Thromboembolic Cerebral Aneurysms

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Abstract

Intracranial hemorrhage is the most common outcome of cerebral aneurysms, and, therefore, clinical guidelines for the management of patients with cerebral aneurysms are primarily based on assessing the risk of their rupture. Brain ischemia due to the cerebral aneurysms occur significantly less frequently (i.e. in 3–5% of cases), and, in most cases, it is caused by distal embolism with thrombotic masses from large and giant thrombosed aneurysms. A conclusion that ischemic stroke is associated with embolism of thrombi from the aneurysm sac can be made only after ruling out other risk factors, primarily cardioembolism and stenosis of intracranial and extracranial arteries. The management of patients with thromboembolism from aneurysms who developed ischemia is challenging because these patients require antithrombotic agents, which can result in recanalization and rupture of the aneurysm. In addition, the optimal timing for surgery for the aneurysm in the event of acute ischemia has not been determined, given the high risk of recurrent embolism and aneurysm rupture. We present an overview of recent studies on this issue and our experience in managing 4 patients with thromboembolic stroke caused by cerebral aneurysms.

Keywords: *cerebral aneurysm; ischemic stroke; embolism; thrombosis*

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Тромбоэмболический тип течения церебральных аневризм

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Аннотация

Геморрагический тип течения церебральных аневризм встречается наиболее часто, в связи с чем клинические рекомендации по ведению пациентов с аневризмами головного мозга в первую очередь основываются на оценке риска их разрыва. Ишемический тип течения церебральных аневризм встречается значимо реже (в 3–5% случаев) и чаще всего связан с дистальной эмболией тромботических масс из больших и гигантских тромбированных аневризм. Сделать заключение о том, что ишемический инсульт ассоциирован с эмболией тромбов из мешка аневризмы, можно только при исключении других факторов риска, в первую очередь кардиоэмболии и стенозов интра- и экстракраниальных артерий. Имеются сложности в определении тактики ведения пациентов с тромбоэмболией из аневризм и развитием ишемии, т. к. требуется назначение антитромботических препаратов, которые могут способствовать реканализации аневризмы и её разрыву. Кроме того, в настоящее время не определены оптимальные сроки для выполнения хирургического вмешательства на аневризме при развитии острой ишемии, учитывая высокий риск повторных эпизодов эмболии, а также риск разрыва аневризмы. В статье представлены краткий обзор современных исследований по данной проблеме и собственный опыт ведения 4 пациентов с тромбоэмболическим вариантом течения церебральных аневризм.

Ключевые слова: *церебральная аневризма; ишемический инсульт; эмболия; тромбоз*

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

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

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Introduction

Every year, 10–14 aneurysmal subarachnoid hemorrhages occur per 100 thousand population in Russia [1, 2]. Cerebral aneurysms (CAs) are found in 2–5% of the population, with most of them being small and asymptomatic [3]. The incidence of ischemic stroke (IS) is much higher (i.e. up to 2.5–3.0 cases per 1000 population per year) [2]. Based on these data on the prevalence aneurysm in the population and the annual incidence of IS, we can assume that some strokes and transient ischemic attacks occur in patients with asymptomatic CAs.

A history of aneurysm is a contraindication for systemic thrombolysis in patients with IS. However, angiography is not required to perform systemic thrombolysis, and most patients do not know if they have asymptomatic aneurysms. There have been isolated reports describing ruptures of asymptomatic aneurysms during thrombolytic therapy in patients with IS [4, 5]. However, these could be not asymptomatic aneurysms but those ruptured and partially thrombosed in the past, so the administration of a fibrinolytic agent could promote lysis of the thrombus and re-rupture of the aneurysm. A large cohort study did not detect any cases of rupture of asymptomatic aneurysms during thrombolytic therapy [6].

Therefore, in most cases CAs are a concomitant condition in IS patients. However, in 3–5% of cases, thrombosed aneurysms (TAs) can themselves cause IS, most often due to the embolism with thrombotic masses from the sac into the distal branches [7–9]. The rate is even higher in large and giant aneurysms. According to V.V. Krylov et al., in a group of patients with giant aneurysms of the middle cerebral artery, 6.9% of patients had embolism resulting in cerebral ischemia [10].

