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Intracranial arachnoid cyst in a 28-year-old man. A clinical case with a fatal outcome

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ABSTRACT

Most intracranial arachnoid cysts are thought to be non-tumorous, congenital, intra-arachnoid cerebrospinal fluid collections that account for about 1% of all intracranial space-occupying lesions. In children, the prevalence of this pathology is 2.6%; in adults, it reaches 1.4%. The disease is more often registered in men. Most often arachnoid cysts are supratentorial. Their most common locations are in the middle cranial fossa and the retrocerebellar cistern. Less often they can be detected on the convexity of the brain hemispheres; however, cases of arachnoid cysts at more unusual sites have also been described, including in newborns. The pathology is often characterized by an asymptomatic course, while certain symptoms may have an acute onset, which is due to compression of brain structures caused by the large cyst size.

This article describes a clinical case of a large intracranial arachnoid cyst in a 28-year-old man. It was not verified in the antemortem diagnosis, but was revealed according to the autopsy findings (macroscopic features of the cyst, histologic presentation with specific morphological changes, and findings of computed tomography of the cerebral hemispheres).

Keywords: intracranial arachnoid cyst, morphology

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Арахноидальная внутримозговая киста у мужчины 28 лет. Клинический случай с летальным исходом

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РЕЗЮМЕ

Арахноидальные внутримозговые кисты представляют собой чаще врожденные, отграниченные листками паутинной оболочки пространства (образования кистозного строения), заполненные спинномозговой жидкостью, распространенность их составляет до 1% от всех объемных образований данной локализации, процесс в большинстве случаев имеет доброкачественный характер течения. У детей частота выявления патологии составляет 2,6%, этот показатель у взрослых пациентов соответствует значению 1,4%, несколько чаще заболевание регистрируется среди мужчин. Наиболее часто подобные кисты имеют супратенториальное расположение, преимущественно они диагностируются в средней черепной ямке, в ретроцеребеллярной области, реже их можно обнаружить в конвекситальных отделах больших полушарий, также описаны случаи более редких особых локализаций, в том числе у новорожденных. Патология часто характеризуется бессимптомным течением, при этом возникновение определенной симптоматики может иметь острое начало, обусловлено крупными размерами кист со сдавлением структур головного мозга.

В статье представлено описание случая внутримозговой арахноидальной кисты больших размеров у мужчины 28 лет, не верифицированной прижизненно и выявленной лишь посмертно на основании данных патологоанатомического исследования (макроскопические особенности образования, гистологическая картина с характерными морфологическими изменениями и данные компьютерной томографии больших полушарий головного мозга).

Ключевые слова: арахноидальная внутримозговая киста, морфология

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии финансирования при проведении исследования.

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INTRODUCTION

Intracranial arachnoid cysts (IAC) are most often congenital collections of cerebrospinal fluid delimited by the layers of the arachnoid mater. They account for about 1% of all intracranial space-occupying lesions. The process in most cases is benign. There is a classification according to which primary (developmental cysts) and secondary cysts are distinguished. The former are caused either by splitting of the arachnoid membrane during fetal development with the subsequent formation of sacs and abnormal accumulation of cerebrospinal fluid in them or by a failure of the frontal and temporal embryonic meningeal merging, resulting in a duplication within the Sylvian fissure [1]. Secondary cysts are more likely to be a consequence of previous infections or traumatic damage to brain structures. They can also result from surgical interventions and intracranial hemorrhages, while the communication with the ventricular system of the brain can be preserved but is often absent [1–4].

The literature presents data on cases of multiple IACs in one patient [5]. There is also information that congenital IACs can be associated with a mutation of the *FOXC2* gene on chromosome 16 [6] and can also cooccur with Arnold – Chiari malformation [7]. Medical cases of IACs were first described by R. Bright in 1831 [8]. The research results obtained by W.N. Al-Holou et al. report that the incidence of the pathology is about 2.6 and 1.4% in children and adult patients, respectively [5, 9]. It is known that the disease is detected somewhat more often in men [5, 8].

