

ACUTE CONGESTIVE HEART FAILURE AFTER LAPAROSCOPIC CHOLECYSTECTOMY: A CASE REPORT

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Compared with open procedures, laparoscopic surgery is safe with a low incidence of complications. In rare circumstances, however, intraoperative complications such as acute pulmonary edema have been reported. The patient described herein is a 59-year-old woman with obesity, gastroesophageal reflux disease, and chronic obstructive pulmonary disease who developed acute congestive heart failure (CHF) and cardiomegaly immediately following laparoscopic cholecystectomy. She required emergent reintubation, diuresis, and admission to the intensive care unit for postoperative mechanical ventilation.

Potential causes of pulmonary edema associated with laparoscopic surgery (extreme Trendelenburg position, venous carbon dioxide embolism, absorption of crystalloid irrigation fluid, cardiopulmonary disease, adverse

drug reactions, negative pressure [postobstructive pulmonary edema]) were considered. A process of exclusion revealed that the hemodynamic changes induced by insufflation with an intra-abdominal pressure of 20 mm Hg were the most likely causes of the CHF. Suggestions to prevent occurrence of CHF are tight control of hemodynamics with use of invasive monitoring in high-risk patients and gentle, slow insufflation of the abdomen to an intra-abdominal pressure of 15 mm Hg or less. Intraoperative and/or postoperative CHF should be treated with diuretics, intravenous nitroglycerin, arterial vasodilators, and/or inotropic agents as needed.

Key words: Cholecystectomy, congestive heart failure, laparoscopic surgery, pulmonary edema.

The popularity of laparoscopic surgery has greatly increased during the last few years. Compared with open procedures, laparoscopic surgery is associated with less tissue trauma, a shorter recovery time, and improved postoperative respiratory muscle function. Respiratory muscle function returns more rapidly than in open surgery, particularly in upper abdominal procedures such as laparoscopic cholecystectomy.¹ Although rare, pulmonary edema during laparoscopic surgery has been cited in the literature.²⁻⁵ The present report is about a patient with no cardiac history who developed acute congestive heart failure (CHF) with severe respiratory compromise after a laparoscopic cholecystectomy. The patient required emergent reintubation in the postanesthesia care unit (PACU) and admission to the intensive care unit for postoperative ventilation and aggressive diuresis.

Case summary

The patient was a 59-year-old woman who was admitted as a surgical day patient for a laparoscopic cholecystectomy. Her medical history included obesity, gastroesophageal reflux disease (GERD), and chronic obstructive pulmonary disease (COPD). The patient

was 162.6 cm tall, weighed 112 kg, and had a body mass index of 40 (morbid obesity). The COPD was secondary to a 63-pack-year smoking history. She quit smoking 10 months before surgery. The patient stated that she usually felt short of breath and tired after climbing 1 flight of stairs. She also reported occasional use of an albuterol inhaler and was never hospitalized for COPD. She denied having any chest pain, chest pressure, diabetes, and a family history of cardiac disease. A preoperative electrocardiogram (ECG) showed normal sinus rhythm, no left ventricular hypertrophy, no axis deviation, and no evidence of previous infarction. The preoperative chest roentgenogram displayed clear lungs without infiltrates and normal heart and pulmonary vasculature.

Before arriving in the preoperative holding area, the patient received the institution's standard preoperative medications for GERD prophylaxis, which consisted of metoclopramide, 10 mg, and ranitidine, 150 mg, both by mouth. The patient was very anxious and received 5 mg of diazepam orally, 1 hour preoperatively, with minimal effect. While in the preoperative holding area, an 18-gauge intravenous catheter was inserted. To provide adequate anxiolysis, 2 mg of midazolam were administered intravenously with good effect. She was

calm and cooperative and only mildly sedated. Because the patient was obese, she was preoxygenated with use of a simple facemask at 6 L/min. The patient had been intubated easily according to past anesthetic records, and physical airway assessment revealed that she had a visible soft palate and uvula. Since the patient had tolerated morphine for past anesthetics and required more narcotics for past procedures than other patients, intravenous morphine (10 mg total) was titrated for preemptive analgesia. The patient was noted to have clear lungs to auscultation with a continuous oxygen saturation of 98% to 99%. Sodium citrate, 30 mL, was administered 5 minutes before the patient was taken into the operating room.

