CASE REPORTS

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Tension Hydrothorax during Laparoscopy in a Patient with Ascites

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LAPAROSCOPIC surgery has become an increasingly accepted form of surgical therapy for many disorders. More patients with underlying diseases are undergoing laparoscopy, which presumably imparts less risk to the patient than conventional surgery and allows for faster recovery. As experience with this type of surgery accumulates, so will the complications specific to it.

As this case illustrates, the unique features of laparoscopic surgery, including patient positioning and the use of intraperitoneal carbon dioxide for visualization, may promote the development of tension hydrothorax in patients with ascites and pleural effusions.

Case Report

A 50-yr-old woman, 77 kg in weight and 163 cm tall, with a history of stage III, grade 3 ovarian carcinoma and recurrent malignant ascites was scheduled to undergo a laparoscopic drainage of ascites, lysis of adhesions, and placement of an intraperitoneal Infuse-a-Port (Norwood, MA). The patient's past medical history was notable for ovarian adenocarcinoma, which had been diagnosed 2 yr previously by exploratory laparotomy. She was noted to have extensive intraabdominal metastases and was initially treated with several courses of chemotherapy. The management of her tumor was complicated by the development of recurrent tense ascites and bilateral pleural effusions requiring regular paracentesis and thoracentesis on a weekly basis. Other conditions included hypothyroidism, mild renal insufficiency, and a previous episode of supraventricular tachycardia.

The patient had been admitted to the hospital 2 days before surgery with increasing dyspnea secondary to a large left pleural effusion and tense ascites. Her symptoms were relieved by thoracentesis, and she did not complain of any dyspnea preoperatively. Her postthoracentesis chest radiograph, done 1 day before surgery, demonstrated normal lung fields with a moderate residual left-sided pleural effusion (fig. 1).

On arrival to the operating room, the patient's hemoglobin oxygen saturation was measured at 99% while she was administered oxygen at 4 l/min *via* nasal cannula. Anesthesia was induced by rapid-sequence induction with etomidate and succinylcholine. The trachea was intubated with a 7.0-French endotracheal tube. Initial end-tidal carbon dioxide tension was 38 mmHg, tidal volume 500–700 ml, respiratory rate 8 breaths/min, peak inspiratory pressure 32 cmH_2O, and fraction of inspired oxygen 1.0. Anesthesia was maintained with isoflurane (0.5–2.0%), fentanyl, and vecuronium.

The patient was placed in the dorsal lithotomy position in slight Trendelenburg position for the 45-min procedure. Surgery included a small midline incision through which a laparoscope was inserted and 1.0 l ascites drained. Subsequently, carbon dioxide was instilled into the peritoneal cavity with inflating pressures of 12–14 cmH_2O for better surgical visualization, and lysis of adhesions and placement of a Port-a-Cath in the left colic gutter proceeded.

After induction and throughout the operation, the hemoglobin oxygen saturation intermittently decreased to 88% but with manual

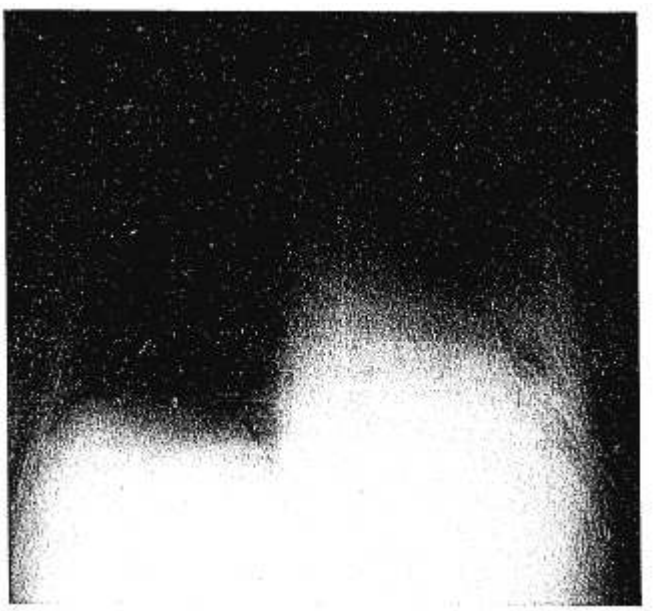

Fig. 1. Preoperative chest radiograph of the patient after thoracentesis, 1 day before surgery.

