is rather related to activity of physicians when activity is lower in patients without congestion (6).

The mechanism of sudden decompensation or death is still controversial. Various theories have been proposed. A possible mechanism is pressure of the cyst on the hypothalamus, resulting in the disruption of autonomic cardiac reflex control, leading to acute cardiac arrest, with or without a pre-existing cardiac disease. Another possible mechanism is based on the relative movement of the cyst on its pedicle with attachment to the tela choroidea, causing intermittent obstruction of the foramen of Monro and sudden intracranial hypertension. Another mechanism is acute haemorrhage in the cyst, causing progressive expansion of the cyst and subsequent obstruction of the foramen (7).

Conservative care is recommended only for clinically stable patient. Cerebrospinal fluid diversion, open surgical approaches, and neuroendoscopy have been applied to the management of colloid cysts (11). The conservative approach in asymptomatic patients is based on head position elevated to 30 degrees, securing of the airways, relaxation, sedation, serum osmolality 295–305 mosm, control of hypertension, maintaining central perfusion pressure above 60 mmHg, normothermia, use of hyperosmolar solutions. Acute external drainage is indicated in severe and rapidly progressing cases.

Surgical treatment of colloid cysts include craniotomy with excision via transcallosal or transcortical route, endoscopic removal, or stereotactic aspiration, and external ventricular drains (or rarely bilateral drains) could also be performed as bridge therapy in life-threatening hydrocephalus (12). External ventricular drainage (EVD) enables the monitoring of intracranial pressure, reduction of the cerebrospinal fluid volume, and temporary treatment of intracranial hypertension. Another option is surgical reduction of the enlarged volume (usually a tumour or haematoma) responsible for the growth of intracranial pressure. The neurosurgeon may opt for decompressive craniectomy in certain cases (6).

This case report describes an acute condition (with a rapidly deteriorating clinical condition and limited options for conservative management), requiring urgent management to affect intracranial hypertension and focus on its cause – introduction of external ventricular drainage. The important role of cerebrospinal fluid on the development of malignant brain oedema is an important pathogenic mechanism in acute obstructive hydrocephalus leading to bad prognosis (8, 13).

Surgical management depends on the fact of whether the colloid cyst is found incidentally or is symptomatic and whether risk factors are present. High mortality rate was observed in patients with neurological deficits. The mortality rate is close to 100% without surgical intervention. Mortality rate reaches only 48% in patients who underwent a surgical procedure (6). Acute conditions are always managed by emergent procedures -external ventricular drainage and cyst resection is primarily approached only rarely (9). After ruling out an acute condition, the colloid cyst may be treated using various surgical methods. For this definitive treatment the approach type is chosen according to the location and size of the cyst in the third ventricle. Transcortical approach using endoscopic technique and navigation is currently utilised. Previously used stereotactic aspiration of the cyst is of marginal use (10).

In our patient with peracute development of intracranial hypertension with biventricular hydrocephalus and malignant brain edema the brain CT and then CT angiography were completed. Because further worsening of consciousness and symptoms with bilateral mydriasis the

neurosurgeon (and neurologist on ICU) insisted on further brain CT to diagnose the evolving pathology, that causes the clinical worsening.

CONCLUSION

It is necessary to consider the risk of the malignant progress (MRI scale) in CC cases to improve the prognosis of patients with CC and peracute intracranial hypertension syndrome (due to biventricular hydrocephalus associated with development of malignant brain oedema). The incidence of peracute progress of intracranial hypertension syndrome requires a rapid diagnosis (CT, MRI) and acute consultation of a neurosurgery workplace. The primary logical surgical solution is introduction of external ventricular drainage and administration of antioedematous treatment. On the example of our patient, we are showing pitfalls of CC management, with a high mortality rate despite rapid diagnosis and treatment.

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