Congestive heart failure associated with endoscopic cholecystectomy: A case report

KEN SPAIN, CRNA, MSEd
Hutchinson, Kansas

Endoscopic cholecystectomies or laparoscopic cholecystectomies are becoming relatively commonplace in major medical centers throughout the United States. Very few anesthesia complications have been attributed to the procedure. This is a case report of congestive heart failure occurring during the procedure itself, which could have implications for patient management and selection.

Key words: Congestive heart failure, endoscopic cholecystectomy, laparoscopic cholecystectomy.

Review of the literature

With more than 500,000 open cholecystectomies being performed annually in the United States, there is little doubt that the number of endoscopic cholecystectomies will continue to increase nationally. Laparoscopic removal of the gallbladder was first performed in the United States by Saye and McKernan in 1988. That same year, Reddick and Olsen first reported a series of laparoscopic cholecystectomies (LPC).2

Most major hospitals in the United States now have or are working on obtaining equipment and expertise necessary to remove gallbladders endoscopically.

Few complications have been associated with LPC. Only the most recent literature has even hinted at associated complications. A recent abstract did, however, report that LPC was associated with atelectasis and a cephalad shift of the diaphragm; its sample size was 33 ASA physical status I or II patients. All other reports in current literature have been favorable and in contradiction to the previously cited study. Therefore, it must be concluded that, to date, LPC has received a preponderance of favorable reports in the literature.

This is a case report of congestive heart failure that occurred during LPC.

Case history

A 58-year-old male with a history of congestive heart failure (CHF) and coronary artery disease presented for LPC. In 1989, the patient had heart surgery involving seven bypass grafts. Two months later, he also had a cerebrovascular accident, as well as chronic atrial fibrillation, right-sided weakness, and chronic left pleural effusions. A preoperative chest x-ray, taken the day before surgery, showed cardiomegaly with left pleural effusions. The patient's height was 71 inches, and his weight was 80 kg.

Induction was uneventful. The patient was induced with sufentanil 20 µg, etomidate 10 mg, lidocaine 100 mg, and vecuronium 8 mg intravenously (IV). Just prior to incision, the patient developed a rapid ventricular response, which abated after verapamil 2.5 mg was administered. The patient's vital signs were stable throughout the operative course, and he was maintained with sufentanil .5 µg/kg,
vecuronium 0.04-0.06 mg/kg as required, isoflurane titrated at 0.5-1.25%, and 50% oxygen:air.

The total volume of intravenous fluids administered throughout the operative course was 1,200 mL of lactated Ringer's over 4 hours. The procedure lasted 2½ hours; another 90 minutes was spent in induction, emergence, and transport. During the last hour of the case, the patient's urinary output was 40 mL, or ½ mL/kg/hr. Furosemide 5 mg IV was given, and his urinary output increased to 1-2 mL/kg/hr.

When surgery was concluded, the patient was reversed with neostigmine and glycopyrrolate, after exhibiting a strong train-of-four. The patient's eyes were open, he exhibited strong motor movements, and head lift was sustained as extubation was accomplished. One hundred percent oxygen, delivered by face mask, was instituted for about 5 minutes, and the SaO₂ measured 99% by pulse oximeter.

With the patient in the semi-Fowler's position, respiratory effort and the work of breathing were increasing at the time of transport, which was then aborted to further assess and improve his respiratory status. With 100% oxygen and assisted respiration via mask, the patient's SaO₂ deteriorated to 96% over the next 10 minutes. The patient exhibited frothy secretions on expiration, was suctioned several times, and the reintubated after receiving etomidate 10 mg IV.

Breath sounds revealed coarse rales and rhonchi throughout with poor compliance as demonstrated by the feel of the reservoir bag. A tentative diagnosis of pulmonary edema was made. A chest x-ray revealed bilateral pulmonary congestion with a marked increase in the cardiac silhouette; the patient had developed marked CHF.

