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Cerebral amyloidoma

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Amyloidoma is a non-tumor lesion characterized by local deposits of insoluble protein aggregates of κ/λ immunoglobulin light chains in tissues. This condition is also known as AL-amyloidosis and it is not associated with systemic amyloidosis. The usage of term "amyloidoma" is due to the fact that this lesion acts like a tumor according to clinical and neurovisualization signs but it is not a tumor.
The correct diagnosis is difficult due to small number of described cases as well as absence of specific clinical symptoms and tumor-like natural history. The most reliable diagnostic method is histological examination. We present 2 clinical cases of cerebral amyloidoma, confirmed histologically.

Keywords: amyloidoma, amyloidosis, biopsy, microsurgical resection, cerebral mass lesion

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BACKGROUND

Amyloidoma is a non-tumor lesion characterized by local deposits of insoluble protein aggregates of κ/λ immunoglobulin light chains in tissues. This condition is also known as AL-amyloidosis and it is not associated with systemic amyloidosis. Amyloidoma is not a tumor, but this term is used because of the fact that this lesion acts like a tumor according to clinical and neurovisualization signs [1].

Other morphological forms of amyloid deposits in the brain may include cerebral amyloid angiopathy and neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, etc. [2].

Besides amyloid deposits in the brain, there are also many systemic forms of amyloidosis. According to the current classification of the World Health Organization (2016), there are more than 30 amyloid proteins. The diagnostics includes detecting amyloid deposits in tissues using Congo red staining. Due to its cross-β-folded structure, amyloid is capable of birefringence, which is why it acquires a yellow-green fluorescence in polarized light.

Depending on the amyloidosis form (AA, AL amyloidosis, Aβ, APrPScr), the clinical signs and treatment methods differ radically – from damage of the peripheral and central nervous system to amyloid deposits in organs (heart muscle, rectal mucosa, kidneys, etc.) [3].

The first mention of amyloidoma was recorded in 1935 by S. Saltykow, during a pathological examination of brain matter the small macroscopically distinguishable formations were revealed in the white matter. These formations were histologically identified as amyloid deposits [4].

Since then, less than 100 cases of amyloid deposition in the nervous system have been described in the world literature. This disease is classified as an orphan disease, affecting a small part of the population. Its diagnostics is complicated by the absence of specific signs of the process and tumor-like natural history.

We present 2 clinical cases of histologically confirmed cerebral amyloidoma as well as literature review.

CLINICAL CASE 1

***The female patient**, 50 years old, was admitted to the Federal Center of Brain Research and Neurotechnologies of the Federal Medical Biological Agency of Russia on May 2, 2023, for elective surgery – biopsy of an intracerebral lesion. From the patient's medical history, it is known that since 2019, the patient has been suffered from decreased sensitivity and impaired fine motor skills in the left limbs, as well as episodic headaches.*

Magnetic resonance imaging (MRI) of the brain performed on 25.12.2019 revealed an area of altered signal with indistinct boundaries in the deep area of the right parietal lobe, measuring 25 × 18 × 16 mm, hyperintense on T2 and FLAIR and hypointense on T1 sequences, with heterogeneous accumulation of contrast agent and without perifocal edema (Fig. 1).

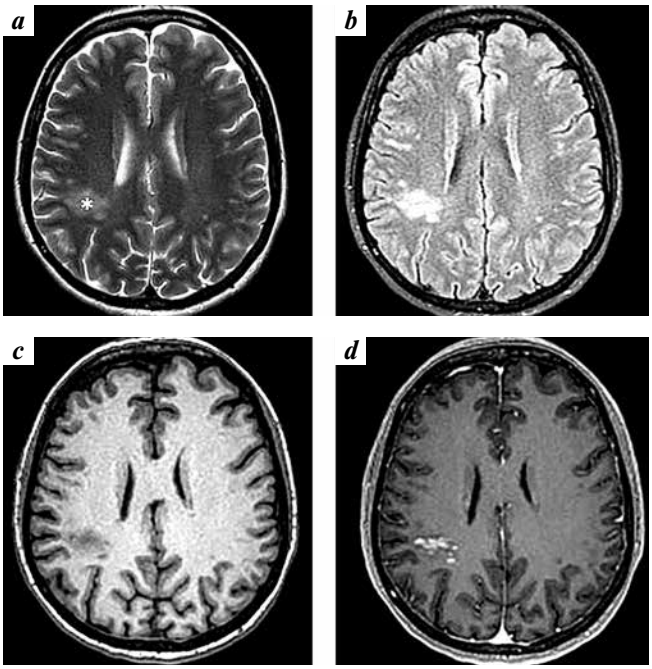

Fig. 1. *Clinical case 1. Brain magnetic resonance imaging of a patient at 2019: an area (marked with a star) of an altered signal with indistinct boundaries in the deep area of the right parietal lobe, hyperintense on T2 (a) and FLAIR (b) and hypointense on T1 (c); with heterogeneous accumulation of contrast agent (d), without perifocal edema*

