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Case Report

INTRACRANIAL EMBOLIZATION: A RARE COMPLICATION

Igor Lazić[1], Marko Todorović[2], Saša Knežević[1], Ivan Vukašinović[3], Žarko Nedeljković[3], Vladimir Jovanović[2,4]

[1]Center for Anesthesiology and Reanimatology, University Clinical Center of Serbia, Belgrade, Serbia
[2]Clinic for Neurosurgery, University Clinical Center of Serbia, Belgrade, Serbia
[3]Center for Radiology, University Clinical Center of Serbia, Belgrade, Serbia
[4]Medical school, University of Belgrade, Serbia

Abstract. *We report a case of a rare complication: aortic pseudoaneurysm rupture after embolization of an intracranial aneurysm in a 62 –year- old patient with spontaneous subarachnoid hemorrhage, presented with chest pain and shortness of breath the fourth day after the procedure. The intracranial aneurysm was embolized with no intraoperative complications, and a postprocedural CT (computed tomography) scan showed no signs of intracranial rebleeding, ischemic lesions, or hydrocephalus. Cardiosurgical treatment was not indicated for the aortic pseudoaneurysm rupture.*

Key words: *intracranial embolization, complication, pseudoaneurysms.*

Introduction

Cerebral aneurysmal rupture almost always results in subarachnoid and/or intracerebral, intraventricular hemorrhage.

Subarachnoid hemorrhage (SAH) is the pathologic condition that exists when blood enters the subarachnoid space. Ruptured aneyrisms represent the most common cause of spontaneous SAH , accounting for 85% of spontaneous SAHs [1]. SAH affects six to nine people per 100,000 per year, has a 35% mortality. Both measures are bigger when -standardized by age.

It has been shown that smoking, high blood pressure, excessive alcohol consumption ,and female gender are the factors to increase the risk of aneyrismal formation and potential ruptures [2–9].

Numerous heritable systemic disorders, mainly connective tissue diseases, include brainaneurysms such as: autosomal dominant polycystic kidney disease [10], *Ehlers-Danlos* syndrome [11], *Marfan* syndrome [2, 13], *Loeys-Dietz* syndrome [14], *Alagille* syndrome [15, 16] and neurofibromatosis Type 1 [17,18].

Rehemorrhage is imminent danger so the first goal is occlusion of the ruptured aneurysm. Early repair of the ruptured aneurysm by endovascular coiling or neurosurgical clipping is essential, because it stops rehemorrhage occurring and limits the early brain injury from the hemorrhage and also delayed cerebral ischemia, which is reflected in neurological condition [19].

Pseudoaneurysm, or false aneurysm, of the thoracic aorta results from transmural disruption of the aortic wall, with the leak contained by surrounding mediastinal structures. Although it can be secondary to trauma [20] or infection, [21] previous cardiac surgery is the most frequent cause [22].

Most common complications include intraoperative bleeding and thromboembolic complications, less common ones being postembolization rebleeding [23,24] coil migration and vessel injury [25].

Correspondence to: Marko Todorović
Center for Anesthesiology and Reanimatology, University Clinical Center of Serbia, 2 Pasterova, 11000, belgrade, Serbia
E-mail: markotodorovic993@gmail.com
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Case

A 62 year-old patient was admitted to the emergency room, presenting with symptoms of spontaneous subarachnoid hemorrhage.The onset of symptoms started two days ago, including the loss of consciousness, which reoccurred on the day of hospital admission.

On admission, there was no focal neurological sign and no neck stiffness, the patient was grade I Hunt & Hess. The cerebrospinal fluid was bloody, and the super- natant xanthochromic.

CT scan showed subarachnoid hemorrhage (Figure 1) and angiography showed a saccular aneurysm on the bifurcation of left ACM 7,1x8,7x7,6mm in size. (Figure 2)

Thoracic radiography showed no signs of aneurismal aortic dilatations in mediastinum.