According to several studies, embolism of thrombi from the aneurysm sac was associated with an increased risk of rupture in the near future [9, 11–13].

Aim. The study aimed to review cases of thromboembolic CAs.

Materials and methods

We reviewed medical records of 240 patients with CAs who were treated at the regional vascular center of the Krasnoyarsk Regional Clinical Hospital in 2022–2023. Of these, 136 patients had surgery in the acute period of aneurysm rupture, 56 had a history of rupture, and 48 had unruptured aneurysms. In 4 (1.6%) patients, CAs manifested as IS.

A causal relationship between an aneurysm and stroke can be established based on the following:
- a large partially or completely TA;
- ischemia in the branches distal to the TA;
- no other risk factors for stroke, mainly cardioembolism and hemodynamically significant stenoses of extracranial and intracranial arteries.

We reviewed history, clinical, and neurovisualization data of patients with IS associated with TA.

Results

Our patients with thromboembolic cerebral aneurysms included 2 men and 2 women. Two patients were aged 22 and 24 years, and the other two were older (57 and 62 years old).

All patients had aneurysms larger than 10 mm (i.e. 13, 15, 15, and 20 mm). TAs were located in the middle cerebral artery (MCA) or internal carotid artery (ICA) in 3 and 1 cases, respectively. In 3 cases, the aneurysms had a small functioning part; in 1 case, the aneurysm was completely thrombosed.

One patient developed lacunar stroke in the internal capsule, and another had a large ischemic lesion in subcortical structures. One patient developed ischemia in the territory of the frontal branch of MCA M2, and another patient had multiple foci of subcortical and cortical ischemia.

In 3 patients with acute ischemia, native multislice computed tomography (MSCT) showed a hyperdense signal from a part of the TA, which could suggest acute formation of a thrombus, which partially migrated to the distal branches and caused cerebral infarction.

All patients were prescribed antiplatelet agents to treat acute IS. There were no recurrent ischemia episodes. Three patients with partially TAs underwent elective surgery (osteoplastic craniotomy, microsurgical clipping of the aneurysm) 3 months after IS or later. The patient with completely TA and MCA thrombosis did not have any surgery.

We present clinical cases of the patients with thromboembolic CAs.

Clinical case 1

Patient B., a 57-year-old woman, was admitted to the primary vascular department with severe right-sided hemiparesis (up to score 3 in the arm and leg). She had severe neurological deficit with an NIHSS score of 14. MSCT showed an ischemic area in the putamen, internal capsule, and head of the caudate nucleus on the left (Fig. 1, *A*). We can see a hyperdense area in the Sylvian fissure, which should be differentiated from hemorrhage and TA (Fig. 1, *B*). MSCT angiography showed a functioning part of the TA of MCA M1 (Fig. 1, *C*). After 3 months, the patient was hospitalized for planned surgery for MCA aneurysm. Follow-up native MSCT showed isodense signal from the aneurysm; follow-up MSCT angiography showed a functioning part of the aneurysm of up to 3.5 mm (Fig. 1, *D*, *E*).

In the acute period of IS, the thrombosed part of the aneurysm had an hyperdense signal, suggesting a fresh thrombus (Fig. 1, *B*), and after 3 months the signal from the thrombus became isodense (Fig.1, *D*). Based on the areas of ischemia in the territory of the lenticulostriate branches and a fresh thrombus in the aneurysm, we can assume that the ischemia was caused by thrombosis of the striatal branches of the MCA by an embolus from the aneurysm sac. The patient did not have any other IS risk factors. Microsurgical clipping of the aneurysm was performed. A small functioning part of the aneurysm and a large thrombosed part (13 mm) were identified during the surgery. The sac was opened, the blood clots were removed, and a permanent clip was placed on the neck formed. The patient was discharged with preoperative functional status of modified Rankin score 3.