Since the initial diagnosis of the lesion (2019), the patient has undergone the additional examination to exclude parasitic invasion, sarcoidosis, tuberculosis and demyelinating diseases. Based on the results of the analysis of cerebrospinal fluid and brain MRI, computed tomography (CT) of the chest organs, serum and cerebrospinal fluid studies using the polymerase chain reaction method, the above-described diseases were excluded and a decision was made to observe the tumor dynamically.

During the period from 2019 to 2021, according to brain MRI, a growth of the lesion was noted, while left-sided hemiparesis up to 4 points remained.

Four years after the initial diagnosis (March 27, 2023), due to a sudden episode of increasing weakness in the left limbs, hyperkinesis in the form of rhythmic twitching in the fingers of the left hand, in the left half of the face, the patient was admitted to Federal Center of Brain Research and Neurotechnologies of the Federal Medical Biological Agency of Russia by ambulance team with suspected acute cerebrovascular accident. Upon admission, the patient underwent a standard examination with an incoming diagnosis of "stroke", including brain CT and MRI.

According to the CT data on 03/27/2023 (Fig. 2) an area of pathological changes of irregular shape is visualized in the right parietal lobe without clear boundaries, partially surrounded by an area of perifocal edema, without calcifications; there were no signs of hemorrhages and infarctions.

According to brain MRI data on 03/27/2023 (see Fig. 2) an intracerebral formation measuring 33 × 35 × 18 mm in the deep areas of the white matter of the right frontal and parietal lobes is determined, with indistinct boundaries. Thise lesion has a heterogeneous structure with numerous small hemorrhagic inclusions, with vasogenic edema up to 1.5 cm, spreading to the subcortical areas of the central gyri as well as heterogeneously accumulating contrast agent. According to perfusion maps, the lesion demonstrates an indistinct increase in T_{max} indicators (see Fig. 2).

Comparing with MRI data of 2021, a significant increase in the size of the lesion as well as the contrast area, and the appearance of perifocal edema were noted.

The neurological status assessment revealed a decrease of muscle strength in the right hand to 4 points, anisoreflexia (D <S), sensory ataxia, and "high gloves and golf type

