Asystole during microvascular decompression in case of trigeminal neuralgia—A case report—

Su Yeon Lee*, Duk-Hee Chun, Taekyu Lee, Min-young Kim, and Soo-yeun Park

Department of Anesthesiology and Pain Medicine, CHA Bundang Medical Center, CHA University, Seongnam, *CHA Gangnam Medical Center, CHA University, Seoul, Korea

Manipulation of the sensory branches of the trigeminal nerve is known to cause autonomic changes, such as bradycardia or asystole, known as the trigemino-cardiac reflex. In this case, the patient underwent microvascular decompression due to trigeminal neuralgia and developed sudden bradycardia, followed by abrupt asystole with a concurrent fall in the systolic blood pressure. There was spontaneous return of cardiac rhythm and blood pressure, but two more episodes of sinus bradycardia occurred during the surgery. (Anesth Pain Med 2014; 9: 58-60)

Key Words: Microvascular decompression, Trigeminal neuralgia, Trigemino-cardiac reflex.

The trigemino-cardiac reflex (TCR) is a phenomenon consisting of bradycardia, arterial hypotension, apnea, and gastric hypermotility and is thought to occur via stimulation of one or more of the sensory branches of the trigeminal nerve [1,2]. There is little information regarding the occurrence of TCR during microvascular decompression (MVD) in cases of trigeminal neuralgia and we experienced sudden bradycardia followed by abrupt asystole during MVD.

CASE REPORT

A 71-year-old female patient was diagnosed with trigeminal neuralgia 10 years ago and received occasional treatment including radiofrequency rhizotomy. Surgical MVD was scheduled due to severe right maxillary and mandibular area pain. The patient’s past medical history revealed a ten-year history of well-controlled hypertension. Other routine preoperative laboratory tests showed values within normal range.

The patient was not premedicated but given glycopyrrolate 0.2 mg and midazolam 2.5 mg IV after routine monitoring at the operating room. Anesthesia was induced and maintained using a target controlled infusion pump (Orchestra® Base Primea, Fresenius Vial, Brezins, France) with propofol (target effect-site concentration range: 3.0–4.0 μg/ml) and remifentanil (target effect-site concentration range: 3.0–4.0 ng/ml). To facilitate tracheal intubation, rocuronium 40 mg IV was given and the patient was mechanically ventilated with oxygen and air (FiO2 = 0.5). Atracurium 4 ug/kg/min was also continuously IV administered. Intraoperatively the patient had electrocardiogram, end-tidal CO2 (ETCO2) detector, oxygen saturation (SpO2), bispectral index (BIS), esophageal temperature, and an indwelling radial artery catheter to monitor vital signs throughout the surgery. The patient was turned to the left lateral decubitus position for the surgery.

A retrosigmoid craniotomy was performed and the tortuous right superior cerebellar artery compressing the right 5th cranial nerve at the root exit zone was observed. The arterial blood gas analysis showed pH 7.462, PaCO2 34.2 mmHg, PaO2 202.9 mmHg, Hb 10.2 g/dl, and electrolytes within normal range. Ninety minutes after surgery began, while decompressing the offending vessel from the right 5th cranial nerve, the patient’s heart rate suddenly decreased from 62 to 42 beats/min. Then the patient developed abrupt, sustained asystole with a concurrent fall in systolic blood pressure from 130 mmHg to almost zero. The surgeon was notified immediately and surgical manipulation was halted. There was a spontaneous return of a sinus bradycardia with the return of systolic blood pressure to 130 mmHg upon cessation of manipulation (Fig.
Asystole during MVD

Fig. 1. The schematic representation of ECG and arterial line blood pressure change during asystole. There was sudden decrease in heart rate followed by asystole and then return of the rhythm after manipulation halted. ABP: arterial blood pressure.

1). Asystole lasted about 5 s and there was no need for pharmaceutical intervention. Over the next 20-30 s, a sinus rhythm with a heart rate of 60-70 beats/min returned and the procedure was resumed. After asystole, SpO₂ and ETCO₂ values remained the same. SpO₂ was maintained at 100% and ETCO₂ was maintained at 29 mmHg. There were two more episodes of bradycardia during the decompression but no medication was required. The surgery lasted 175 min and anesthesia lasted 285 min. The patient had an uneventful recovery and the later follow-up was also uneventful.

DISCUSSION

The TCR is a reflex response that can cause sudden onset of bradycardia or asystole with arterial hypotension during manipulation of the sensory branches of the trigeminal nerve [1,2]. Lee et al. [3] reported a case of oculo-cardiac reflex (OCR) manifested as high degree atrioventricular block during endoscopic sinus surgery. Unlike the OCR which has undergone extensive investigation, the exact mechanism of TCR is still unclear even though OCR is a variant of the TCR. The definition of TCR is controversial that no strict inclusion criteria for TCR are present yet. Only clinical features are described, including sudden-onset of sinus bradycardia, bradycardia terminating asystole, asystole with no preceding bradycardia, arterial hypotension, apnea, and gastric hypermotility [4].

It is thought that the TCR pathway begins with the sensory nerve endings of the trigeminal nerve sending neuronal signals via the Gasserian ganglion to the sensory nucleus of the trigeminal nerve. Then it continues along the internuncial fibers in the reticular formation which connect with the motor nucleus of the vagus nerve. Vagal stimulation via these connections provokes the negative chronotropic and inotropic responses [5,6] like bradycardia, hypotension, and asystole observed in this patient.

In cases of trigeminal neuralgia undergoing MVD, there is little information about the occurrence of TCR. In one study, Schaller [7] defined the TCR as a drop in mean arterial blood pressure and heart rate of more than 20% compared with the baseline values before the stimulus and coinciding with the manipulation of the trigeminal nerve. This retrospective study over the period of 8 years studied 28 consecutive cases where 5 patients developed TCR and one of them developed asystole. Therefore, one must recognize that TCR is not an uncommon event during MVD.

Intraoperative factors such as light anesthesia, hypercapnia, and hypoxemia as well as young age, the nature of the provoking stimulus, and drugs are known to increase the risk of occurrence of TCR [6,8,9]. Also abrupt and sustained traction is more prone to elicit TCR than gentle traction [8]. During the surgery, there was no evidence of hypercapnia (ETCO₂ = 29 mmHg), hypoxemia (SpO₂ = 100%), or light general anesthesia (BIS was maintained between 40-60). There was no additional medication given to the patient. Therefore, the only possible explanation is the provoking stimulus, the stimulation of the trigeminal nerve. Removing the triggering factor is often known to cause cessation of the reflex; therefore the surgeon must be notified to withhold further maneuvers upon occurrence of the TCR. Administration of the anticholinergic atropine may help in cases where bradycardia is severe or it persists despite cessation of the stimulus. Prophylactic uses of the anticholinergic drugs are not recommended because they can cause refractory cardiac arrhythmias [8]. Use of vasopressors and cardiac life support (like temporary pacing) should be considered for those who are hemodynamically insufficient [10].

Owing to the course of the trigeminal nerve, the TCR has been reported to occur during neurosurgical interventions, craniofacial, skull base, and ophthalmologic surgeries [11]. As the TCR may occur without prior hemodynamic changes [12] and patient’s characteristics do not influence the occurrence of this reflex [11], all patients undergoing surgeries on the head must be monitored vigilantly and thoroughly during operation to recognize TCR and treat TCR.

REFERENCES

1. Bainton R, Lizi E. Cardiac asystole complicating zygomatic arch