DOI: 10.17650/1683-3295-2023-25-1-85-94

RECONSTRUCTIVE ENDOVASCULAR TREATMENT OF DISTAL CEREBRAL ANEURYSM ASSOCIATED WITH CARDIAC MYXOMA

R.S. Martynov[1], A.V. Savello[1], A.N. Savchuk[1], K.N. Babichev[1], A.I. Kiskaev[2], D.V. Svistov[1]

[1]S.M. Kirov Military Medical Academy, Ministry of Defense of Russia; 6 Akademika Lebedeva St., Saint Petersburg 194044, Russia
[2]City Hospital of the Holy Martyr Elizabeth; lit. A,14 Vavilovykh St., Saint Petersburg 193312, Russia

Contacts: Martynov Roman Sergeevich *Rs_martin@mail.ru*

Introduction. The etiology and pathogenesis of cerebral aneurysms are diverse. The rare cause of cerebral aneurysms occurrence is cardioembolism in cases of heart tumors, in particular in myxoma. Cardiac myxoma (from *Latin* muxa, mucus) is the most common (about 50 %) benign tumor of heart. With untimely diagnosis and absence of the disease treatment, embolisms in cerebral vessels may occur with typical symptoms of ischemic stroke as well as in some cases the myxomal aneurysms may develop. There are several theories of their origin. The myxomal aneurysms of cerebral vessels are more common in women and in the vast majority of cases they develop in carotid basins. In a quarter of cases, the disease onsets as intracranial hemorrhage caused by rupture of an oncotic (myxomal) aneurysm, that significantly exceeds the frequency of ruptures (1–3 % per year) in cases of "normal" bifurcation-hemodynamic intracranial aneurysms. The mortality rate is 3.4 %. The majority (80 %) of ruptures were observed within 2 years after the diagnosis of cardiac myxoma, and in half (48.6 %) of cases clinically significant episodes of embolism were noted. Timely treatment can prevent the hemorrhagic type of course of these aneurysms. Various methods of treatment are described in the literature: different options for surgical eradication as well as the possibilities of chemotherapy and radiation exposure.
Aim. To present a case of successful radical reconstructive endovascular treatment of patient with distal cerebral aneurysm associated with cardiac myxoma, combined with presence of multiple cavernous malformations and also to highlight issues of diagnosis, differential diagnosis and existing treatment options for these diseases.
Clinical observation. The article presents a clinical observation of multiple aneurysms associated with myxoma of the heart in combination with multiple cavernous angiomas of the brain. The possibility of reconstructive eradication of distal aneurysm after its transformation from fusiform to saccular is demonstrated. The etiology and pathogenesis of occurrence, the nuances of differential diagnosis, possible methods and the algorithm for choosing a method for treating brain aneurysms associated with myxomas are reflected.
Conclusion. All patients with cardiac myxomas, both before and in dynamics after their removal, are recommended to perform noninvasive visualization of brain vessels for verification of such complication as metastatic (myxomal) aneurysms. The prognosis in most patients with multiple intracranial aneurysms associated with myxoma is favorable and most aneurysms associated with myxoma are stable. However, in cases with progressive or ruptured aneurysms, surgical treatment options should be considered including microsurgical or endovascular eradication. Radiation and chemotherapy methods may have a certain therapeutic value.

Key words: oncotic aneurysms, myxomatous aneurysms, cardiac myxoma, treatment, embolization

For citation: Martynov R.S., Savello A.V., Savchuk A.N. et al. Reconstructive intravascular treatment of distal cerebral aneurysm associated with cardiac myxoma. Neyrokhirurgiya = Russian Journal of Neurosurgery 2023;25(4):85–94. (In Russ.). DOI: 10.17650/1683-3295-2023-25-1-85-94

INTRODUCTION

The pathogenesis of brain aneurysms is diverse, and the classification of this pathology includes variants of metastatic, or oncological, aneurysms [1]. The cause of such predominantly distal cerebral vascular aneurysms is, among other things, cardioembolism in cases of heart tumors, in particular in myxoma.

Cardiac myxoma (from Latin muxa, mucus) is the most common (about 50 %) benign heart tumor that is usually localized in left or right atrium. Subendocardial multipotential mesenchymal cells are considered to be a source of these tumors. The tumor grows on a pedicle attached to atrial septum. Radical resection of these neoplasms leads to a cure. At the same time, hematogenous metastasis of the tumor is possible both before and during tumor removal [2, 3].

Cardiac myxoma is manifested by the following triad [4]:

1) inflammatory syndrome with myalgia, arthralgia, fever, elevated levels of ESR and C-reactive protein;

2) embolic manifestations, most often in the brain or in the large circle of blood circulation, since tumors are often localized in left chambers of heart;
3) obstruction of heart valves leading to pulmonary edema and less often to right ventricular failure.

Early diagnosis and treatment of these lesions can prevent embolism. One of the most unfavorable targets of embolism by myxoma cells are brain vessels, which occlusion often leads to ischemic stroke. Distal aneurysms associated with myxoma develop much less frequently [5].

