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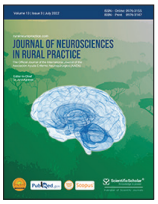

Case Report

Subarachnoid hemorrhage and intracranial hemorrhage from ruptured multiple cerebral mycotic aneurysms managed with neuroendovascular coiling

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ABSTRACT

Inflammatory blood vessel lesions, known as cerebral mycotic aneurysms (CMAs) or infectious intracranial aneurysms (IAAs), represent a small percentage (2.5–4.5%) of all IAAs. Despite their small size, CMAs are prone to rupture due to rapid, dynamic changes in the vessel wall's fragility, resulting in significant morbidity and disability. Authors have described their experience managing a case of ruptured CMAs with subarachnoid hemorrhage and intracerebral hemorrhage using endovascular coils. Although definitive evidence establishing endovascular treatment as the gold standard for ruptured CMAs is lacking, recent studies demonstrate the frequent use of embolization with coils and onyx.

Keywords: Cerebral mycotic aneurysms, Endovascular coils, Intracerebral hemorrhage, Subarachnoid hemorrhage

INTRODUCTION

The term mycotic describes the mushroom-like appearance of the aneurysm.[1] The common causative organisms implicated include *Streptococcus* species, *Staphylococcus* species, fungi, and rarely parasites and viruses.[2] Most commonly affects the anterior circulation, especially in the peripheral branches of the middle cerebral artery although, in some cases, it can affect the posterior circulation, in extracranial, it can involve the aorta, visceral, and peripheral arteries.[3] Ruptured cerebral mycotic aneurysms (CMAs) are associated with mortality of up to 80%, one of the determinant factors is the topography of the hemorrhage where lobar hemorrhage has a higher mortality rate than deep subcortical hemorrhage. In the lobar location, non-hypertensive mechanisms are predominant in intracerebral hemorrhages.[1,4] In terms of management in CMAs, various neurosurgical approaches are performed. Surgical clipping is not easy, due to the fragile wall of the aneurysms. Endovascular management is emerging as the favorite management option, with coiling and onyx, liquid embolic agents for the management of mycotic aneurysms. However, it remains unclear for the safety and efficacy.[5]

CASE REPORT

Natural history and clinical findings

A 20-year-old woman was brought to the emergency department with a chief complaint of sudden onset of severe headache that was described as the worst headache in her life, the patient experienced vomited during the event, and she also complained of weakness in her left extremities. No history of hypertension or head injury, but there was a significant reduction of body weight of around 9 kg in the past three months. Physical examination found paralysis of the left facial and hypoglossal nerve, also paralysis of the left extremity with a motor power of 1/5 and nuchal rigidity.

Laboratory and radiology results

Laboratory examination showed leukocytosis (15,300/mm^3), reactive anti-rubella Immunoglobulin G (IgG) (titer 144, 10 IU/mL), and reactive anti-CMV IgG (titer 214, 70 IU/mL). The echocardiography result was secondary moderate to severe mitral regurgitation due to mitral annular dilatation. No abnormalities were seen on the thorax X-ray, head computed tomography (CT)-scan non-contrast showed subarachnoid hemorrhage (SAH) in parietotemporal and