Clinical case 2

Patient K., a 62-year-old man, was admitted to the primary vascular department with right-sided hemiparesis (score 2 in the arm, score 3 in the leg) and motor aphasia; his NIHSS score was 16. MSCT showed an ischemic area in the left frontal lobe extending to subcortical structures. Native MSCT suspected MCA TA of up to 2 cm (Fig. 2, *A*, *B*). Due to un-

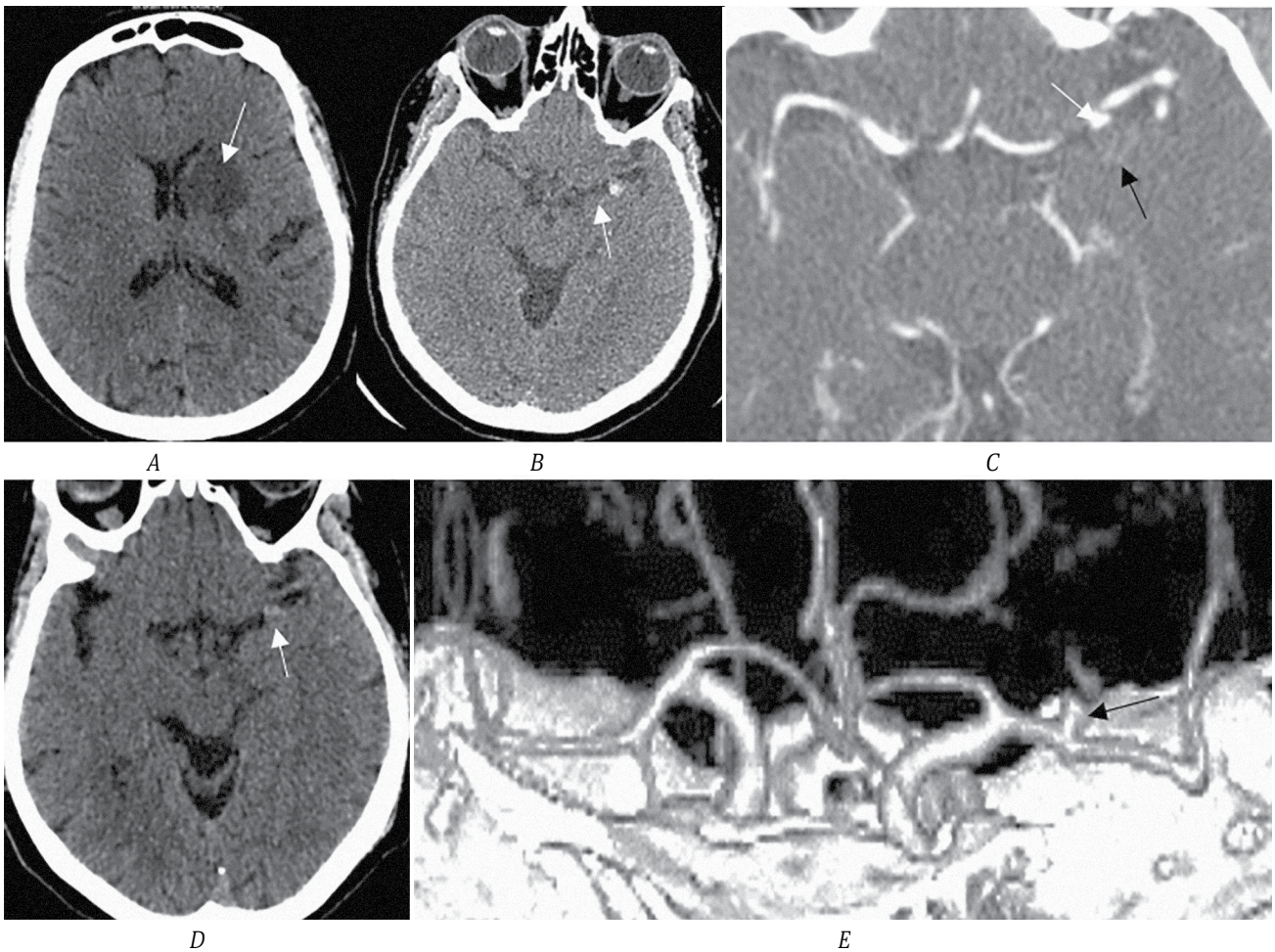

Fig. 1. Brain MSCT of patient B.
A, acute ischemia in the head of the caudate nucleus, internal capsule, and putamen (arrow); *B*, thrombosed part of the aneurysm (arrow); *C*, functioning part of the aneurysm (white arrow); the black arrow indicates the contours of the thrombosed part; *D*, MSCT 3 months after the stroke, the arrow indicates the thrombosed part of the aneurysm; *E*, 3D reconstruction of the functioning part of the aneurysm (arrow).