There are different points of view on the natural course of aneurysms associated with myxoma and the strategy of their treatment, especially in multiple lesions [4, 6, 7]. The prevalence of such aneurysms in patients with cardiac myxoma according to different studies varies in range 12,8–56,0 % [6, 8, 9]. In some cases, aneurysms may be detected before the myxoma is recognized, and in others, decades after its resection [10]. In most cases (67.5 %), aneurysms are detected after the myxoma resection.

Despite the obvious reason for formation of aneurysms associated with cardiac myxomas there are two theories of their pathogenesis. The first is the theory of neoplastic process: myxoma cells are attached to endothelium, penetrate into it, grow in the subintimal layer with subsequent destruction of the entire arterial wall. The second is the theory of microembolic injury: the myxoma embolus causes endothelial damage accompanied by impaired local hemodynamics and followed by aneurysm formation. In the second case, we can talk about the dissection nature of the artery injury. The described theories are similar to pathogenesis of infectious aneurysms which includes inflammatory process in the artery wall associated with infectious embolus [11]. In any case, both the theories reflect the fact that pathological process is initiated by myxoma embolus [6, 12].

Some authors additionally highlight hematogenic dissemination in the vasa vasorum of cerebral vessels as a mechanism for aneurysms' development. This leads to artery wall destruction, especially the middle layer and the inner elastic membrane, and as a consequence to aneurysm formation [5].

At the same time, it is reported in the literature that an important factor in the initiation of aneurysm is interleukin-6, which is secreted by myxoma cells. However, there is no convincing evidence that this protein that belonging to proinflammatory cytokines group can be used as a biomarker for diagnosis of aneurysms associated with myxoma [13–15].

In most cases, the multiple aneurysms associated with myxoma develop before the age of 50 and mainly in women.

The most frequent lesion localization of such aneurysms is anterior part of circle of Willis 89.7 %, and 10.3 % of cases fall on the vertebrobasilar arteries. In 24.4 % of cases, the disease manifests in patients with intracranial hemorrhage caused by aneurysm rupture, which significantly exceeds the frequency of ruptures of 1–3 % per year with "common" bifurcation-hemodynamic intracranial aneurysms. The mortality rate is 3.4 % [7]. The majority (80 %) of ruptures were observed within 2 years after diagnosis of cardiac myxoma [16], and in half (48.6 %) of cases clinically significant episodes of embolism were noted [12].

CLINICAL CASE

Patient E.A., *born in 1981 (38 years old), sought medical help in connection with the appearance of photopsias. Magnetic resonance imaging (MRI) of the brain with intravenous contrast dated 02.04.2019 showed subcortical changes in right parietal and left occipital lobes. Upon further comprehensive examination, the left atrial myxoma was verified in the patient. According to echocardiography data, there was rather mobile formation in the left atrium having cellular structure occupying more than half of the atrium. In systole, the formation was wedged into mitral flaps valve, mitral regurgitation was of 1–2 degree. On* ***04/19/2019****, a myxoma of left atrium measuring 7.0 × 3.0 cm was resected, the myxoma was on pedicle with fixation in the middle third of the atrial septum.*

In the postoperative period, the photopsias still persisted that is why 6 months after cardiac surgery the patient underwent repeated brain MRI with intravenous contrast and signs of "mirror cavernous malformations of both occipital lobes" were diagnosed (Fig. 1).

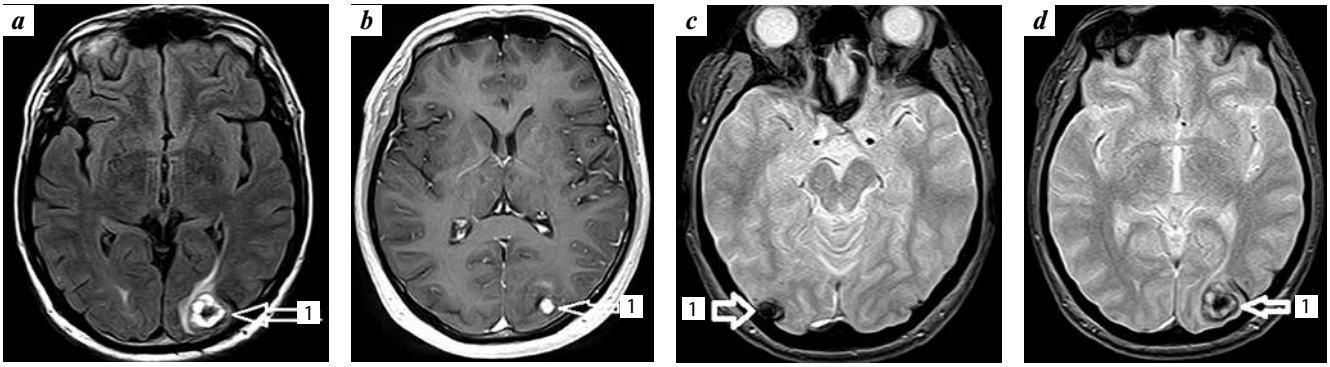

Fig. 1. *Mirror cavernous malformations of occipital lobes (MRI of brain dated 29.10.2019, 6 months after cardiac surgery): a – cavernous angioma (1) of left occipital lobe (MRI in the FLAIR sequence); b – cavernous angioma (1) of left occipital lobe with accumulation of contrast (sequence-WI with contrast enhancement); c – cavernous angioma (1) of right occipital lobe (MRI in sequence T2*); d – cavernous angioma (1) of left occipital lobe (MRI in sequence T2*) FLAIR (Fluid Attenuated Inversion Recovery) – the mode with suppression of free water signal, WI – weighted image, T1 and T2* – modes of sequences of weighted images on MRI.*

