

## **CASE REPORT**

# A 3rd ventricular colloid cyst causing acute hydrocephalus with stunned myocardium: A case report

Mohammed Ayasa, Nissar Shaikh, Marco A.E. Marcus

Address for Correspondence:

## Mohammed Ayasa

Department of Anesthesia, SICU & Perioperative Medicine, Hamad Medical Corporation, Doha, Qatar Email: mayasa@hamad.qa

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## **ABSTRACT**

Background: Third ventricular colloid cysts are benign but may cause acute hydrocephalus, raised intracranial pressure, decreased consciousness level, and sudden death. These ventricular colloid cysts associated with stunned myocardium are rarely reported in the literature. This study reported a case of a third ventricular colloid cyst presented as acute hydrocephalus complicated with severe neurogenic pulmonary edema, stunned myocardium, and heart failure, which survived at the end.

Case presentation: A 29-year-old woman presented to the emergency department with one day history of headache, vomiting, and altered consciousness level. Early brain imaging showed a cyst in the third ventricle. The patient rapidly deteriorated neurologically and developed severe pulmonary edema and heart failure requiring immediate external ventricular drain and heart failure management. Once stabilized, she underwent endoscopic excision of the ventricular cyst. Histopathology confirmed the diagnosis of colloidal cyst. She survived all these acute lifethreatening events, improved, and stabilized, and was discharged home. She was followed up in outpatient clinics after 6 months of discharge with no symptoms or neurological deficit.

Conclusion: A third ventricular colloid cyst can cause acute hydrocephalus leading to stunned myocardium requiring immediate surgical intervention, advanced hemodynamic monitoring, and acute heart failure management.

Keywords: third ventricular colloid cyst, neurological deterioration, hydrocephalus, neurogenic pulmonary edema, stunned myocardium

## INTRODUCTION

The third ventricular colloid cyst is a rare congenital lesion of endodermal origin. It is one of the causes of headache and sudden death<sup>1</sup> and is more common in individuals in the third to fifth decades of life but can occur in children and young adults as well.<sup>2</sup> Radiological imaging such as brain magnetic resonance imaging (MRI) and computed tomography (CT) scans are essential to diagnose these ventricular cysts. The classical location of these cysts makes it essential to recognize, diagnose, and properly manage it early on to avoid potentially life-threatening complications. These third ventricular cysts causing acute obstruction to cerebrospinal fluid (CSF) flow can lead to hydrocephalus, brainstem herniation, and brain death. Several case reports of third ventricular colloid cysts have been reported in the literature presenting with hydrocephalus only without cardiac involvement. However, a third ventricular colloid cyst, which causes stunned myocardium, neurogenic pulmonary edema, and heart failure, is reported only in six cases in the literature.<sup>3 – 7</sup> Five of which had sudden cardiac arrest and died and were diagnosed as colloidal cysts on autopsy. This study presented a young woman with colloid cyst of the third ventricle who developed acute hydrocephalus, severe neurogenic pulmonary edema, and heart failure with a better outcome.

#### **CASE**

A 29-year-old woman, previously healthy, presented to the accident and emergency department with complaints of fronto-occipital headache and decreased consciousness level for the last 24 hours. The symptoms suddenly started and included photophobia, neck pain, and nonprojectile vomiting (four times). She had urine incontinence and drowsiness. No further symptoms or signs were present on admission. Her vital signs were stable, and she was afebrile. Her Glasqow coma score (GCS) was 12/15 (E3 M5 V4), and pupils were 3 mm bilaterally and reactive to light. Her laboratory values showed leukocytosis (white blood cell:  $22.5 \times 10^3/\mu L$ , neutrophils 42%, hemoglobin 13.3 gm/dL, platelet  $318 \times 10^3/\mu$ L), high lactate 4.8 mmol/L, and normal glucose, electrolytes, and liver and kidney function tests. A CT head scan showed isodense rounded lesion seen at the level of the foramen of Monro measuring  $13 \times 15$  mm, which was likely a cyst with secondary significant dilatation of the lateral ventricles with brain edema.

