

ORIGINAL ARTICLE

A. Büttner · P. A. Winkler · W. Eisenmenger · S. Weis

Colloid cysts of the third ventricle with fatal outcome: a report of two cases and review of the literature

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Abstract Two cases of sudden death due to colloid cysts of the third ventricle are presented with a review of the literature. In the first case, a 40-year-old woman suffered an acute onset of severe frontal headache after an intercontinental air flight. The next day, she was found comatous and died 7 h after admission to a hospital. In the second case, a 33-year-old man with a medical history of recurrent headaches was found dead in his car. Autopsy in both cases revealed a colloid cyst of the third ventricle and hydrocephalus involving the lateral ventricles.

These cases demonstrate that fatal cases still occur. Nevertheless, prompt diagnosis using computed tomography (CT) or magnetic resonance imaging (MRI) is essential, since colloid cysts are histologically benign tumors that can be removed safely by neurosurgical intervention.

Key words Colloid cyst · Sudden death · Third ventricle

Introduction

Colloid cysts of the third ventricle are slow-growing benign tumors, the majority of which are located in the anterior third ventricle with an attachment to the roof. They are rare lesions, accounting for 0.2–2% of all intracranial neoplasms (DiMaio and DiMaio 1974; Greenwood 1949; Hernesniemi and Leivo 1996; Leestma and Konakei 1981; Little and MacCarty 1974; Poppen et al. 1953;

Schröder et al. 1990). Colloid cysts occur in all age groups, but the age of onset of symptoms usually ranges between 20 to 50 years of age (Antunes et al. 1980; Batnitzky et al. 1974; Çetinalp et al. 1994; Ferry and Kempe 1968; Grossiord 1941; Kelly 1951; Schröder et al. 1990; Stookey 1934). Some authors found a slight male preponderance (Little and MacCarty 1974; Mathiesen et al. 1997), whereas others found no sex predominance (Batnitzky et al. 1974; Camacho et al. 1989; Çetinalp et al. 1994; Schröder et al. 1990).

Histologically, colloid cysts are composed of an outer fibrous capsule and an inner lining of epithelium usually consisting of a single layer of squamous, cuboidal or low columnar, ciliated and non-ciliated epithelial cells. Mucous goblet cells are commonly present. The cyst content consists of amorphous PAS-positive gelatinous material with some macrophages, debris, and sometimes cell ghosts (Lach et al. 1993; Schröder et al. 1990; Shuangshoti and Netsky 1966).

Since the first description of colloid cysts (Wallmann 1858), sudden deaths attributable to this lesion have only been described occasionally. Despite their easy recognition using modern imaging techniques (CT or MRI) and their safe microsurgical removal, fatal cases do still occur. In the present report we describe the clinicopathological features of two cases of sudden death due to a colloid cyst of the third ventricle and a review of the literature.

Case reports

Case 1

A 40-year-old previously healthy woman suffered an acute onset of severe frontal headache after an intercontinental air flight. Her friend, a general practitioner, sent her home and advised her to rest for some hours. The following day she went to work, but the headache worsened and she had to vomit twice. She was sent home again, where she was found stuporous some hours later by the emergency physician. The pupils were dilated and there was no pain reaction. The initial blood pressure was 200/120 mm Hg. In the emergency room, 44 min later, there was slight decerebrate rigidity on the left side, a decline of blood pressure to 80/50 mm Hg, and a pulse rate of 80 beats/min. Fundoscopic examination

A. Büttner (✉) · W. Eisenmenger · S. Weis
Institute of Legal Medicine, Ludwig-Maximilians University,
Frauenlobstrasse 7a, D-80377 München, Germany
Fax: +49 (89) 5160 5144

P. A. Winkler
Department of Neurosurgery, Klinikum Grosshadern,
Ludwig-Maximilians University, Marchioninistrasse 15,
D-81377 München, Germany

