A diverticulum of the brain ventricle is a pathological condition characterized by an extracerebral cerebrospinal fluid (CSF) filled outpouching of the ventricular wall [17, 18]. The prevalence of a ventricular diverticulum in the case of congenital occlusion hydrocephalus may reach 24% [38]. “Preferable” origins of a diverticulum are believed to be as follows:

1. The choroidal fissure (fissura choroidea) adjacent to the medial wall of the atrium of the lateral ventricle (atrium ventriculi lateralis) with bulging of diverticula through the tentorium into the quadrigeminal and supracerebellar cisterns [30];
2. The posterior wall of the third ventricle through which an outpouching extends into the cavity of the quadrigeminal cistern [2, 12, 26];
3. The ascending portion of the fourth ventricle at the rostral level of the superior medullary velum (velum medullare superius) [27, 30].

The extension route of a lateral ventricular diverticulum to the quadrigeminal cistern is believed to pass through the tela choroidea and the antrum [25]. The most thinned part of the trigone of the lateral ventricle is located between the crus of fornix (crus fornicis) and the occipital forceps (forceps major). An outpouching is directed towards fissure of Bichat past the fimbria hippocampi [5, 18]. The medial choroidal fissure (fissura choroidea) in the cavum velum interpositum may be an important anatomical structure for the extension of a lateral ventricular diverticulum into the supracerebellar cistern [25].

According to pathomorphological studies, cerebrospinal fluid-filled diverticula at the choroidal fissure level are covered by the arachnoid sheath or its fragments and by components of the neuroepithelial tissue [8, 17, 27]. A diverticulum is always associated with the cerebral ventricular system and usually communicates with it [2, 18]. Arachnoid cysts, contrary to a diverticulum, may be located aside the cerebral ventricles and arise from the cerebral cisterns, whereas a diverticulum arises from the cerebral ventricle [3, 9, 19]. The arachnoid sheath is believed to form both the diverticulum and arachnoid cyst walls. Secretory activity of the diverticulum wall, unlike that of the arachnoid cyst walls, is highly questionable, although it has not been scientifically confirmed yet [18].

A diverticulum of dilated cerebral ventricles is occasionally termed in the literature [23] as a “congenital porencephalic cyst of the median line”, and its typical clinical syndromes are described. Some authors [36] noted that a diverticular cyst results from dysgenesia of the brain structures of the median line and is not caused by hydrocephalus.

A distinctive feature of a diverticulum distinguishing it from porencephaly is that brain tissue deficiency (destructive brain lesions) is detected in porencephaly. A disrupted portion of brain tissue (by trauma, ischemia, inflammation, etc) is filled with CSF. In contrast to congenital porencephaly, symptomatic ones develop in patients of all ages due to injuries and brain diseases [7, 13, 23].

As described in the literature [21, 24—26, 33, 34], a cerebral ventricular diverticulum may be associated with cavernous angioma in the fourth ventricle, occipital encephalocele, intraventricular and paraventricular...
tumors, vascular malformations, and congenital obstruction of the ipsilateral foramen of Monro.

A lateral ventricular diverticulum may often present in the form of other nosologies (hydrocephalus, arachnoid cyst, tumor, etc) [31, 32]. The pathognomonic clinical signs of a ventricular diverticulum are most likely lacked or hidden by symptoms of the underlying disease. A ventricular diverticulum presented as cerebellar ataxia was reported [38]. However, the neurological symptoms are always nonspecific and often minimal, which necessitates purposeful examination.

Diagnosis of a cerebral ventricular diverticulum using introspective methods involves the use of both noninvasive (percutaneous sonography, magnetic resonance imaging (MRI), cardiac-gated phase-contrast MRI, and computed tomography (CT) in various modes) and invasive (CT ventriculography, 3D MSCT angiography, and cerebral angiography) techniques [2, 10, 29, 35, 37]. The cumulative information provided by noninvasive methods in most cases replaces the need for contrast enhancement. T. Naidich et al. [32] identified 10 computed tomography features of a cerebral ventricular diverticulum. On a MRI scan, a diverticulum looks like a clearly defined region of a modified signal that has the equal intensity to the CSF signal in all sequences. At the same time, a diverticulum is not clearly delineated from the walls of adjacent lateral ventricles [37]. Therefore, a certain combination of introspective examinations may sufficiently objectively characterize the morphological features of a cerebral ventricular diverticulum. Objectivization of involvement of a diverticulum in the overall CSF circulation is also a significant aspect of the morphological and functional characterization of the pathological process.