known reasons, MSCT angiography was not performed in the primary vascular department. Three months later, the patient was referred to the regional vascular center. MSCT showed a cystic-atrophic area in the area of the previous IS in the left frontal lobe. MSCT angiography showed a small functioning part of the M1 segment aneurysm and patent distal branches of the MCA (Fig. 2, *C*, *D*).

Considering a large thrombosed aneurysm sac in MCA M1 and ischemia in the territory of the frontal M2 branch of the MCA, thromboembolism from the aneurysm sac was considered. Three months later, angiography showed that all MCA branches were patent, which could be related to the recanalization of the thrombosed branch. An additional examination did not show any further risk factors for IS except for essential hypertension. Considering a high risk of recurrent thromboembolic complications, microsurgical clipping of the aneurysm was performed. After the surgery, his cognitive functions declined, and he and was discharged with a modified Rankin score of 4.

Clinical case 3

Patient I., a 22-year-old woman, was delivered by ambulance with complaints of weakness and difficulty to control her movements in the right limbs. The patient presented fully conscious, with right-sided hemiparesis scoring up to 3.5 in the arm and 4 in the leg. MSCT showed a round-shaped lesion of hyperdense density in the projection of the Sylvian fissure with a TA suspected. The lesion had areas of different densities, including a high-density area, which was a possible sign of acute thrombosis (Fig. 3, *A*). MSCT angiography showed no blood flow in the MCA on the left and no functioning part of the aneurysm. Considering the mild neurological deficit (NIHSS score 5), acute thrombosis of the MCA aneurysm with the artery itself was unlikely. Brain MRI was performed. A large MCA aneurysm with thrombosis along the whole MCA was identified. An ischemic lesion was seen in the internal capsule, which corresponded to the neurological deficit. Therefore, thrombosis of the MCA aneurysm and MCA itself was likely to be chronic, and