S. Weis
Institute of Neuropathology, Ludwig-Maximilians University,
Thalkirchnerstrasse 36, D-80337 München, Germany



Fig. 1 **a** Cranial CT scan showing hydrocephalus with periventricular zones of low density suggesting transependymal spread of CSF and obliteration of the subarachnoid spaces. No focal space-occupying lesion can be identified. **b** Coronal brain section at autopsy showing an impacted gelatinous lesion within the third ventricle

was not performed. An emergency CT scan (Fig. 1a) showed obstructive hydrocephalus involving the lateral ventricles with a periventricular zone of low density suggesting transependymal spread of CSF, obliteration of the subarachnoid spaces, and transtentorial herniation. No focal space-occupying lesion could be identified. MRI was not performed. Angiography demonstrated extremely slow intracranial circulation without venous filling. Transcranial doppler sonography showed shuttle flow in the internal carotid and in the middle cerebral arteries. The patient died 7 h after admission. Consent was only given for brain autopsy by the family members.

At autopsy the brain weighed 1350 g. On macroscopic examination, the brain was markedly swollen with gyral flattening, sul-

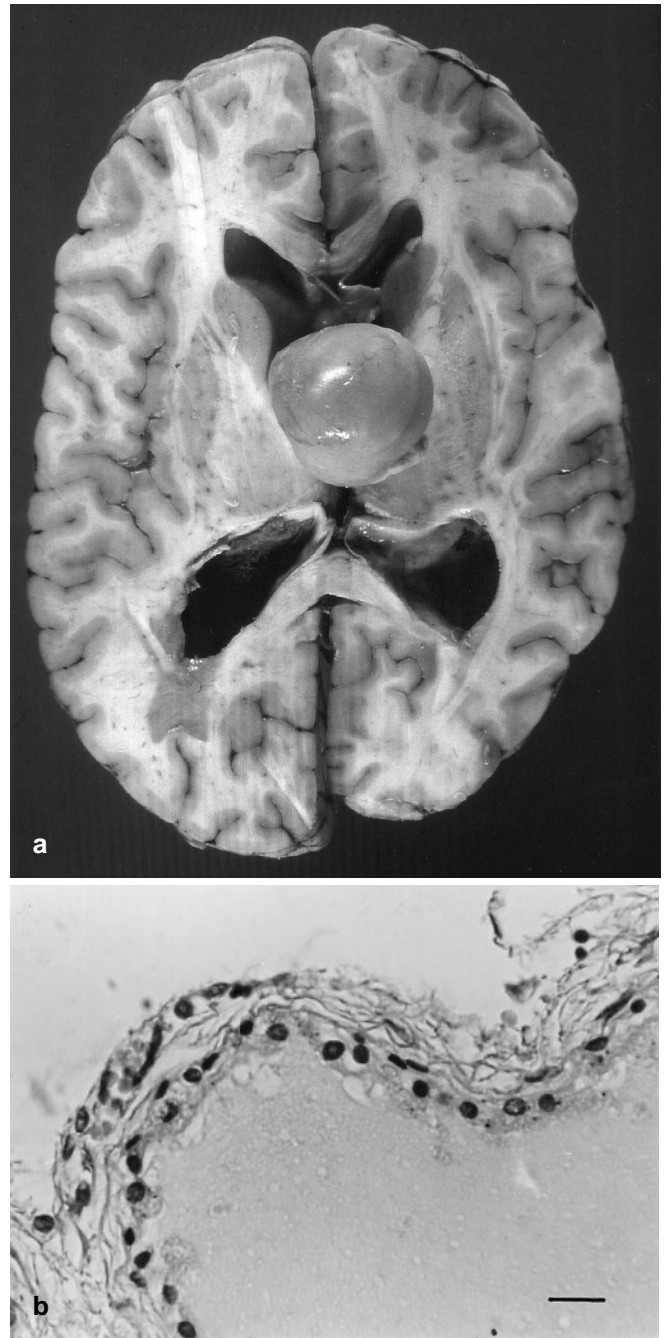


Fig. 2 **a** Horizontal brain section at autopsy with a large space-occupying cystic lesion within the third ventricle. **b** Light microscopic finding of the characteristic features of a colloid cyst showing a thin cyst wall composed of loose connective tissue, lined by a single layer of cuboidal cells and an amorphous content (H&E; magnification (240; bar = 160 μ m)