Differential diagnosis of a ventricular diverticulum has been performed with an arachnoid cyst of the area of the tentorial notch (extending from the quadrigeminal or ambient cistern), a neuroepithelial cyst of the posterior cranial fossa, a cystic tumor of the pineal body, etc [2, 4, 10, 16, 38]. Detection of the diverticulum opening (ostium) or a communication between the cyst and ventricular system is important for diagnosis [18, 30].

Regarding treatment of a ventricular diverticulum, this disease is believed to occur asymptomatically and rarely requires surgical treatment [17]. Most clinicians prefer surgical treatment in the case of symptomatic progression of the disease [18, 34]. A diverticulum regression may occur after placement of a CSF shunt, neuroendoscopy, or tumor resection [25, 27, 30].

In the case of a lateral ventricular diverticulum, certain difficulties in the interpretation of diagnostic indicators may be related to the lack of sufficient theoretical and summarising studies in the special neurosurgical and radiological literature. The reason for this probably is that a diverticulum is not an object of direct treatment. In addition, there is no unified etiologic concept, but there are numerous hypotheses of the pathogenic mechanism. All the above mentioned was the basis for presenting this case report.

A 12-year-old female patient was admitted to the somatic department of a regional multipurpose center of emergency medicine, complaining of convulsive seizures and dizziness.

The past medical history revealed that the patient was detected with an increased head circumference at the age of 1 year. However, adequate attention was not paid to the fact. At the age of 9 years, limb trembling and poor scholarship developed after the emotional stress (due to a hyperactive bladder during a school class). The patient underwent MRI at the age of 11, since the administered treatment had no effect. Based on detected hydrocephalus with the Evans’ index of 0.61 (Fig. 1), a local neurosurgeon suggested surgery for hydrocephalus, but the child’s parents refused the treatment. About a year later, tonic convulsions suddenly developed at night. She was admitted to the pediatric department, but then was transferred to the intensive care unit because of an increased frequency of multiple convulsive attacks. Multiple convulsions were reversed.

Life history: the patient was the child of the first pregnancy. Her mother was treated at a hospital during the fourth month of pregnancy due to threatened miscarriage. No complications at delivery. The girl started walking at the age of 1.5 years; she started school at the age of 7 years. The girl had good scholarship at elementary school.

Objective evaluation of the patient was as follows: the general condition was very poor; impaired consciousness (deep stupor); poorly communicating. Instructions were carried out partially, after a pause. Slight exotropia. Sideward glance was limited. The abdominal reflexes were depressed. The tendon reflexes were brisk, D>S. Muscle tone was spastic. Tetraparesis (muscle grade of 1 or 2). Poor response to painful stimuli. Stiff neck, Kernig’s symptoms. Locally: a hydrocephalic head shape with the head circumference of 59 cm (+4.5 cm).

Electroencephalographic examination (EEG) revealed a decrease in the convulsion threshold and the foci of pathological activity of subcortical structures. Ophthalmoscopy revealed angiopathy of retinal vessels and mild venous stasis. MRI detected a fluid intensity mass lesion above the cerebellum associated with triventricular hydrocephalus (Fig. 2).

Differential diagnosis was performed to differentiate among three nosologies: an arachnoid cyst of the quadrigeminal cistern, a cystic tumor of the pineal region, and a diverticulum of the third or lateral ventricle [6, 10].

Multislice computed tomography (MSCT) with intravenous contrast revealed no tumor mass (Fig. 3). Contrast-enhanced MRI was not conducted due to patient intolerance to a paramagnetic contrast agent.