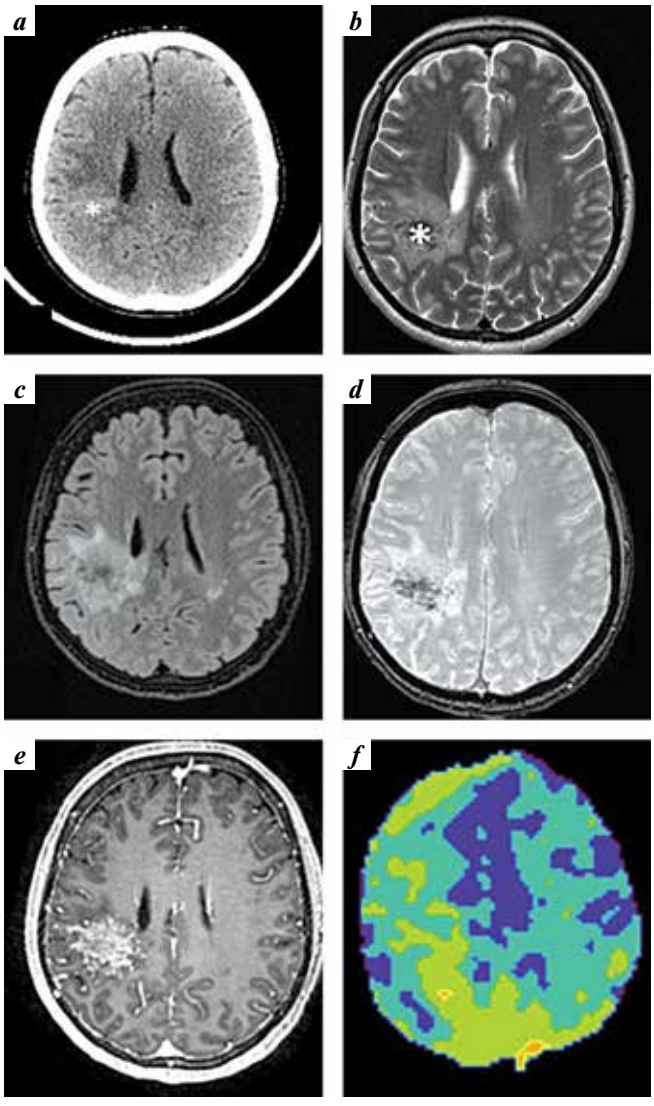

Fig. 2. *Clinical case 1. Brain computed tomography (a) and magnetic resonance imaging (b–f) of patient of 2023: an area (marked with a star) of an altered signal with indistinct boundaries in the deep area of the right parietal lobe, surrounded by minimal perifocal edema (a) hyperintense on T2 (b) and FLAIR (c) with multiple small hemorrhages on the SWAN sequence (d); with heterogeneous accumulation of contrast agent (e) as well as slight increase of T_{max} during cerebral perfusion examination (f)*

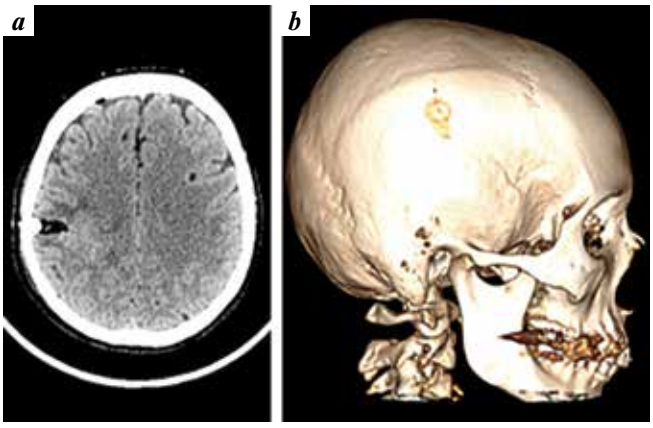

Fig. 3. *Clinical case 1. Brain computed tomography of the patient performed 1 day after biopsy: there are no areas of ischemia and hemorrhages (a); a trefination hole filled with bone powder is determined in the right parietal bone (b)*

hypoesthesia" on the left. The patient was consulted by a neurosurgeon – the elective surgery (biopsy) was recommended.

On May 2, 2023, the elective surgery was performed at the Federal Center of Brain Research and Neurotechnologies of the Federal Medical Biological Agency of Russia – a biopsy of the lesion from the trephination hole. The postoperative period was uneventful. According to the control brain CT scan there were no ischemic or hemorrhagic complications (Fig. 3).

Cerebral amyloid angiopathy was verified histologically (Fig. 4). When staining for amyloid (Congo red), the substance accumulations were found to be red; when examined in polarized light, a yellowish-greenish fluorescence *was noted.*

The patient was discharged on the 3rd postoperative day in a satisfactory condition, without any increase of neurological