cal narrowing, and bilateral uncus grooving. There was transtentorial herniation of the basal ganglia and diencephalon with congestive hemorrhages in the pons. The cerebellar tonsils were grooved and their tips were necrotic. Coronal brain sections revealed a cystic, round-oval lesion, inside the third ventricle near the foramen of Monro (Fig. 1b) measuring 1 cm in diameter. The corpus callosum was partly destroyed due to poor tissue preservation. Death was due to brain-stem compression caused by obstruction of the third ventricle by the cystic lesion. Microscopic examination revealed a

cyst lined by a single layer of flattened, non-ciliated epithelial cells, resting on a collagenous membrane. The epithelial cells were partly vacuolated. The cyst content was strongly positive for the periodic-acid-Schiff (PAS) reaction and contained some macrophages. The other parts of the brain showed extensive edema, hypoxic nerve cell changes, and moderate nerve cell loss.

Case 2

A 33-year-old man with a medical history of recurrent headaches over several years was found dead in his car. Autopsy revealed no relevant findings attributable to the death of the patient in the other organs except the brain.

At autopsy the brain weighed 1740 g. On macroscopic examination, the brain was markedly swollen with gyral flattening, sulcal narrowing, and marked bilateral uncal grooving. Furthermore, a marked narrowing of the IVth ventricle was noted. Coronal brain sections revealed a free-floating, cystic, round-oval lesion, measuring $4 \times 3.5 \times 3.5$ cm inside the third ventricle (Fig. 2a). The basal ganglia and fornices were markedly displaced by the lesion. The lateral ventricles were dilated and the midbrain showed some petechial hemorrhages. On the cut surface, the lesion showed a soft, partially gelatinous content and a thin fibrous capsule. Microscopic examination revealed a cystic lesion lined by a single layer of partially low cuboidal, partially flattened, non-ciliated and ciliated epithelial cells, resting on a thin collagenous membrane (Fig. 2b). The epithelial cells were partly vacuolated and contained some PAS-positive granules. The cyst content was strongly PAS-positive and contained some macrophages. The other parts of the brain showed extensive edema and hypoxic nerve cell changes.

Discussion

Colloid cysts of the third ventricle vary in size from a few millimeters to several centimeters in diameter and the largest cysts reported in the literature measured 9 cm in diameter (Gemperlein 1960; Palacios et al. 1976). Colloid cysts are believed to be congenital in origin, although they usually do not become symptomatic until the third or fourth decade of life (Kelly 1951). Others remain asymptomatic and are incidentally found only at autopsy (Little and MacCarty 1974; Schröder et al. 1990). At present, the exact histogenesis is unclear and is still a matter of debate. They are believed to arise either from the rudimentary paraphysis (Kappers 1955; Sjövall 1910), the neuroepithelium of the choroid plexus (Shuangshoti and Netsky 1966), the ependyma (Kappers 1955), or from the primitive foregut endoderm (Ho and Garcia 1992; Kuchelmeister and Bergmann 1992; Lach et al. 1993; Schröder et al. 1990). The latter possibility of a non-neuroepithelial origin has been advanced by immunohistochemical investigations demonstrating a different immunohistochemical profile of colloid cyst epithelium compared to choroid plexus epithelium or ependyma (Kuchelmeister and Bergmann 1992).

The clinical symptoms in children and adults are attributable to increased intracranial pressure but are non-specific. The most common complaints consist of severe episodic attacks of headache associated with nausea and vomiting. Other symptoms, although seen less frequently, include gait disturbances, syncopal attacks, sudden weakness of the lower limbs, blurred vision, dizziness, tinnitus, hypothalamic dysfunction, seizures, mental changes, im-

pairment of recent memory, progressive or fluctuating dementia, and urinary or rectal incontinence (Antunes et al. 1980; Batnitzky et al. 1974; Cairns and Mosberg 1951; Camacho et al. 1989; Çetinalp et al. 1994; Ferry and Kempe 1968; Hall and Lunsford 1987; Hernesniemi and Leivo 1996; Kelly 1951; Little and MacCarty 1974; Mathiesen et al. 1997; Poppen et al. 1953; Stookey 1934; Yenermen et al. 1958). The association of gait disturbances and urinary incontinence in the absence of headache and papilledema produce a clinical picture closely resembling that of normal-pressure hydrocephalus (Little and MacCarty 1974). The so-called "classical history" with intermittent, position-dependent headaches is the exception rather than the rule (Antunes et al. 1980; Batnitzky et al. 1974; Hall and Lunsford 1987; Kelly 1951; Little and MacCarty 1974; Yenermen et al. 1958). The duration of symptoms range from some hours to several years (Ferry and Kempe 1968; Kelly 1951; Little and MacCarty 1974). Neurological examination may reveal papilledema, cerebellar signs, nystagmus, as well as Babinski's signs.