Two days after the admission the patient was treated endovascularly, the aneurysm was embolized with platinum micorspirals (Cosmos 8x25, Cosmos 6x18, Optima soft 4x13), no complications were observed. (Figure 3)

Postoperatively, the patient was extubated, awake, with no neurological deficit, febrile to 39,5C, control laboratory showed a rise in inflammation factors and antibiotic i.v. therapy was adjusted accordingly.

Control CT scan showed no signs of rehemorrhage, ischemic lesions or hydrocephalus. (Figure 4)

The fourth day after the intervention, the patient reported a strong sternal pain and loss of breath, which was followed by rapid neurological deterioration, loss of consciousness, agonal breathing. The patient was transferred to the ICU and reanimated. In the ICU the patient was intubated, connected to mechanical ventilation, and sedated continuously with propofol, hemodynamically unstable, stimulated with noradrenalin. The patient showed partial epileptic attacks in spite of the sedation and antiepileptic therapy.

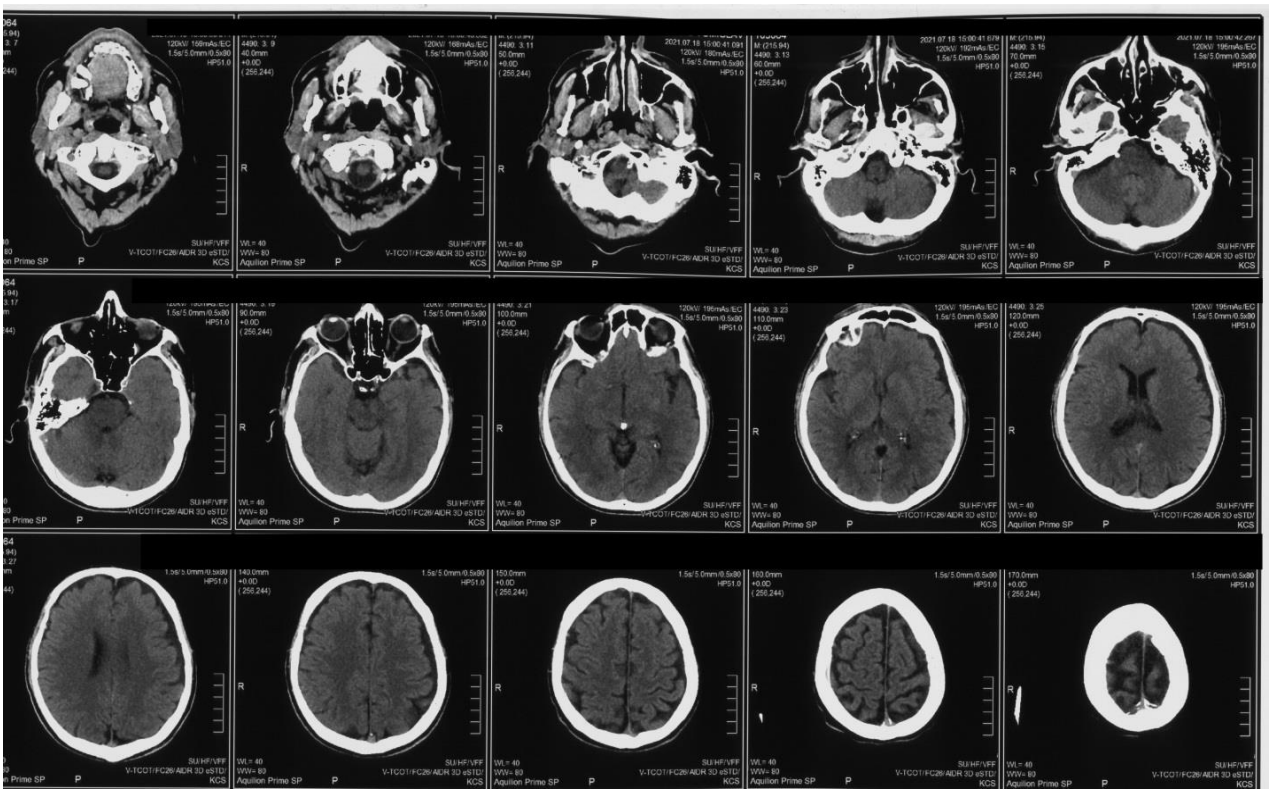

Fig 1 CT scan showed subarachnoid hemorrhage

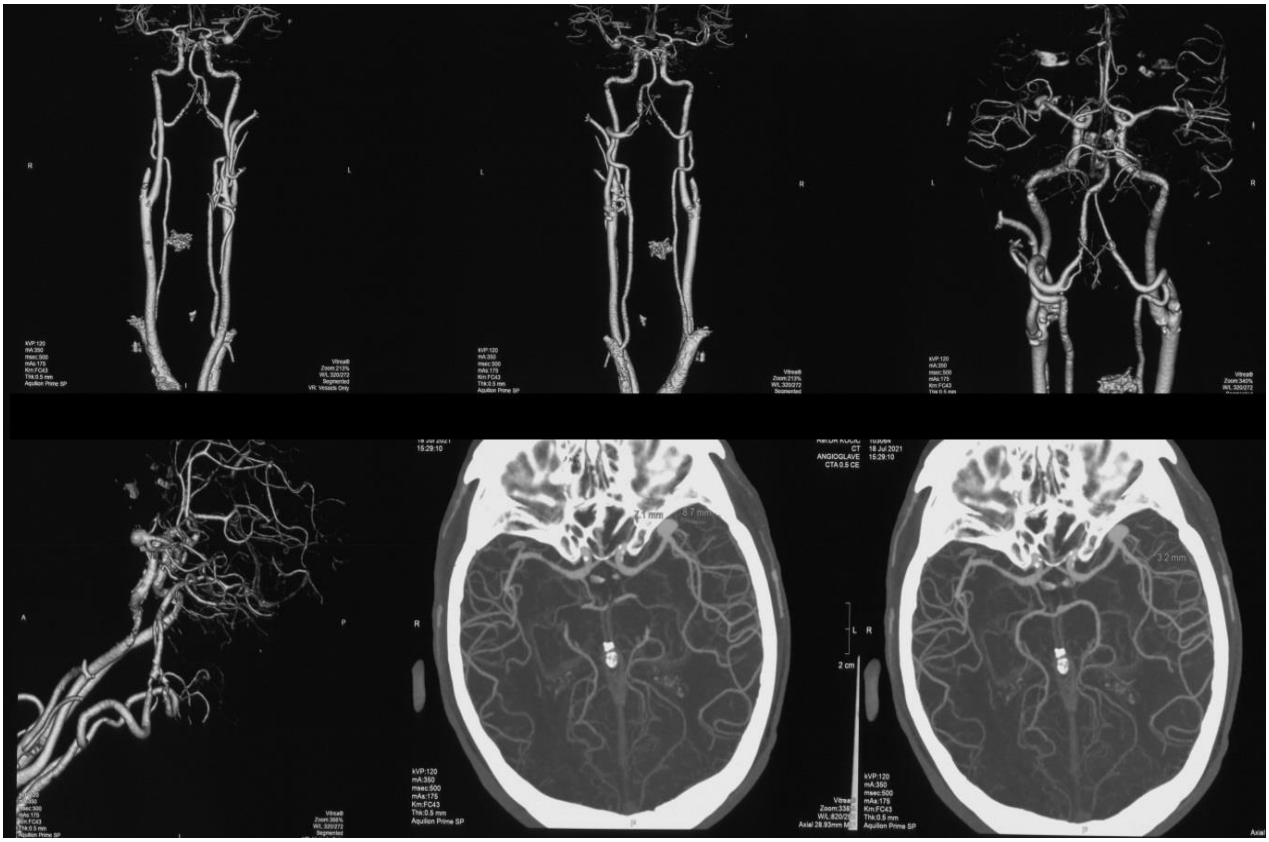

Fig. 2 Angiography showed a saccular aneurysm on the bifurcation of left ACM 7,1x8,7x7,6mm in size

