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Case Report

Risk of fatal sinus arrest induced by low-grade subarachnoid hemorrhage: A case of a young patient with obstructive sleep apnea

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ABSTRACT

Background: Sleep apnea syndrome (SAS) and subarachnoid hemorrhage (SAH) are both considered possible causes of secondary arrhythmias. However, there are limited reports on the increased risk of bradyarrhythmia for arrhythmia-free SAS patients with SAH.

Case Description: A 31-year-old woman with SAS developed low-grade SAH and underwent coil embolization on postbleed day 1. Following a coiling procedure, she experienced worsening episodes of sinus arrest lasting up to 12 s and required a temporary pacemaker. Frequent episodes of sinus arrest were detected for the next 4 days. Thereafter, all types of arrhythmias gradually decreased, and she eventually recovered to be arrhythmia free.

Conclusion: Acceleration of sympathetic nervous activity caused by acute SAH may predispose patients to bradyarrhythmia with SAS and elicit asystole. The coexistence of SAS and SAH should be recognized as a cause of life-threatening sinus arrest, even if the severity of SAH is low grade.

Keywords: Endovascular surgery, Sinus arrest, Sleep apnea syndrome, Subarachnoid hemorrhage

INTRODUCTION

Potential cardiac abnormalities with obstructive sleep apnea syndrome (SAS), including arrhythmias and ischemic heart disease, have been previously reported. An increased risk of complicated cardiac or lung disease in acute subarachnoid hemorrhage (SAH) has also been described. The greater the severity of SAH and SAS, the greater the number and severity of associated complications.[1,4,8] The coexistence of SAH and SAS is expected to further increase the risk of arrhythmia. However, to the best of our knowledge, there are no previous reports describing the complications in such cases. Herein, we report a case of SAH in a patient with SAS who experienced recurrent episodes of bradyarrhythmia, which included periods of sinus arrest requiring temporary pacemaker insertion, after the onset of low-grade SAH.

CASE REPORT

The patient was an obese 31-year-old woman (body mass index: 43.7 kg/m^2). She was examined at our hospital laboratory 1-year before severe SAS, with 132 episodes of apnea/hour and significant hypoxia

in polysomnography. A 24-h electrocardiogram (ECG) detected an inverted T wave, but no arrhythmias. She denied a history of hypertension, diabetes mellitus, or dyslipidemia. She had no habit of smoking or a family history of SAH or intracranial aneurysm. She presented with a worsening headache, but was alert and oriented, with no neurological deficit (World Federation of Neurosurgical Societies [WFNS] Grade 1). Head computed tomography revealed SAH, and computed tomography arteriogram showed a carotid-posterior communicating artery aneurysm with a bleb [Figure 1]. Continuous ECG monitoring on admission showed sinus tachycardia (mean: 120 beats/min) with frequent ventricular premature complexes [Figure 2]. However, a transient bradycardia (mean: 30 beats/min) was occasionally observed, especially during sleep apnea.

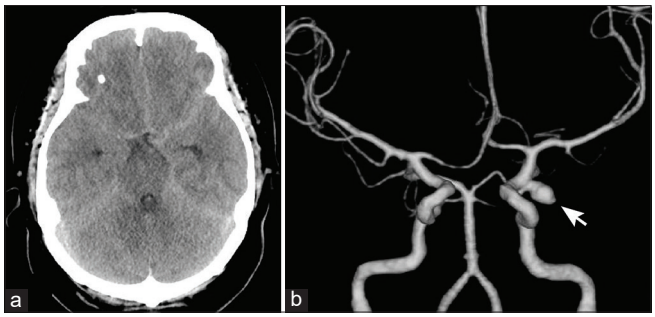

Figure 1: Preoperative axial computed tomography (CT) (a) demonstrating subarachnoid haemorrhage. Three-dimensional reconstructed CT angiogram (b) showing a large internal carotid-posterior communicating artery aneurysm with a bleb (arrow).