Few hours later, the patient became unresponsive with drop of GCS to 3/15, tachycardic (heart rate 160 beat/min), and hypertensive (blood pressure 150/110 mm Hg). Pupils were unequal in size; right 8 mm nonreactive and left 6 mm reactive to light. The patient was immediately intubated and escorted to the operating room (OR) for emergency external ventricular drain (EVD) insertion. Upon connecting her to the anesthesia ventilator in the OR, she had pink frothy secretions from the endotracheal tube, high peak airway pressures of 40 cm H2O, and oxygen saturation (SO2) 92%, and she needed fraction of inspired oxygen (FiO2) of 100%. The clinical diagnosis of pulmonary edema was made, and she was given furosemide and morphine and was escorted to the surgical intensive care unit where she was sedated with remifentanil and dexmedetomidine infusions. Her pupils were 2 mm bilaterally equal and reactive to light after the EVD insertion. Noradrenaline infusion was required to maintain adequate hemodynamics. Her chest had bilateral crepitations, and chest X-ray showed bilateral infiltrates (Figure 1). Her arterial blood gas showed metabolic acidosis (pH 7.26, PCO2 43 mm Hq, PO2 74 mm Hq, lactate 2.5 mmol/L, SO2 94%, BE: -7 mmol/L, HCO3 18 mmol/L, and PO2/ Fio2 148 mm Hg). Cardiac biomarkers were elevated (pro B-type natriuretic peptide 3825 pg/mL and highly sensitive troponin T: first set 1917, second set 1085, and third set 756 ng/L). Electrocardiogram (ECG) showed T-wave inversion in leads I. aVL. V2. and V3 (Figure 2).

A right femoral arterial pulse-induced continuous cardiac output (PiCCO®) catheter was inserted for



Figure 1. Chest X-ray showing pulmonary edema

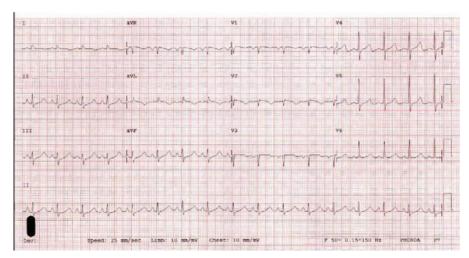


Figure 2. Electrocardiogram on surgical intensive care unit admission

advanced hemodynamic monitoring. Its findings were in line with the diagnosis of heart failure: cardiac index 1.85 L/min/m<sup>2</sup>, cardiac functional index 3.2, systemic vascular resistance index 2710 DS/cm5m<sup>2</sup>, extravascular lung water index 16.2 mL/kg, intrathoracic blood volume index 854 mL/m<sup>2</sup>, global end-diastolic volume index 623 mL/m<sup>2</sup>, and stroke volume variation 10 L/min/m<sup>2</sup>. She was started on dobutamine infusion and boluses of furosemide in addition to noradrenaline infusion. Transthoracic echocardiogram (ECHO) showed global hypokinesia of the left ventricle with low-moderate systolic function impairment (ejection fraction of 30% – 35%) and no diastolic dysfunction or valvular abnormalities or any changes in wall thickness. The right ventricle systolic pressure was 25 mm Hq. Lisinopril and carvedilol were added to the therapy as anti-failure medications. Brain MRI on day 3 showed a midline anterior third ventricle and foramen of Monro well-defined globular cyst measuring about 1.5 cm that causes CSF obstruction with mild-moderate hydrocephalus of the lateral ventricles (Figure 3). Noradrenaline infusion was stopped on day 3, and the patient was kept on dobutamine infusion and furosemide. Daily PiCCO results are shown in Table 1.

The patient showed improvement in clinical, hemodynamic, and neurological status by day 5 and was extubated and put on noninvasive ventilation. Dobutamine infusion was discontinued on day 6. After a multidisciplinary team meeting, patient was brought to the OR on day 17 for endoscopic excision of the cystic lesion. The patient came back from the OR extubated with GCS 15/15. Final histopathology results of the cystic lesion on day 21 were

consistent with colloid cyst (no hemorrhage in the cyst). She was transferred to a neurosurgical ward on day 25. From there, she was discharged home on day 32. Her follow-up in outpatient clinics for the next 6 months of discharge showed improvement in her cardiac status. Her ECG and ECHO findings were improved and within the normal range. Follow-up brain MRI showed complete excision of the cyst.