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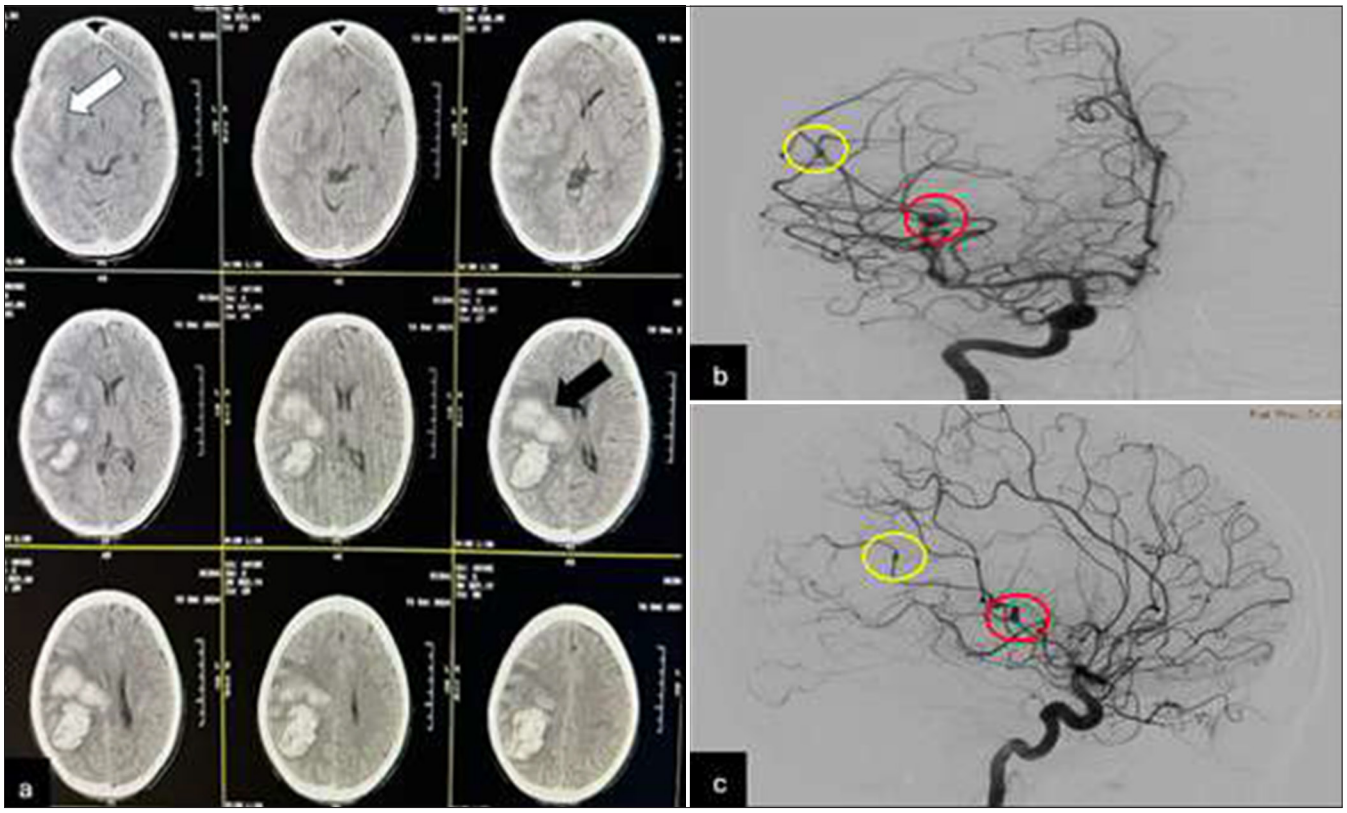

Figure 1: (a) Head non-contrast computed tomography scan showed subarachnoid hemorrhage in the right parietotemporal lobe (white arrow) and intracerebral hemorrhage in the right parietal lobe with volume estimation ±57 cc (black arrow). (b and c) Digital substraction angiography showed ruptured cerebral mycotic aneurysms on distal right MCA, postero-parietal branch (M3) (red circles) and angular branch (M4) (yellow circles).

intracranial hemorrhage in the right parietal lobe [Figure 1a]. Digital subtraction angiography (DSA) showed ruptured CMAs on the distal right middle cerebral artery (MCA), postero-parietal branch (M3) and angular branch (M4) [Figure 1b and c]. A blood sample was taken from inside the aneurysm sac during cerebral angiography, but there was no anaerobic bacterial growth in blood culture tests.

Pharmacology and intervention management

The patient was then treated with endovascular coiling, occlusion was performed on the first aneurysm on the M3 segment of the right MCA postero-parietal branch using axium prime 3D bare platinum 5 mm × 8 cm and hydrosoft-10 helical 4 mm × 8 cm. The second aneurysm on the M4 segment of the right MCA angular branch using hydrosoft-10 helical 3 mm × 6 cm [Figure 2a and b]. After the procedure, coils appear to be securely filling the aneurysm [Figure 2c and d]. During the therapy, the patient received analgesics, nimodipine, mannitol 20%, and broad-spectrum antibiotics, including 1 g of ceftriaxone twice a day and 600 mg of clindamycin every 3 times a day.

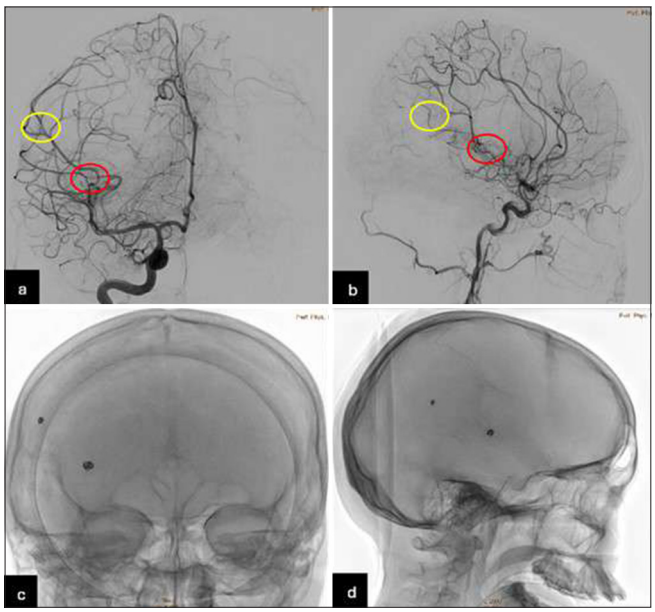

Figure 2: (a and b) Coils placed on the M3 segment postero-parietal branch (red circle) and M4 segment angular branch (yelllow circle). (c and d) Coils were attached to aneurysms.