For the purpose of differential diagnosis of an arachnoid cyst, the patient underwent MSCT
ventriculography through the anterior horn of the right lateral ventricle. MSCT ventriculography confirmed the diverticulum of the lateral ventricle (Fig. 4). CSF pressure measured in the course of ventricular puncture was 240 mm w.g. Laboratory CSF parameters were as follows: cytosis was 5/3 (under the equal neutrophil-lymphocyte ratio); the protein content was 0. Removal of a certain amount of CSF for the “relief” purpose during ventriculography had an effect—the patient consciousness level improved to moderate stupor.

The patient was prepared for CSF shunt surgery, but unfortunately, the plan was never implemented. The patient died due to an idiopathic acute intestinal disease accompanied by severe diarrhea and complicated by a hypovolemic shock. No autopsy study was carried out. It is likely that hydrocephalus complicated by the diverticulum, as a competitive disease, also played a role in the lethal outcome.

Case features:
1. The diverticulum was an acquired disease.
2. The diverticulum development rate, i.e. the time during which the diverticulum grew up to a giant size and contributed to decompensation, was approximately 14 months.
3. The development of a lateral ventricular diverticulum in symmetric hydrocephalus.

The poor clinical and past medical history data (due to a decompensated patient condition at admission) confine the objective evaluation of the pathological process progression. However, it may be assumed that an outpouching of the ventricular wall in one of the morphologically complex regions near the choroid glomus (glomus chorioideum) occurred due to progressive CSF hypertension in the cerebral ventricular system [1, 14]. In this region, CSF “found” a weak spot by stretching and pulling out tissues along the pathway of least resistance towards the supracerebellar cistern [28]. It was most likely the pulsion diverticulum (diverticulum e pulsione verum) [15, 31] formed due to pressure from within a hollow organ on its altered wall.

A detailed retrospective study of initial MRI images (of 2012, when the diverticulum had no obvious outpouching) revealed tomographic signs of a developing diverticulum (Fig. 5). The typical tomographic features of a diverticulum were also detected by repeated MRI

Fig. 1. MRI (2012).

a – axial sections, view inverse. Lateral ventricles are enlarged and the periventricular edema is determined; the third ventricle of the brain is enlarged as well; b – sagittal sections. The third and the fourth ventricles of the brain are clearly visible. Sylvian aqueduct is occluded.

Fig. 2. MRI (2013).

a – axial sections. Lateral ventricles are enlarged; the periventricular edema is evident. An irregularly oval-shaped pathological structure is determined in the projection of the quadrigeminal cistern; b – sagittal sections. Cerebellum is compressed from above by liquid mass. No the fourth ventricle and Sylvian aqueduct are visible.

Fig. 4. MSCT ventriculography.

CSF pressure measured in the course of ventricular puncture was 240 mm w.g. Laboratory CSF parameters were as follows: cytosis was 5/3 (under the equal neutrophil-lymphocyte ratio); the protein content was 0. Removal of a certain amount of CSF for the “relief” purpose during ventriculography had an effect—the patient consciousness level improved to moderate stupor.

The patient was prepared for CSF shunt surgery, but unfortunately, the plan was never implemented. The patient died due to an idiopathic acute intestinal disease accompanied by severe diarrhea and complicated by a hypovolemic shock. No autopsy study was carried out. It is likely that hydrocephalus complicated by the diverticulum, as a competitive disease, also played a role in the lethal outcome.

Case features:
1. The diverticulum was an acquired disease.
2. The diverticulum development rate, i.e. the time during which the diverticulum grew up to a giant size and contributed to decompensation, was approximately 14 months.
3. The development of a lateral ventricular diverticulum in symmetric hydrocephalus.

The poor clinical and past medical history data (due to a decompensated patient condition at admission) confine the objective evaluation of the pathological process progression. However, it may be assumed that an outpouching of the ventricular wall in one of the morphologically complex regions near the choroid glomus (glomus chorioideum) occurred due to progressive CSF hypertension in the cerebral ventricular system [1, 14]. In this region, CSF “found” a weak spot by stretching and pulling out tissues along the pathway of least resistance towards the supracerebellar cistern [28]. It was most likely the pulsion diverticulum (diverticulum e pulsione verum) [15, 31] formed due to pressure from within a hollow organ on its altered wall.