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ventilation and application of positive end-expiratory pressure could be increased to 93%. Later, the heart rate increased to 145 beats/min, associated with a variable decrease in mean arterial pressure to a nadir of 50 mmHg. During the course of the surgery, a total of 2.2 l ascites was removed and an estimated 50 ml blood was lost. The patient received a total of 500 ml 5% albumin and 2,100 ml crystalloid during the operation.

By the end of surgery, the patient's heart rate was 145 beats/min, with a mean arterial pressure of 50 mmHg, a spontaneous respiratory rate of 30 breaths/min, and an end-tidal carbon dioxide tension of 60 mmHg. Her skin was mottled, with inadequate capillary refill. The patient was initially treated with ephedrine and fluid, and blood pressure increased transiently. She was moved to the postanesthesia care unit with the trachea still intubated, and an arterial blood gas analysis showed a *p*H of 7.08, carbon dioxide tension of 59 mmHg, and oxygen tension of 61 mmHg with a fraction of inspired oxygen of 1.0. The patient again became hypotensive, and a dopamine infusion was started. A chest radiograph demonstrated a massive left pleural effusion with displacement of the mediastinum to the right (fig. 2).

A chest tube placed emergently into the left pleural cavity yielded more than 1.75 l straw-colored fluid under pressure. There was an immediate improvement in the patient's hemodynamics, with an increase in mean arterial pressure of 20 mmHg, a decrease in heart rate, an increase in hemoglobin oxygen saturation, and improvement in peripheral perfusion evidenced by improved capillary refill and disappearance of skin mottling. An arterial blood gas analysis after thoracostomy demonstrated a *p*H of 7.30, carbon dioxide tension of 35 mmHg, and oxygen tension of 172 mmHg with a fraction of inspired oxygen of 1.0. A pulmonary artery catheter placed after the tube thoracostomy demonstrated a cardiac output of 4.2 l/min, central venous pressure of 18 mmHg, and a pulmonary artery occlusion pressure of 17 mmHg. A follow-up echocardiogram demonstrated a very small pericardial effusion without evidence of tamponade.

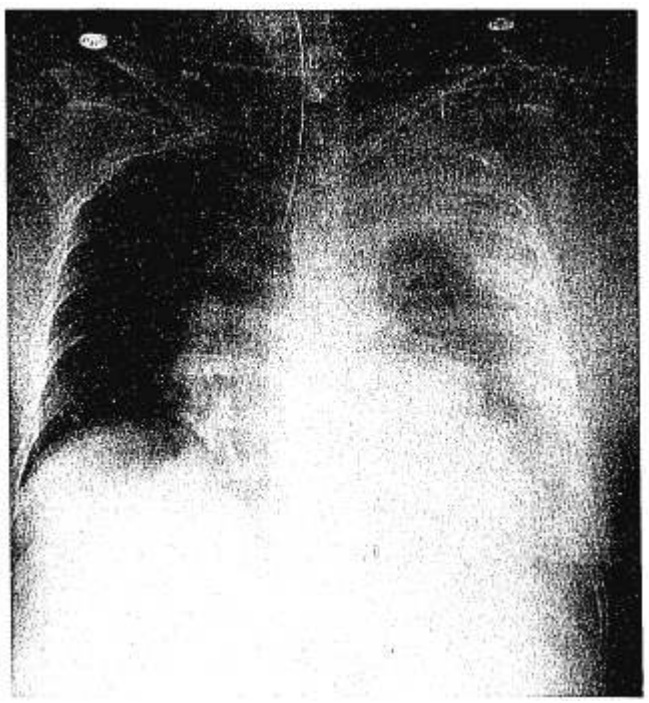

Fig. 2. Postoperative chest radiograph of the patient, demonstrating massive pleural effusion with shift of the mediastinum to the right.

