

## Case Report

Open Access, Volume 4

# Delayed cerebral ischemia after coil embolization of an unruptured giant intracranial aneurysm: A case report

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Received: Apr 04, 2023

Accepted: Apr 24, 2023

Published: May 01, 2023

Archived: www.jcimcr.org

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DOI: www.doi.org/10.52768/2766-7820/2394

**Keywords:** Unruptured aneurysm; Coil embolization; Delayed cerebral ischemia; Complication.

### Introduction

Delayed Cerebral Ischemia (DCI) is a frequent and serious complication after Subarachnoid Hemorrhage (SAH), the leading cause of disability and fatality in survivors of SAH, the most important complications of stent-assisted coil embolization of cerebral aneurysms, and one of the complications after clipping surgery of aneurysm. However it has been rarely reported after coil embolization of unruptured cerebral aneurysms without stent assistance. Here we report a 72-year-old patient presenting with DCI, which occurred 6 days after coil embolization for the treatment of a giant unruptured communicating segment aneurysm of internal carotid without stent assistance. The symptom of DCI showed rapid spontaneous remission with the National Institute of Health Scale Score (NIHSS) changing from 27 to 9. He recovered gradually to a score of 0 in NIHSS and the Magnetic Resonance Imaging (MRI) of the brain revealed watershed infarction after intravenous thrombolysis with rt-PA. The primary thrombotic embolism may be generated in the neighborhood of the giant aneurysm, and its pathophysiology is perhaps as follows: the incomplete occlusion influenced the local hemodynamic effect that caused turbulence and the presence of thrombus. The thrombolysis was made partly because of its

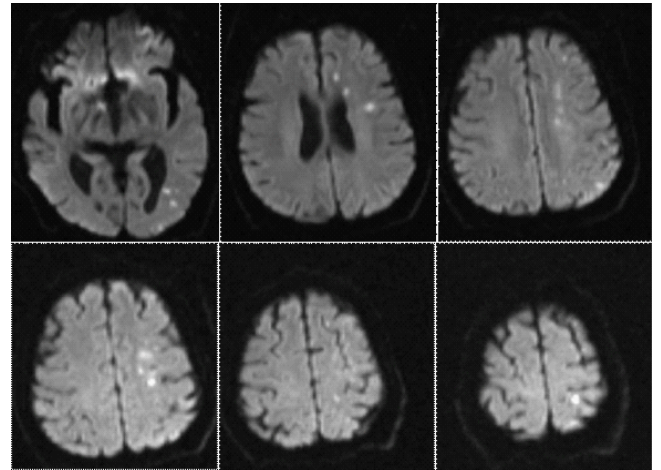
instability and partly because of the effect of the drug. Both the compressive stenosis of the initial segment of bilateral Anterior Cerebral Arteries (ACA) and the hypovolemia followed diarrhea accelerated the fragment of thrombus to flow to the watershed.

### Case report

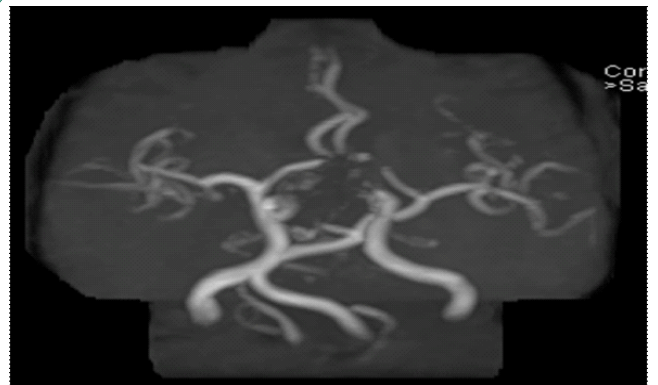
A 72-year-old man was admitted due to his being found in an unconscious state for 1 hour. He was normal 3 hours ago before his admission. He was lying on the sofa, showing no reaction to others voice. Neither seizures nor gaze of the double eyeballs was found. He vomited once during his being transported to the hospital and the color of the vomitus was brown. He had a history of hypertension for many years and was administered with 2.5 mg levoamlodipine once daily recently. He has a giant unruptured communicating segment aneurysm of internal carotid (Figure 1) and accepted endovascular coil embolization without stent assistance 6 days ago. The grade was III according to the Raymond-Roy classification (class I, complete obliteration; class, residual neck, and class III, residual aneurysm) (Figure 2) [1]. He suffered for acute diarrhea in the past 2 days. Physical examination on admission was as follows: T 36.7, P 64 bpm, R 16 bpm, BP 130/70 mmHg. He was in a sopor state. His pupils

**Citation:** Wang yan, Delayed cerebral ischemia after coil embolization of an unruptured giant intracranial aneurysm: A case report. J Clin Images Med Case Rep. 2023; 4(5): 2394.