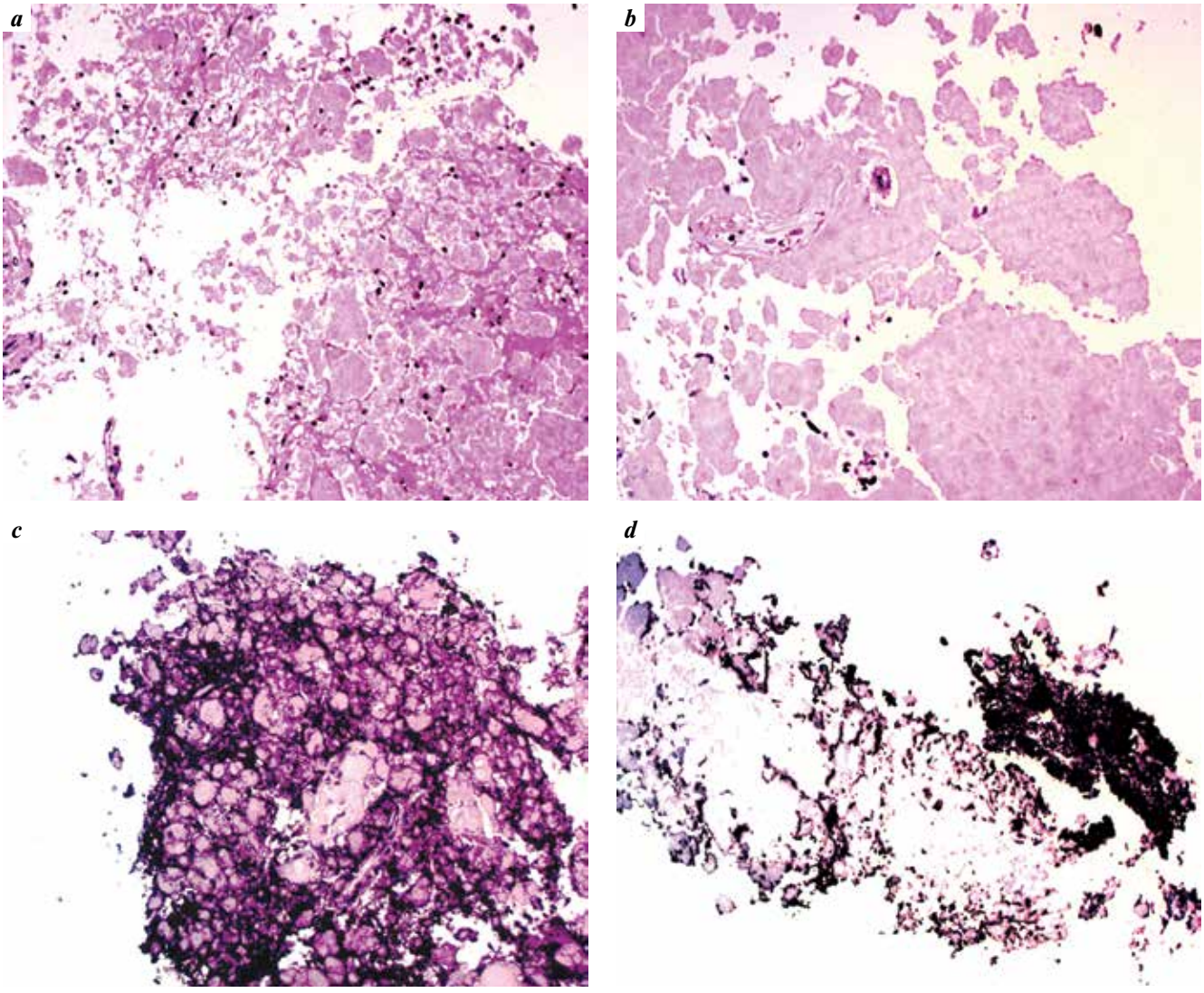

Fig. 4. *Clinical case 1. Histological (a) and immunohistochemical (b–d) examination of patient's biopsy material (×200): fragments of brain tissue with multiple accumulations of amorphous eosinophilic structureless substance, reactive glial changes and focal gliosis (hematoxylin and eosin staining) (a); immunohistochemical examination revealed diffuse expression of S100 (c), GFAP (d), synaptophysin in brain tissue, negative expression of p53; Ki-67 in brain tissue was not detected (b), positive nuclear expression – only in leukocytes*

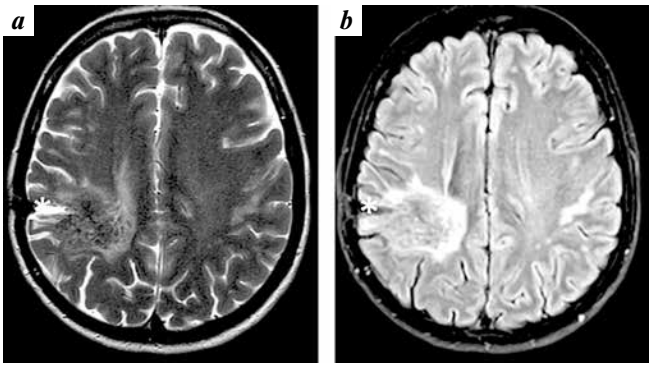

Fig. 5. *Clinical case 1. Brain magnetic resonance imaging of patient performed 8 months after the biopsy: the biopsy area is marked with a star. The increase of the volume of lesion and postoperative changes on T2 (a) and FLAIR (b) sequences is not determined*

deficit. During follow-up (8 months), no further growth of the lesion was recorded (Fig. 5).