Discussion

This case reveals a noteworthy cause of intraoperative cardiovascular and respiratory deterioration in a patient undergoing laparoscopy. In this patient with malignant ascites, the unanticipated rapid accumulation of pleural fluid caused a tension hydrothorax with deterioration of respiratory and cardiovascular function.

The association of ascites with pleural effusions has been described for many years. In 1937 Meigs and Cass described a syndrome of ascites and hydrothorax in patients with ovarian fibromas.[1] Hydrothorax also is a well-known complication of cirrhosis with ascites.[2,3] The pathogenesis of this type of pleural effusion associated with ascites was not established until 1966, when Lieberman and colleagues demonstrated transmigration of fluid and air through the diaphragm in a series of tests in patients with cirrhosis and ascites complicated by hydrothorax.[4] After draining the hydrothoraces by thoracentesis and injecting radioactive albumin intravascularly and intraperitoneally, they were able to demonstrate a gradient of radioactivity from the peritoneum into the pleural space. They also injected air within the peritoneum and later documented development of pneumothoraces.

Post mortem examinations of some of their patients revealed small diaphragmatic defects and blebs within the tendinous portion of the hemidiaphragm that were determined to be the sites of fluid transmigration. These defects act as one-way valves that favor the movement of fluid from the peritoneum to the pleural cavity as the diaphragm descends and creates a negative intrapleural pressure and a more positive intraperitoneal pressure.[5] In addition, large diaphragmatic defects and diaphragmatic eventration have been described as sites of fluid movement from the peritoneum into the pleural space.[6–9]

As the use of continuous ambulatory peritoneal dialysis has increased, numerous cases of associated pleural effusions and tension hydrothorax have been reported.[10] The mechanism of pleural fluid accumulation in these patients is likely similar to that described in patients with ascites from other causes and in our patient. The empty peritoneal cavity has a pressure of

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0.5–2.2 cmH_2O.[10] Factors known to alter this pressure include weight, age, abdominal girth, and body mass index.[11] Intraabdominal pressure increases linearly in proportion to the intraperitoneal volume.[10] In patients undergoing continuous ambulatory peritoneal dialysis, intraperitoneal pressures usually range between 2 and 10 cmH_2O, to a maximum of 12 cmH_2O with large volume (3-l) infusions.[12]

In our patient, intraperitoneal pressures increased during laparoscopic surgery. After some ascites was drained from her abdomen, a carboperitoneum was produced to permit better intraoperative visualization. In addition, the patient was obese, with a large abdominal girth, and was maintained in slight Trendelenburg position during the procedure. All of these factors caused an acute increase in intraperitoneal pressure that favored the mass movement of fluid across the diaphragm, presumably through a small defect, and into the pleural space despite the use of positive-pressure ventilation.

The consequence of this rapid accumulation of pleural fluid is initially an impairment of respiratory function on the affected side, progressing to respiratory and cardiovascular deterioration due to tension hydrothorax. Increased intraperitoneal pressures cause a decrease in functional residual capacity. Further decrease in functional residual capacity occurs as the lung collapses under increasing intrapleural pressures causing small airway collapse, ventilation–perfusion mismatch, and subsequently arterial hypoxemia.[10,13–15]

As intrapleural pressure increases, cardiovascular function may deteriorate. The increased intrapleural pressure decreases venous return; increases resistance to pulmonary blood flow[14]; and reduces stroke volume, leading to decreased cardiac output, hypotension, and decreased systemic oxygen delivery.[15] These consequences of tension hydrothorax were manifested in our patient by hypotension, hypoxemia, and tachycardia, which were relieved by tube thoracostomy.