Most often arachnoid cysts are supratentorial. Their most common locations are in the middle cranial fossa (34%) and the retrocerebellar cistern (33%). Less often they can be detected on the convexity of the brain hemispheres (14%) [5]. However, cases of arachnoid cysts at more unusual sites have also been described, in particular, in prepontine cisterns, quadrigeminal plate, and between the cerebral hemispheres [10–12].

This pathology is registered in various age groups. Such cysts are often asymptomatic for a long time and become an incidental finding during structural and functional neuroimaging for unrelated symptoms and complaints [1]. A symptomatic IAC is a rare case and more often recorded in children [13]. However, researchers have described giant arachnoid cysts in young men who had acute neurological symptoms caused by severe compression of brain structures; the sizes of the cysts were 12.3 × 16. × 7.9 cm and 6.5 × 11.5 × 12.5 cm [14, 15].

There have been few studies addressing the morphology of arachnoid cysts. The main studies date back to 1972–2000. So, K. Rabiei et al. (2014) presented a detailed description of the morphological features of these formations and showed possible differences in the structure of the epithelial lining and extracellular components in the walls of the studied cystic brain lesions, including the presence of squamous and ciliated epithelium, as well as glial and neuronal components in typical arachnoid tissue. The discovered structural features of IACs allowed the authors to suggest that they have different barrier properties and characteristics of fluid flow, which may be a determining factor in assessing growth rates and susceptibility to relapse [16].

This article describes a clinical case of a large intracranial arachnoid cyst in a 28-year-old man. It was not verified in the antemortem diagnosis, but was revealed according to the autopsy findings (macroscopic features of the cyst, histologic presentation with specific morphological changes, and findings of computed tomography of the cerebral hemispheres).

CLINICAL CASE

On May 26, 2021, Patient F., 28 years old, was admitted to the University Clinics of Siberian State Medical University (Tomsk) via the emergency room. According to the medical history, the patient complained of the body temperature rising to 37.2˚C, severe weakness, and headache. These symptoms appeared acutely on the day of hospitalization. He came to the pharmacy, where his condition deteriorated significantly. The pharmacy staff called an ambulance.

The medical history reports that the patient had several tick bites in May. The patient was not examined for this reason and did not take any preventive measures from tick-borne encephalitis and Lyme disease (tick-borne borreliosis). Taking into account the symptoms and medical history, the patient was transported to the Infectious Disease Clinic of Siberian State Medical University. Upon admission, the clinical examination revealed that the patient was in a severe condition, body temperature was 37.3˚C, blood pressure – 100/60 mm Hg, heart rate – 88 bpm, and saturation rate (SpO_2) – 98%. The patient was conscious but had difficulty communicating. Facial hyperemia and sweating were noted. Neurological status during the examination in the emergency room revealed hyperesthesia, nuchal rigidity (four fingerbreadths), severe pain at the cranial nerve exits, palpebral fissure S > D, tongue deviation to the right, and decreased muscle strength.

Given such symptoms as the acute onset of the disease, the presence of severe weakness, fever, and headaches, high tick activity, and episodes of tick bites in history, upon admission to the clinic, the patient was diagnosed with A84.0 Far Eastern tick-borne encephalitis (Russian spring – summer encephalitis), form of neuroinvasive encephalitis, acute onset, severe condition. In accordance with this diagnosis, an examination and treatment plan were determined. Given the severe condition, the patient was immediately transported to the intensive care unit.

