Management of tracheal compression that’s caused by an innominate artery aneurysm
— A case report —

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An aneurysm of the innominate artery could compress the tracheal lumen and airway management needs special care. Intubation without intensive monitoring and antihypertensive agents could aggravate the hypercapnia and completely rupture the aneurysm. There are few reports on the airway management of tracheal compression that’s caused by an innominate artery aneurysm. We report here on a patient who had a severe hypercapnia after endotracheal intubation above the stenotic area of the tracheal compression, which was caused by an artery aneurysm. Permissive hypercapnia was inadvertently enforced without our knowledge, but the patient recovered without any neurologic problems. (Korean J Anesthesiol 2009; 57: 762~)

Key Words: Hypercapnia, Innominate artery aneurysm, Tracheal compression.

An aneurysm of the innominate artery could compress the tracheal lumen and airway management needs special care. Endotracheal intubation may not resolve the dyspnea and rather be dangerous. So intensive monitoring for the rupture of aneurysm and advanced knowledge about the airway management are required. There are few reports about the airway compression caused by an artery aneurysm. We report a patient who had a severe hypercapnia after endotracheal intubation in tracheal compression caused by an artery aneurysm, but recovered without neurologic sequelae.

CASE REPORT

A 50-year-old man, 170 cm tall and weighing 69 kg, presented with severe dyspnea. He had been treated unsuccessfully for asthma for 3 months at another hospital. A computed tomographic (CT) scan of the chest showed a 50 mm aneurysm of the innominate artery with a large thrombus from rupture of the medial wall of the proximal innominate artery, causing compression of the trachea (Fig. 1). The left common carotid artery branched from the innominate artery (Fig. 2).

The patient was a chronic alcoholic and heavy smoker. He had a history of a motor vehicle accident 9 years ago and a screw was inserted into the right clavicle for a fracture 18 months ago. He had a successful nasal bone reduction after a fracture under general anesthesia 1 year ago.

In the emergency room, the patient presented with a blood pressure of 122/77 mmHg, heart rate of 108 beats/min, respiratory rate of 26 breaths/min, oxygen saturation of 96%, and arterial blood gas analysis showed a pH 7.472, PaCO₂ 35.7
mmHg, and PaO₂ 92.8 mmHg in oxygen 3 L/min with the nasal cannula. While waiting for the emergency operation, the respiratory rate increased up to the 30 breaths/min, and dyspnea and chest pain increased as well. Although breathing with an oxygen mask, dyspnea aggravated. By the surgeon, 5 mg midazolam was injected and the patient was intubated with a 7.5 mm tracheal tube above the stenotic area and spontaneous breathing permitted (Fig. 3). Fifteen minutes after intubation, a blood gas analysis showed a pH 7.066, PaCO₂ 90.2 mmHg, and PaO₂ 187.4 mmHg in the FiO₂ of 0.6. After a few minutes, a subsequent blood gas analysis showed a pH 6.692, PaO₂ 343 mmHg, and PaCO₂ -- (not checked). Upper limit of a PaCO₂ is 150 mmHg in our blood gas analyzer.

The patient was transferred to the operating room assisted with self-inflating resuscitation bag (Ambu) and showed drowsy mental state. Initial vital signs showed a blood pressure of 120/80 mmHg and a heart rate of 120 beats/min. After anesthetic induction with 3 mg midazolam, 100 μg fentanyl, and 30 mg rocuronium, it was maintained with target controlled infusion of propofol and remifentanil. Intraoperative monitoring included invasive arterial (right and left radial) pressure, jugular central venous (right internal) pressure, pulse oximetry, 5-lead electrocardiogram, temperature (nasopharyngeal and rectal) and end-tidal carbon dioxide. Pressure controlled ventilation with the airway pressure of 30 cmH₂O and a respiratory rate 30 breaths/min in the FiO₂ 1.0 showed a tidal volume of 150 ml and considering the lung injury by the barotraumas, the airway pressure was maintained below the 30 cmH₂O. After thirty minutes, the end-tidal CO₂ was 84 mmHg and arterial blood gas was checked with a pH 6.842, PaCO₂ 170.1 mmHg, and PaO₂ 480.1 mmHg. Cardiopulmonary bypass of the femoral artery and vein decreased the blood CO₂. A right axillary artery was cannulated and the patient was cooled to 18°C for total circulatory arrest. Innominate artery was incised and the beginning of it from the aorta was clamped. A graft was inserted in the right common carotid artery (RCCA) and antegrade cerebral perfusion was initiated. The graft from the innominate artery to the RCCA and subclavian artery was interposed. The total circulatory arrest time was 10 minutes and the antegrade cerebral perfusion time was 65 minutes. During the antegrade cerebral perfusion, transcranial Doppler was performed and the blood flow was well preserved.