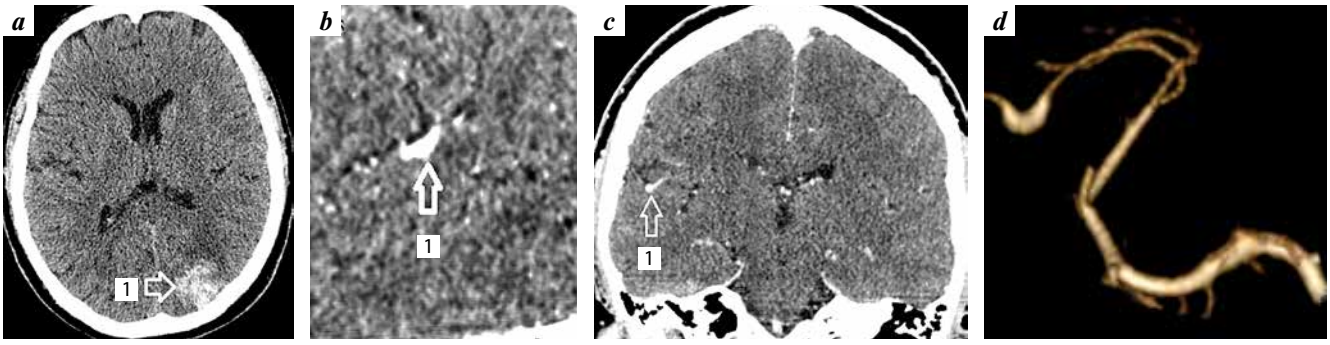

Fig. 2. *Spiral computed tomography (CT) and spiral computed tomography angiography (SCTA) of the brain: a) CT-signs of hemorrhage (1) in the projection of cavernous angioma of left occipital lobe; b, c) fusiform expansion of cortical segment of posterior parietal branch of the right medial cerebral artery in sagittal and frontal planes of multiplanar reconstruction; d) three-dimensional CT-reconstruction of the affected artery segment with fusiform aneurysm*

Computed tomography (CT) and spiral computed tomographic angiography (SCTA) of the brain revealed parenchymal hemorrhage in left occipital lobe in projection of previously diagnosed cavernous malformation. There were no signs of arteriovenous malformation or distal aneurysms in the projection of the hemorrhage focus, but there was a fusiform expansion of cortical segment of posterior parietal branch of right middle cerebral artery (Fig. 2).

At this stage of examination, the local expansion of cortical artery was not given importance as not requiring specific treatment.

The patient underwent surgical intervention on 01/28/2020: bone-plastic skull trepanation in left parietal-occipital region and microsurgical removal of cavernous malformation. Histological conclusion No. 5403–07: cavernous "angioma" (cavernoma). The course of postoperative period is favorable. A slight increase in the area of absolute and relative scottomas was noted.

In March of 2022, the patient underwent a planned comprehensive medical examination. Taking into account the anamnesis the brain CT scan was performed, according its results an aneurysm of posterior parietal branch of right middle cerebral artery was diagnosed. A significant transformation of the aneurysm during the 2-year follow-up period is noteworthy (see Fig. 2, d). In this regard, the patient received consultation by neurosurgeon and was hospitalized to neurosurgery clinic for further examination.

Selective cerebral angiography confirmed the presence of aneurysm of posterior parietal branch of right middle cerebral artery having a size of 4 mm. At the same time, along the length of the artery distal to the aneurysm there were uneven contours of areas alternating in narrowing and expansion that suggesting vasculitis type damage to artery wall (Fig. 4).

Several *surgical treatment options were considered to prevent the aneurysm rupture: 1) microsurgical reconstructive aneurysm clipping, 2) trapping of the affected artery segment, 3) "end-to-end" extra-intracranial bypass with shutdown of the affected artery segment proximal to the aneurysm, 4) "end-to-end" intra-intracranial bypass excision of the artery segment carrying aneurysm, 5) endovascular occlusion of the parent artery at the aneurysm level, 6) endovascular*

Fig. 3. *Spiral computed tomographic angiography of the brain: three-dimensional reconstruction of the affected segment of artery with aneurysm*

reconstructive intervention by embolization of the aneurysm with detachable microspirals. The use of assisting techniques or stent implantation at the aneurysm level due to difficult access and small caliber of cortical arteries was not expected.