CLINICAL CASE 2

***The female patient**, 41 years old, was schedule admitted to the Federal Center for Medical Science of the Federal Medical and Biological Agency of Russia on 10.04.2023 with complaints on constant pressing headache inthe frontal-temporal area, nausea, photophobia, unsteadiness when walking, as well as weakness and numbness in the right limbs. From the medical history it is known that in 2014 the patient first noted the appearance of increasing weakness in the limbs, numbness in the limbs of the "stocking" type.*

The patient was admitted to neurological department with lower paraparesis up to 3 points, numbness from the level of the Th_{12} dermatome. The patient was diagnosed with multiple sclerosis at the place of residence. The one course of plasmapheresis and hormone therapy was performed with positive dynamics – an increase in muscle strength in the lower limbs up to 5 points on the right and up to 4 points on the left with improving sensitivity.

During a scheduled brain MRI in 1 year (in 2015), the structural changes in the left temporal lobe, insular lobe and in the area of the basal nuclei of the left hemisphere were first detected. A preliminary diagnosis was made as arteriovenous malformation. A decision was made to observe the patient in dynamics.

In 2023, during repeated brain MRI with contrast enhancement, the changes were assessed as a tumor of the left insular lobe. The patient was consulted by a neurosurgeon – the surgical treatment was recommended.

During admission hypoesthesia of the right half of the face, smoothing of the right nasolabial fold, decreased muscle strength in the right limbs to 4 points, lively asymmetric tendon and periosteal reflexes from the limbs D >S, right-sided hemihypesthesia, and ataxic gait were observed.

The brain CT scan with contrast enhancement was performed/The calcifications were found in the area of the left basal ganglia. According to the brain MRI with contrast enhancement, a pathological area of structural changes (hyperintense signal on T2, FLAIR sequences) was determined in the cortical-subcortical areas of the left insular lobe with spread to the external capsule and adjacent areas of the lenticular formation, as well as in direct adjacency to the temporal horn of the left lateral ventricle (Fig. 6).

On 11.04.2023, the microsurgical removal of the lesion was performed under neurophysiological control. The tumor fragment was left in the area of the corticospinal tract along its medial border due to the intimate adjacency of the lesion to the corticospinal tract (see Fig. 6).

After the operation, the patient's condition was stable, the neurological status was at the preoperative level. The course of the postoperative period was uneventful. The control brain MRI in 8 months did not reveal any data indicating the growth of the lesion (Fig. 7).

Histological examination revealed no signs of neoplastic or demyelinating process in the samples obtained. The histomorphological signs corresponded to cerebral amyloid angiopathy (Fig. 8).

DISCUSSION

According to the literature, the manifestation of amyloidoma most often occurs at the age of over 45 years, however, in one of the cases presented by us, the disease debuted earlier (32 years at the time of the symptoms onset) [5–7]. The most common symptoms are seizures, headache, visual impairment, dizziness, focal neurological

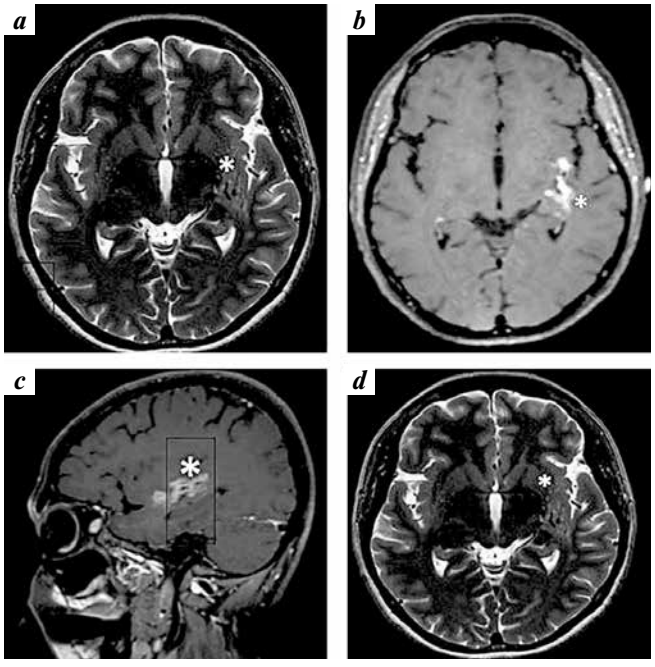

Fig. 6. *Clinical case 2. Brain magnetic resonance imaging of patient with contrast enhancement: an area (marked with stars) of an altered signal with indistinct boundaries in the area of the left insula surrounded by minimal perifocal edema, slightly hyperintensive on T2 (a, d) with heterogeneous accumulation of contrast agent (b, c)*

