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□ Case Report □

Occurrence of iatrogenic pneumothorax during laparoscopy-assisted distal gastrectomy

— A case report —

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The occurrence of a pneumothorax during laparoscopy-assisted distal gastrectomy (LADG) is rare. A pneumothorax was developed during a LADG under general anesthesia in a 67-year-old woman with gastric cancer. About 140 minutes after CO_2 insufflation, sudden hemodynamic collapse occurred. A defect was noted in the diaphragm. After immediate repair under laparoscopy, hemodynamic stability was achieved within several minutes. In the anesthetic management of a LADG, the anesthesia provider should be aware of the possible occurrence of a pneumothorax. **(Korean J Anesthesiol 2009; 57: 765～7)**

Key Words: Iatrogenic diaphragmatic injury, Laparoscopy-assisted distal gastrectomy (LADG), Pneumothorax.

Recently, the use of laparoscopy-assisted distal gastrectomy (LADG) has increased in patients with gastric cancer. The insufflation of CO_2 and operative position may cause physiologic changes in the patient and complications during the procedure of LADG. The complete understanding of several complications that might occur during LADG is therefore important for appropriate management of the anesthesia.

We experienced a case of CO_2 pneumothorax due to iatrogenic diaphragmatic injury which occurred during a LADG. Herein we report this case with a review of the literature.

CASE REPORT

The patient was a 67-year-old woman with a body weight of 66 kg and a height of 167 cm, who was diagnosed with gastric cancer and then was hospitalized for a LADG. She had an admission history of acute pancreatitis and no other significant medical problem. The preoperative laboratory findings were all within normal ranges.

The patient was premedicated with midazolam 2 mg and glycopyrrolate 0.2 mg intramuscularly 30 minutes before anesthesia. In the operating room, routine monitoring (electrocardiogram, non-invasive blood pressure, and pulse oximetry) was initiated. The pre-induction vital signs were as follows; blood pressure 138/89 mmHg, heart rate 88 beats/min and SpO_2 99%. General anesthesia was induced with lidocaine 20 mg, propofol 120 mg, and vecuronium 8 mg. After preoxygenation with 100% oxygen, the patient was intubated with 7.0 sized endotracheal tube (ETT). The position of ETT was confirmed by the auscultation of both lung sounds. The anesthesia was maintained with oxygen (2 L/min), N_2O (2 L/min), and sevoflurane (2.0－3.0 vol%, end-tidal concentration). Following the induction of anesthesia, mechanical ventilation was initiated at a tidal volume of 8 ml/kg and a respiratory rate of 12 breaths/min, which produced a peak airway pressure 12－13 cmH_2O and an end-tidal CO_2 of 32－35 mmHg. After CO_2 insufflation (2 L/min, the intra-abdominal pressure was automatically maintained at 12 mmHg) and trocar insertion, the patient was placed in a 30° reverse Trendelenbug position to assist surgical exposure.

About 140 minutes after insufflation, O_2 saturation suddenly decreased to 93－95% and the lung compliance decreased to 19 ml/cmH_2O. The end-tidal CO_2 increased to 49－50 mmHg and the peak airway pressure increased to 30－32 cmH_2O. The

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blood pressure and heart rate were not changed. On chest auscultation, the breath sounds were decreased in the left lung field. To exclude endobronchial intubation, a fiberoptic bronchoscopy was inserted via ETT. Correct position of the ETT was confirmed by visualization of the carina. Therefore, we thought that the cause of hypoxemia might be due to acute left lung atelectasis. For the treatment of atelectasis, alveolar recruitment (manual ventilation to an airway pressure of 40 cmH_2O for 10 breaths over one minute) with 100% oxygen and a 5 cmH_2O positive end expiratory pressure (PEEP) were applied. Then, breath sounds were heard on the left side and O_2 saturation was increased to 99%. The end-tidal CO_2 decreased to 35－36 mmHg and the peak airway pressure decreased to 23－25 cmH_2O. For continuous blood pressure monitoring and arterial blood gas analysis (ABGA), a 20 G catheter was placed in the left radial artery. The results of the ABGA were as follows: pH 7.18, $PaCO_2$ 67 mmHg, PaO_2 130 mmHg, BE －3.4 mM/L, and SaO_2 98%.

About 150 minutes after insufflation, O_2 saturation decreased to 93－96% and the end-tidal CO_2 concentration increased to 40－42 mmHg. The peak airway pressure increased to 30 cmH_2O. At this time, the systolic and diastolic pressures decreased to 30 mmHg and 20 mmHg, respectively. Immediately, phenylephrine 100 μg was injected twice and the blood pressure returned to 120/55 mmHg. Using a double lumen central venous catheter, the right subclavian vein was cannulated and the central venous pressure was 9 mmHg. For the rapid detection of life-threatening cardiac problems, a transesophageal echocardiography probe was inserted. Myocardial dysfunction, cardiac output, and left ventricular end-diastolic pressure were evaluated and all of the findings were normal. At that point, a defect by surgical injury to the left diaphragm (3 cm in length) was noted by the surgeon. This defect was immediately repaired by laparoscopy-assisted thoracosynthesis.

During the surgical repair of the diaphragmatic perforation, the nitrous oxide was discontinued, the fraction of inspired O_2 (FiO_2) was maintained at 1.0, and a 5 cmH_2O PEEP was applied. These treatments led to correction of the hypoxic state, re-expansion of the lung, and pleural drainage of the CO_2. After such treatments, the vital signs and peak airway pressure were stabilized. The arterial blood gas parameters were also normalized.