The patient underwent endovascular surgery on ***04/18/2022****. Under general anesthesia with systemic heparinization (4000 units) subcranially, a guide catheter was inserted into the right internal carotid artery. Microcatheter was inserted into the aneurysm dome by the use of microconductor 014". The aneurysm was turned off from bloodstream by the sequential introduction of 2 separable ultra-soft microspirals with dimensions of 3 × 20 and 2 × 60 mm. During implantation of the 2nd microspiral, the prolapse of the coils into the supporting artery was noted with their location along the circumference of the cross-section of the artery without impairing of its patency. Control angiography showed that the aneurysm was completely turned off from the bloodstream (type A according to Raymond–Roy classifications) while retaining the carrier artery patency at the level of aneurysm and in the distal segment. In order to prevent early postoperative ischemic complications, antiplatelet therapy with IIb/IIIa inhibitors of platelet glycoprotein receptors was performed in the form*

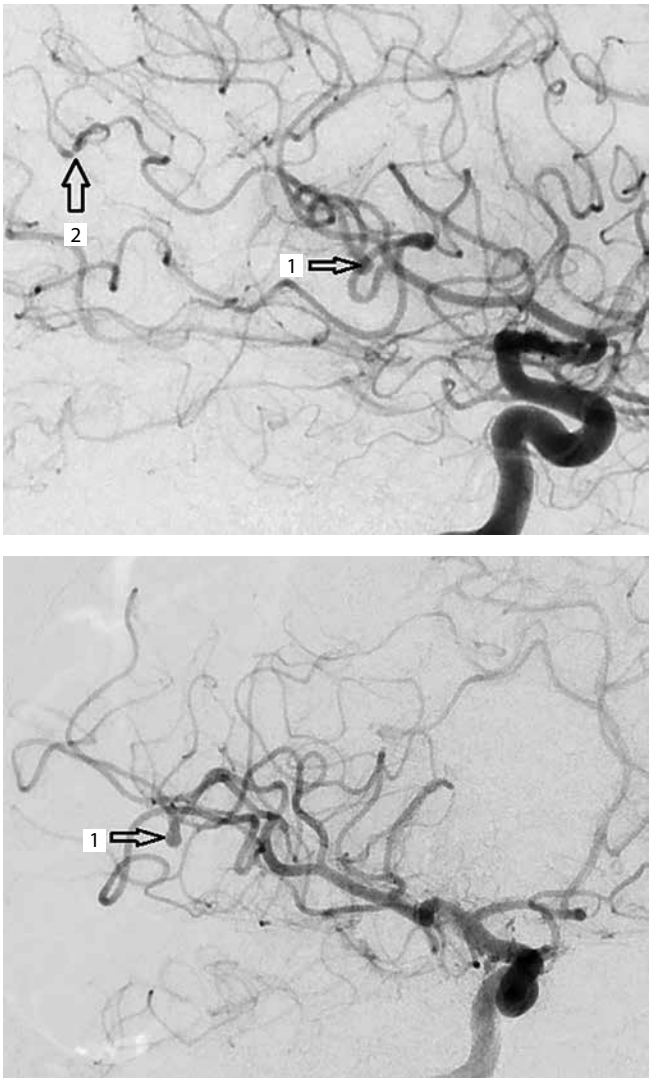

Fig. 4. *On cerebral angiogram in lateral and oblique projections visualization of aneurysm 1 and arterial contour defect in distal segment 2*

of an extended intravenous infusion of eptifibatide in the calculated dosage for 12 hours. In control selective cerebral angiography performed 24 hours later it was observed preservation of the lumen of the supporting artery throughout and the absence of aneurysm contrast (Fig. 5). Due to the peculiarities of the microspiral coil location in the artery lumen, the patient was prescribed antiplatelet monotherapy with ticagrelor 180 mg/day for a month to prevent delayed ischemic complications until completion of endothelization of the spiral coils.

DISCUSSION

Connection between myxoma of heart left chambers and intracranial aneurysm was first described by German pathologist F. Marchand back in 1894. Intracranial aneurysms are usually detected accidentally during examination for other diseases or in stroke patients [17].

Differential diagnosis of myxoma aneurysms may be difficult in some cases. On a contrast-free computed tomogram (CT), myxomal aneurysms have increased density due to the accumulation of myxoid matrix or petrificates in their walls. At the same time, abnormal changes are detected on MRI around myxoid aneurysms, such as signal loss on T2-weighted images, signal gain on T1-weighted images with contrast enhancement due to myxoids, angiogenesis or granulation tissue. These changes make difficulties in differentiating these aneurysms from other diseases, such as cavernous malformation or mycotic aneurysms.

Cavernomas are characterized by absence of pathological changes during angiographic examination. However, on MRI, both cavernoma and myxoid aneurysm can manifest as foci surrounded by an irregular hemosiderin ring; in both cases swelling of the brain tissue around the injured area is detected; and in both cases the effect of "flowering" is detected on T2-weighted images with gradient echo. But only myxoid aneurysms can be clearly identified on T1-weighted images [18].

Angiographic signs of myxoid aneurysms do not differ from the most common mycotic (septic) aneurysms. Recognition of increased density in the aneurysm area on CT without contrast enhancement may indicate myxoid origin (this is evidenced by histopathological studies that showed accumulation of myxoid, hemosiderin and iron due to recurrent chronic bleeding, but not a calcification [19]). Septic aneurysms have a greater tendency to rupture with the formation of hematomas around the injury zones [20].