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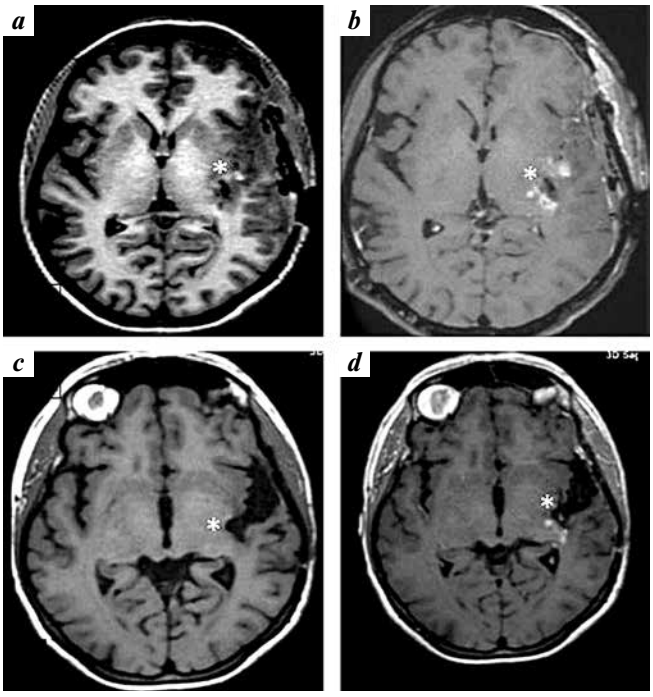

Fig. 7. *Clinical case 2. Brain magnetic resonance imaging of patient: a, b – on 3rd postoperative day: the residual fragment (marked with stars) on T1-waited images without (a) and with (b) contrast enhancement; c, d – 8 months after surgery: residual fragment (marked withstars) on T1-waited images without (c) and with (d) contrast enhancement. Without dynamics*

deficit in the form of muscle strength and sensitivity disorders (hemiparesis and hemiparesthesia) [5].

The high variability of symptoms is associated with the different localization of the lesion. Most often, when performing CT or MRI, the lesion is revealed in the supratentorial white matter, with a periventricular and perivascular location [8]. The several cases of amyloidoma detection in the trigeminal nerve (usually in the ganglion) have been described in the world literature, and this fact is important in the differential diagnostics of the trigeminal neuralgia causes [9].

It is difficult to identify the characteristic neuroimaging signs of amyloidoma, since there are signs similar to glioma, lymphoma and metastases to the brain [10]. For examination in case of suspected amyloidoma, it is recommended to perform brain MRI with contrast enhancement, magnetic resonance perfusion, and additionally, it is prescribed to perform positron emission tomography combined with computed tomography (PET-CT), with aminoacid radiomarkers such as methionine, tyrosine and their analogue F-fluoro-L-phenylalanine (FDOPA).

The following features are often seen during brain MRI:

- on T1-weighted images, the signal from the amyloidoma varies from iso- to hyperintense, with intermediate signal intensity most likely corresponding to uneven amyloid deposition;
- on T2-weighted images, the signal often varies in the range from intermediate to hyperintense;
- a linear pattern of contrast is observed at the periphery of the lesion, which may result from amyloid deposition in the surrounding vessels;
- perfusion MRI shows decreased perfusion in the area of amyloidoma;
- the constant presence of perifocal edema is not typical, but it may be presented [6, 11].

The brain CT typically reveals an area of increased density with accumulation of contrast agent [12]. Given the non-tumor nature of the disease, the question of treatment tactics remains disputable and its choice depends on the presence of a mass effect as well as the dynamics of lesion growth and neurological deficit. The small amount of available information and the absence of clear guidelines complicate the treatment of this group of patients.

The single cases of lesion growth during follow-up are described in literature. The systematic literature review by D.P. Bray et al. included data of 39 patients, whose average age was 49.4 years; 54 % of patients were female, the main debut symptoms of the disease were epileptic seizures and headache. During the observation period, the lesion growth was noted in 1 patient with performed biopsy.

Among 11 patients with total amyloidoma removal, the positive dynamics including absence of attacks and stabilization of the condition were noted in 4 patients over a period of 3 to 24 months; the progressive neurological deficit in the form of vision deterioration and weakness in the right hand was observed in 3 cases; there was no follow-up in 4 patients. The partial resection of lesion was performed in 5 patients, only one of them had a neurological deficit after surgery [6].