Following completion of about 7 h surgical procedure, a chest tube was inserted. The patient completely recovered from anesthesia in the operating room with no signs of respiratory failure. The patient was therefore transferred to the recovery room. A chest radiograph on the second post-operative day revealed no abnormal findings, and the chest tube was removed. Without any complication during the post-operative course, she was discharged on the 13th post-operative day in a stable condition.

DISCUSSION

Laparoscopic surgery has gained in popularity and the indications have increased due to the diagnostic and therapeutic usefulness. In laparoscopic surgery, a CO_2 pneumoperitoneum is essential to achieve surgical vision and a sufficient degree of procedural space. A CO_2 pneumoperitoneum produces respiratory complications due to the direct absorption of CO_2, abdominal distension, and controlled ventilation. Respiratory complications due to pneumoperitoneum include ventilation/perfusion mismatch, subcutaneous emphysema, pneumothorax, pneumomediastinum, and massive CO_2 embolism. A complete understanding of these complications which might occur during laparoscopic surgery is therefore important for appropriate management of the anesthesia.

Pneumothorax is very rare, but life-threatening complication related to endoscopic procedures, and may occur at any time during a laparoscopic procedure [1]. It is also noteworthy that the occurrence of a pneumothorax is not related to the surgical site. Of the endoscopy-assisted surgeries that have been reported, the occurrence of a pneumothorax has been observed in cardiovascular surgery via thoracoscopy, abdominal surgery (hepatectomy and cystectomy), head-and-neck surgery (thyroidectomy), and urogenital surgery (nephrectomy and prostatectomy). Based on these cases, laparoscopy-related pneumothoraces occur independent of the type of surgical approach (transperitoneal or retroperitoneal approaches). Wolf et al. [2] reported that the rate of CO_2 absorption was higher in cases in which a procedure was performed via a retroperitoneal approach than those performed via a transperitoneal approach. These authors also noted that the incidence of complications, such as subcutaneous emphysema, pneumothorax, and pneumomediastinum, was higher in cases in which surgery was performed via a retroperitoneal approach. According to the study of Ng et al. [3], however, the retroperitoneal approach is not associated with increased CO_2 absorption. Therefore controversial opinions exist regarding the correlation between a retroperitoneal approach and the occurrence of a pneumothorax.

A pneumothorax can occur during all laparoscopic surgery. However, there are no reports of a pneumothorax developing during LADG. In 2007, Kim et al. [4] reported that complications occurred in 128 patients who underwent LADG. According to the report, the intra-operative complications include bleeding and damage to adjacent organs. The authors also noted that the post-operative complications were bleeding, subcutaneous emphysema, atelectasis, acute pancreatitis, and dumping syndrome. Of note, pneumothorax was not one of the complications that occurred in association with LADG.

The known risk factors for a pneumothorax in laparoscopic surgery include a long surgical time (>200 minutes), end-tidal CO_2 > 50 mmHg, old age, and unskilled surgeons [5]. The causes of a pneumothorax during laparoscopy include barotrauma due to mechanical ventilation, dissection of intra-pulmonary vessels, rupture of the mediastinal pleura due to an influx of CO_2 infused into the mediastinum along the musculofascial planes, the insertion of a trocar during surgery, and dissection of adjacent organs [6]. Even though there have been no reports of a pneumothorax during a LADG, the longer surgical time of a LADG and the dissection of organs around the diaphragm make it possible that a LADG may also cause a pneumothorax.

The first physiologic changes which occur in the case of a pneumothorax are the increased end-tidal CO_2, increased airway pressure, decreased O_2 saturation, and decreased blood pressure [1,7]. Ludemann et al. [1] pointed out that changes in EKG findings may be another predictive indicator of a pneumothorax. In this case, when the hypoxia was observed for the first time, it was suspected that a bronchial intubation was the cause of hypoxia because pnumothorax was not the known complication of LADG. Therefore, a bronchial fiber-optic assessment was done to confirm the position of ETT. After confirmation of correct position of ETT, we assumed that the cause of hypoxia might be atelectasis. For this reason, alveolar recruitment maneuver was performed. When the cardiovascular collapse was occurred, an intraoperative TEE examination was done to detect any possible life-threatening cardiac events. At this moment, we recommended the surgeon to find out any surgical problems. The surgeon reported a diaphragmatic injury. Therefore, it was verified that the sequential changes in the vital signs arose from the pneumothorax secondary to a diaphragmatic perforation.

If a diagnosis of a pneumothorax is made during a laparoscopic procedure, the first-line treatment would be to discontinue the use of nitrous oxide (which can increase the size of the pneumothorax), reduce the intra-abdominal pressure as much as possible, increase the FiO_2 to correct the hypoxia, apply 5 cmH_2O PEEP for the purpose of inflating the collapsed alveoli, and prevent further absorption of CO_2 [6-8]. In addition, because the diffusion coefficient of CO_2 is greater than O_2, CO_2 can be promptly absorbed into the tissue. It can therefore be inferred that thoracostomy or thoracecentesis would be avoided if the patient is in a stable condition [9].

In conclusion, the surgeon should consider the possibility of a pneumothorax caused by injury to the diaphragm during laparoscopy, especially procedures performed around the diaphragm. For the anesthesia provider, it is important to make a prompt determination of the possibility of a pneumothorax, and then perform the appropriate management as promptly as possible. It is also important that anesthesia providers communicate and collaborate with surgeons, which is essential in resolving intra-operative complications as soon as possible.

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