



Case Report

# Dual antiplatelet therapy in a patient with simultaneous aneurysmal subarachnoid hemorrhage and myocardial infarction

Abolghasem Mortazavi<sup>1</sup>, Sina Jelodar<sup>1</sup>, Keyvan Edraki<sup>1</sup>, Sima Narimani<sup>2</sup>, Mohammad Ghorbani<sup>3</sup>, Koroush Karimi-Yarandi<sup>1</sup>, Sina Asaadi<sup>3</sup>

<sup>1</sup>Department of Neurosurgery, Sina Hospital, <sup>2</sup>Tehran Heart Center, Department of Interventional Cardiology, Tehran University of Medical Sciences,

<sup>3</sup>Department of Neurosurgery, Division of Vascular and Endovascular Neurosurgery, Firoozgar Hospital, Iran University of Medical Sciences, Tehran, Iran.

E-mail: Abolghasem Mortazavi - [sgmortazavi@sina.tums.ac.ir](mailto:sgmortazavi@sina.tums.ac.ir); Sina Jelodar - [sinajelodar@gmail.com](mailto:sinajelodar@gmail.com); Keyvan Edraki - [kedraki@hotmail.com](mailto:kedraki@hotmail.com);

Sima Narimani - [sima\\_narimani100@yahoo.com](mailto:sima_narimani100@yahoo.com); Mohammad Ghorbani - [ghorbani.m@iums.ac.ir](mailto:ghorbani.m@iums.ac.ir); Koroush Karimi-Yarandi - [kouroshkarimi\\_ir@yahoo.com](mailto:kouroshkarimi_ir@yahoo.com);

\*Sina Asaadi - [s.asaadi90@gmail.com](mailto:s.asaadi90@gmail.com)



**\*Corresponding author:**

Sina Asaadi,  
Department of Neurosurgery,  
Division of Vascular and  
Endovascular Neurosurgery,  
Firoozgar Hospital, Iran  
University of Medical Sciences,  
Tehran, Iran.

[s.asaadi90@gmail.com](mailto:s.asaadi90@gmail.com)

Received : 31 August 19

Accepted : 05 February 20

Published : 21 March 20

DOI

10.25259/SNI\_472\_2019

Quick Response Code:



## ABSTRACT

**Background:** Electrocardiography (ECG) changes after subarachnoid hemorrhage (SAH) are well described. However, concurrent myocardial infarction (MI) and SAH are rarely reported, and its management remains a dilemma. We report a patient with traumatic SAH concurrent with acute MI that managed successfully by endovascular intervention and dual antiplatelet therapy.

**Case Description:** A 47-year-old man was admitted to the emergency department with a complaint of severe headache. Diffuse SAH, with a Hunt and Hess score of 5, was noticed. ECG showed ST elevation in anterior leads, and cardiac troponin became positive. On brain computed tomography angiogram, a 6 mm anterior communicating artery aneurysm was seen. Considering the possibility of MI and SAH simultaneously, endovascular obliteration of the aneurysm was done, and then, the patient received dual antiplatelet medications until coronary angiography was done. Coronary angiography revealed normal epicardial coronary arteries. The patient was discharged with a Glasgow Coma Scale score of 15 and was visited 2 months after discharge without any new episodes of intracranial hemorrhage with a modified Rankin scale score of 2.

**Conclusion:** Cerebral aneurysm coiling could be considered as the first choice of treatment in the case of acute MI with hemodynamic stability, before carrying out cardiac endovascular intervention or antiplatelet medication to reduce the risk of rebleeding from a brain aneurysm.

**Keywords:** Antiplatelet therapy in subarachnoid hemorrhage, Myocardial infarction, Subarachnoid hemorrhage

## INTRODUCTION

Electrocardiography (ECG) and echocardiographic changes such as nonspecific ST deviations, T-wave inversion, prolonged QT interval, and transient segmental hypokinesia of the left ventricular wall frequently report in accompany with subarachnoid hemorrhage (SAH). However, the association between SAH and acute myocardial infarction (MI) is rarely reported.<sup>[1]</sup>

We report a patient with traumatic SAH concurrent with acute MI that managed successfully by endovascular intervention and dual antiplatelet therapy.