On May 27, 2021, the neurological examination showed that the patient was in a poor condition, which deteriorated gradually; neurological deficit increased; there was a trend toward hypotension. The patient was unconscious. He did not answer questions and did not follow commands, which made it impossible to conduct coordination and other tests. The following was observed: palpebral fissures and nasolabial folds D = S, eye pupils D = S narrowed, gaze fixed in the center. There were nuchal rigidity (four fingerreadths) and positive Kernig and Brudzinski signs. Reflexes and tone of

the upper and lower extremities were preserved. Abdominal reflexes were preserved. Pathological foot and hand signs were not detected. There were no reliable signs of pelvic disorders. On May 27, 2021, mechanical ventilation was used to support the patient's breathing. The patient started receiving inotropic therapy with a gradual increase in the dose of administered drugs.

Upon emergency admission, a lumbar puncture, magnetic resonance imaging (MRI) of the brain, as well as an enzyme-linked immunosorbent assay (ELISA) of blood were conducted to detect antibodies (Ab) and antigens (Ag) to tick-borne encephalitis and borreliosis virus in order to clarify the diagnosis and chose the treatment strategy. The result of cerebrospinal fluid examination (lumbar puncture): colorless, slightly turbid liquid (volume 2.5 ml); protein 0.2 g / l (reference 0.22–0.33 g / l); cytosis 26 in 1 μl (reference 4–5 in 1 μl); glucose 3.4 mmol / l. Cerebrospinal fluid (CSF) analysis: neutrophils 89%, lymphocytes 8%, monocytes 3%, plasma cells 0, eosinophils 0, basophils 0. Blood ELISA result was as follows: anti-tick-borne encephalitis IgM (–), IgG 160 U / ml; Lyme IgM (–) and IgG (–) antibodies.

MRI of the brain revealed the presence of an oval-shaped intracranial cystic lesion in the left frontal lobe (66 × 48 × 55 mm). There was expansive growth of a heterogeneous structure with signs of high protein content (the doctor speculated about the dysembryogenetic origin of the identified neoplasm). The contours of the cystic tumor were uneven and unclear. The cyst had a capsule of 2–5 mm thick. Areas of calcification were identified in the lower part of this formation. The anterior horn of the lateral ventricle was deformed and compressed. In the anterior sections, the interventricular septum was displaced to the right up to 6.8 mm. The sella turcica had a regular shape and size. The suprasellar cistern prolapsed into the sellar cavity, the pituitary gland was pushed to the bottom and flattened to 22 mm (changes similar to the empty sella syndrome). The intracranial segment of the right vertebral artery was narrowed to 1 mm; the left vertebral and basilar arteries were 2.5 mm.

The MRI protocol described the presence of areas of acute brain ischemia in the cortical parts of the right parietal lobe and in the cortical parts of the left parasagittal parietal lobe; their dimensions were up to 4 × 6 mm. The convexity subarachnoid space in the left parietal – occipital region was narrowed and was not distinct during the examination. Figure 1 shows MRI images of the brain. The changes in blood parameters, including those with a significant deviation from the reference values, are presented in Table. The patient's coagulogram and other blood biochemistry and general urinalysis parameters were within the normal range. The electrocardiogram (ECG) dated May 28, 2021 revealed the vertical axis of the heart, sinus rhythm of 96 beats per min, incomplete right bundle branch block; increased load on the right atrium; disruption of ventricular repolarization in leads 3 and aVF; changes in the ST segment and T wave with weak elevation in the precordial leads (ECG data suggest the possible presence of pericarditis, myocarditis, and / or cerebrovascular disorders); QT interval was within the normal range.

After conducting brain MRI and receiving an impression about the presence of a volumetric neoplasm in the left frontal lobe, a telemedicine consultation with a neurosurgeon was requested. Taking into account the symptoms, MRI results, and results of additional laboratory and instrumental research, the neurosurgeon diagnosed an abscess of the frontal lobe of the left hemisphere as the