Follow up and outcomes

The DSA evaluation showed the ruptured CMAs in the right MCA segment M2 and M3 were no longer visible [Figure 2c and d], indicating successful treatment. The headache was reduced, and there were no new neurologic deficits or complaints after the procedure. The National Institutes of Health Stroke Scale (NIHSS) during hospital admission was 11 and 8 at discharge from hospital.

DISCUSSION

The patient experienced sudden severe headaches and vomited during the event, weakness in her left extremities, and nuchal rigidity which are common clinical manifestations of SAH. Head CT scan non-contrast showed SAH in parietotemporal and intracranial hemorrhage in the right parietal lobe. CMAs are often identified after rupture of the aneurysm and become SAH with or without accompanying intraparenchymal and intraventricular hemorrhage.[5] DSA showed ruptured CMAs on distal right MCA, postero-parietal branch (M3) and angular branch (M4).

DSA is used as the gold standard in detecting CMAs, with its location more often in distal vessels and small in size. Since DSA is an invasive procedure, CT angiography and magnetic resonance angiography are more commonly used for infective endocarditis (IE) patients with neurologic disorders. However, both can only detect under 45% of all aneurysms detected by DSA. Some findings on angiography that point toward CMAs are the fusiform shape, multiplicity, distal location, and change in size on follow-up angiography.[1,3]

CMAs, also called infective intracranial aneurysms, are uncommon but described mostly as complications of IE. The vasa vasorum theory is the most commonly observed pathological process. Bacteria from septic emboli penetrate the vasa vasorum and cause significant inflammation and necrosis of the tunica media and adventitia.[6] Pressure by pulsation on the weakened arterial wall eventually leads to the formation and enlargement of aneurysms.[2,6]

Bacterial infections are said to account for 72.8% of these aneurysms. In comparison, in 12.7%, no organism was found on blood cultures (either due to empirical antibiotic treatment, inadequate investigations, or rupture after the underlying pathogen had been cleared from the blood).[2] In our case, there was no bacterial growth in blood culture tests.

Our patient satisfied the criteria of clinically definite CMAs, the score is more than 3 (age under 45 years, intraparenchymal hemorrhage on imaging, on angiographic features the aneurysms multiple and distal location) as proposed by Kannoth *et al.* have high specificity and sensitivity (96%) for the diagnosis of CMAs [Table 1].[7]

For the unruptured aneurysms, antibiotic therapy continued if the aneurysms disappeared or shrunk. Positive blood or cerebrospinal fluid cultures and antibiotic administration are tailored to the type of microorganism. For patients with negative cultures, broad-spectrum antibiotics should be given for 4–6 weeks in most cases. However, surgical and endovascular therapy should be considered in aneurysms that are progressively enlarging despite adequate antibiotics.[1,3] Coil embolization has been shown to be an effective and durable management for ruptured aneurysms despite risks such as vasospasm, rebleeding, and recanalization of the aneurysms.[5]

Table 1: Proposed diagnostic criteria for CMAs.

Presence/ recent history of predisposing infection	1. Infective endocarditis
	2. Meningitis
	3. Orbital cellulitis
	4. Cavernous sinus thrombophlebitis
Angiographic features	1. Multiplicity
	2. Distal location
	3. Fusiform shape
	4. Change is the shape and size of a new aneurysm on follow-up imaging
Other contributory features	1. Age <45 years
	2. Fever/history of fever >7 days
	3. Recent lumbar puncture
	4. Intraparenchymal hemorrhage in brain imaging
Diagnosis of CMAs based on the above criteria:	
Definitive	Mandatory criteria plus 3 or more supportive criteria satisfied
Probable	Mandatory criteria plus 2 or more supportive criteria satisfied
Possible	Mandatory criteria plus one supportive criterion satisfied

CMAs: Cerebral mycotic aneurysms

Ruptured CMAs are associated with mortality up to 80%, the adequate therapy, immunology status, bacteria virulence, pathogen within the vessel walls, aneurysms location, and topography of the hemorrhage where lobar hemorrhage has a higher mortality rate than deep subcortical hemorrhage and intraventricular extension of the cerebral hematoma as the determinant of poor outcome.[1,4,8]

Limitations

Due to loss of contact with the patient, we were unable to follow up and perform control angiography on the patient.

Opinion on the future lines

MCAs are very rare, a larger sample size is needed to determine which treatment modality is superior in terms of safety and outcomes.

CONCLUSION

CMA is a rare type of aneurysm, most often caused by bacterial microorganism infection, commonly diagnosed after aneurysms ruptured, endovascular is an effective therapy in cases of ruptured CMAs; in these recent years, the employed modality that has been frequently used is coil embolization.

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