The patient agreed to endovascular treatment to prevent rebleeding, and coil embolization for the ruptured aneurysm was performed under general anesthesia on postbleed day (PBD 1) [Figure 3]. Worsening bradycardia and repetitive transient asystole lasting 3–12 s with hypotension occurred during postoperative care in the intensive care unit [Figure 4]. To treat the recurrent severe bradycardia, our cardiologists performed temporary pacemaker insertion (back-up VVI pacing at 40 beats/min) the day after surgery. The patient, who was postoperatively receiving mechanical ventilation under sedation to control her unstable circulatory status, was able to be weaned from ventilation and extubated on the same day. Temporary pacing was induced once per hour to prevent severe asystole until 2 days after insertion, regardless of the presence of sleep apnea. At PBD 5, the back-up pacing was decreased to once per 4 h, and bradycardia only occurred during sleep apnea. The following day, there were only three requirements for back-up pacing, and the bradycardia and ventricular premature complexes were rarely detected, even during sleep apnea. Thereafter, temporary pacing was not required. All types of arrhythmia decreased, and a final 3 s episode of sinus arrest was observed 7 days later. The 24-h ECG monitoring was continued until 21 days after admission. She did not experience any other complications such as cerebral vasospasm or hydrocephalus caused by SAH and was discharged home with no neurological deficit at 30 days after admission.

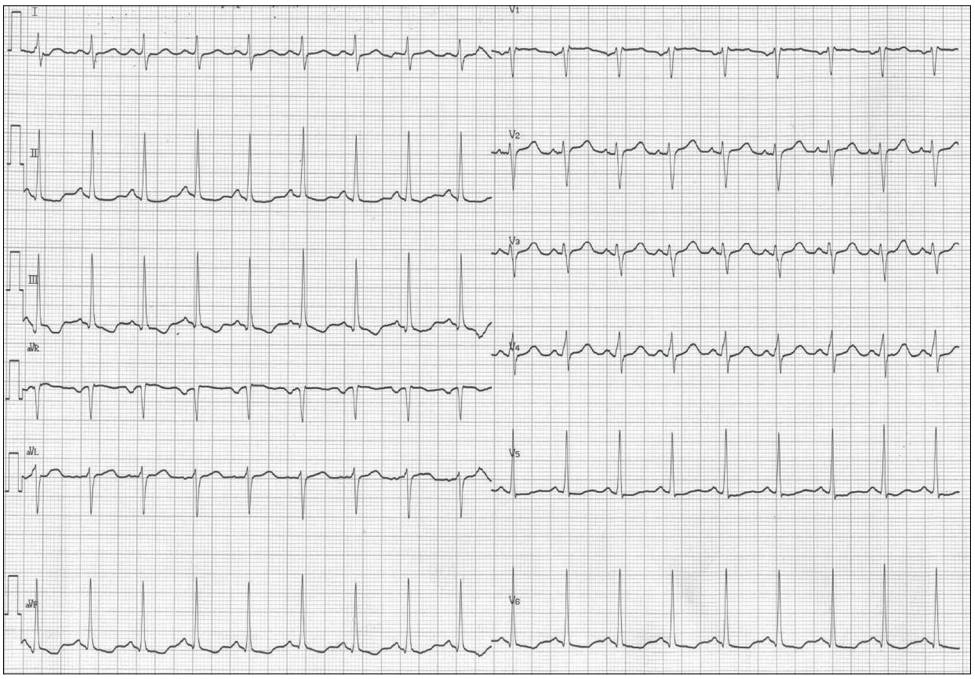

Figure 2: Electrocardiogram on admission.

DISCUSSION

Cardiac abnormalities are very common in patients with acute SAH and include electrocardiographic abnormalities,[6] cardiomyopathy,[2] and arrhythmias.[5] Several studies have proposed that cardiac abnormalities result from excessive catecholamine release from activated sympathetic nerves onto the myocardium.[3,9] Increased sympathetic nervous system activity is associated with a poor neurologic grade on admission[8] and occurs immediately at the onset of SAH. Thus, cardiac abnormalities are often detected during the first few days after SAH. Frontera *et al.* prospectively evaluated the cardiac arrhythmias after SAH in 580 spontaneous patients.[5] In that study, arrhythmias occurred in 4.3% of SAH patients. Atrial fibrillation or atrial flutter was the most common arrhythmias (76%) followed by junctional rhythm, while asystole was less frequent (12%).