In another study conducted by B. Fischer et al., during follow-up of 27 patients for a period of 18 to 48 months, the dynamic growth of amyloidoma was observed in 3 cases. Nine surgical resections were performed, in 3 cases during observation from 3 months to 2 years no neurological deficit or lesion growth was recorded; the information about 6 patients was not presented [12].

The usage of PET-CT is recommended for differential diagnostics, especially when biopsy is not possible. This examination usually reveals the decreased metabolism, which is not typical, for example, for high-grade tumors and allows the differential diagnostics with brain tumors, in particular with glioma [13, 14].

The histological examination is a key of confirming the diagnostics. The histological signs are characterized by:

- congophilic large clusters of pale eosinophilic deposits;
- amyloid deposits surrounded by infiltrates consisting of lymphocytes, macrophages and plasma cells;
- characteristic yellow-green birefringence in polarized light;
- perivascular and intravascular distribution of amyloid;
- possible focal calcification in deposits [2, 10, 11].

During immunohistochemical examination, it is important to determine whether these deposits belong

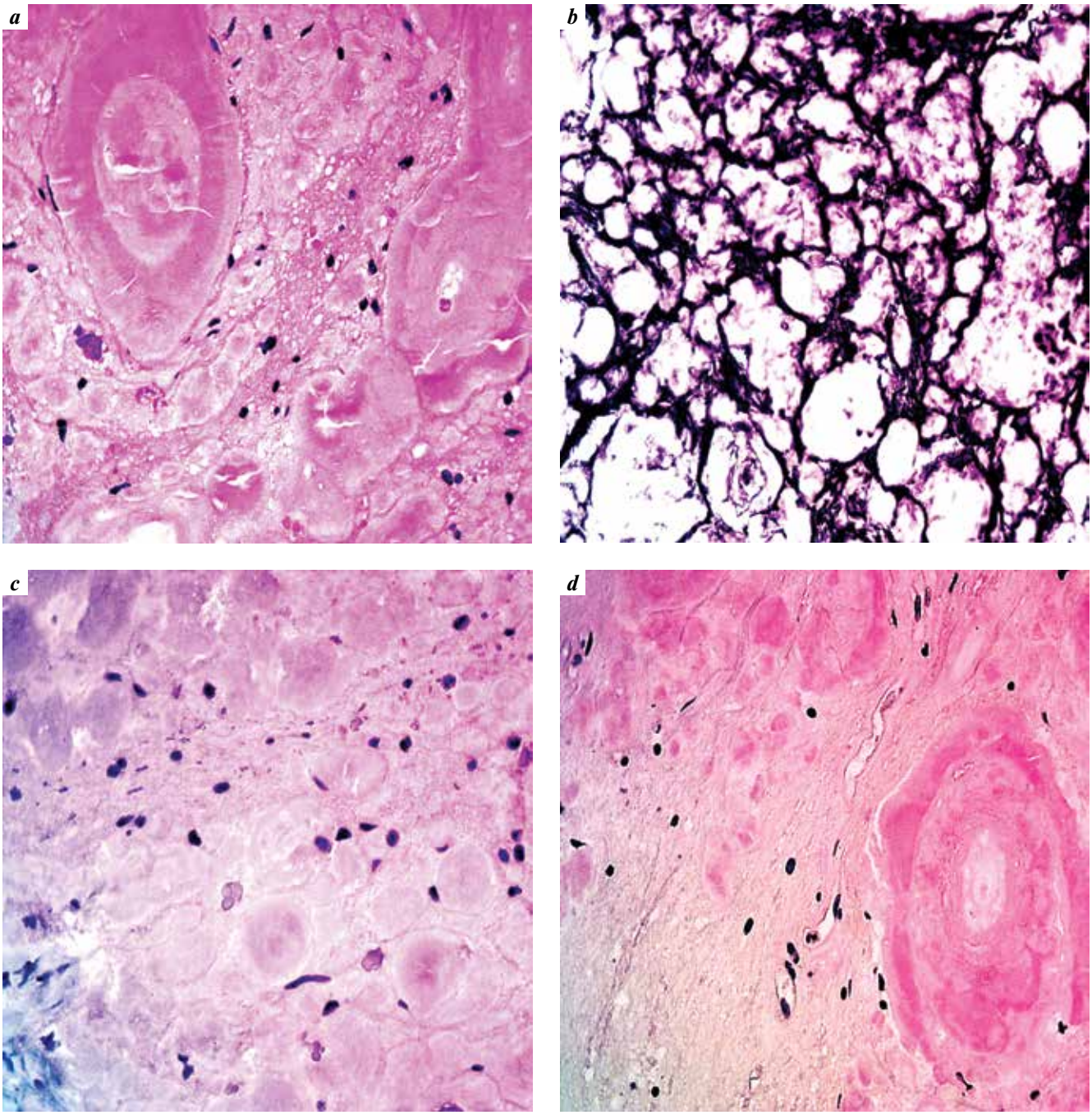

Fig. 8. *Clinical case 2. Histological (a, d) and immunohistochemical (b, c) examination of patient's biopsy material (×400): microscopic examination visualizes pathological tissue represented by variously rounded and ovoid pale eosinophilic amyloid-like structures (hematoxylin and eosin staining) (a). In some of these structures, gaps are visible, and small-caliber vessels are visualized in some of them; somehow these structures resemble the "shadows" of small and medium-sized vessels; the spaces between the described amyloid-like structures are made of narrow strips of glial tissue with signs of reactive piloid gliosis; no nuclear atypia has been detected. Immunohistochemical examination revealed the expression of GFAP (b), synaptophysin and myelin protein in the residual areas of the central nervous system tissue; the proliferative index (Ki-67) is about 0 % (c). When stained with Congo red, deposits of an amyloid-like substance were detected both in the walls of blood vessels and in the intervascular space (d). When examined in polarizing light, a greenish-yellow fluorescence characteristic of amyloidosis was revealed*

to the immunoglobulin κ or λ light chain. The additional molecular methods such as chromogenic *in situ* hybridization can also be used to determine them [5, 15].

The treatment methods include partial or radical resection of the lesion and in radiation therapy a small number of cases, which is relevant when the amyloid is located in a functionally significant areas and it is impossible to safely perform a resection or biopsy of the lesion.