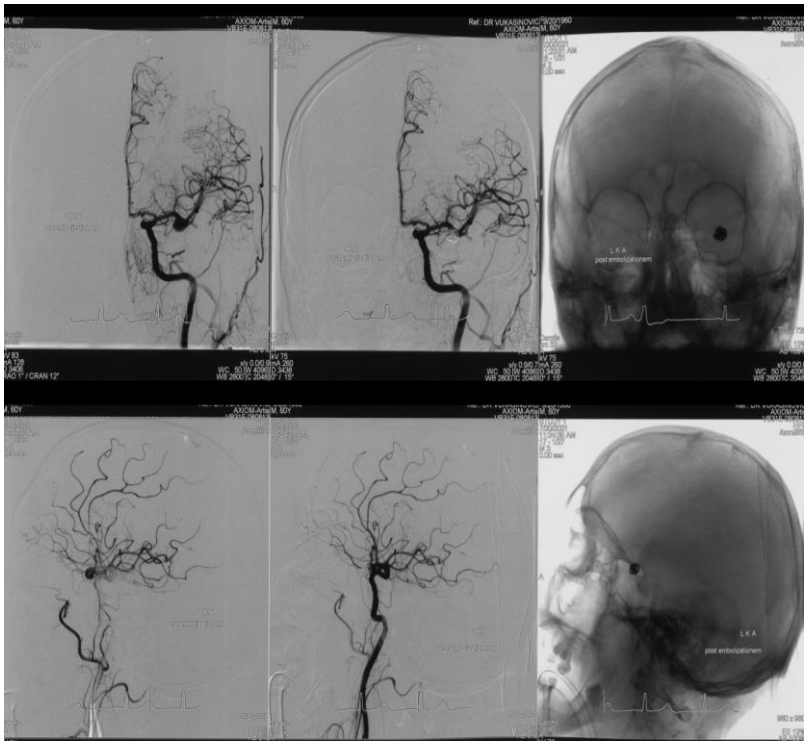

Fig. 3 Aneurysm was embolized with platinum micorspirals (Cosmos 8x25, Cosmos 6x18, Optima soft 4x13)