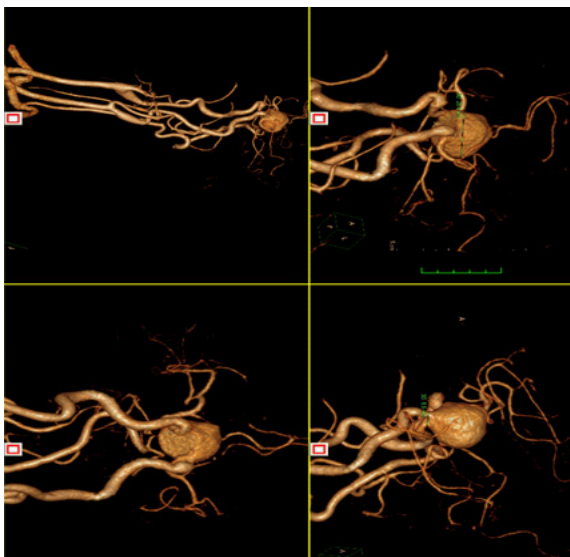
was in the normal size and reflexed sensitively to the light. He showed no neck resistance, negative Kernig sign, and negative Bruzinski sign. His left limbs showed involuntary movement while the right ones didn't move at all. The National Institute of Health scale score (NIHSS) was 27. Color Doppler ultrasound of the heart and the cervical arteries was taken 10 days ago which showed a normal heart and there was no arterial stenosis. His electrocardiogram was normal. The preliminary diagnosis was made as follows: hemorrhagic stroke? ishchemic stroke? Epilepsy mimicking stroke ? CT scan of the brain showed no obvious abnormality. After the CT examination he recovered a lot with the NIHS slowed down to 9 gradually. After the occult blood testing of vomitus gave a negative result we gave the patient rt-PA intravenous thrombolysis (0.9 mg/kg) which result in an perfect clinal manifestation and the NIHSS became 0 the next day. Subsequently, the Magnetic Resonance Imaging (MRI) of the brain was performed and it revealed that there were acute cerebral infarction lesions in the watershed area (Figure 3). Magnetic Resonance Angiopathy (MRA) showed compressive stenosis in the initial segment of the bilateral Anterior Cerebral Arteries (ACA) (Figure 4). The patient was then treated with 100mg aspirin once daily, and was discharged 1 week later.



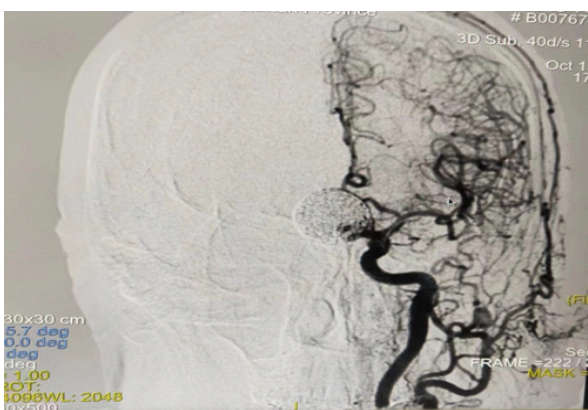
**Figure 3:** DWI shows multiple high signal in the frontal, parietal and the occipital lobe. All of them were scattered in the watershed area between the left ACA and the left middle cerebral artery.



**Figure 4:** Magnetic Resonance Angiopathy (MRA) of the brain shows compressive stenosis in the initial segment of the bilateral Anterior Cerebral Arteries (ACA).



**Figure 1:** CTA of the brain : A giant unruptured aneurysm in the communicating segment of internal carotid. It's about 24 mm x24 mm x27 mm (the neck was abo).



**Figure 2 DSA:** The giant aneurysm was incompletely occluded and it's in class III according to the Raymond-Roy classification.