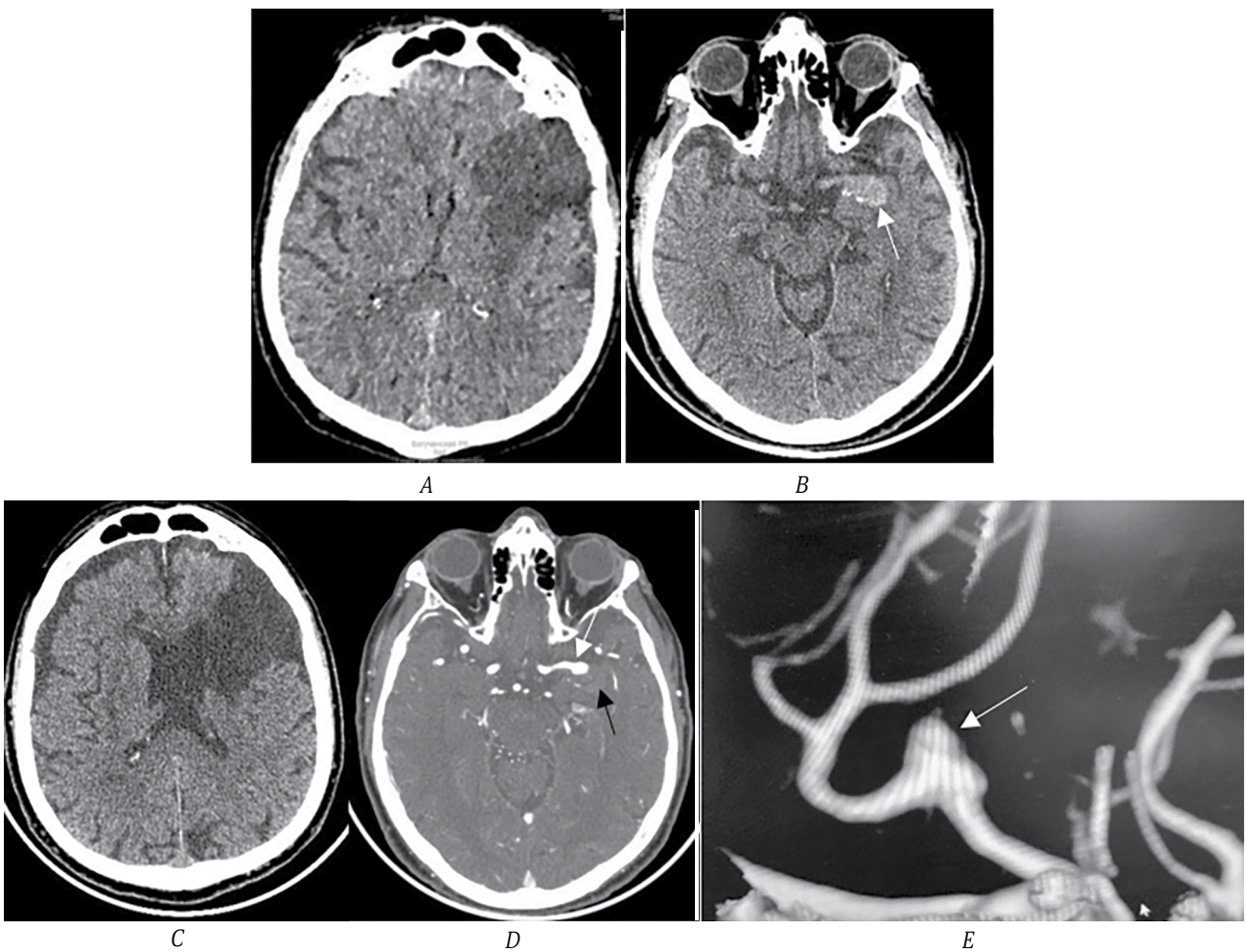

Fig. 2. Brain MSCT of patient K.
A, native MSCT, an ischemic area is seen in the left frontal lobe; *B*, the arrow indicates MCA TA with calcification of up to 2 cm; *C*, MSCT 3 months after IS, an area of cystic-atrophic changes in the left frontal lobe is seen; *D*, MSCT angiography, the white arrow indicates the functioning part of the aneurysm, the black arrow indicates the contour of the thrombosed part; *E*, 3D reconstruction of MSCT angiography. The arrow indicates the functioning part of the aneurysm.

the current clinical picture was caused by ischemia in the subcortical structures supplied with blood by the lenticulostriate arteries. Ischemia could be associated either with thromboembolism or enlargement of the aneurysm (a fresh thrombus) leading to an arterial occlusion.

This young patient did not have any other stroke risk factors. Cerebral angiography and MSCT perfusion were performed to determine treatment strategy. Angiography showed good collateral blood flow. MSCT perfusion did not show any significant differences in capillary blood flow in both hemispheres. Non-surgical treatment was chosen with follow-up angiography. The patient was followed up 6 months after; she had no recurrent ischemia episodes and had a modified Rankin score of 0. There were no MSCT angiography findings suggesting recanalization of the aneurysm and MCA during the administration of the antiplatelet therapy.

Clinical case 4

Patient A., a 24-year-old man, was admitted to the emergency room with acute right-sided hemiplegia. Brain MSCT showed an area of increased density in the chiasmatic-sellar region on the left, which had to be identified as a hemorrhagic focus or TA of the ICA (Fig. 4, *A*). MSCT angiography showed a small functioning part of the aneurysm in the left ICA. The ICA and MCA were patent along their entire length.