His transport to the recovery room was uneventful. After arrival in the recovery room, the patient was treated aggressively with furosemide 20 mg IV, and an infusion of dopamine 10 µg/kg/min was begun. A Swan-Ganz catheter was inserted, and the following parameters were obtained: central venous pressure 23 mmHg, right ventricular pressure 58/13 mmHg, pulmonary artery pressure 68/43 mmHg, with a pulmonary artery occlusion pressure of 30 mmHg.

Thirty minutes later, the central venous pressure was 13 mmHg, pulmonary artery pressure 32/18 mmHg, and pulmonary artery occlusion pressure 16 mmHg.

Repeat x-rays showed dramatic improvement 1 hour after intervention and on the following morning. The patient was extubated and had an uneventful course with 100% oxygen via a nonre-breathing mask, which was later replaced with a regular oxygen mask. The patient was discharged from the intensive care unit to the ward without subsequent incidents and returned home without further sequelae.

**Discussion**

Endoscopic cholecystectomies have received mostly favorable reports in the literature. No reports of CHF have been associated with LPC to date.

In view of the fact that the patient's preoperative x-ray was negative for CHF, he received fewer total fluids than the nothing by mouth (NPO) deficit, and he showed no overt clinical signs of CHF immediately preoperatively, the cause of CHF and pulmonary edema can only be hypothesized.

Fluid administration should be scrutinized first. The patient's NPO deficit was 80 kg × 2 mL/hr × 10 hours of NPO status, or 1,600 mL. Hourly replacement was calculated at 3-4 mL/kg/hr, or 240 mL/hr. Accordingly, the patient could receive 1,600 + 960 (240 × 4 hours of surgery) or 2,560 mL. The fact that 1,200 mL were infused over 4 hours (one half the calculated requirements) rules out excess fluid administration as a precipitating cause of the CHF and pulmonary edema; this patient had received less than his NPO requirement over 4 hours, and his preoperative chest x-ray was negative for both CHF and pulmonary edema.

Patient positioning could be another precipitating factor. Prolonged Trendelenberg positioning could have contributed to the CHF and pulmonary edema, because many patients with cardiovascular disease cannot tolerate the increased venous return imposed on their already compromised cardiac output. However, it should be noted that the patient was not placed in a head-down tilt at any time.

Two hours of positive intraperitoneal pressure in a patient with limited cardiac reserve could precipitate CHF. The proposed mechanism is similar to the effects found with the administration of positive end expiratory pressure.9 The cephalad displacement of the diaphragm could encroach on the mediastinum and lungs, resulting in decreased venous return, increased intrapleural pressure, and a secondary reduction in cardiac output. Conceivably, the increased intrathoracic pressure on the pulmonary vasculature could have led to pulmonary hypertension, with transudation or ultrafiltration of fluid, and the resultant pulmonary edema.

The increased pressures on the patient's dia-
phragm and mediastinum could have produced a number of effects. The increased intrathoracic pressures could have caused an increase in pulmonary pressure, or increased mediastinal pressure could have been reflected on the myocardium and caused a decrease in cardiac output similar to that exhibited in cardiac tamponade, where there is a compression of the heart structures. Finally, decreased cardiac output in the presence of increased pulmonary pressures is a universal finding in CHF.

Conclusion

Hypotheses regarding the physiological events precipitating CHF during LPC have been presented. Further reports and studies must be conducted to support or refute the assertions made. Clinicians may wish to exercise caution in selecting patients as candidates for LPC because of the unique and significant physiological burdens that it may impose.

If patient selection for LPC includes those with limited cardiovascular reserve, then perhaps more aggressive hemodynamic monitoring is warranted. Minimally, anesthetists should be cognizant of the potential for development of CHF in such patients.

REFERENCES


AUTHOR

Ken Spain, CRNA, MSEd, received his nursing degree from Washburn University, Topeka, Kansas, and his anesthesia degree from Kansas Newman College in affiliation with the Wichita Clinic School of Anesthesia. In addition, he also has an MSEd from the University of Kansas and is a staff anesthetist with Hutchinson Anesthesia Associates, PA, in Hutchinson, Kansas.

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