Several studies have reported the risk factors for complicated arrhythmias in SAH, which include age >53 years,[5] hypokalemia,[6] and use of angiotensin receptor antagonists.[4] However, to the best of our knowledge, there are no reports evaluating the increased risk of dangerous arrhythmia with a comorbidity of SAS. A previous study reported repetitive oxygen desaturation and bradyarrhythmias in SAS; 20% of patients with severe SAS developed heart block, while 7% presented with sinus arrest.[1] In that study, the bradyarrhythmias typically occurred during sleep. Nevertheless, Ki-Hwan *et al*. reported a case of severe obstructive SAS requiring pacemaker implantation for daytime bradycardia. The cause of bradyarrhythmias in SAS may relate to increased vagal tone caused by hypoxia following cessation of air flow,[11] and the occurrence of bradyarrhythmias was linked to the severity of sleep apnea.[1] Marked and sudden hypertension was also reported as an additional factor in SAS-related bradyarrhythmias,[10] although there are no reports of SAH. An increased risk of severe sinus arrest would be expected by the coexistence of SAS and SAH.

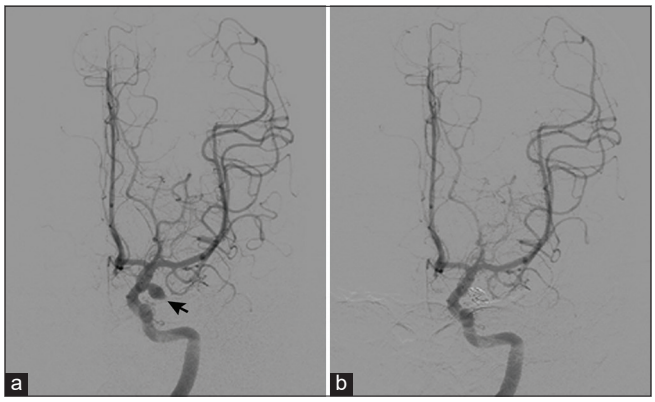

Figure 3: Preoperative (a) and postoperative (b) left internal carotid artery angiogram (frontal view). The low flow in the internal carotid-posterior communicating artery aneurysm (arrow) disappeared after endovascular treatment.

Our case was a young woman without any medical history apart from SAS. Her recent examination for SAS and associated arrhythmia did not detect any bradyarrhythmia during the day or night. Her SAH was low grade (WFNS Grade 1), although she experienced life-threatening severe asystole with a long pause. With respect to the cause of the unexpected cardiac event from the baseline clinical state, the potential risk of serious bradyarrhythmias was likely caused by the severe SAS, which was exacerbated by SAH, despite being low grade. Increased cardiac arrhythmias were observed until PBD 4 and decreased thereafter. After PBD 14, we observed no sinus arrest on 24-h ECG monitoring. Horie *et al*. reported that the inflammatory response peaked at 24–48 h after surgery for SAH,[7] which supports the hypothesis that fluctuation of the inflammatory response may have influenced the clinical course of arrhythmia in our case.

With respect to the influence of additional stress induced by surgery, the treatment should be as minimally invasive as possible. Horie *et al*. described a lower postoperative inflammatory response in patients receiving coiling compared with clipping, regardless of the WFNS grading of SAH. Thus, the selection of less invasive surgery, such as endovascular treatment, may help to decrease the arrhythmias associated with the catecholamine surge during the acute phase of SAH.[7]

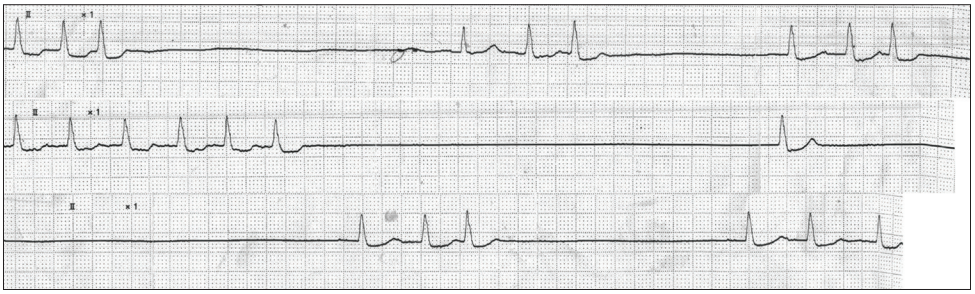

Figure 4: Electrocardiogram in the intensive care unit showing bradycardia and repetitive transient severe asystole.

CONCLUSION

We report a case of low-grade SAH with SAS, who experienced a life-threatening sinus arrest and required temporary pacemaker insertion. It is important to recognize the potential increased risk of bradyarrhythmia with the coexistence of these diseases.

Declaration of patient consent

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

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Nil.

Conflicts of interest

There are no conflicts of interest.

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