Respiratory and Circulatory Compromise Associated with Acute Hydrothorax during Operative Hysteroscopy

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OPERATIVE hysteroscopy is a relatively safe procedure with few major associated complications reported. Perforation, hemorrhage, fluid overload, and bowel and urinary tract injuries occurred in less than 1% of procedures in one survey and in less than 3% in another. Complications related directly to uterine distension media occur in less than 4% of procedures and vary according to the medium used. Carbon dioxide may cause sympathetic stimulation, dysrhythmias, or venous air embolism. Dextran 70 (Kabi Pharmacia, Piscataway, NJ) may cause fluid overload or anaphylaxis. Fluids such as sorbitol and glycine can lead to fluid overload, hyponatremia, and, in the case of glycine, the potential for glycine toxicity. We report in this paper a previously unrecognized complication of the use of a fluid for uterine distension: hydrothorax. This complication presented as respiratory distress in a patient undergoing operative hysteroscopy under epidural anesthesia.

Case History

The patient was a 48-yr-old woman with a history of menorrhagia who was scheduled for hysteroscopic resection of uterine fibroids. Her past medical history included depression and a history of a heart murmur. Echocardiography had demonstrated normal valvular anatomy and left ventricular function. She previously had undergone several surgical procedures on the uterus, including a caesarean section, two dilations and curetages, and a diagnostic hysteroscopy. Physical examination was unremarkable and laboratory values and electrocardiogram were normal. The patient had requested epidural anesthesia for her procedure.

On the day of surgery, an epidural catheter was inserted without difficulty via the third lumbar interspace. After a negative test dose, anesthesia was established with a mixture of 2% lidocaine, 0.5% bupivacaine (25 ml total volume) and 100 µg fentanyl. The patient received 2 mg of midazolam during epidural placement and another 2 mg in divided doses during the first hour of the procedure. She was comfortable, hemodynamically stable, and drowsy but spontaneously verbal during this time. Hemoglobin oxygen saturation (SpO₂) was 100% during room air breathing. One hour after the final dose of local anesthesia was given through the epidural catheter and one half hour after the operation had begun, the anesthetic level was at the seventh thoracic level. Fifteen minutes later the patient started to shiver and complain of being cold. These symptoms were attributed to the epidural anesthesia, and this phenomenon was explained to the patient. Warm blankets were placed around her head and shoulders and the room temperature was increased to 22°C, but these measures did not relieve her sensation of cold or her shivering. Fifteen minutes after voicing the aforementioned complaints, the patient stated she was having difficulty breathing. Lung auscultation was unremarkable, the level of sensory anesthesia remained at the seventh thoracic level, and SpO₂ remained 100% while the patient breathed room air. Inquiries to the surgeons elicited the information that they were having "equipment problems, but that otherwise things appeared all right". The patient continued to experience a sensation of dyspnea and then complained of right shoulder discomfort. The surgeons were informed of the patient's symptoms, particularly the complaint of shoulder pain, but again could not provide a cause for the problem. Shortly thereafter, the patient's SpO₂ decreased to 94-96% and her heart rate decreased from 80 to 48. Her systolic blood pressure, which had been in the range of 140 mmHg declined to 110 mmHg. Oxygen (100%) was administered via mask with 0.3 mg intravenous glycopyrrolate. Vital signs and SpO₂ returned to their baseline values. Further inquiries to the surgeons now elicited the information that over 2 l of distention medium (1.5% glycine) were unaccounted for and, given the patient's current symptoms, were presumed to have been absorbed. A Foley catheter was placed and 20 mg intravenous furosemide was administered. A decision was made to perform a laparoscopy. Because of the potential for additional respiratory compromise during this procedure, general anesthesia was induced using 450 mg intravenous thiopental and 100 mg intravenous succinylcholine. After tracheal intubation was completed, breath sounds seemed equal and clear bilaterally, with peak inspiratory pressures of 40-50 cm of water at a tidal volume of 10 ml/kg. Anesthesia was maintained with isoflurane, nitrous oxide, and oxygen. A blood gas sample drawn immediately after induction, while the fraction of inspired oxygen was 0.5 (Pap), revealed an arterial