Taking into account the possibilities of modern technologies, most patients can be offered effective and safe treatment options. Various options for invasive treatment of oncotic (myxomal) aneurysms are presented in literature. Microsurgical interventions are represented by such methods as aneurysm resection [7, 12, 13], aneurysm resection in combination with anastomosis [13], aneurysm clipping [14, 21]. In these cases, the anastomoses imposition is considered if the affected artery supplies blood to a functionally significant area of the brain. The choice of this method is due to the fact that reconstruction of lumen of the parent artery with clips is impossible due to looseness of the aneurysm walls [13, 22]. Switching off the myxomal aneurysms from bloodstream during endovascular operations can be performed by embolization of the aneurysm itself or destructive occlusion of the aneurysm parent artery. M.B. Yilmaz et al. in 2003 presented a case of successfully performed endovascular embolization of giant aneurysm of the left medial cerebral artery [10].

A variant of destructive shutdown of a giant partially thrombosed fusiform aneurysm from the bloodstream is described by T.J. Sorenson et al. (2019). The proximal embolization with microspirals was performed after assessment of collateral cortical blood flow using balloon occlusion test [23].

J. Sedat et al. in 2007 presented a case of radiation therapy with a total dose of 46 Gy. Occlusion of the aneurysm parent artery was observed a year later [24]. Despite the unproven clinical efficacy of radiation therapy, K. Khatibi et al. (2020) concluded that low-dose targeted radiation

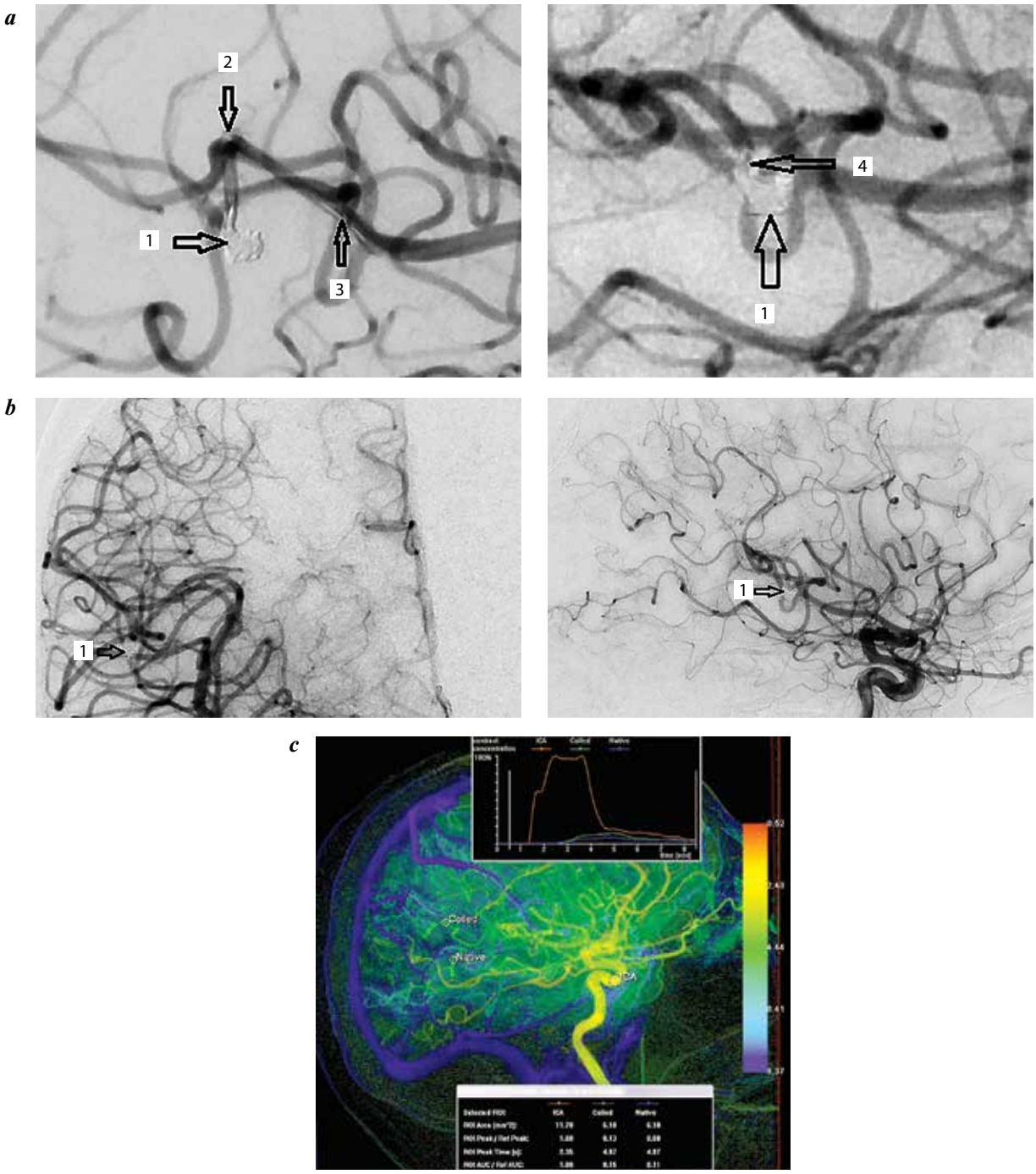

Fig. 5. *Stages of endovascular treatment and control selective cerebral angiography: a – on stage and control angiograms during the intervention, a tightly packed tangle of microspirals are visualized 1 in aneurysm with coils of microspirals in the carrier artery, in distal 2 and proximal 3 microcatheter marks, the prolapse of the coil of the microspiral along the contour of the aneurysm-bearing artery 4 with preservation of its patency; b – on control angiogram 24 hours after surgery a tangle of tightly packed microspirals is visualized in direct and lateral projection 1; the aneurysm-bearing artery is contrasted throughout, no-vascular zones are not observed; c – the perfusiogram shows the correspondence of blood flow in the affected artery (Coiled) distal to the embolized aneurysm and the intact (Native) artery*

