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Postoperative left recurrent laryngeal nerve palsy possibly caused by coincidental swelling of the metastatic mediastinal lymph node

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Introduction

Postoperative recurrent laryngeal nerve palsy (RLNP) is mainly caused by direct surgical injury to the nerve [1]. Although tracheal intubation represents one of the other causes of postoperative RLNP, the incidence of postoperative RLNP as a result of tracheal intubation is very rare (less than 0.1%) [1]. Generally, most cases of RLNP due to tracheal intubation recover spontaneously within 2–3 months [1–3].

This report describes a patient with prolonged postoperative RLNP. This patient underwent surgery at a site far from the anatomical course of the left recurrent laryngeal nerve, and the tracheal intubation was completely uneventful. The cause of the postoperative RLNP was unknown until 4 months after the operation when a swollen lymph node was clearly detected by computed tomography (CT) in the aortopulmonary window.

Case report

A 67-year-old, 52-kg woman was admitted to our hospital with an abdominal tumor. Previously she had undergone sigmoidectomy and partial resection of the right lung after a diagnoses of primary sigmoid colon cancer and metastatic lung cancer. This time, abdominal total hysterectomy and bilateral adnexectomy were scheduled after a diagnosis of metastases to the uterus and bilateral lungs. Before the operation, she had not complained of any symptoms of recurrent laryngeal nerve palsy (RLNP). The patient was premedicated with 0.01 mg·kg^{-1} atropine sulfate and 0.5 mg·kg^{-1} hydroxyzine intramuscularly. General anesthesia was then induced with fentanyl (2 μg·kg^{-1}), propofol (1 mg·kg^{-1}, and subsequently continuous infusion at a rate of 10 mg·kg^{-1}·h^{-1}) and vecuronium (0.15 mg·kg^{-1}) intravenously. Tracheal intubation was performed gently, smoothly, and atraumatically, using a 7.5-mm (internal diameter) tube with a high-volume, low-pressure cuff (Fuji Systems, Tokyo, Japan). No abnormalities of the vocal cords were detected by visual inspection. The cuff was inflated with air to the minimum occlusive pressure. As the patient's lungs were ventilated with a combination of 33% O_2 and 67% N_2O, we frequently adjusted the cuff pressure to keep it below 20 mmHg. The patient's head and neck were kept in a neutral position. The operation lasted 3.5 h and was uneventful. There was no transesophageal echocardiography or esophageal stethoscope monitoring during the operation. The trachea was extubated smoothly 15 min after the operation.

From the second postoperative day, the patient complained of symptoms of RLNP, hoarseness and dysphagia. For a definitive diagnosis of RLNP, an otolaryngological examination was performed on the 10th postoperative day. The left vocal cord was in the paramedian position with no signs of hematoma or lesions. Computed tomography (CT) of the anatomical course of the left recurrent laryngeal nerve showed nothing that could cause RLNP during the perioperative period. The dysphagia disappeared but the hoarseness still remained on the 29th postoperative day when the patient was discharged. Four months after the operation, CT clearly showed a swollen lymph node (12 mm in diam-

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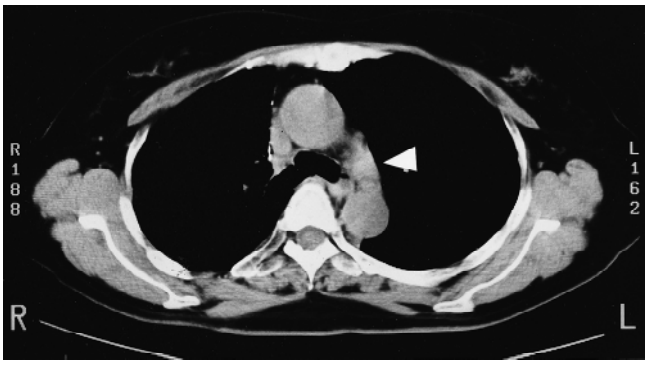

Fig. 1. A swollen lymph node in the aortopulmonary window was detected by computed tomography (CT) 4 months after the operation. This is a plain CT film; the *arrow* points to the swollen lymph node (12 mm in diameter)

eter) in the aortopulmonary window in the follow-up examination (Fig. 1). At this point, the hoarseness still remained.

Discussion

Patients undergoing tracheal intubation frequently experience postoperative hoarseness and a sore throat (4–42%) [1,2]. On the other hand, the incidence of postoperative RLNP is rarely described. Postoperative RLNP is mainly caused by direct surgical injury to the nerve [1]. It also occurs as a result of traumatic tracheal intubation, sustained high pressure in the cuff of the tracheal tube (higher than 25 mmHg), inappropriate positioning of the tracheal tube, and malpositioning of the patient's head and neck [1,4,5]. The use of transesophageal echocardiography as well as the use of an esophageal stethoscope also increase the risk of postoperative RLNP [5]. The incidence of postoperative RLNP as a result of tracheal intubation, however, is very rare (less than 0.1%) [1].

Generally, most cases of RLNP due to tracheal intubation recover spontaneously within 2–3 months [1–3]. In this case, one symptom of RLNP (hoarseness) lasted until 4 months after the operation. Owing to functional compensation by the right vocal cord, the dysphagia disappeared, but the hoarseness remained.

In this case, the tracheal intubation was performed gently, smoothly, and atraumatically. It appears less possible that the postoperative RLNP could be attributed to the tracheal intubation in this case. In support of this is the fact that at 4 months after the operation, we clearly detected a swollen lymph node in the aortopulmonary window, i.e., in the anatomical course of the left recurrent laryngeal nerve. Although we could not identify any positive finding from the CT films taken during the perioperative period, retrospective reanalyses of those films show, although not very clearly, something that could be the swollen lymph node in the same location. Because those CT films were taken of 10-mm-thick sections, the slightly swollen lymph node was not visible enough for a definite diagnosis because of the partial volume effect. If we had used finer collimated sections (5 or 7 mm thick), we might have been able to detect the swollen lymph node distinctly at that time.

It is impossible to prove a definite causal relationship between the RLNP and the swollen lymph node in this case, since it is unrealistic to remove the lymph node and observe any changes in the symptoms of RLNP. It is difficult to exclude a causal relationship between the general anesthesia and the neurological deficit [6], because either general anesthetics or tracheal intubation might directly impair the nerve. Nevertheless, the overall information in this case supports the possibility that the swollen lymph node was the etiology of the postoperative RLNP. The swollen lymph node might have directly (or indirectly through the edematous tissues around it) compressed the nerve, thus resulting in the permanent palsy.

In conclusion, our experience with this patient suggests that postoperative RLNP can be caused by coincidental swelling of the metastatic lymph node. Complete examinations should be performed to detect any pathological changes in the anatomical course of the recurrent laryngeal nerve in a patient with prolonged RLNP.

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