Brain MRI was performed. A 15-mm TA of the left ICA was seen. In DWI mode, areas of acute ischemia (restricted diffusion) were seen in the head of the caudate nucleus on the right, putamen, insular cortex, and lateral and medial regions of the right frontal lobe. Given this multifocal nature of ischemia and the presence of a TA of the ICA, embolism from the sac was likely to lead to the formation of multiple ischemic

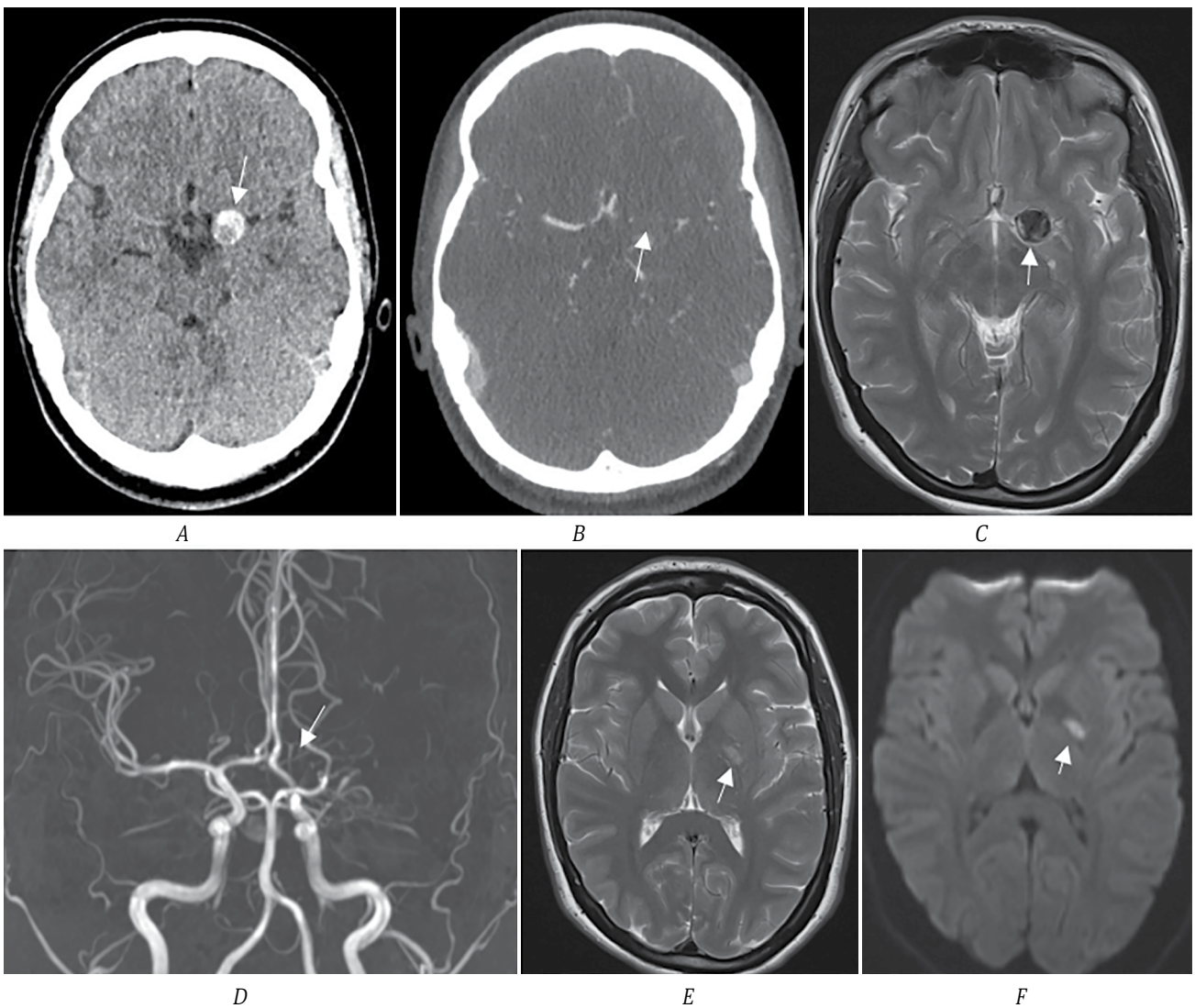

Fig. 3. Brain MSCT of patient I.
A, TA is indicated with the arrow; *B*, MSCT angiography, the white arrow indicates no blood flow in the left MCA; *C*, T2-WI MRI, the arrow indicates MCA TA; *D*, MR angiography, no blood flow in the left MCA (shown with the arrow); *E*, *F*, T2-WI and DWI, an ischemic lesion in the internal capsule is shown with the arrow.

areas. No other risk factors for IS were found in this young patient. The patient's neurological status was assessed an NIHSS score of 11. The patient had elective microsurgical clipping of the ICA aneurysm. He was discharged with a modified Rankin score of 2.