Note. *The time to reach peak of contrast enhancement in both arteries estimated by Peak Time (sec.) is identical, the area under the curve of contrast enhancement AUC is comparable, which indicates the preservation of perfusion in the embolized artery*

therapy is safe and effective on the basis of treatment of multiple myxoma aneurysms of cerebral vessels [25].

The role of chemotherapy for metastatic myxoma aneurysms of cerebral vessels as well as the role of radiation therapy is poorly understood. In the case of multiple aneurysms, M. Branscheidt et al. (2014) have used to stabilize their growth carboplatin (240 mg/m^2 of body surface on day 1) and etoposide (100 mg/m^2 of body surface, days 2 and 3) with dexamethasone (4 mg/day). However, 2 months after completion of 6 cycles (with 4-week intervals between

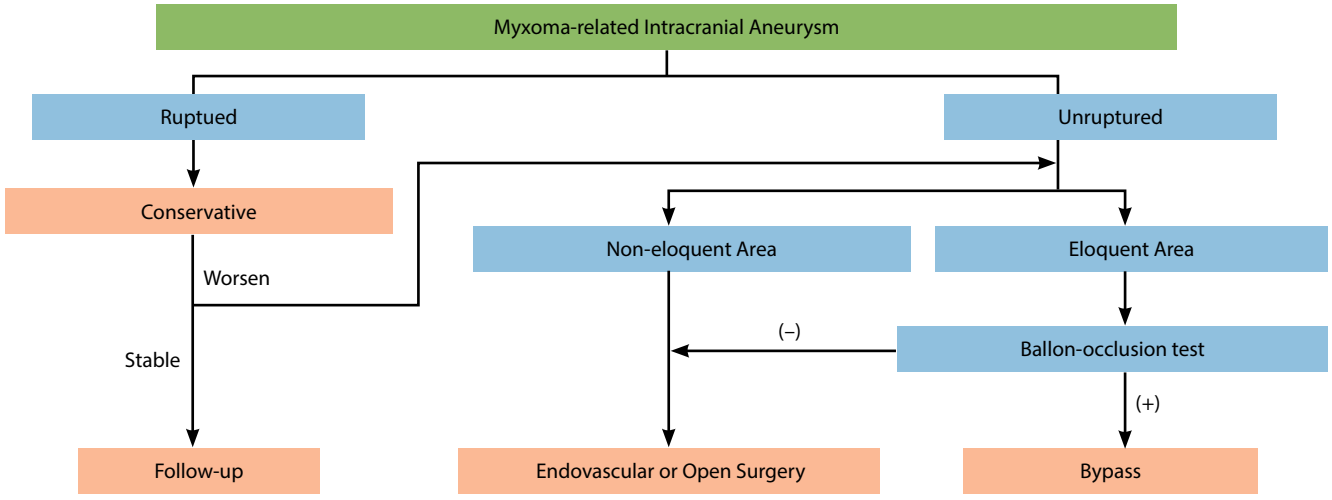

Fig. 6. *Algorithm for choosing a treatment method for a patient with intracranial aneurysms associated with cardiac myxoma (cit. according to [12])*

cycles), control MRI showed an increase in size, contrast and progression of edema. As a result, the aneurysm was resected in the patient. Nevertheless, the authors concluded that in cases of severe neurological disorders chemotherapy can stabilize the growth of aneurysms when waiting tactics are not a reasonable therapeutic solution [26].

The authors of one of the latest (2020) literature reviews with multiple myxoma brain aneurysms showed that in 19 of 41 cases patients were treated conservatively. All aneurysms were stable in size during the observation period (from 3 months to 11 years, mean is 40 months), their growth was noted in 3 observations [12].

There are no clear recommendations regarding the choice of treatment method for patients with aneurysms associated with myxomas in actual literature, in each case the decision is made individually. A. Santillan et al. (2019) proposed recommendations for monitoring such patients on the basis of the longest (21 and 22 years) observations described in the literature for 2 patients with multiple myxoma aneurysms:

- Patients with embolic cerebral infarction associated with atrial myxoma or with history of atrial myxoma resection are undergoing diagnostic cerebral angiography and subsequently a careful monitoring is carried out using non-invasive imaging studies, such as magnetic resonance angiography or SCTA, which should be performed every 12–15 months for the first 3–5 years, and then every 2 years [17].
- If the aneurysm is treatable, endovascular embolization is recommended [27]. But if a fusiform aneurysm is detected, which is asymptomatic and has a stable size, it is indicated a dynamic noninvasive imaging constant disaggregant therapy to prevent thromboembolism. If a patient has a fusiform aneurysm that is progressed or ruptured, the preferred method of treatment is microsurgical or endovascular aneurysm trapping [10, 22, 28–31].