After surgery, a tidal volume of 450 ml and a respiratory rate 16 breaths/min in the FiO₂ 1.0 showed a pH 7.214, PaCO₂ 32.2 mmHg, and PaO₂ 204.1 mmHg. The trachea was expanded in a 3-dimensional chest CT. The dyspnea symptom was improved and postoperative neurological problems or abnormalities in the brain magnetic resonance imaging were not identified.

**DISCUSSION**

Airway management of a patient with tracheal compression caused by a major artery aneurysm is challenging to the anesthesiologist. Fearing muscle fatigue and severe dyspnea, the surgeon intubated the patient with a sedative drug without intensive monitoring, but this is very dangerous. This procedure
could cause total airway obstruction, respiratory arrest and aneurysmal rupture [1]. Endotracheal intubation of tracheal compression caused by a major artery aneurysm should be performed under intensive monitoring and drugs that could avoid hypertension. Before intubation, whether improvement of dyspnea should be considered cautiously. Airway managements in tracheal stenosis include intubation above or below the stenotic area, laryngeal mask airway, high frequency ventilation, cardiopulmonary bypass and endotracheal stent insertion. Each method has advantages and disadvantages. Endotracheal intubation and laryngeal mask airways may not provide a successful conduit [2]. High frequency ventilation can develop barotraumas and is difficult to monitor the respiratory pattern [3]. In cardiopulmonary bypass, it is important that the patient cooperates and that a suitable posture can be adopted. With the use of heparin, bleeding can increase. In endotracheal stent insertion, restenosis and an aneurysm rupture could occur and stent should be removed afterwards [4]. But in author’s case, endotracheal stent following CPB might have been better safe. Intubation above the stenotic area may not improve dyspnea symptoms; on the contrary hypercapnia could occur. But stenotic area was near the carina, we didn’t insert the small endotracheal tube below it. In this case, after intubation, a PaCO₂ increased rapidly more than 10 mmHg per hour and persisted for several hours. A PaCO₂ over 170 mmHg increases intracranial pressure (ICP) and makes cerebral edema, myocardial depression and arrhythmia. We considered the permissive hypercapnia for preventing the lung injury, but it was very difficult to maintain the pH.

Initially, we ventilated the patient with volume controlled mode but the airway pressure was over 40 cmH₂O and end-tidal CO₂ was checked above 90 mmHg. Manual ventilation didn’t ameliorate the situation, so we hurried to change the ventilation with pressure controlled mode and hyperventilated the patient. If we had decreased the respiratory rate and increased the expiratory time, carbon dioxide might have eliminated more. Although permissive hypercapnia was allowed, too rapid elevation and very high CO₂ levels are dangerous [5]. In the permissive hypercapnia, allowable PaCO₂ is difficult to present exactly, but the speed which is instituted is important [6].

Because the origin of the innominate artery from the aorta was clamped and the left common carotid artery branched from the innominate artery, total circulatory arrest and selective cerebral perfusion was required. Transcranial Doppler monitoring was used during total circulatory arrest and antegrade cerebral perfusion, but this method shows only blood flow and whether cerebral edema or elevated ICP are not known. Subarachnoid pressure monitoring and drain of cerebrospinal fluid (CSF) would be a safer method, but we wised after that event.

The causes of aneurysms are bacterial infection, iatrogenic, chest trauma, syphilis, atherosclerosis and collagen disorders [7,8]. In this case, a traumatic aneurysm was suspected. A right clavicle fracture 18 months prior is a possible culprit and small multiple traumas caused by heavy drinking could have instigated the arterial injury. In published report, trauma 30 years previous caused the injury [9]. But in this case, a successful nasal bone reduction after a fracture, under general anesthesia 1 year prior without special event is doubt.

Fortunately, the patient recovered without neurological problems. Acute lung injury by barotraumas and the idea of permissive hypercapnia should be considered in the management of dyspnea in tracheal compression caused by a major artery aneurysm. And further studies about the permissive hypercapnia are warranted.

**REFERENCES**