Discussion

Neurologists and neurosurgeons are aware of embolic CAs; however, there have been only isolated clinical cases described in scarce scientific publications. L. Calviere et al. [14] described one of the largest case series of embolic CA. They followed up 15 patients with IS and transient ischemic attack associated with thromboembolism from the CA. The mean age of patients was 49.7 years. The mean aneurysm diameter was 7.5 mm. During antiplatelet therapy, no patient had recurrent episodes of ischemia. However, the aneurysms ruptured in 2 patients.

In the near future, we can expect increased CA detectability in patients with IS due to the development and increasing use of the thrombectomy technique, which requires angiography. When identifying CAs in patients with IS, doctors should not only assess the risk of its rupture or determine contraindications to thrombolytic therapy but also assume whether the aneurysm, especially a large one, could contribute to the development of cerebral ischemia.

IS in patients with unruptured CAs may be explained by several mechanisms. Firstly, spontaneous thrombosis of the aneurysm can be associated with inflammatory changes in its wall [13]. A. Fomenko et al. presented a clinical case of a 56-year-old man with spontaneous thrombosis of the MCA aneurysm spreading into the lumen of the artery, which led to ischemia throughout the MCA territory [15]. In some cases, with giant aneurysms, adjacent branches can be mechanically compressed, resulting