In the operating room, the patient received a smooth, rapid-sequence induction with propofol, a defasciculating dose of rocuronium, and succinylcholine. The patient was intubated without difficulty, with an oxygen saturation of 100% and a clear pharynx. Anesthesia was maintained with 1% isoflurane and 50% nitrous oxide/50% oxygen. When the neuromuscular block from the succinylcholine resolved, rocuronium was administered before abdominal insufflation.

During laparoscopic insufflation, the surgeons had difficulty creating a pneumoperitoneum (adequate to conduct the procedure) owing to the patient's obese abdomen. The surgeons increased the flow on the insufflator to 40 L/min, with a resultant intra-abdominal pressure (IAP) of 20 to 23 mm Hg, to improve visualization. The IAP was stable at 20 mm Hg for the remainder of the case.

The patient required a respiratory rate of 12 with an inspiratory:expiratory ratio of 1:1.5 and a tidal volume of 750 mL to maintain an end-tidal carbon dioxide of 35 torr and a peak inspiratory pressure of 30 cm H₂O. After a stable anesthetic and a 40-minute surgical course, the patient received a full muscle relaxant reversal with neostigmine. She exhibited 4 twitches and a sustained tetanus after reversal. Since the patient was obese and had GERD, she was not extubated until she was fully awake with a positive gag reflex. She opened her eyes to verbal command with sustained head lift. Before extubation, the patient had an SaO₂ of 97% and an end-tidal carbon dioxide of 45 torr (owing to the respiratory depressant effects of general anesthesia and underlying COPD history) with adequate respiratory mechanics (a respiratory rate of 15 breaths per minute, a tidal volume of 400 to 500 mL, and a negative inspiratory force of -33 cm H₂O). During awakening, nitroglycerin paste was applied to the patient's chest wall because the systolic blood pressure was trending upward from the 120s to the 160s (in mm Hg). After extubation, the patient

received flumazenil, 0.1 mg, administered twice to reverse the benzodiazepines administered preoperatively, to optimize the patient's laryngeal reflexes and respiratory drive. The patient was awake and responding appropriately; a nonbreather 100% facemask was used with the head of the stretcher at 45° with an SaO₂ of 97% before transport to the PACU.

Several minutes later, while in the PACU, the patient suddenly verbalized feeling short of breath and developed acute respiratory distress with a respiratory rate of 32 breaths per minute and an SaO₂ of 71%. The patient went from calm and responding appropriately to restless and unresponsive, requiring assisted ventilation of 100% oxygen via bag valve mask. Because the patient was in acute distress, a trial dose of naloxone, 0.04 mg (to aid in a differential diagnosis for the cause of the respiratory failure), was administered over 1 minute without improvement. The blood pressure (160-170/80s, in mm Hg) and heart rate (88-100 beats per minute) did not change after the naloxone was administered. To ensure adequate ventilation and airway protection, the patient received propofol and succinylcholine and was intubated immediately. A copious amount of frothy pink-tinged sputum was noted in the endotracheal tube, and rales were auscultated throughout all lung fields.

Results of a chest roentgenogram were consistent with CHF and revealed cardiomegaly. STAT arterial blood gas measurements showed a pH of 7.27, a PaCO₂ of 61, and a PaO₂ of 421 on 100% FIO₂. No changes were noted on the postoperative ECG when it was compared with the preoperative ECG. The patient's primary care physician was notified, and further consultations were obtained from pulmonary and cardiology medicine.