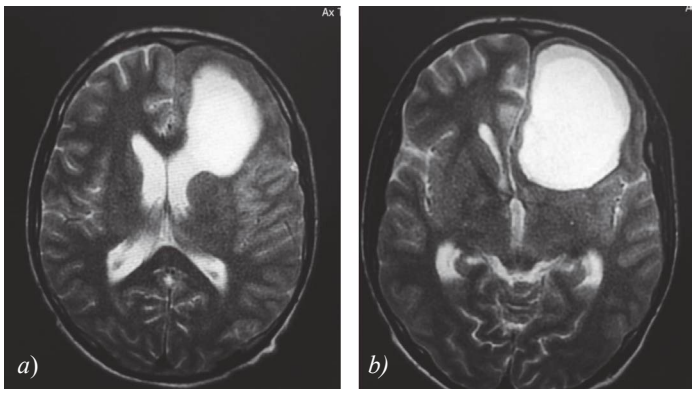


Fig. 1. Brain MRI. A cystic lesion in the left frontal lobe pressing against the adjacent brain tissues and adjacent structures (*a*, *b*)

underlying disease. It was recommended to transfer the patient for further treatment to the Neurosurgery Department. However, it was impossible to transport the patient and perform surgery due to the severity of the patient's condition. Therefore, it was decided to hold a second consultation when the hemodynamics stabilized.

Table

Patient's blood parameters

Blood counts	Changes in blood parameters		
	May 26, 2021	May 28, 2021	May 29, 2021
Red blood cells	4.24×10^{12} / l	4.86×10^{12} / l	4.9×10^{12} / l
Hemoglobin	133 g / l	155 g / l	162 g / l
White blood cells	9×10^{9} / l	19.2×10^{9} / l	18.5×10^{9} / l
Hematocrit	40%	45%	45%
Platelets	245×10^{9} / l	339×10^{9} / l	268×10^{9} / l

Over the next 3 days, the patient's condition was assessed as severe with negative dynamics (stage 3 coma). Heart rate was 126 beats per minute. Eye pupils D = S did not respond to light. On May 28, 2021, the body temperature rose to 37.6˚C; leukocytosis dramatically increased (19.2×10^{9} / l); a pronounced trend toward hypotension appeared; blood pressure was 80–60 / 50–40 mm Hg; hypernatremia of 160.2 mmol / l and hypokalemia of 2.32 mmol / l were detected. According to the progress notes, the condition was assessed as extremely severe and terminal with a pronounced trend toward hypotension.

On May 30, 2021, asystole was recorded. Medical staff started to perform cardiopulmonary resuscitation (CPR). Although CPR was effective and the heart rhythm restored in 5 minutes, blood pressure remained extremely low and unstable. Fifteen minutes later, with a newly emerging episode of asystole and signs of severe hypotension, medical staff started performing CPR which lasted for 30 minutes. CPR was not successful. The biological death was pronounced. The final clinical diagnosis was formulated as follows:

Underlying disease: an abscess in the frontal lobe of the left brain hemisphere.

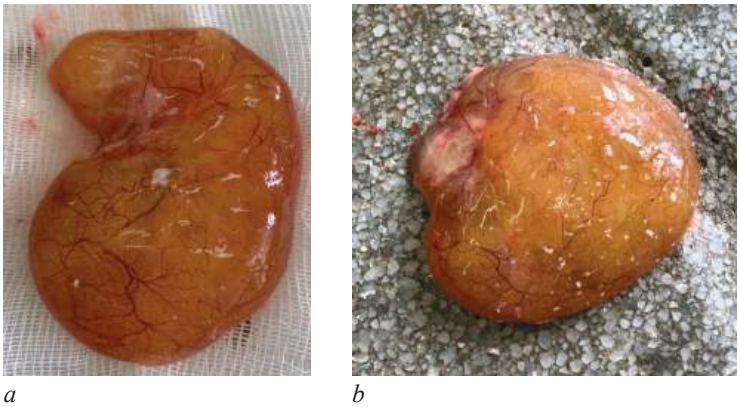
Complications: cerebral edema. Multiple organ dysfunction syndrome. Asystole. Cardiopulmonary resuscitation: mechanical ventilation, indirect heart massage, norepinephrine injection.