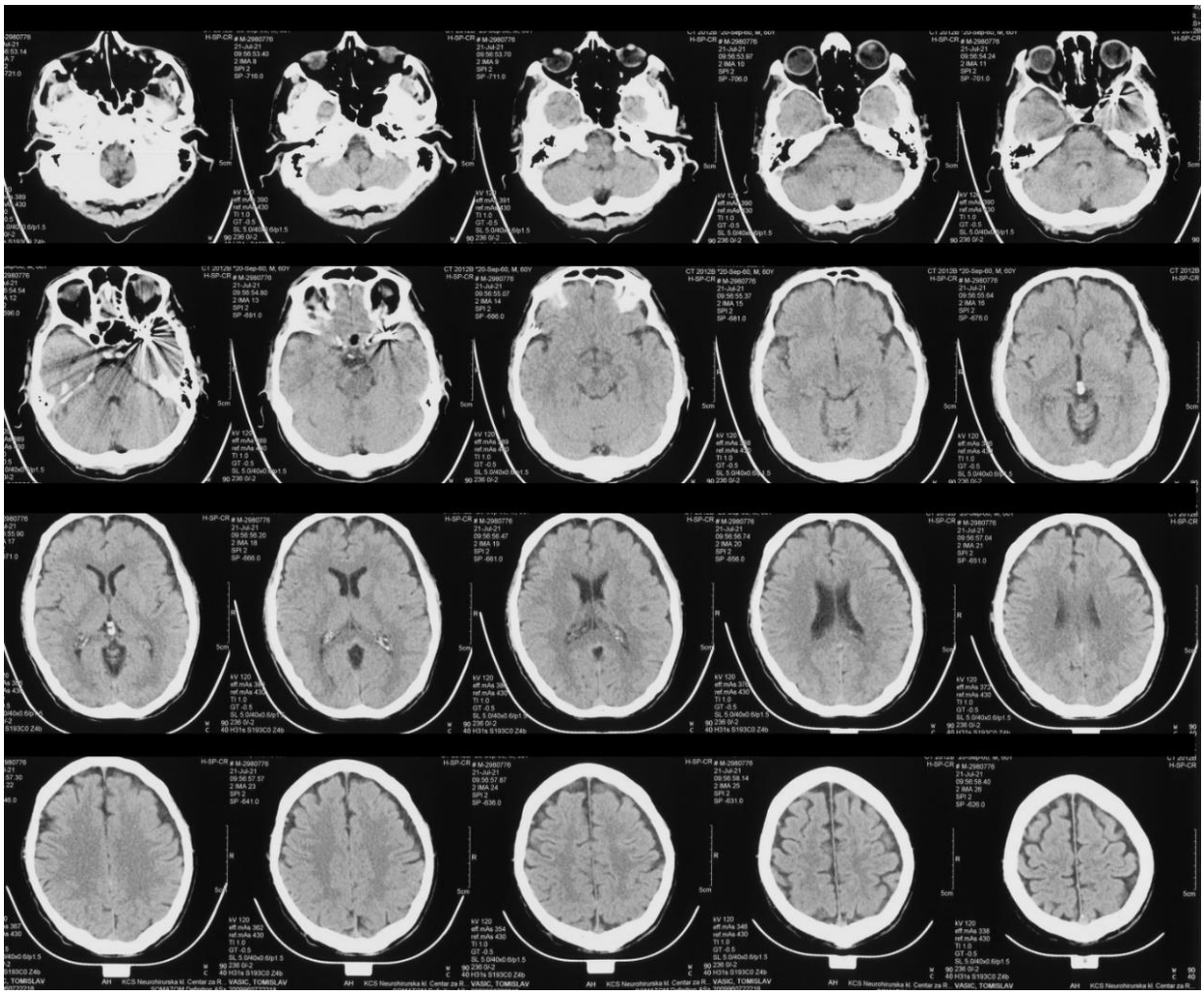

Fig. 4 Control CT scan showed no signs of rehemorrage, ischemic lesions or hidrocephalus.

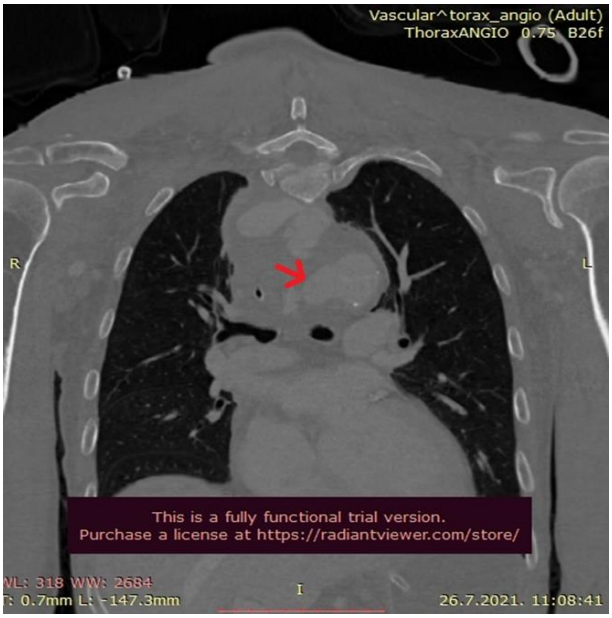

Fig. 5 There was a compressive hematoma spreading retropharingealy, dimensions 54,3x75,1 mm, ending in the posterior mediastinum i the height of tracheal bifurcation, bilateral pleural effusions with consequential subatalectasis and pericardial effusion, there were no signs of embolism.

