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Cardiac Arrest during Temporary Clipping for **Ruptured Internal Carotid Artery Aneurysm: A Case Report**

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Abstract	Objectives Mechanical stimulation of the trigeminal nerve during craniofacial, skull
	base, or dental surgeries may cause bradycardia, hypotension, or cardiac arrest. This
	phenomenon is called trigeminal cardiac reflex (TCR). We encountered a rare case of a
	patient who experienced sinus arrest due to temporary clipping of the intracranial
	carotid artery during the clipping of a ruptured aneurysm. We discuss possible reasons
	for the occurrence of TCR in this case.
	Materials and Methods A man in his 30s with no medical history presented with a
	sudden-onset headache. Computed tomography revealed a subarachnoid hemorrhage
	in the basal cistern and left Sylvian fissure. Angiography revealed a saccular aneurysm
	of the left internal carotid-anterior choroidal artery. A left frontotemporal craniotomy
	and dural incision were performed, followed by a trans-Sylvian approach. Cardiac arrest
	occurred twice during the temporary clipping of the intracranial carotid artery. After
	surgery, we performed a cardiac ultrasound echo and a 1-week Holter electrocardio-
	gram. Neither showed abnormalities. No arrhythmia or cardiac events were observed
Keywords	over a one and half-year follow-up period.
 trigeminal cardiac 	Results The cardiac arrest might have been triggered by the stimulation of the
reflex	trigeminal nerve in the internal carotid artery. The repeated and anatomical features of
 subarachnoid 	this case suggest that TCR triggered cardiac arrest.
hemorrhage	Conclusion The high probability that cardiac arrest was induced by trigeminal nerve
 temporary clipping 	stimulation should be considered during the temporary clipping of the internal carotid
 aneurysmal clipping 	artery. However, the predisposing factors and exact underlying mechanisms for these

sinus arrest

arrhythmias remain unknown and require further investigation.

Case Report

A man in his 30s with no medical history presented with a sudden-onset headache. Computed tomography revealed a

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subarachnoid hemorrhage (SAH) in the basal cistern and left Sylvian fissure. The patient was then referred to our hospital. Angiography revealed a saccular aneurysm of the left internal carotid-anterior choroidal artery. The aneurysm neck and

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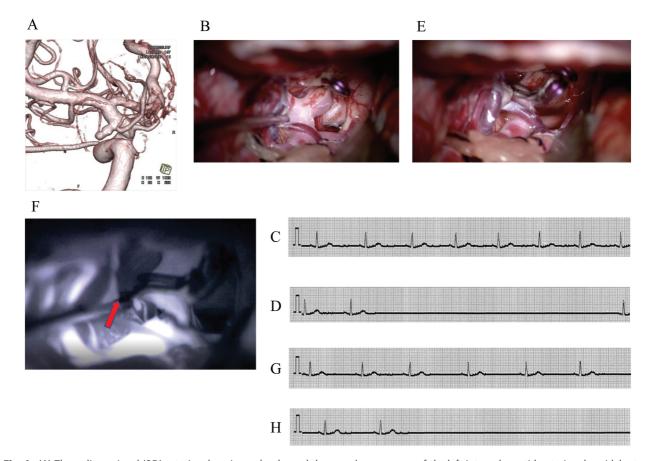


Fig. 1 (A) Three-dimensional (3D) rotational angiography showed the saccular aneurysm of the left internal carotid-anterior choroidal artery aneurysm. (B) Temporary clip was applied proximally to the internal carotid artery. (C) Clipping of the aneurysm was performed. (D) Intraoperative indocyanine green fluorescence video angiography showed a slight influx of the fluorescent dye into the dome of the aneurysm (red arrow). (E) Prior to the first temporary clipping, the electrocardiogram (ECG) showed sinus rhythm. (F) Upon the first temporary clipping, sinus arrest occurred for ~5 seconds. (G) Before the second temporary clipping, the ECG showed sinus rhythm. (H) When the second temporary clip was applied, sinus arrest occurred for 7 seconds.