Concomitant diseases: incomplete bundle branch block.

The body of the deceased was sent for autopsy to the Anatomic Pathology Department of Siberian State University clinics.

PATHOLOGY EXAMINATION

The gross examination showed significant morphological changes in the brain and lungs. The meninges had a typical anatomical structure. The brain was enlarged due to edema (1,600 g). The brain was soft. The furrows and convolutions were unevenly smoothed. On the surface of the cerebellar hemispheres there was a distinct occlusal groove caused by the herniation into the foramen magnum. The cerebellum was flabby and watery; the structure was preserved on sections. The pons and brainstem were characterized by soft, flabby consistency; the anatomical structure remained intact. In the horizontal section of the white matter of the frontal lobe of the left hemisphere, a large (6.8 × 5 × 5 cm), clearly demarcated cystic structure with a thin transparent smooth capsule was found. It was located subcortically and had an oval shape. This cyst was represented by jelly-like masses of soft and elastic consistency of a yellowish-brown color with the presence of thin, dense layers of whitish tissue (Fig. 2). In the areas adjacent to the cyst, the brain



a *b*

Fig.2. Arachnoid cyst in the brain (macroscopic changes). The cyst appears as an oval-shaped, soft, elastic, yellowish-brown jelly-like masses with a thin, lucent capsule (*a*, *b*)

was flabby. The boundary between the white and the gray matter was not distinct. The cyst compressed the brain tissue. It made the shape of the anterior horn of the lateral ventricle slit-like; its cavity was not visually determined.

During the autopsy and examination of the chest organs, it was noted that the layers of the pleura were not changed; the lungs had the correct anatomical shape and were of normal size. The rest of the lung tissue was homogeneous, elastic, dark red in color; foamy, slightly pinkish liquid and liquid dark cherry-colored blood flowed abundantly from the surface of the sections. Gross autopsy did not reveal any significant pathological changes in other organs and tissues. Tissue fragments of internal organs were taken for the microscopic examination. The brain tissue fragments and fragments of the cystic lesion in the left frontal lobe of the brain and lungs were taken with specific marking.

The microscopic examination of brain tissue specimens revealed pronounced edema with the formation of areas of pronounced rarefaction of the neuropil, congestion in intra-organ vessels, diapedesis-type perivascular hemorrhage in single fields of vision, and leukostasis in the lumen of some capillaries. Pronounced edema was also detected throughout tissue fragments taken from the cerebellum and brain stem. The histologically described cystic formation in the frontal lobe of the left hemisphere was represented by abundance of chaotically located small-cell fibrous eosinophilic structures. The structures resembled multiple cystic cavities with deformed lumens and thin walls. Some cavities had homogeneous eosinophilic content.

In the peripheral areas of this cyst, an unevenly expressed inflammatory infiltrate was found with the presence of lymphoid cells, large cells like monocytes and macrophages with abundant cytoplasm; segmented leukocytes predominated in the cellular composition. The cyst was covered with one layer of closely adjacent columnar epithelial cells, on the apical part of which, multiple cilia were clearly detected. It also contained a large number of blood vessels, such as arteries, veins, as well as capillary-like vessels with thin walls in a state of pronounced congestion. In some fields of vision, only few large cells were identified, whose cytoplasm contained multiple round nuclei (multinucleate cells); small basophilic calcifications were also found in the cyst wall (Fig. 3). In the sections of the brain tissue adjacent to the cyst in the left frontal lobe, foci of acute ischemic damage were found with disruption of the matter structure. Signs of edema were significantly more pronounced; unchanged red blood cells were minimally detected in the foci.