Based on their own clinical observation and literature review, S. Gai et al. in 2019 have presented an algorithm for choosing treatment method for patients with multiple aneurysms depending on aneurysm rupture presence, localization, results of test balloon occlusion and taking into account of the aneurysm size stability (Fig. 6).

The presented case is characterized by observation of formation of distal cerebral aneurysm associated with myxoma, as well as, probably, an aneurysm being formed distally along the artery along with multiple clinically manifested caverns of the brain. Hemorrhagic manifestation of cavernous malformations preceded formation of aneurysm of cortical branch of the middle cerebral artery on the background of its fusiform expansion. An assumption of a diagnostic error, overdiagnosis of cavernoma on the background of parenchymal hemorrhage is refuted by the results of histological examination of the removed sample.

The patient underwent reconstructive aneurysm shutdown by embolization with microspirals which became possible due to the anatomical shape that favored this type of treatment. Conversion to destructive intervention was not required.

Due to the natural course of the disease, the patient remains at risk of progression of a distal aneurysm in the basin of the same artery, and therefore it will be observed in dynamics.

CONCLUSION

All patients with cardiac myxomas, both before and in dynamics after their removal, are recommended to perform noninvasive visualization of brain vessels to verify such complication as metastatic (myxomal) aneurysms. The prognosis in most patients with multiple intracranial aneurysms associated with myxoma is favorable, and most aneurysms associated with myxoma are stable. But in cases with progressive or ruptured aneurysms, surgical treatment options should be considered, including microsurgical or endovascular eradication. Radiation and chemotherapy methods may have a certain therapeutic value

