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CLINICAL REPORT

Two cases of massive pleural effusion noted only after induction of anesthesia in living donor liver transplantation

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Abstract Two adult patients who underwent living donor liver transplantation with acute accumulation of right-side pleural effusion are reported. The chest X-ray of patient 1 showed no specific finding 3 days before the operation, and patient 2 was known to have pleural effusion and underwent pigtail drainage before transplant. After anesthesia induction and insertion of central venous catheters, a portable chest radiograph was taken to confirm the positions of the central venous catheters and endotracheal tube. A massive right-side pleural effusion was noted unexpectedly in both patients. Approximately 2,000 ml transudative fluid was surgically drained through the right diaphragm in patient 1 upon opening of the abdominal cavity. The acute accumulation of massive pleural fluid in patient 2 was caused by clamping of the pigtail drainage tube during patient transfer to the operating room; upon unclamping of the tube, 2,000 ml fluid was drained. The intraoperative and postoperative transplant courses of both patients were uneventful. Both were discharged from the hospital in stable condition. Our cases suggest that chest X-ray after induction of the anesthesia and before liver transplantation surgery is recommended. In addition to documenting the positions of the central venous catheters and endotracheal tube, a potential life-threatening pleural effusion requiring appropriate management may be detected.

Keywords Organ · End stage liver disease · Anesthesia · General · Monitoring · Chest X-ray · Surgery · Liver transplant

Introduction

The cost–benefit ratio and usefulness of a preoperative chest X-ray (CXR) are controversial [1]. Joo et al. [2] suggested that routine preoperative CXR should not be performed in asymptomatic patients regardless of age. However, in the setting of liver transplantation, CXR before the operation is necessary [3]. The incidence of pleural effusion in end-stage liver disease patients is high. It is also difficult to determine how many days before the transplant operation the CXR of those patients with pleural effusion can be regarded as representative in terms of being pleural effusion free or having a small effusion not requiring intervention. Herein, we present two cases of massive pleural effusion noted only after induction of anesthesia in the setting of living donor liver transplantation (LDLT).

Case reports

Case 1

A 49-year-old man with end-stage liver disease secondary to primary biliary cirrhosis was scheduled to undergo LDLT. Preoperative liver transplant evaluation was conducted by protocol [4]. Physical examination showed massive ascites. Laboratory results were hemoglobin = 11.2 g/dl,

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hematocrit = 32.9%, and platelet count = 19,000/mm^3. His CXR showed no specific finding 3 days before the planned operation (Fig. 1a). The patient walked from the ward to the operation room on the day of operation without remarkable dyspnea. Anesthesia was induced with fentanyl, propofol, and atracurium. After intubation, both lungs were listened to by stethoscope during breathing to rule out bronchial intubation, and no sounds suspicious of right pleural effusion were heard. Anesthesia was further maintained with isoflurane in an oxygen–air mixture. Fentanyl was given whenever necessary, and atracurium was used as muscle relaxant. Continuous monitoring by ECG, pulse oximetry, arterial blood pressure, central venous pressure, end-tidal CO_2, urine output, and nasopharyngeal temperature was performed. After induction of general endotracheal anesthesia and insertion of the central venous catheters, a routine portable CXR was taken to confirm the positions of the central venous catheters and endotracheal tube. The CXR showed a massive right-side pleural effusion (Fig. 1b). Approximately 2,000 ml transudative fluid was surgically drained through the right diaphragm upon opening of the abdomen. When and how this much pleural effusion accumulated is not determined; anesthesia was started after routine checking of the identification of the patient, without physical auscultation and percussion of the chest. PaO_2 (FiO_2, 0.9) after induction of anesthesia was 239 mmHg, which then decreased to 178 mmHg when pleural effusion was noted; it increased to 278 mmHg after drainage of the pleural effusion. Heart rate and blood pressure of the patient could be maintained within acceptable ranges, even after drainage of the pleural effusion and ascites. Ascites and blood loss were 1,350 and 1,210 ml respectively. The losses were replaced with 1,200 ml 5% albumin, 375 ml leukocyte-poor red blood cells, and 6,715 ml crystalloid. The transplant operation proceeded unremarkably.