### Discussion

Clipping surgery and endovascular coil embolization are two different methods for the treatment of cerebral aneurysms. It is still controversial about which is the best choice for treatment of unruptured cerebral aneurysms. Many researches show that coil embolization is of great advantage due to its low fatality and disability rate [2,3]. It was managed by coil embolization in this case. As for unruptured cerebral aneurysms, perforation occurs in 0.5-2.4% of the patients treated through endovascular method [4], both intra procedural rupture and delayed rupture are included [5,6]. Besides, remote intraparenchymal hemorrhage can also happens after end ovascular treatment [6]. Due to the symptoms of unconsciousness and vomitting which may indicate high intracranial pressure, delayed ruputure of the aneurysm, remote intraparenchymal hemorrhage and intraprocedural rupture which led to the advance of clinical features were first suspected. However CT imaging of the brain denied all of them. Then Acute Ischemic Stroke (AIS) was considered. But regrettably, we didn't apply MRI immediately because there was risk of apnea caused by vomitting. In addition epilepsy which mimicked stroke couldn't be excluded based on the mass effect of the giant aneurysm and unclear onset of clincial manifestations, while the improving consciousness and the neurological function defect which may indicate TODD paralysis could be the evidence of epilepsy. It was difficult to make an exact di-

agnosis at that time. In consequence of the idea that time is brain, considering the potential disability of AIS, intravenous thrombolysis with rt-PA (0.9 mg/kg) was exerted to him. Further improvement after thrombolysis supports the effectiveness of thrombolytic therapy for acute ischemic stroke on one hand, but does not rule out the natural outcome of Todd paralysis after epileptic seizures on the other hand.

MRI was examined later. The compressive stenosis of the initial segment of the bilateral ACA showed by MRA provides the anatomical basis for cerebral infarction in the watershed area theoretically. And the diarrhea before the neurological deficit may cause hypovolemia and cerebral hypoperfusion which can be the pathophysiological basis of watershed cerebral infarction. However, AIS in the watershed does not explain the disturbance of consciousness. Therefore, we inferred that the cerebral infarction lesions in the watershed area are secondary changes after thrombus. The primary lesion should be locating in the upstream artery. Combined with the coil embolization 6 days ago, we think it may be in the neighborhood of the giant aneurysm.

So what is the etiology of AIS in this case? According to the modified TOAST system [7], the most common etiology is atherosclerotic, followed by cardioembolism. However, neither the result of color Doppler ultrasound nor the imaging of DSA during the interventional procedure supports them. Therefore, we think its essential etiology is DCI. DCI was first described by Gull in 1859, based on the symptoms of neurological deficit in a patient with Subarachnoid Hemorrhage (SAH). Different literatures illustrate various definitions of DCI, the most appropriate one is suggested to be cerebral infarction by Mervyn et al. in 2011 [8]. It is a frequent and serious complication after SAH, the leading cause of disability and fatality in survivors of SAH [9,10], the most important complications of stent-assisted coil embolization of cerebral aneurysms [11], and one of the complications after clipping surgery of aneurysm [12,13]. However, it's a relatively rare complication after coil embolization without stent assistance of unruptured cerebral aneurysms. ARETA, a prospective multicenter study in endovascular treatment for unruptured aneurysms, shows that all the DCI events happened in the weeks following the procedure [14]. In this case it occurred 6 days after treatment of endovascular coil embolization, which coincides with the aforementioned study. Since no stent was used to assist the coil embolization, what's the pathophysiology of the DCI in this case? The aneurysm can be recanalized, grow, and rupture if it is incompletely occluded. Thus it's a useful way to assess the risk of recurrence and reperforation for treated aneurysms by assessing the occlusion degree and their parent arteries [15].

However, there is still not enough attention on the DCI after incomplete occlusion nowadays. It was reported the incidence rate of DCI was 9.4% in patients with post-procedure aneurysm remnant while it's only 1.6% in those with adequate occlusion [14]. Raymond-Roy classification is widely used to assess the degree of aneurysm occlusion. We think that incomplete occlusion leads to the formation of a dead space, which in turn causes blood flow disturbance, stagnation, and eventually leads to the DCI event although no stent had been used during the interventional procedure. So the grade of Raymond-Roy classification may be helpful to estimate the possibility of DCI event. Advances in interventional embolization techniques and materials may be expected to improve the degree of embolization and reduce DCI in such patients.

DCI is a common complication in patients with SAH and aneurysm stent-assisted interventional embolization. Prevention is more important than treatment in some scholars' opinions [16]. Dual antiplatelet therapy has become the standard for prevention of these diseases, but it is still not standardized [17]. In the circumstances such as this case when the aneurysm was unruptured, and the stent-assisted technique was not employed during the endovascular treatment, it seems to be inappropriate for routine antiplatelet therapy unless there are other diseases that require antiplatelet therapy. It needs further research to clarify whether there would be potentially fatal complications such as thrombocytopenia and bleedings if dual anti platelet therapy is applied.