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oxygen tension of 110 mmHg, an arterial carbon dioxide tension of 46 mmHg, a pH of 7.34, a sodium concentration of 155 mEq/l, a potassium concentration of 3.8 mEq/l, and a hematocrit of 45. Peak inspiratory pressures remained high. Reassuclitation of the lungs revealed decreased breath sounds on the right. The position of the endotracheal tube was unchanged. The FiO₂ was increased to 1.0. A suction catheter passed through the endotracheal tube demonstrated the tube's patency and lack of airway secretions. There were no signs of subcutaneous emphysema. Breath sounds continued to be diminished on the right, and the lungs were becoming increasingly difficult to ventilate. The laparoscopy had been started and intraabdominal pressures had increased transiently to 30–40 cm of water. The surgeons were informed that ventilation was becoming progressively more difficult and that a pneumothorax was suspected, but that it was difficult to rule out an abdominal etiology for the problem without more information from them. A chest x-ray was ordered to rule out pneumothorax. Twenty minutes after induction of general anesthesia, SpO₂ decreased to 96%, while FiO₂ was 1.0. Systolic blood pressure decreased to 90 mmHg. The chest film revealed a large right hydrothorax with mediastinal shift (Fig. 1). A chest tube was placed, and 2200 ml of clear fluid were drained from the right chest cavity. The patient's systolic blood pressure stabilized at 140–160 mmHg after drainage and peak inspiratory pressures decreased to 26–35 cm of water. With FiO₂ still 1.0 and isoflurane at 1%, a blood gas sample drawn. This time, the sample revealed an arterial oxygen tension of 363 mmHg, an arterial carbon dioxide tension of 45 mmHg, a pH of 7.34, a sodium concentration of 135 mEq/l, a potassium concentration of 4.3 mEq/l, and a hematocrit of 44. The surgeons reported a probable perforation in the left cornual region of the uterus, the presence of approximately 500 ml of fluid in the peritoneal cavity, and no apparent damage to other intraabdominal structures. The remainder of the case was uneventful and the patient was exsufflated in the operating room. Follow-up chest film in the postanesthesia care unit revealed drainage of the right hydrothorax, return of the heart and mediastinum to the midline position, full reexpansion of the right lung, and no residual pneumothorax. The chest tube was removed 24 h after it was placed. The patient was discharged from the hospital 48 h after her procedure.

Discussion

Negative fluid balance (i.e., the movement of more fluid in than out) occurs routinely when 1.5% glycine is used for uterine distension in operative hysteroscopy. In one study involving 250 patients, the average volume absorbed by each patient was 480 ml, but 3% of women gained over 2 l of fluid.² Five of seven of these women required treatment for symptoms of acute left heart failure. Decreases in serum sodium and hematocrit were proportional to the amount of fluid absorbed.⁴ No cases of TURP-like syndrome were recorded. Intraoperative fluid balance was influenced by the status of the endometrium, tubal patency, the need for myomectomy, and the duration of surgery. The irrigant was thought to be absorbed via open myometrial vessels or through transstubal loss with rapid peritoneal absorption. Uterine perforation could provide another route by which distension media could gain intraperitoneal access. Given the location of the perforation, we postulate that, in the case presented above, this complication provided a route for the rapid deposition of over 2 l of fluid into the intraabdominal cavity. With the stable hematocrit and serum sodium values, we conclude that most of this irrigant was not absorbed, but ended up in the intrapleural space. How could this have occurred?

Hydrothorax occurring as a result of intraabdominal processes has been documented well in the literature. Mcigs described the syndrome of ovarian fibroma, ascites, and hydrothorax in the 1930s. In a later paper,⁵ he attempted to determine how this fluid crossed the diaphragm and reached the pleural cavity. Although some of his cases had obvious diaphragmatic defects, most did not. In two cases in which India ink was introduced into the ascites, equilibration quickly oc-

Fig. 1. Anteroposterior chest radiograph taken before placement of chest tube, demonstrating right hydrothorax and mediastinal shifted to the left.

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curred between the fluids in the two cavities. No ink was detected in the blood, suggesting to these authors that the passage of fluid was not from abdomen to blood to chest, but that movement was either directly across the diaphragm or via the lymphatics. This question was readdressed in a study that examined the pathogenesis of the hydrothorax, which complicates up to 6% of cases of cirrhosis with ascites. Using radiolabeled albumin, the authors of this study duplicated Meigs' results and also demonstrated that a gradient of radioactivity existed from pleural fluid to thoracic duct. These data, they argued, were evidence against the diaphragmatic and thoracic duct lymphatics as the pathway for the movement of ascites into the pleural space. The authors postulated the existence of a direct passage across the diaphragm. Post mortem examinations performed on nine patients who died with cirrhosis, ascites, and hydrothorax failed to reveal gross diaphragmatic defects. However, two patients demonstrated small (<1 mm) communications when water was introduced under pressure into the abdominal cavity.

Hydrothorax complicating peritoneal dialysis is well documented, and cases of pneumothorax associated with therapeutic pneumoperitoneum, menstruation, and laparoscopy have been described. These phenomena may occur on the right or left sides only, or bilaterally. They may occur in the presence of low or moderate intraabdominal pressures and may be abetted by the negative intrathoracic pressure of the spontaneously breathing patient. In many of the individuals in which these problems have occurred, no major diaphragmatic defect was detectable.

In summary, we have described acute right-sided hydrothorax associated with uterine perforation during operative hysteroscopy. We postulate that the uterine distension medium that was deposited in the intraabdominal cavity moved across microscopic defects in the diaphragm by a combination of the effects of high intraabdominal pressure, negative intrathoracic pressure, and by the slight Trendelenberg's position of the patient. We have not ruled out a congenital right-sided diaphragmatic defect (foramen of Morgagni defect) as the pathway in this case, nor have we eliminated trauma to the diaphragm as a route, although laparoscopy revealed no damage to intervening bowel or liver. Previous evidence suggests, however, that there exists a small but definite number of people who have congenital or acquired microscopic diaphragmatic defects. Given the growing number of hysteroscopic and laparoscopic procedures being performed, an increased appreciation for the possibility of respiratory and cardiovascular compromise from hydro- or pneumothorax in the healthy patient seems warranted.

References