A detailed retrospective study of initial MRI images (of 2012, when the diverticulum had no obvious outpouching) revealed tomographic signs of a developing diverticulum (Fig. 5). The typical tomographic features of a diverticulum were also detected by repeated MRI
(of 2013) on the background of a pronounced cystic outpouching (Fig. 6). The firm knowledge of reference tomographic signs of a ventricular diverticulum would probably enable prognosing for diverticulum progression [32].

Taking into account all the above mentioned, certain questions and suggestions arise. Answering these questions could reveal the morphological and functional structure of cerebral ventricular diverticula:

1. The association of a ventricular diverticulum with hydrocephalus. Is a diverticulum always associated with hydrocephalus or it may be an independent disease?
2. Is a ventricular diverticulum a definite sign of occlusive hydrocephalus, or it may also develop in the association with communicating hydrocephalus? [22]
3. Is a ventricular diverticulum a sign of liquor hypertension, or it may occur under normal CSF pressure?
4. Is the persistent diverticulum localization in certain anatomical parts of cerebral ventricles due to the structural features of the liquor system of the brain or due to other causes?
5. What exactly a ventricular diverticulum is? Is it a compensatory mechanism for tension hydrocephalus (similar to spontaneous ventriculocisternostomy [13, 20, and 28]), a genetically determined process [23], or a combination of these phenomena?
6. How much stable is compensation of hydrocephalus in the case of diverticulum formation (by analogy with the Bechtereva’s mechanism of steady compensation states)?
7. Which intervention is effective in the case of a ventricular diverticulum (CSF shunt surgery, internal cyst-cisternal shunt, endoscopic surgery, or their combination) [16, 38]?
8. What are the duration and degree of regression for a lateral ventricular diverticulum after surgery [38]?
9. What is the probability of diverticulum recurrence after surgery?
10. How much is it clinically and practically reasonable to distinguish a cerebral ventricular diverticulum upon hydrocephalus (the impact of diverticulum detection on the treatment tactics?)

![Fig. 3. MSCT with intravenous contrast enhancement, axial sections. Regions accumulating a contract agent are not determined, which confirm the absence of tumor.](image_url)
Fig. 4. MSCT ventriculography. The cavity of diverticulum and the ventricular system are filling with contrast agent simultaneously.

a – axial sections; b – sagittal sections; c – frontal sections; the opening (ostium) of diverticulum is visualized (*).
According to the described case, it is clear that a lateral ventricular diverticulum is a disease that, under a particular clinical situation, requires a neurosurgical set of diagnostic (for indications) and invasive measures for the purpose of differential diagnosis [39]. It may be assumed that the use of an extensive complex of introscopic and neurophysiological examinations for each particular case will make possible to gain the morphological and functional characteristics and pathophysiological aspects of a cerebral ventricular diverticulum in detail. This, in turn, will facilitate systematization and classification of diverticula and, most importantly, will determine the right choice of the disease management. According to the literature [38], there have been attempts to classify ventricular diverticula by size, relationship with the tentorial notch, and relation to the superior cerebellar cistern.

The purpose of this report was not so much to describe a particular case of a cerebral ventricular diverticulum as to demonstrate and set out, if possible, promising directions in systematization of information on this phenomenon. The issues related to the features of surgical treatment of diseases associated with a diverticulum may help outline and correct possible directions of further research.
REFERENCES


Commentary

The article describes a rare case of a 12-year-old child with hydrocephalus complicated by the development of a diverticulum of the lateral ventricle. The article is well documented; the disease development is followed up over time clinically and by MRI data.

A very detailed analysis of the literature data is conducted that is dedicated to the anatomical features and pathogenesis of cerebral diverticula in children.

The article points out the difference in the origins of cerebral diverticula, arachnoid cysts, and porencephaly; differential diagnosis of these lesions is discussed, which is very important for the practitioner.

Diverticula of the cerebral ventricular system are a rare and still underexplored phenomenon. The article not so much solves the specified problem as brings up the right questions and makes one think about the principles of diagnosis and treatment of the disease.

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