## DISCUSSION

The ventricular colloid cysts are benign cysts filled with mucin and covered by epithelial cells. They usually arise from the roof of the third ventricle immediately posterior to the foramen of Monro.8

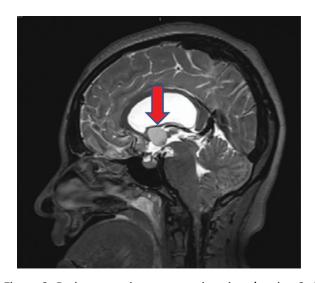


Figure 3. Brain magnetic resonance imaging showing 3rd ventricular cyst, shown on T1 sequence

Table 1. PiCCO results and days of admission

Day	CI (L/min/m <sup>2</sup> )	EVLWI (mL/kg)	SVRI (DS/cm <sup>5</sup> m <sup>2</sup> )	ITBVI (mL/m <sup>2</sup> )	GEDVI (mL/m²)
1	1.85	10.3	2770	712	570
1	2.68	10	2625	595	475
2	2.38	6.8	2414	645	517
2	3.04	8.6	2185	661	529
3	3.17	8.7	2560	767	614
3	3.17	8.7	2025	767	614
4	3.07	11.3	2139	819	656
4	3.53	7.9	2543	760	609
5	4.12	8.3	2139	793	634
6	2.92	6.7	1879	644	515

PiCCO, pulse-induced continuous cardiac output; CI, cardiac index; EVLWI, extravascular lung water index; SVRI, systemic vascular resistance index; ITBVI, intrathoracic blood volume index; GEDVI, global end-diastolic volume index.

Depending on their site, size, and CSF obstruction degree, they can cause various neurological symptoms, ranging from headache to sudden death due to brainstem herniation.<sup>1</sup> Several case reports of colloid cyst presenting with headache, hydrocephalus, or increased ICP but without cardiac involvement have been reported. However, only six cases of a third ventricular colloid cyst associated with neurogenic pulmonary edema and cardiac complications were reported in five articles and found in the literature.  $^{3-7}$ Table 2 summarizes these cases with their outcomes. Only one of them survived and had a resection of the cyst surgically with only mild memory disturbance on follow-up. This case was a young female presented with a third ventricular colloid cyst causing acute hydrocephalus leading to severe pulmonary edema, stunned myocardium, and heart failure. The survival of this case was attributed to early detection and proper intracranial pressure (ICP) management with early bilateral EVD insertion and heart failure control with inotropic support and vasopressors guided by invasive cardiac output monitoring and PiCCO line measurements. The patient survived and underwent complete endoscopic resection of the colloid cyst and discharged home. Previously reported colloid cyst cases and sudden cardiac death diagnosed on autopsy mentioned the histopathological appearance of coagulative myocytolysis and contraction band necrosis (CBN) of myocardial specimens.<sup>3</sup> CBN is observed as a histological hallmark of an adrenerqic overstimulation. Neurogenic myocardial stunning due to hyperacute sympathetic overstimulation may represent the cardiac events that occur because of increased ICP<sup>9</sup>. As the hypothalamic cardiovascular

regulatory centers are located close to the walls of the third ventricle, these structures can be compressed by the growing cyst and can explain the reflex cardiac events associated with it. The resultant neurological crisis that occurs because of hypothalamus compression and high ICP stimulates the vasomotor centers, which leads to massive autonomic sympathetic discharge, pulmonary edema, and stunned myocardium. The increased catecholamine levels can lead to an increase in the left atrial pressure and systemic hypertension, which negatively affects left ventricular performance. Sympathetic discharge can also cause pulmonary venoconstriction, increase vascular permeability, and develop pulmonary edema. 10 This patient was followed up in outpatient clinics for 6 months. She had no symptoms and no neurological deficit. Follow-up head MRI showed complete removal of the cyst, and follow-up ECHO was within normal limits. The limitation of this study is that this neurogenic pulmonary edema and stunned myocardium were presented in a single case. A case series is required for better data and conclusion.