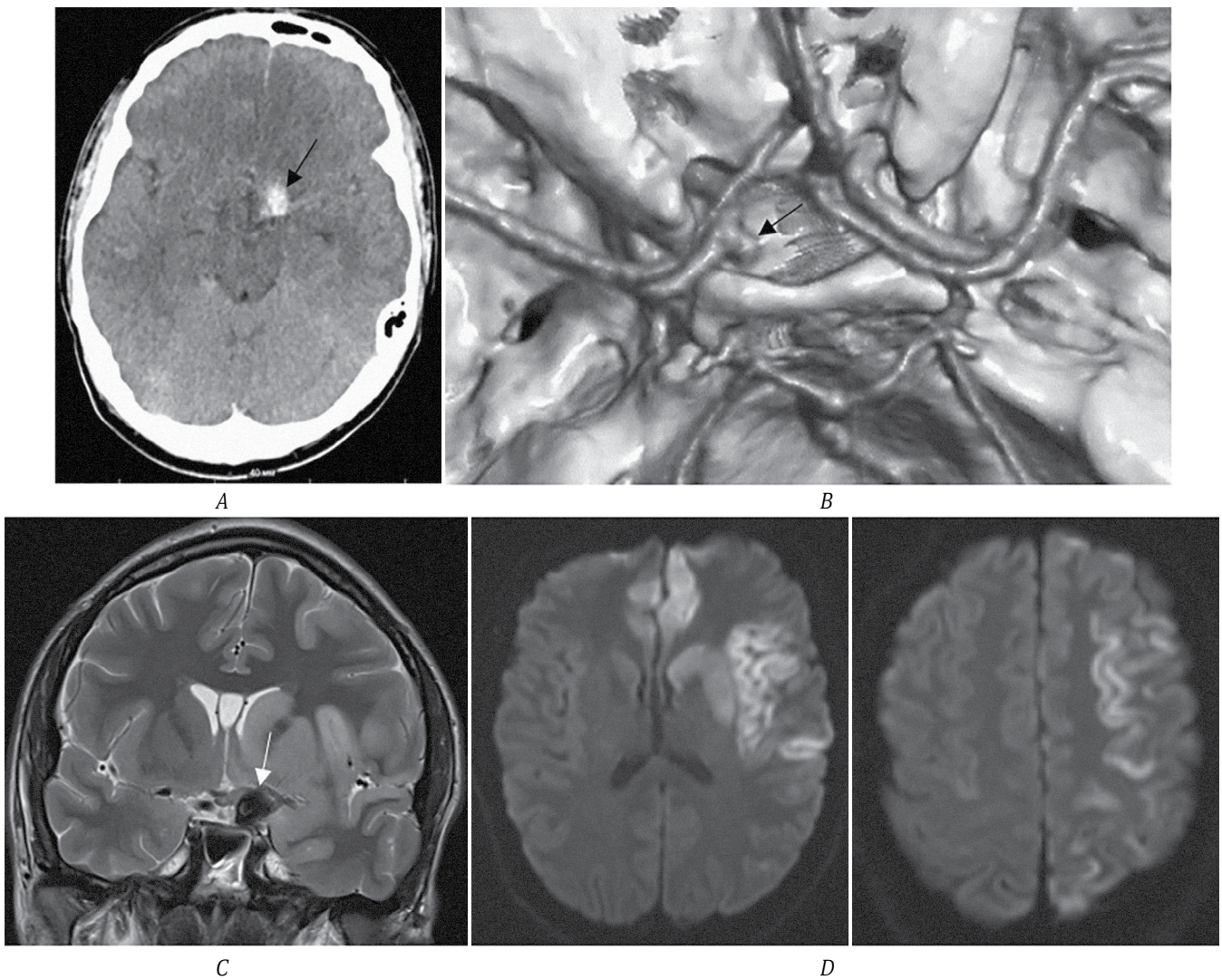

Fig. 4. Brain MSCT of patient A.
A, the arrow indicates the TA of the left ICA; *B*, MSCT angiography, 3D reconstruction, the arrow indicates the functioning part of the aneurysm of the left ICA; *C*, brain T1-WI MRI, the arrow indicates the TA of the left ICA; *D*, MRI in DWI mode, areas of acute ischemia in the subcortical structures of the left brain hemisphere.

in ischemia. However, thromboembolism most often occurs from a completely or partially thrombosed aneurysm sac into the distal cerebral branches [16].

It is not always possible to prove embolism of thrombotic masses from the aneurysm sac. To do so, it is necessary to verify the aneurysm with signs of thrombosis and exclude all other risk factors for IS, primarily cardioembolism and stenoses of extracerebral and intracerebral arteries. Paradoxical embolism through a patent foramen ovale should be ruled out in young patients.

Therefore, TA-associated stroke is more likely to be assumed in patients without other risk factors and with an infarction in the area of the blood supply of the artery carrying the aneurysm. However, as shown in our cases, this may also occur in patients of older age groups.

Thromboembolism can occur both from large/giant aneurysms and aneurysms of up to 1 cm in size [17–19]. All aneurysms in our case series were more than 1 cm in size.

However, what is more important is not the size of the aneurysm itself but whether it contains blood clots. Partial thrombosis of the aneurysm sac can be detected by MSCT angiography or cerebral angiography only based on indirect signs, such as uneven contour or unusual flow of contrast around the thrombosed part. Therefore, it is recommended to perform brain MRI, which can identify the thrombosed part of the aneurysm.

Completely TAs are especially challenging because they are not contrasted during angiography but can also be a source of thromboembolism. In these cases, completely TAs can be detected only by MRI, especially in T2-WI and SWI.

Considering that embolism from the CA may increase the risk of rupture in the near future [9, 11, 12], it is not known if such patients can be safely prescribed antithrombotic therapy, as required by the standards of care for IS. N. Kuroda et al. described a case of TA rupture on the fourth day of IS treatment with antithrombotic therapy [11].

There are no guidelines on the optimal timing of open or endovascular surgical management of the CA after stroke, including that to prevent recurrent episodes of thromboembolism from the aneurysm sac [20, 21].

Conclusion

Cases of cerebral thromboembolism due to CAs are rare and occur mainly in patients with large or giant TAs. In some cases, TA can be suspected based on native MSCT, especially if the patient has a fresh hyperdense thrombus. However, sometimes the aneurysm remains undetected, and recurrent embolism can develop from the sac followed by recurrent strokes and transient ischemic attacks. There are no current guidelines for the management of such patients, such as whether they can be administered with antithrombotic agents and what time is optimal for surgery, so further multicenter studies are needed.

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