This is an open-access article distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

©2020 Published by Scientific Scholar on behalf of Surgical Neurology International

## CASE REPORT

A 47-year-old cocaine-addicted man with a history of severe headache following falling was referred to the emergency department with a complaint of decreased level of consciousness and intubated with a Glasgow Coma Scale (GCS) score of 4. No lateralizing signs were observed, and the pupils were symmetrical and midsized. Brain computed tomography (CT) scan showed diffuse SAH with a Hunt and Hess score of 5 and the World Federation of Neurosurgical Societies (WFNS)<sup>1</sup> score of 5. On further workup, the patient's ECG showed ST elevation in V1-4 and ST depression in inferior leads. Cardiac troponin also raised significantly along with the evidence of wall motion abnormality on echocardiography. According to the patient's neurologic condition, the treatment of possible MI was started with the dual oral antiplatelet drug.

Brain CT angiogram that performed after admission of the patient to the neurosurgical intensive unit, revealed a 6 mm AComA aneurysm. Single antiplatelet therapy was started on the 1<sup>st</sup> day after the ictus with no new episode of rebleeding from the AComA aneurysm. Given the evidence of SAH and aneurysm on CT, the administration of dual antiplatelet drugs and anticoagulants imposed a high risk of rebleeding and deteriorating neurologic condition on our patient. Therefore, considering the stability of our patient's hemodynamic, we decided to close the aneurysm first. Cerebral angiography was performed to obtain anatomical data required for endovascular treatment [Figure 1a and b]. The endovascular intervention was done, and complete occlusion was achieved [Figure 1c].

After successful endovascular closure of brain aneurysm, dual antiplatelet therapy was started with no evidence of rebleeding on follow-up brain CT. The patient underwent coronary angiography that revealed normal epicardial coronary arteries that result in discontinuing antiplatelet therapy. Five days later, the patient was discharged with a GCS score of 15 and was visited 2 months after discharge

without any new episodes of intracranial hemorrhage and modified Rankin scale score of 2.

## DISCUSSION

SAH can be associated with ECG changes.<sup>[6]</sup> The most common ECG changes in SAH include nonspecific ST deviation, T-wave inversion, and prolonged QT interval caused by cardiocerebral interaction.<sup>[2]</sup> ECG changes followed by SAH can be mistaken for MI. However, cardiac changes in SAH are not limited to ECG changes and cardiac motility, and even enzymes could occur. Cardiac dysfunction following aneurysmal SAH includes dysrhythmias, infarction, ischemia, and stunned myocardium with ventricular dysfunction.<sup>[4,10]</sup>

It is reported that elevated cardiac markers may be seen in patients with SAH.<sup>[8]</sup> The etiologies for these changes are not well known. Takotsubo cardiomyopathy, acute pericarditis, and MI are the proposed mechanisms for these changes.<sup>[3]</sup> Diagnosis of the etiology of ECG changes is of great importance because the mainstay of treatment in MI is fibrinolytic and/or antiplatelet medications, which are both contraindicated in acute SAH. In our case, the patient was presented with loss of consciousness and admitted due to SAH. However, evidence of ST elevation was observed on ECG after admission. We propose two possibilities for ECG changes in our patient; the first was ECG changes secondary to the cerebral hemorrhagic event, and the second was coronary artery spasm secondary to cocaine abuse in our patient.

A review of the literature showed only three cases of concomitant MI and SAH in patients with a decreased level of consciousness.<sup>[7,9]</sup> In Tan *et al.* report, the patient admitted with cardiac manifestation and then developed SAH. However, they believe that SAH was the initial event that causes secondary coronary plaque rupture. Unlike our management strategy, they chose dual antiplatelet therapy in the first step due to the high risk of coronary stent thrombosis.<sup>[7]</sup> van der Velden *et al.* discussed a treatment protocol for patients with concomitant MI and SAH. Platelets and their coagulation factors play a significant role in the development of clinical vasospasm.<sup>[9]</sup> They showed

1 WFNS



**Figure 1:** Angiography of anterior communicating artery aneurysm (a), roadmap image of AComA aneurysm on angiography (b) and postcoiling image of aneurysm (c).