The case of radiation therapy with positive dynamics in the form of regression of symptoms such as imbalance, headache, vomiting, weakness in the right half of the face

is described in literature [16]. No growth of the lesion or increase of neurological deficit was observed during follow-up for 48 months in 2 clinical cases after focal radiation therapy [7].

The therapy goal for systemic amyloidosis is to suppress the synthesis of immunoglobulin light chains that form amyloid, which improves the function of the affected organs. Laboratory, instrumental, and clinical indicators are used to evaluate the effectiveness of therapy.

The first line of therapy for systemic AL amyloidosis was proposed in 1978 by R.A. Kyle and P.R. Greipp, who demonstrated the effectiveness of chemotherapy (melphalan and prednisolone) in reducing amyloid deposition in the liver by repeated biopsies. However, due to the high risk (up to 20 %) of developing myelodysplastic syndromes and secondary leukemia, the search for a more effective and safe line of therapy continued [17].

In another clinical study at the National Amyloidosis Centre (UK), the standard chemotherapy (cyclophosphamide, thalidomide, dexamethasone) was used, in which the median survival exceeded 6.3 years, and complete hematological remission was achieved in 100 % of patients.

The combination of autologous hematopoietic stem cell transplantation with standard chemotherapy has demonstrated its advantage over other monotherapy methods in the form of an increase of overall survival by 59 % [17].

In the presence of epileptic seizures, the antiepileptic therapy (levetiracetam) is indicated, and in case of cephalgic syndrome, pain therapy is selected (non-steroidal anti-inflammatory drugs, pain-relieving antidepressants, triptans).

CONCLUSION

The cerebral amyloidoma is a very rare pathology, and therefore making a correct diagnostic without histological verification is a difficult task. Despite the fact that it is difficult to diagnose amyloidoma at the pre-hospital stage, it must be taken into account during the differential diagnosis of various nervous system diseases. The only method that guarantees the reliability of the diagnostics is histological examination of the lesion.

PET-CT is also recommended if biopsy is not possible. When choosing a particular treatment method, the individual patient symptoms and the degree of their deterioration over time should be taken into account. Initially, a biopsy may be recommended, and if there is no neurological deficit, further monitoring of the disease dynamics is prescribed. The lesion resection is performed in the presence of a mass effect, neurological deficit and seizures.

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O.O. Kordonskaya, S.A. Melchenko, I.V. Senko: scientific editing of the article;
O.I. Patsap: analysis of the data obtained, article writing;
M.B. Dolgushin: analysis of the data obtained.

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