Plethora and pronounced alveolar edema were observed throughout the fragments of lung tissue. In sections of the lower lobe of the left lung, the structure of the lung tissue was disrupted due to the presence of abundant purulent inflammation foci with a large number of segmented leukocytes and purulent bodies; erythrocytes were also detected in the exudate. Numerous red blood cells were also detected in the exudate. In some fields of vision, the interalveolar septa were not differentiated due to the described purulent exudates. In some fields of vision, the alveolar septa were not differentiated due to this exudate, which was most pronounced and had a predominant localization around the bronchi. The walls of the bronchi were unevenly thickened due to edema and pronounced inflammatory infiltrate. The boundaries of the bronchi in some fields of vision were not clearly differentiated, the walls were destroyed, the bronchial epithelium was absent, diffuse necrotic areas of the epithelium were identified. Masses of detritus and abundance of neutrophils with purulent bodies were found in the lumens of the bronchi. Leukostasis was detected in the capillaries of the lungs.

The morphological changes in the lung tissue described in the microscopic examination corresponded to focal purulent hemorrhagic bronchopneumonia of the indicated localization accompanied by alveolar edema and severe acute congestion with the presence of leukostasis. It should be noted that only microscopy revealed significant histologic changes in the myocardium: unevenly expressed stromal edema, partial fragmentation of muscle fibers with the presence of inflammatory infiltrate with lymphocytes, single plasma cells, and larger cells like macrophages and segmented leukocytes in the stroma between the myocytes. This infiltrate was detected in the interstitium and extended to cardiomyocytes in many fields of vision. In the zones with a more pronounced inflammatory infiltrate, the contours of myocytes were unclear, foci with partial destruction of muscle fibers and leukostasis in the capillaries were detected. In the