REFERENCES

1. Medvedev Yu. A., Zabrodskaya Yu. M. A new conception of the origin of bifurcational aneurysms of brain stem arteries. St. Petersburg, 2000. (In Russ.).
2. Sandok B.A., von Estorff I., Giuliani E.R. Subsequent neurological events in patients with atrial myxoma. Ann Neurol 1980;8:305–7. DOI: 10.1002/ana.410080314
3. Vaideeswar P., Butany J.W. Benign cardiac tumors of the pluripotent mesenchyme. Semin Diagn Pathol 2008;25:20–8. DOI: 10.1053/j.semdp.2007.10.005
4. Pinede L., Duhaut P., Loire R. Clinical presentation of left atrial cardiac myxoma. A series of 112 consecutive cases. Medicine (Baltimore) 2001;80: 159–72. DOI: 10.1097/00005792-200105000-00002
5. Wen X.Y., Chen Y.M., Yu L.L. et al. Neurological manifestations of atrial myxoma: A retrospective analysis. Oncol Lett 2018;16: 4635–9. DOI: 10.3892/ol.2018.9218
6. Brinjikji W., Morris J.M., Brown R.D. et al. Neuroimaging findings in cardiac myxoma patients: a single-center case series of 47 patients. Cerebrovasc Dis 2015;40(1–2):35–44. DOI: 10.1159/000381833
7. Zheng J., Li S., Cao Y. et al. Multiple cerebral myxomatous aneurysms: what is the optimal treatment? J Stroke Cerebrovasc Dis 2015;24:232–8. DOI: 10.1016/j.jstrokecerebrovasdis.2014.08.017
8. Sabolek M., Bachus-Banaschak K., Bachus R. et al. Multiple cerebral aneurysms as delayed complication of left cardiac myxoma: a case report and review. Acta Neurol Scand 2005;111:345–50. DOI: 10.1111/j.1600-0404.2005.00413.x
9. Viganò S. Papini G.D., Cotticelli B. et al. Prevalence of cerebral aneurysms in patients treated for left cardiac myxoma: a prospective study. Clin Radiol 2013 Nov;68(11):e624–8. DOI: 10.1016/j.crad.2013.06.010
10. Yilmaz M.B., Akin Y., Güray Ü. et al. Late recurrence of left atrial myxoma with multiple intracranial aneurysms. Int J Cardiol 2003;87:303–5. DOI: 10.1016/s0167-5273(02)00348-0
11. Efimov V.V., Fedorchenko A.N., Porkhanov V.A. et al. Cerebral infectious aneurysm as a complication of bacterial endocarditis. E'ndovaskulyarnaya khirurgiya = Russian Journal of Endovascular Surgery.2021;8:228–36. (In Russ.). DOI: 10.24183/2409-4080-2021-8-2-228-236
12. Gai S., Lv N., Zhang Q. et al. Multiple intracranial aneurysms caused by myxoma: case report and literature review. World Neurosurg 2020;133:e376–84. DOI: 10.1016/j.wneu.2019.09.014
13. Saffie P., Riquelme F., Mura J. et al. Multiple myxomatous aneurysms with bypass and clipping in a 37-year-old man. J Stroke Cerebrovasc Dis 2015;24:e69–e71. DOI: 10.1016/j.jstrokecerebrovasdis.2014.10.017
14. Ezerioha N., Feng W. Intracardiac myxoma, cerebral aneurysms and elevated interleukin-6. Case Rep Neurol 2015;7(2):152–5. DOI: 10.1159/000437256
15. Morgan L., Cooper J., Montgomery H. et al. The interleukin-6 gene –174G>C and –572G>C promoter polymorphisms are related to cerebral aneurysms. J Neurol Neurosurg Psychiatry 2006;77(8):915–7. DOI: 10.1136/jnnp.2005.081976
16. Steiner T., Juvela S., Unterberg A. et al. European Stroke Organization guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. Cerebrovasc Dis 2013;35(2):93–112. DOI: 10.1159/000346087
17. Santillan A., Knopman J., Patsalides A., Pierre Gobin Y. Delayed intracranial aneurysm formation after cardiac myxoma resection: Report of two cases and review of the literature. Interv Neuroradiol 2019;25(2):177–81. DOI: 10.1177/1591019918808026
18. Albuquerque-Peres C.M., Castro de Brito A., Mesquita-Peres C.H. et al. Oncotic (myxomatous) aneurysms: a review of management aneurismas oncóticos (mixomatosos). Brazilian Neurosurgery 2022; (02): e167–73. DOI: 10.1055/s-0041-1734013
19. Hwang B.J., Connelly M.M., Lev M.H. Distinctive MR imaging appearance of hemorrhagic cerebral aneurysms associated with atrial myxoma. AJR Am J Roentgenol 2001;177:925–7. DOI: 10.2214/ajr.177.4.1770925
20. Liao W.H., Ramkalawan D., Liu J.L. et al. The imaging features of neurologic complications of left atrial myxomas. Eur J Radiol 2015;84:933–9. DOI: 10.1016/j.ejrad.2015.02.005
21. Sen'ko I.V., Krylov V.V. Microsurgery of distal brain aneurysms. Neyrokhirurgiya = Russian Journal of Neurosurgery 2016;1:98–103. (In Russ.).
22. Eddleman C.S., Gottardi–Littell N.R., Bendok B.R. et al. Rupture of cerebral myxomatous aneurysm months after resection of the primary cardiac tumor. Neurocrit Care 2010;13(2):252–5. DOI: 10.1007/s12028-010-9400-z
23. Sorenson T.J., Brinjikji W., Lanzino G. Giant fusiform intracranial aneurysm in patient with history of myxoma. World Neurosurg 2019;128:200–1. DOI: 10.1016/j.wneu.2019.05.051
24. Sedat J., Chau Y., Dunac A. et al. Multiple cerebral aneurysms caused by cardiac myxoma. A case report and present state of knowledge. Interv Neuroradiol 2007;13(2):179–84. DOI: 10.1177/159101990701300209
25. Khatibi K., Ponce Mejia L.L., Kaneko N. et al. Targeted radiation therapy can treat myxomatous cerebral aneurysms. World Neurosurg 2020;143:332–5. DOI: 10.1016/j.wneu.2020.08.022
26. Branscheidt M., Frontzek K., Bozinov O. et al. Etoposide/carboplatin chemotherapy for the treatment of metastatic myxomatous cerebral aneurysms. J Neurol 2014;261(4):828–30. DOI: 10.1007/s00415-014-7281-3
27. Lazarow F., Aktan S., Lanier K., Agola J. Coil embolization of an enlarging fusiform myxomatous cerebral aneurysm. Radiol case reports 2018;13:490–4. DOI: 10.1016/j.radcr.2017.11.015
28. Sveinsson O., Herrman L. Multiple cerebral aneurysms in a patient with cardiac myxoma: what to do? BMJ Case Rep 2015;2015: bcr2013200767. DOI: 10.1136/bcr-2013-200767
29. George K.J., Rennie A., Saxena A. Multiple cerebral aneurysms secondary to cardiac myxoma. Br J Neurosurg 2012;26:409–1. DOI: 10.3109/02688697.2011.626877
30. Flores P.L., Haglund F., Bhogal P. et al. The dynamic natural history of cerebral aneurysms from cardiac myxomas: A review of the natural history of myxomatous aneurysms. Interv Neuroradiol 2018; 24(3):277–83. DOI: 10.1177/1591019917754070
31. Penn D.L., Lanpher A.B., Klein J.M. et al. Multimodal treatment approach in a patient with multiple intracranial myxomatous aneurysms. J Neurosurg Pediatr 2018;21:315–21. DOI: 10.3171/2017.9.PEDS17288

Author's contributions
R.S. Martynov: literature review, article writing;
A.V. Savello: scientific editing of the article;
A.N. Savchuk: writing an article;
K.N. Babichev: writing an article;
A.I. Kiskaev: article writing;
D.V. Svistov: scientific editing of the article.

ORCID of authors
R.S. Martynov: https://orcid.org/0000-0002-2769-3551
A.V. Savello: https://orcid.org/0000-0002-2465-4856
K.N. Babichev: https://orcid.org/0000-0002-4797-2937
A.I. Kiskaev: https://orcid.org/0000-0002-6936-3040
D.V. Svistov: https://orcid.org/0000-0002-3922-9887

Conflict of interest. The author declares no conflict of interest.

Financing. The study was performed without external funding.

Article submitted: 19.08.22. **Accepted for publication:** 24.11.2022