that antiplatelet therapy could be started as soon as 72 h after SAH or after coiling of the aneurysm.<sup>[9]</sup>

Nagahama *et al.* showed that treatment with an antiplatelet agent led to a decrease of vasospasm and poor outcome. Their results strongly suggest that antiplatelet reduces the risk of clinical vasospasm in patients with SAH without an increased risk of hemorrhagic complications.<sup>[5]</sup>

According to our experience, cerebral aneurysm coiling could be considered as the first choice of treatment in the case of confronting the possibility of MI and hemodynamic stability, before carrying out cardiac endovascular intervention or antiplatelet medication to reduce the risk of rebleeding from a brain aneurysm, and it should be performed in the first 72 h after the ictus. We followed this treatment strategy successfully, and our patient did not have any evidence of vasospasm. However, further studies with more patients are needed to identify actual outcomes and potential risks.

## CONCLUSION

Discontinuation of life-saving anti-thrombotic agents or life-threatening worsening of neurological conditions without discontinuing these medications is a challenge in the management of myocardial infarction coincidence with subarachnoid hemorrhage. Our experience provides a suggestion that proceeding with anti-thrombotic agents after invasive management of brain pathology might show promising results.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

## Financial support and sponsorship

Nil.

## Conflicts of interest

There are no conflicts of interest.

## REFERENCES

1. Bárcena JP, Rota JI, Ramírez JH, Sala MF, Juve JI. Subarachnoid hemorrhage and acute myocardial infarction. *Intensive Care Med* 2000;26:1160-1.
2. Davies KR, Gelb AW, Manninen PH, Boughner DR, Bisnaire D. Cardiac function in aneurysmal subarachnoid haemorrhage: A study of electrocardiographic and echocardiographic abnormalities. *Br J Anaesth* 1991;67:58-63.
3. Deshpande A, Birnbaum Y. ST-segment elevation: Distinguishing ST elevation myocardial infarction from ST elevation secondary to nonischemic etiologies. *World J Cardiol* 2014;6:1067-79.
4. Horowitz MB, Willet D, Keffer J. The use of cardiac troponin-I (cTnI) to determine the incidence of myocardial ischemia and injury in patients with aneurysmal and presumed aneurysmal subarachnoid hemorrhage. *Acta Neurochir (Wien)* 1998;140:87-93.
5. Nagahama Y, Allan L, Nakagawa D, Zanaty M, Starke RM, Chalouhi N, *et al.* Dual antiplatelet therapy in aneurysmal subarachnoid hemorrhage: Association with reduced risk of clinical vasospasm and delayed cerebral ischemia. *J Neurosurg* 2018;129:702-10.
6. Sommargren CE, Zaroff JG, Banki N, Drew BJ. Electrocardiographic repolarization abnormalities in subarachnoid hemorrhage. *J Electrocardiol* 2002;35 Suppl:257-62.
7. Tan JH, Uddin A, Fernandez JP. Concurrent subarachnoid haemorrhage and ST elevation myocardial infarction. *JRSM Open* 2017;8:2054270416685207.
8. Tung P, Kopelnik A, Banki N, Ong K, Ko N, Lawton MT, *et al.* Predictors of neurocardiogenic injury after subarachnoid hemorrhage. *Stroke* 2004;35:548-51.
9. van der Velden LB, Otterspoor LC, Schultze Kool LJ, Biessels GJ, Verheugt FW. Acute myocardial infarction complicating subarachnoid haemorrhage. *Neth Heart J* 2009;17:284-7.
10. Zouaoui A, Sahel M, Marro B, Clemenceau S, Dargent N, Bitar A, *et al.* Three-dimensional computed tomographic angiography in detection of cerebral aneurysms in acute subarachnoid hemorrhage. *Neurosurgery* 1997;41:125-30.

**How to cite this article:** Mortazavi A, Jelodar S, Edraki K, Narimani S, Ghorbani M, Karimi-Yarandi K, Asaadi S. Dual antiplatelet therapy in a patient with simultaneous aneurysmal subarachnoid hemorrhage and myocardial infarction. *Surg Neurol Int* 2020;11:49.