Colloid cysts may produce an increased intracranial pressure by intermittent obstruction of the passage of cerebrospinal fluid at the level of the interventricular foramina, with the cyst serving as a ball-valve, or by impaction of the cyst in the third ventricle. If the obstruction is complete and prolonged, the acute decompensated obstructive hydrocephalus may lead to sudden death of the patient. As another mechanism, reflex effects involving the cardiovascular centers near the third ventricle have been discussed (Ryder et al. 1986). Sudden death has been reported to occur in about 10% of the patients with colloid cysts of the third ventricle (Little and MacCarty 1974) and sudden deterioration may be preceded by longer symptomatic periods. Table 1 gives a review of 98 cases of sudden death due to a colloid cyst of the third ventricle as reported in the literature. The age of the patients ranged from 6 to 79 years (mean 29.6 years) in 40 females and 41 males. In 18 cases the age was not given and in 17 cases the sex was not given. The cyst size ranged from 0.8×0.8 cm to 7.9×6.5 cm in diameter. The onset of symptoms prior to death ranged from 17 years to only a few hours. As seen in Table 1, patients deteriorating rapidly have usually been symptomatic for a considerable time before deterioration and death.

In case 1 of our study an acute onset of headache after an intercontinental air flight is similar to the three cases reported by Nelson and Haymaker (1957). In one of these cases, a pilot died from acute hydrocephalus due to impaction of a colloid cyst in the third ventricle soon after the flight was grounded. In the other two cases, symptoms of acute hydrocephalus developed during flight. The authors pointed out that hypoxidosis may be encountered in aircraft manoeuvres as a result of negative as well as positive gravitational forces which increase the intracranial pressure and induce impaction of colloid cysts in the third ventricle.

The diagnosis of a colloid cyst is suggested by CT or MRI. The characteristic CT appearance shows a round or ovoid lesion inside the third ventricle, which may be hy-