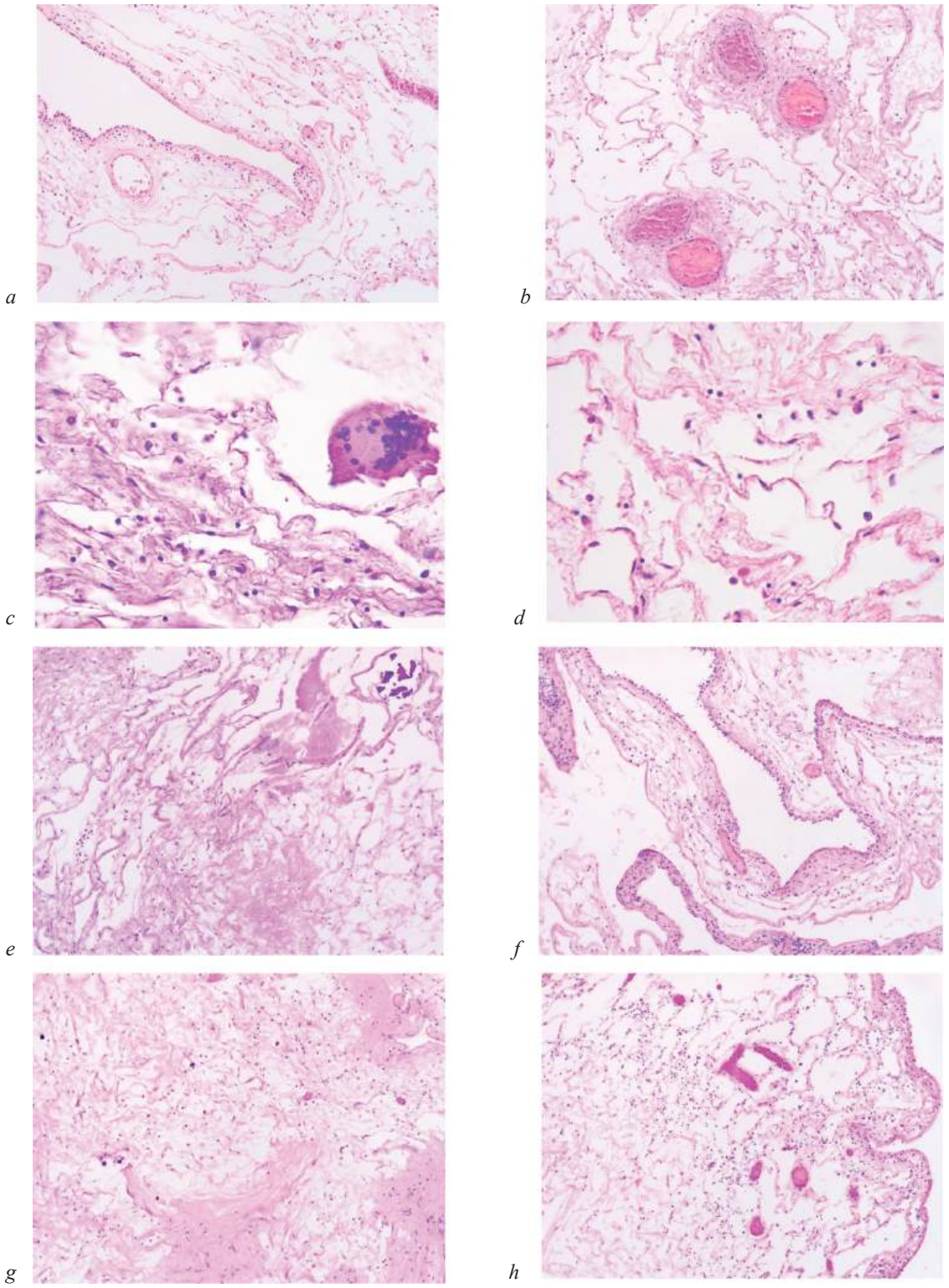


Fig.3. Arachnoid cyst in the brain (microscopic images). Staining with hematoxylin and eosin;
a–b, *e–h* ×10/0.22; *c–d* ×40/0.65

myocardium, uneven blood supply to the vessels, mild arteriosclerosis, and small foci of adipose tissue growth in the stroma were also identified.

The described pathomorphological changes corresponded to acute diffuse focal serous myocarditis with the morphological signs of acute heart failure. Based on the analysis of the medical history and taking into account the clinical presentation, gross changes, and microscopy results of organ tissue fragments, a pathology diagnosis was formulated.

PATHOLOGY DIAGNOSIS

Underlying disease: intracranial arachnoid cyst of the brain (cyst size 6.8 × 5 × 5 cm), type III according to the Galassi classification, with predominant localization in the frontoparietal region of the left hemisphere with foci of acute cerebral ischemia.

Complications: cerebral edema with dislocation and herniation of the cerebellar trunk into the foramen magnum. Coma. Hospital-acquired left-sided lower lobe focal purulent hemorrhagic bronchopneumonia. Sepsis (leukocytosis 19.2 × 10^9 /l, neutrophils 87.6%; lymphopenia 1.1 × 10^9 / l, lymphocytes 5.9%; leukostasis in the capillaries of the myocardium, lungs, brain). Acute diffuse focal serous myocarditis. Acute heart failure. Pulmonary edema.

Concomitant diseases: incomplete right bundle branch block (based on clinical data). Atherosclerosis of the aorta with the presence of type II–IV plaques, prevalence 10%.

CONCLUSION

In our article, we presented a clinical case of a 28-year-old male patient with IAC in the anterior horn of the left lateral ventricle. The cyst was discovered in a young man only posthumously. The diagnosis was established based on the results of a pathology examination. The case shows the presence of certain difficulties in diagnosing the pathology, including differential diagnosis in the presence of various factors in a particular patient.

In this case, the following aspects were of particular importance: no history of previous neurological symptoms, the acute onset of the disease during the peak seasonal incidence of tick-borne encephalitis and Lyme disease in the endemic area, extremely rapid development of another pathology in a hospital setting, in particular, pneumonia with changes in laboratory parameters typical of acute inflammation, (leukocytosis). The factors described above in the clinic resulted in an incorrect diagnosis of the pathological process, but the outcome of the disease most likely could not have been changed.

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