The drained pleural fluid was clear in appearance. Analysis of this fluid showed protein = 1.8 g/dl (serum protein, 5.7 g/dl), glucose concentration = 135 mg/dl (serum glucose, 91 mg/dl), and amylase concentration = 57 IU/l (serum amylase, 82 IU/l). The pleural fluid cell count was not increased (red blood cells, 580/mm^3; white blood cells, 240/mm^3; neutrophils, 1%). The cytology report was negative for malignancy, and cultures did not show any bacterial growth. An acute onset of hepatic hydrothorax was entertained as to the cause of the massive unilateral pleural effusion.

A postoperative CXR taken in the liver intensive care unit showed a small right pneumothorax without effusion (Fig. 1c). The postoperative transplant course was uneventful. The patient was discharged from the hospital in stable condition and has had regular follow-up in our hospital to present date.

Case 2

A 62-year-old man with end-stage liver disease secondary to alcoholic liver cirrhosis complicated by massive ascites, esophageal varices bleeding, hepatic encephalopathy, and diabetes mellitus was scheduled for LDLT. Preoperative liver transplant evaluation was performed by protocol [4]. A right-side pleural effusion was noted, and 1,500 ml was subsequently drained using a pigtail tube (Fig. 2a). On the day of the transplant operation, the patient was brought to the operating room with the pigtail tube clamped. Anesthesia induction and maintenance was similar to that described in case 1. After induction of general endotracheal anesthesia, a routine portable CXR was taken to confirm the positions of the central venous catheters and endotracheal tube. A massive right-side pleural effusion was seen (Fig. 2b). It was then noted that the pigtail tube had remained clamped and was not opened when the patient arrived in the operating room and throughout anesthesia induction. Upon unclamping, approximately 2,000 ml fluid was drained from the tube. PaO_2 after anesthesia was 110 mmHg (FiO_2 0.5), after increasing FiO_2 to 0.75, PaO_2 was 206 mmHg, and after drainage of pleural effusion,

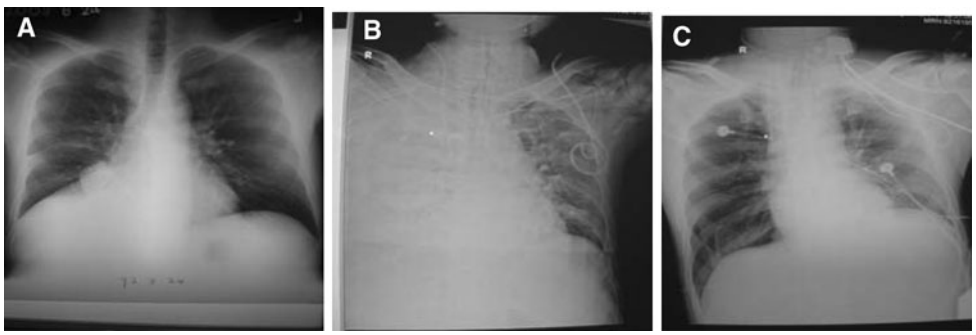

Fig. 1 A 49-year-old male patient with end-stage liver disease secondary to primary biliary cirrhosis. **a** Chest X-ray 3 days before the operation showed no significant pulmonary findings. **b** Chest X-ray in the operating room after induction of anesthesia showed massive right-side pleural effusion. **c** Drained right pleural cavity after transplant operation showed only a small pneumothorax