## CONCLUSION

The proximity of the third ventricular colloid cyst to the hypothalamus and vasomotor centers of the brainstem can cause sympathetic overstimulation and lead to life-threatening pulmonary edema, stunned myocardium, and heart failure, which requires immediately relieving raised ICP, advanced hemodynamic monitoring, taking anti-pulmonary edema measures, and continuing heart failure management, which can lead to survival.

Table 2. Total number of reported cases of 3rd ventricular colloid cyst with cardiac complications

Outcome	Died	Survived (cyst resected surgically)	Died	Died	Died	Died
Cardiac Complications	Sudden cardiac arrest (histopathology: contraction band necrosis)		Sudden cardiac arrest	Sudden cardiac arrest	NPE, sudden cardiac arrest	Sudden cardiac arrest
Age (years), Sex	10, boy	33, wотап	40, woman	33, man	45, man	18, girl
Diagnosed by imaging or Autopsy	Autopsy	Imaging	Autopsy	Autopsy	Autopsy	Autopsy
Diagnosis	3rd ventricle colloid cyst	3rd ventricle colloid cyst	3rd ventričle colloid cvst	3rd ventricle	3rd ventricle	3rd ventricle colloid cyst
Reported cases	Turillazzi et al. 2012 [3]	Jarquin et al. 2005 [4]	Buttner et al. 1997: case 1 [5]	Buttner et al. 1997: case 2 [5]	Belhadj et al. 2015 [6]	Skerbinjek et al. 2004 [7]

NSM, neurogenic stunned myocardium; NPE, neurogenic pulmonary edema.

## **CONSENT**

Written informed consent was obtained from the patient for publication of this case report and any relevant investigations and imaging studies.

## **ETHICS APPROVAL**

We obtained permission from the Medical Research Center (Project Ref. No: MRC-04-18-224) of Hamad Medical Corporation for the publication of this case report on the condition of no patient identifiable data (including patient name and photograph).

## **REFERENCES**

- 1. Demirci S, Dogan KH, Erkol Z, Gulmen MK. Sudden death due to a colloid cyst of the third ventricle: report of three cases with a special sign at autopsy. Forensic Sci Int. 2009;189(1-3):e33-e36.
- 2. Kapu R, Symss NP, Pande A, Vasudevan MC, Ramamurthi R. Management of pediatric colloid cysts of anterior third ventricle: a review of five cases. J Pediatr. Neurosci. 2012;7(2):90 - 95.
- 3. Turillazzi E, Bello S, Neri M, Riezzo I, Fineschi V. Colloid cyst of the third ventricle, hypothalamus, and heart: a dangerous link for sudden death. Diagn Pathol. 2012;7(1):144.
- 4. Jarquin-Valdivia AA, Rich AT, Yarbrough JL, Thompson RC. Intraventricular colloid cyst, hydrocephalus and neurogenic stunned myocardium. Clin Neurol Neurosurg. 2005;107(5):361 - 365.
- 5. Büttner A, Winkler PA, Eisenmenger W, Weis S. Colloid cyst of the third ventricle with fatal outcome: a report of two cases and review of the literature. Int J Legal Med. 1997;110(5):260 - 266.

- 6. Belhadj M, Thaljawi W, Chkirben Y, Jedidi M, Souguir MK, Masmoudi T, et al. Sudden death due to intracranial colloid cyst: about three cases. J Clin Pathol Forensic Med. 2015;6(1):1 - 5.
- 7. Kavalar MS, Kavalar R, Strojnik. A colloid cyst of the third ventricle – the cause of episodic headache and sudden unexpected death in an adolescent girl. Wien Klin Wochenschr. 2005;117:837 - 840.
- 8. Osborn AG, Preece MT. Intracranial cysts: radiologicpathologic correlation and imaging approach. Radiology. 2006;239(3);650-654.
- 9. Johnson J, Ragheb J, Garg R, Patten W, Sandberg DI, Bhatia S. Neurogenic stunned myocardium after acute hydrocephalus: report of 2 cases. J Neurosurg Pediatr. 2010;5(5):428-433.
- 10. Khademi S, Frye MA, Jeckel KM, Schroeder T, Monnet E, Irwin DC, et al. Hypoxia mediated pulmonary edema: potential influence of oxidative stress, sympathetic activation and cerebral blood flow. BMC Physiol. 2015;15(1):4.