## Conclusion

In conclusion, the occurrence of DCI in this case may be related to clinical, technical and anatomical reasons. It seems that we still have a long way to go in the identification, prevention and treatment of DCI after coil embolization of unruptured aneurysms especially when no stent is used.

## References

1. Roy D, Milot G, Raymond J. Endovascular treatment of unruptured aneurysms. *Stroke*. 2001; 32 : 1998-2004.
2. Pierot L, Barbe C, Nguyen HA, et al. Intraoperative Complications of Endovascular Treatment of Intracranial Aneurysms with Coiling or Balloon-assisted Coiling in a Prospective Multicenter Cohort of 1088 Participants: Analysis of Recanalization After Endovascular Treatment Of Intracranial Aneurysm (ARETA) Study. *Radiology*. 2020, 295: 381-389.
3. Pierot L, Barbe C, Herbreteau D, et al. Rebleeding and bleeding in the year following intracranial aneurysm coiling: analysis of a large prospective multicenter cohort of 1140 patients-Analysis of Recanalization after Endovascular Treatment of Intracranial Aneurysm (ARETA) Study. *J NEUROINTERV SURG*. 2020; 12 : 1219-1225.
4. Fiehler J, Byrne JV. Factors affecting outcome after endovascular treatment of intracranial aneurysms. *CURR OPIN NEUROL*. 2009; 22: 103-108.
5. Kawabata S, Imamura H, Adachi H, et al. Risk factors for and outcomes of intraprocedural rupture during endovascular treatment of unruptured intracranial aneurysms. *J NEUROINTERV SURG*. 2018; 10: 362-366.
6. Jee TK, Yeon JY, Kim KH, et al. Treatment Outcomes After Single-Device Flow Diversion for Large or Giant Aneurysms. *WORLD NEUROSURG*. 2021; 153: e36-345.
7. Han SW, Kim SH, Lee JY, Chu CK, Yang JH, Shin HY, et al. A new subtype classification of ischemic stroke based on treatment and etiology. *Eur Neurol*. 2007; 57: 96-102.
8. Vergouwen MD. Vasospasm versus delayed cerebral ischemia as an outcome event in clinical trials and observational studies. *NEUROCRIT CARE*. 2011; 15: 308-311.
9. Caricato A, Silva S. Delayed cerebral ischemia: is it time to change our practice? *MINERVA ANESTESIOLOGIA*. 2020; 86: 1266-1268.
10. Cremers CH, Cos PC, van der Schaaf IC, et al. CT perfusion during delayed cerebral ischemia after subarachnoid hemorrhage: distinction between reversible ischemia and ischemia progressing to infarction. *NEURORADIOLOGY*, 2015; 57: 897- 902.
11. Song J, Yeon JY, Kim JS, et al. Delayed thromboembolic events more than 30 days after self expandable intracranial stent-assisted embolization of unruptured intracranial aneurysms. *CLIN*

12. Campe C, Neumann J, Sandalcioglu IE, et al. Vasospasm and delayed cerebral ischemia after uneventful clipping of an entrapped intracranial aneurysm - A case report. *BMC Neurol.* 2019; 19: 226.
13. Goertz L, Krischek B, Reiner M, et al. Penumbra salvage by delayed clip reposition 19 hours after cerebral aneurysm clipping - induced ischemia results in neurologic restitution - correlation with indocyanine green videoangiography and FLOW 800 measurements. *WORLD NEUROSURG.* 2020; 138: 61-67.
14. Pierot L, Barbe C, Herbreteau D, et al. Delayed thromboembolic events after coiling of unruptured intracranial aneurysms in a prospective cohort of 335 patients. *J NEUROINTERV SURG.* 2021; 13: 534-540.
15. Etminan N, de Sousa DA, Tiseo C, et al. European Stroke Organisation (ESO) guidelines on management of unruptured intracranial aneurysms. *EUR STROKE J.* 2022; 7: V.
16. Koenig HM, Chen J, Sleg EP. Delayed cerebral ischemia: Is prevention better than treatment? *J NEUROSURG ANESTH.* 2021; 33: 191-192.
17. Cheddar El Aouni M, Magro E, Abdelrady M, et al. Safety and efficacy of cangrelor among three antiplatelet regimens during stent-assisted endovascular treatment of unruptured intracranial aneurysm: A single-center retrospective study. *Front Neurol.* 2022; 13: 727026.