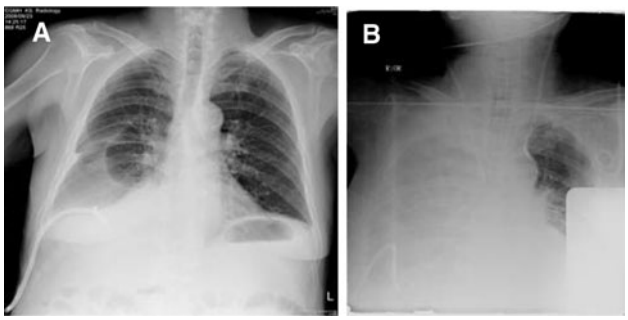

Fig. 2 A 62-year-old male patient with end-stage liver disease secondary to alcoholic cirrhosis **a** Preoperative chest X-ray with pigtail in right lung. **b** Massive right-side pleural effusion was noted after induction of anesthesia with clamped pigtail drain in place

PaO_2 was 266 mmHg with the same FiO_2. Blood and ascites loss were 2,000 and 2,600 ml, respectively; the patient received 2,800 ml 5% albumin, 2,370 ml RBC, and 8,510 ml crystalloids. Despite initial pulmonary concern, both LDLT and postoperative course were uneventful.

Discussion

We presented two cases of massive pleural effusion noted after induction of anesthesia in the liver transplant setting. Three days before the operation, the CXR of case 1 was totally free from pleural effusion; the massive right-side pleural effusion was noted only after a routine CXR to check positions of central venous catheters and endotracheal tube. Similarly, the massive pleural effusion in case 2 was again noted after routine CXR following anesthesia induction. Both cases represent instances where pleural effusion in end-stage liver patients may develop rapidly over a short period of time. The amount of pleural effusion may be massive.

Hepatic hydrothorax in a patient with liver cirrhosis without a primary cardiac or pulmonary disease was first described in the 1950s and defined as significant when the pleural effusion is greater than 500 ml [5]. The incidence of hepatic hydrothorax is approximately 5–12% in patients with liver cirrhosis [3]. It often occurs in the right side, but left-side and bilateral effusions are also reported: frequency is 85% for right-side, 13% for left-side, and 2% for bilateral effusions [3]. Several mechanisms of hepatic hydrothorax are postulated. The most likely cause is direct passage of peritoneal fluid into the pleural cavity via diaphragmatic defects [6], either grossly or microscopically [7]. The other possible causes include passage of fluid from the peritoneal into the pleural cavity via lymphatics, decreased colloid osmotic pressure related to hypoalbuminemia, azygous vein hypertension with plasma leakage, and thoracic duct lymphatic leakage [8].

Patients with hepatic hydrothorax may or may not have ascites [9]. Both our patients had massive ascites: 1,350 and 2,600 ml was drained from the abdomen for case 1 and case 2, respectively. Pleural effusion may limit lung expansion and subsequently cause respiratory syndrome such as dyspnea in a conscious patient, or decrease tidal volume and increase airway pressure. Massive pleural effusion may cause atelectasis, pneumonia, and poor weaning in a ventilator-dependent patient [10]. Severe arterial hypoxemia was reported in a preeclampsia patient with unrecognized pleural effusion when under general anesthesia but who did not manifest dyspnea before the operation. Oxygenation in this patient improved after draining the pleural effusion [11]. Any of the aforementioned respiratory impairments may influence the perioperative LDLT course in patients whose immunity has to be deliberately suppressed by drugs to prevent organ rejection. Therefore, pleural effusion in a transplant candidate should be managed appropriately.

Most pleural effusions resolve with appropriate treatment of the underlying cause. Fluid removal through pigtail drains or chest tube is necessary in patients with massive pleural effusion presenting with respiratory distress [10]. Removal of the effusion was necessary in both our cases to avoid possible intra- or postoperative respiratory complications.

As stated earlier, pleural effusion in end-stage liver patients may develop rapidly over a short period of time. A normal CXR taken a few days before the transplant operation may not be reliable, as exemplified by case 1. Further, in case 2, almost 2 l fluid accumulated within less than 2 h upon clamping of the drainage tube. These observations provide a rationale for obtaining routine CXR after induction of anesthesia and immediately before a long-duration major operation such as liver transplantation in end-stage liver patients.

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