Table 1 Sudden death in patients with colloid cysts of the third ventricle

Authors	Age (years)/ Sex	Size (cm)	Symptoms	Onset of symptoms prior to death
Batnitzky et al. (1974)	79/M	NM	Found in coma at home	–
Beutler (1921)	41/M	1.7 × 1.9	Headache, vomiting	2 years
Boldrini (1931)	24/M	NM	Vomiting, unconsciousness	Few hours
Brun & Egund (1973)	16/F	1.5	Headache, nausea, vomiting	1 month
	26/F	1.4	Headache, nausea, vomiting	9 years
	16/F	1.5	Headache, nausea, tiredness	2 months
	43/F	1.7	Headache, vomiting, tiredness	10 days
Busch (1917)	28/M	NM	Dizziness, nausea	Sudden attack
Byard & Moore (1993)	9/M	1.0	Headache, vomiting	2 days
Cairns & Mosberg (1951)	27/M	NM	Headache, vomiting, paresis	9 months
	46/F	NM	Headache	1–2 days
	48/F	NM	Headache, vertigo, memory loss	6 months
Chan & Thompson (1983)	29/M	1.1	Headache	3 months
	62/F	1.5	Headache, nausea, vomiting	3 days
DiMaio & DiMaio (1974)	14/M	5.0 × 3.8 × 3.8	Headache	1 day
	23/M	3 × 1 × 1	Headache	Recent onset
	19/F	1.5	Headache	10 months
DiMaio et al. (1980)	39/M	2.2 × 1.7	Headache, vomiting, unconsciousness	3 days
Drennan (1929)	19/F	NM	Headache	2 years
	23/M	NM	Vomiting, rectal incontinence	8 hours
Filkins et al. (1996)	13/F	NM	Headache, lethargy, double vision	3 months
	26/F	NM	Headache	NM
	30/F	NM	Headache	2 days
	33/F	NM	Headache, photophobia, unconsciousness	6 years
Greenwood (1949)	8/M	1.0	Seizures, unconsciousness	1 day
	25/F	1.2	Headache, seizures, unconsciousness	1 day
	26/M	2 × 2.5	Headache, seizures, unconsciousness	2 years
Grondahl (1908)	23/M	NM	Headache	Years
Grossiord (1941)	14/72	No further information		
Hall (1913)	18/F	NM	Headache	6 months
Heaven & Young (1959)	19/M	1.9 × 1.6	Sudden collapse	NM
	26/M	1.4 × 1.5 × 1.4	Headache	NM
	30/M	0.8 × 0.8	Vomiting, seizures	2 days
	36/M	1.8 × 1.8 × 2.2	Headache, nausea, vomiting, visual disturbances	8 months
	42/M	2.4 × 1.7 × 1.3	Sudden death	NM
Hernesniemi & Leivo (1996)	25/F	NM	Sudden death	NM
	36/F	NM	Admitted in coma	NM
	36/F	NM	Admitted in coma	NM
	39/F	NM	Sudden death	NM
	46/F	NM	Sudden death	NM
Humphreys et al. (1993)	13/F	NM	Headache, vomiting	7 days
Kelly (1951)	52/F	NM	Headache, vomiting, leg weakness, lethargy	5 months
	48/F	NM	Headache	10 days
Kuchelmeister (1992)	23/M	1.0	Headache, nausea, vomiting, seizures	5 years
	32/F	0.9	Headache, nausea, vomiting	9 years
	34/F	1.3	Headache, nausea, vomiting	Several days
Leestma & Konakei (1981)	17/F	1.8	Headache, nausea, vomiting	1 day
	30/F	1.5	Headache, nausea, vomiting	3 days
	30/M	2.0	Headache, vomiting	17 years
	33/F	1.5	Headache, nausea, vomiting, collapse	2 years
Little & MacCarty (1974)	4/38	No further information		
Lu (1961)	26/F	7.9 × 6.5	Headache, blurred vision	12 years
	35/F	5.4 × 4.5	Seizures, coma	13 days
Mathiesen et al. (1997)	18/M	1.8	Headache, nausea	4 months
	40/M	1.3	Headache	5 years
McDonald (1982)	6/F	1.2	Vomiting, unconsciousness, seizures	24 hours
	21/M	NM	Headache	4 days

Table 1 (continued)

Authors	Age (years)/ Sex	Size (cm)	Symptoms	Onset of symptoms prior to death
Nelson & Haymaker (1957)	31/M	3.0	Headache	Several years
	35/M	2.8 × 2.1 × 1.8	Headache	7 months
	41/M	2.0	Headache	NM
Opeskin et al. (1993)	13/F	1.5	Headache, vomiting, photophobia	24 hours
Read (1990)	6/F	20.0 × 1.8 × 1.2	Headache, vomiting, seizures, neck stiffness	3 days
Rinder & Cannon (1933)	47/F	1.5	Headache	9 years
Saulsbury et al. (1981)	6/F	3.0	Headache, vomiting, gait disturbances, lethargy	1 day
Schröder et al. (1990)	18/M	NM	NM	NM
Shaver (1940)	19/F	2.0	Headache	2 months
	NM/F	2.0	Headache	Several months
Sigrist (1943)	22/M	2.5	Headache, nausea, vomiting	Several hours
	46/F	2.0	Headache, dizziness	Several hours
Sjövall (1910)	53/M	1.8 × 1.5	Headache	4 months
Stedman (1883)	28/M	NM	Headache	1 year
Sundberg (1923)	32/F	1.2 × 0.8	Headache, vomiting, unconsciousness	10 months
Wallmann (1858)	51/M	3.2 × 1.6	Memory disturbances	14 years
Weisenburg (1910)	27/M	NM	Headache, nausea, vomiting, poor vision	NM
Yenerman et al. (1958)	19/M	2.0	Headache, nausea, vomiting, seizures	4 days
	22/M	1.1 × 0.9	Headache	44 hours
	22/M	2.0 × 1.2 × 1.2	Headache, nausea, vomiting, neck stiffness	4 days
	25/M	1.4	Unconsciousness	Few hours
	27/M	2.5	Headache, nausea, vomiting	41 hours
	29/M	1.5	Headache, deterioration of memory	Sudden onset
	40/M	1.9 × 1.3 × 1.2	Headache, vomiting	3 days
	42/M	3.0 × 2.5 × 1.5	Headache, vomiting	50 hours