Cervical bilateral edema was observed and neck, thoracic, and pulmoangiography CT was performed which showed a ruptured saccular pseudoaneurysm located on the caudomedial wall, sizing around 2cm, orientated medially, on the aortic arc after the branching of left subclavian artery. There was a compressive hematoma spreading retropharyngealy, dimensions 54,3x75,1 mm, ending in the posterior mediastinum i the height of tracheal bifurcation, bilateral pleural effusions with consequential subatalectasis and pericardial effusion, there were no signs of embolism. (Figure 5)

The cardiosurgical council was consulted, There were no indications for surgical treatment.

Inflammation factors continued to rise and *Staphylococcus aureus* was is isolated from hemoculture, antibiotic therapy was administered according to the antibiogram.

Two weeks after the procedure, the patient passed away.

Discussion

SAH remains a highly fatal disease; However, in recent years, improvements in outcomes have been observed. Diagnosing SAH and uncovering its etiology relies largely on a CT scan with DSA for localization of aneurysms. The early repair of aneurysms and the rise of endovascular aneurysm repair have likely contributed to these improvements in outcomes, and providers treating SAH are now able to select patient-centered treatment strategies based on the patient-leveland aneurysmal factors to optimize outcomes for individual patients. Regardless of this fact, the most feared complication after the securing of the aneurysm is delayed cerebral ischemia. The second most common is hydrocephalus.

Medical complications are a common occurrence after SAH, with some reports suggesting that all patients will experience at least one complication: fever, anemia treated with transfusion, hyperglycemia, treated hypertension, hypernatremia, pneumonia, hypotension treated with vasopressors, pulmonary edema, and hyponatremia [26].

Cardiac and respiratory dysfunctions are common after SAH, and manifestations include cardiomyopathy, electrocardiogram abnormalities, arrhythmias, pneumonia, pulmonary edema, and acute respiratory distress syndrome [27,28]. Furthermore, cardiac arrest at the time of SAH is not uncommon and reports of good functional outcomes after cardiac arrest in SAH patients are rare [29].

To our knowledge, a rupture of a thoracic aortic aneurysm after embolization of an intracranial aneurysm has been described for the first time in the literature in this paper.,. Hereditary risk factors for aneurysms were not reported in this case, and chest radiography did not indicate thoracic aortic aneurysm, although later analysis of chest CT scans clarified that it would have been difficult to detect thoracic aortic aneurysm on radiography due to possible superposition with thoracic vertebrae. However, the analysis of the position and direction of the thoracic aortic aneurysm (on the caudomedial wall of the aortic arch, directed medially, after the branch of the subclavian artery on the left side) indicates that it is not in the path during endovascular access.

Also, the initial CT angiography of the intracranial vascular vessels (Figure 2), in its phase, shows the beginning of the common carotid arteries, without the aortic arch.

Given that this is a rare and potentially fatal complication, future research could clarify whether angiographic examinations should include broader anatomical structures.

Conclusion

The results obtained in this study indicated a high frequency of joint manifestations in SSc, which was confirmed by clinical and radiographic examination. Joint involvement in SSc was underestimated in clinical trials, as it occurred more frequently than expected. Radiographic hand findings in tested SSc patients indicated the presence of arthritis, erosions, joint space narrowing, radiological demineralization, acro-osteolysis, flexion contractures, and calcinosis. Hand involvement wasan important cause of morbidity, which seriously affected the quality of life in patients with SSc.Various forms of joint and bone involvements presented in this paper, from arthralgia to arthritis and deformities, point out the need ofapplying innovative approachesand options for treating patients with systemic sclerosis.

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