dome sizes were 2 and 6 mm, respectively (> Fig. 1A). He had no medical history of heart disease and was not prescribed any medication. On the day after onset, we performed aneurysmal clipping. Anesthesia was induced using a combination of propofol, remifentanil, fentanyl, and rocuronium. Propofol and remifentanil were used to maintain the anesthesia. A left frontotemporal craniotomy and dural incision were performed, followed by a trans-Sylvian approach. The arachnoid around the internal carotid artery was carefully dissected and a temporary clip was inserted proximally to the internal carotid artery to dissect and expose the aneurysm (►Fig. 1B). During temporary clipping, sinus arrest occurred for approximately 5 seconds (**~Fig. 1C, D**), and the normal sinus rhythm spontaneously resumed. Clipping of the aneurysm was performed (**Fig. 1E**) and the temporary clip was removed. Intraoperative indocyanine green (ICG) fluorescence video angiography showed a slight influx of fluorescent dye into the dome of the aneurysm (**Fig. 1F**). Therefore, we decided to reclip the aneurysm. When the temporary clip was reinserted, sinus arrest occurred for approximately 7 seconds (**Fig. 1G, H**). Following subsequent removal of the temporary clip, the aneurysmal clip was reapplied, and neck clipping was confirmed via ICG. Sinus arrest was not observed. Postoperative magnetic resonance imaging showed no ischemic lesions. Additionally, postoperative cerebral three-dimensional rotational angiography confirmed that the anterior choroid plexus artery was preserved and the aneurysm was completely closed off. After surgery, we performed a cardiac ultrasound echo and a 1week Holter electrocardiogram. Neither showed abnormalities. He was discharged on postoperative day 25 with a modified Rankin Scale score of 0. No arrhythmia or cardiac events were observed over a one and half-year follow-up period.

Discussion

Trigeminal cardiac reflex (TCR) is defined as parasympathetic and sympathetic arrhythmia, hypotension, apnea, and gastrointestinal hyperactivity that suddenly occurs during stimulation of the trigeminal branch, resulting in a 20% drop in mean arterial blood pressure and bradycardia below 60 beats per minute.¹ TCR involves the sensory afferent pathway of the trigeminal nerve that enters the spinal trigeminal tract and nucleus via the Gasserian ganglion and then centrifugally via the rostral ventrolateral medulla and the intermediate lateral nucleus of the spinal cord, causing a vagal reflex that results in cardiovascular depression.² In the existing literature, we found three cases in which patients developed TCR during aneurysm clipping.^{3–6} Shibata et al reported that cardiac arrest due to the trigeminal vagal reflex occurred in 2 (2.3%) of 86 aneurysmal clipping surgeries.⁶ There is no case of a patient who developed cardiac arrest during the temporary clipping of an SAH aneurysm, possibly due to TCR. In the present case, cardiac arrest occurred on both occasions of temporary clipping. The repeated and anatomical features of this case suggest that TCR triggered cardiac arrest. TCR is usually resolved within tens of seconds by interrupting the intraoperative procedure.⁷ Therefore, the first step is to terminate the intraoperative procedure causing the reflex.

The majority of intracranial blood vessels and dura are innervated by the ophthalmic division of the trigeminal nerve.⁸ The nasociliary nerve, which originates from the ophthalmic branch of the trigeminal nerve, innervates the middle cerebral artery to induce major vasodilation.⁹ Experimental studies have identified trigeminal nerve endings throughout the extravascular membrane, often in close proximity to the tunica media but never in contact with smooth muscle cells.¹⁰ Therefore, TCR should be considered in cranial craniotomy, not only during dural manipulation of the skull base, but also when directly manipulating intracranial blood vessels, and it is important to cooperate with anesthesiologists by deepening the depth of anesthesia.¹¹ However, the predisposing factors and exact underlying mechanisms for these arrhythmias remain unknown and require further investigation.

Triggers that predispose patients to TCR during general anesthesia include hypoxia, hypercarbia, preoperative β -blockers, calcium blockers, shallow general anesthesia, and pediatric cases.¹²

In the present case, the anesthetics consisted of propofol, remifentanil, fentanyl, and rocuronium. Remifentanil is an ultrashort-acting opioid-receptor agonist that is continuously administered intravenously for analgesia and sedation. Remifentanil has been reported to cause bradycardia and cardiac arrest¹¹ and lower the threshold for vagal excitation.¹³ The incidence of TCR in the surgery for the cerebellopontine angle was shown to increase upon changing the anesthetics from fentanyl and nitrous oxide gas to remifentanil.¹⁴ In the present case, no intraoperative hypoxemia or hypercarbia was observed. Additionally, preoperative βblockers or calcium blockers were not administered. Hence, the anesthetic remifentanil, most likely, may have triggered the reflex. When the temporary clip was applied, the depth of anesthesia used, remifentanil, was possibly not enough to avoid TCR.

Few studies have shown that anticholinergic agonists are effective in preventing cardiovascular depression caused by TCR.^{15–17} However, some reports have also indicated that anticholinergic agonists can induce serious arrhythmias¹⁸; hence, the agents should be administered cautiously. TCR is usually resolved within tens of seconds by interrupting the

intraoperative procedure; therefore, the immediate first step is to stop the intraoperative procedure that causes the reflex.

In conclusion, we encountered a case of cardiac arrest during temporary clipping of the internal carotid artery. The cardiac arrest might have been triggered by the stimulation of the trigeminal nerve in the internal carotid artery. The high probability that cardiac arrest was induced by trigeminal nerve stimulation should be considered during the temporary clipping of the internal carotid artery.

Conflict of Interest None declared.

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