NM not mentioned, *F* female, *M* male

perdense, isodense, or hypodense, and occasionally with enhancement after contrast medium administration (Antunes et al. 1980; Camacho et al. 1989; Çetinalp et al. 1994; Hall and Lunsford 1987; Maeder et al. 1990; Mathiesen et al. 1997; Michels and Rutz 1982). In rare instances, calcifications might be detected inside the cyst (Hall and Lunsford 1987; Michels and Rutz 1982). MRI displays a large variety of signal characteristics, but most frequently, colloid cysts are hyperintense on T1-weighted images and hypointense on T2-weighted images (Levrier et al. 1992; Maeder et al. 1990; Wilms et al. 1990). The associated, variably severe hydrocephalus of the lateral ventricles is not proportional to the cyst size (Weisz and Fazal 1983). In the differential diagnosis, ependymoma, intraventricular glioma, craniopharyngioma, choroid plexus papilloma or, rarely, meningioma, arteriovenous malformations, or aneurysms of the tip of the basilar artery must be considered (Antunes et al. 1980).

The clinical signs and symptoms of colloid cysts are non-specific and may be misinterpreted as migraine headaches, transient ischemic attacks, or other brain tumor entities. Furthermore, neither the cyst size, nor the degree of ventricular dilatation as assessed on CT or MRI scans, nor the duration of symptoms prior to the patient's collapse can be reliably used to indicate the risk of sudden neurological deterioration or death of the patient (Ryder et al. 1986). Due to this inability to predict the patients who

will deteriorate and the potential of colloid cysts for sudden death, even asymptomatic colloid cysts discovered incidentally on CT or MRI scans should be removed surgically (Hall and Lunsford 1987; Little and MacCarty 1974; Ryder et al. 1986). Despite their unfavourable location within the third ventricle, most of these lesions can be removed successfully by various neurosurgical approaches, e.g. the transcortical or transcallosal operations or by stereotactic or endoscopic aspiration (Apuzzo 1987; Hall and Lunsford 1987; Hernesniemi and Leivo 1996; Lewis et al. 1994). Recurrence of the lesion is the exception rather than the rule (Antunes et al. 1980).

The emergency management of choice for colloid cysts is ventricular shunting (Weisz and Fazal 1983). Lumbar puncture before CT scan should be strictly avoided as this may contribute to death by accelerating the cerebellar tonsillar herniation and brain-stem compression (Brun and Egund 1973; Little and MacCarty 1974; Opeskin et al. 1993). Prompt diagnosis and early surgical intervention are mandatory in order to prevent or to minimize the damage that may be caused by an acute increase in intracranial pressure (Weisz and Fazal 1983).

A broad spectrum of neuropathological alterations is encountered at medicolegal autopsies e.g. brain tumors, trauma, intoxication, infectious diseases, or vascular diseases (Betz and Eisenmenger 1995; Neuen-Jacob et al. 1993; Skullerud et al. 1991). The present cases are illus-

trative examples of sudden death due to colloid cysts of the third ventricle. Both patients had a long-standing history of intermittent headache with an acute deterioration, and finally died. To avoid such fatal complications prompt diagnosis using CT or MRI is essential, since colloid cysts are histologically benign lesions that can be removed safely by neurosurgical intervention.

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Note added in proof Two further cases has been reported recently (Macaulay RJB, Felix I, Jay V, Becker LE (1997) Histological and ultrastructural analysis of six colloid cysts in children. *Acta Neuropathol* 93:271–276): In the first case, a 15-year-old girl with a 1-week history of intermittent headaches died due to a colloid cyst of the third ventricle measuring 2 × 1 cm. In the second case, a 13-year-old girl experienced drowsiness, headache and vomiting while airborne. She died 24 hours after onset of symptoms and autopsy revealed a 2-cm diameter colloid cyst in the third ventricle.