Negative Pressure Pulmonary Edema Following Foramen Magnum Decompression for Chiari Malformation Type I

—Case Report—

Yoshitaka HIRANO, Taku SUGAWARA, Yoshiharu SATO*, Koji SATO*, Tomoya OMAE, Toshio SASAJIMA, and Kazuo MIZOI

Departments of Neurosurgery and *Anaesthesia, Akita University School of Medicine, Akita

Abstract

A 57-year-old obese female presented with vagal and hypoglossal nerve pareses, and magnetic resonance imaging revealed Chiari malformation type I. Standard surgical treatment for Chiari malformation type I was successfully performed. However, immediately after the patient was extubated, she developed signs of upper airway obstruction and chest radiography revealed pulmonary edema. Her ventilation was assisted by maintaining positive end-expiratory pressure at 8 cmH₂O. Intravenous furosemide and hydrocortisone were administered. Her respiratory status improved 12 hours later, and she was extubated 3 days after the operation. Postextubational course was uneventful, and the patient was discharged 2 weeks after extubation. The initial neurological deficits had mostly disappeared by 10 months after the operation. This unusual case of negative pressure pulmonary edema indicates that obesity and lower cranial nerve paresis are further risk factors for pulmonary edema as a postextubational complication of surgical treatment.

Key words: Chiari malformation, foramen magnum decompression, negative pressure pulmonary edema, perioperative complication

Introduction

Laryngospasm is a common complication after tracheal extubation, occurring in 8 to 237 of every 1000 anesthetic procedures. Laryngospasm may indicate laryngeal hypersensitivity, which may be caused by superior laryngeal nerve injury. Negative pressure pulmonary edema (NPPE) has been reported in 11% of healthy young patients who experienced laryngospasm. Negative thoracic pressure results in capillary failure and will sometimes cause alveolar hemorrhage in association with NPPE.

We describe a case of NPPE which occurred after standard surgical treatment for Chiari malformation type I.

Case Report

A 57-year-old obese female, with weight 70 kg and height 151 cm, had suffered from dysphagia and dysarthria for 6 months. Magnetic resonance (MR) imaging obtained at a local hospital revealed Chiari malformation type I with basilar impression. She was referred to our hospital in September 2005. On admission, neurological examination detected moderate pareses of the left vagal and hypoglossal nerves, and bronchoscopic inspection revealed medially fixed left vocal cord. No evidence of respiratory infection was found, although she had a past medical history of bronchial asthma and diabetes mellitus. Foramen magnum decompression was planned based on the findings of MR imaging. Endotracheal general anesthesia was induced, and the patient was placed in the prone position.
Foramen magnum decompression (suboccipital craniectomy) was performed. C1 and upper C2 laminectomies were added for posterior decompression. The operative procedure was completed by duroplasty with artificial dural substitute, and cranioplasty with hydroxyapatite plate. The operative course was uneventful. She was repositioned supine, the anesthesiologist confirmed complete recovery from the general anesthesia, and she was extubated without difficulty. However, she developed signs of upper airway obstruction immediately after extubation, and pulse oximetry showed a marked decrease from 100% to 22%. Electrocardiography showed bradycardia, and the patient was reintubated. Pinky frothy fluid filled the endotracheal tube. Immediate chest radiography revealed pulmonary edema (Fig. 2).

The patient was admitted to our intensive care unit (ICU), where her ventilation was assisted by maintaining positive end-expiratory pressure at 8 cmH₂O. Intravenous furosemide and hydrocortisone were administered. Her respiratory status improved 12 hours after admission to the ICU, and she was extubated 3 days after the operation. Postextubational course was uneventful, and the patient was discharged 2 weeks after extubation. Her initial neurological deficits had mostly disappeared by 10 months after the operation. Successful decompression of the foramen magnum was confirmed by the MR study on discharge.

Discussion

NPPE is described as a perioperative non-cardiogenic complication. Although various factors are considered in development of this condition, NPPE is basically caused by excessive aspiratory force. The aspiratory force during upper airway obstruction is reported to reach as high as \(-100\) mmHg. This excessive negative pressure is thought to cause in disruption of the alveolar capillaries to cause pulmonary edema, and sometimes alveolar hemorrhage.

The pulmonary edema in the present case was compatible with NPPE. Postextubational NPPE is a rare complication with a reported incidence of 0.05–0.1%, and occurs predominantly in muscular young male patients. The present case suggests that postextubational NPPE may also occur in obese middle-aged female patients. The pathogenesis of the present case of NPPE remains unclear, but we suspect a combination of several factors, such as the patient’s obesity and preexistent vocal cord paralysis. Anesthetic agents absorbed into the dense fat tissue of the obese patient may have delayed recovery from general anesthesia, and combined with the decurarized state of the patient, may have caused severe laryngospasm. The paralyzed vocal cord may have caused upper airway obstruction.

Foramen magnum decompression for Chiari malformation is a safe and well-established surgical procedure, and has never been associated with postoperative pulmonary edema. A review of 146 cases of NPPE found that 50% of patients had undergone surgery on the upper aerodigestive tract, but none had surgery on the upper cervical spine, craniovertebral junction, or cranium.

NPPE can usually be successfully treated by ventilation with PEEP and intravenous furosemide, but every neurosurgeon and anesthesiologist must be aware of this rare complication. Obesity and vocal
cord paresis seem to be further risk factors for postextubational NPPE after surgical treatment for Chiari malformation.

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References


Address reprint requests to: Yoshitaka Hirano, M.D., Department of Neurosurgery, Southern TOHOKU General Hospital, 1–2–5 Satonomori, Iwanuma, Miyagi 989–2483, Japan.