The rapid accumulation of pleural fluid under pressure in this patient with ascites was most likely secondary to the position and pressure required to perform laparoscopy. Although tension hydrothorax has been described in association with abdominal fluid under pressure in patients with ascites or undergoing peritoneal dialysis, we are unaware of any previous reports of its occurrence during laparoscopy. A recent case report[16] describes a similar occurrence of tension hydrothorax after hysteroscopy complicated by occult uterine rupture. This report differs from our case because liquid distension media (1.5% glycine) under pressure was directly forced through a uterine defect into the peritoneal and pleural cavities in a patient without preexisting ascites. Hydrothorax developed during laparoscopy in our patient with ascites in an otherwise routine procedure and did not require the occurrence of a complication like uterine rupture. Although the mechanism of transdiaphragmatic flow of fluid is likely similar in both cases, of note is that in one patient a right hydrothorax developed, whereas in the other a left hydrothorax developed. Right hydrothoraces are more common in the setting of transdiaphragmatic flow of fluid from the peritoneum: in two studies, 67% occurred on the right.[4,17] The explanation for this difference is that fluid transmigration occurs through the tendinous portion of the diaphragm, which is mostly covered by heart and pericardium on the left and is relatively uncovered on the right.[10]

Because increasingly large numbers of procedures are being performed laparoscopically, it is important for anesthesiologists and surgeons to recognize tension hydrothorax as a potential complication in any patient with ascites who undergoes abdominal laparoscopy. Otherwise, this potentially life-threatening complication could be easily overlooked.

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Successful Percutaneous Drainage of Epidural Abscess with Epidural Needle and Catheter

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EPIDURAL abscess associated with an indwelling epidural catheter is a rare but serious complication requiring early diagnosis and treatment.[1] Both antibiotic therapy and surgical laminectomy have been successfully used to treat epidural abscess.[2,3] We report two cases in which epidural abscess was successfully treated with percutaneous drainage by epidural needle and catheter.

Case Reports

Case 1

A 72-yr-old man was referred to our pain clinic because of acute herpes zoster with burning pain in the right L2 dermatome. An epidural catheter was inserted at the L3–L4 interspace without difficulty. Bacteriocidal ointment and a sterile dressing were applied and were changed every 2 days. Six milliliters 0.25% bupivacaine was injected 3 times per day through the catheter, which was protected with a micropore filter.

On the 3rd day, the catheter was replaced with one at the L1–L2 interspace because pain relief had been unsatisfactory and because epidurography revealed insufficient cephalad spread of the contrast medium. The next day, the patient complained of low back pain during injection of local anesthetics, and on the 5th day he had a fever of 37.8°C, general fatigue, and low back pain at rest. A subcutaneous abscess was noticed at the catheter insertion site, and compression of the skin caused oozing of a light-yellow fluid containing *Staphylococcus aureus*. The catheter was removed, and cephazolin 3 g/day and fosfomycin 2 g/day were administered intravenously. Fever and swelling subsided by the next afternoon, but the patient still complained of low back pain upon flexing of the neck, and the following day he complained of pain in the left thigh on walking and of motor weakness of the left lower limb without sensory disturbance. Emergency magnetic resonance imaging (MRI) disclosed an epidural abscess in the left posterolateral epidural space at T11–L2.

Percutaneous drainage of the abscess was attempted. A Tuohy needle was inserted into the left posterolateral epidural space *via* the L1–L2 interspace under fluoroscopic control. Intense pain lasting 2 min was felt in the left thigh when the needle reached the epidural space. One milliliter bloody fluid was aspirated through the needle. A second Tuohy needle was inserted *via* the T11–T12 interspace, but nothing emerged upon aspiration. A catheter was threaded 2 cm cephalad into the epidural space through the second needle, but because only 2 ml yellow pus was aspirated, a third Tuohy needle was inserted *via* the T12–L1 epidural space. The first and third needles were positioned so that the bevels of the needles were facing each other, and the epidural space was irrigated with 2 ml saline through the T12–L1 needle, which washed out pus containing blood through the L1–L2 needle. Irrigation with 1–2 ml saline was repeated until the washout became clear, after which all needles and the cath-

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