The patient's respiratory status improved with diuretic administration (furosemide, 40 mg) and she was weaned from the ventilator and extubated 3 hours postoperatively, based on improved clinical status, in the intensive care unit. The next day, a myocardial infarction was ruled out by enzymes and ECG. An echocardiogram revealed no cardiac abnormalities and an ejection fraction of 65%. A subsequent chest film revealed no cardiomegaly or CHF. The patient was discharged to home with inhalers and instructions for pulmonary and cardiology follow-up. An outpatient stress test 1 month postoperatively revealed no ischemic ECG changes, no chest pain, no significant arrhythmias, and normal variations in heart rate and blood pressure.

Discussion

Pulmonary edema after laparoscopic cholecystectomy

is a rare occurrence, reported in fewer than 1% of patients.² Reported causes of pulmonary edema related specifically to laparoscopic surgery, as cited in the literature, include extreme Trendelenburg positioning,³ venous carbon dioxide embolism from direct insufflation of gas into a vein,⁴ and fluid overload from absorption of crystalloid irrigation fluid.⁵ In this particular case, the aforementioned causes of pulmonary edema were ruled out. On the contrary, the patient was in reverse Trendelenburg position during the procedure. There were no clinical signs of venous carbon dioxide embolism (millwheel murmur, hypotension, tachycardia, arrhythmias, oxygen desaturation, and decreased carbon dioxide by capnography), which would most likely manifest perioperatively instead of postoperatively. Doppler ultrasound was not used during the procedure to detect air embolism. A millwheel murmur was not noted on auscultation by precordial stethoscope at any time during the procedure. The intake and output of crystalloid irrigation fluid was monitored carefully, and an excess of absorption of the irrigation fluid was not noted. Because the patient had nothing by mouth for 14 hours before surgery, the fluid deficit was partially replaced with 1,300 mL of intravenous lactated Ringer's solution over a 1.5-hour period.

Other causes of pulmonary edema considered were underlying cardiopulmonary disease, adverse drug reactions, negative pressure (postobstructive pulmonary edema), and physiological changes induced by insufflation. The medications the patient received preoperatively, without signs of an adverse reaction, were diazepam, midazolam, morphine, and cefazolin. Intraoperatively, the patient received succinylcholine, rocuronium, ketorolac, neostigmine, and glycopyrrolate. Postoperatively, once the patient developed signs of respiratory difficulty, naloxone was administered, but no improvement occurred.

Prough et al⁶ reported that 2 healthy teenagers developed pulmonary edema after receiving general anesthesia and various medications. The narcotics were antagonized with intravenous naloxone. This case report⁶ did not show a direct cause-and-effect relationship between pulmonary edema and naloxone because multiple medications combined with general anesthesia were administered. Specific details of postoperative airway management were not discussed in either case. For example, the possibility of postobstructive pulmonary edema was not considered. One of the 2 patients, after an open appendectomy and prior to extubation, received naloxone, 100 µg, intravenously for pinpoint pupils and apnea. After extubation, in the recovery room, the patient developed

tachypnea (24 breaths per minute) and tachycardia (140 beats per minute) and appeared to be having minor symptoms of respiratory distress. Ten minutes later, he received additional naloxone, 100 µg intravenously and 300 µg intramuscularly, for continued lethargy. Supplemental oxygen was not administered until 1 hour postoperatively, when an arterial blood gas revealed a pH of 7.31, a PCO₂ of 49, and a PaO₂ of only 32 mm Hg. Perhaps prolonged hypoxia from inadequate postoperative oxygen supplementation was the cause of the teenager's pulmonary edema and was not the direct result of naloxone administration.

Michaelis et al⁷ reported 2 cases in which naloxone, 0.1 to 0.4 mg, was administered to reverse morphine anesthesia (morphine/N₂O technique) after tricuspid valve and aortic graft surgery. Both patients immediately developed ventricular fibrillation or tachycardia after receiving naloxone. The issue at hand is that open-heart surgery patients usually are high risk (ASA physical status III-IV) and are undergoing invasive and painful surgery. Naloxone acutely reversed a primarily morphine-based anesthetic, which most likely caused severe pain, theorized to release massive amounts of endogenous catecholamines⁸ and caused the patients to experience cardiac arrest. Therefore, the case study demonstrates that naloxone itself probably is not a direct cause of adverse cardiac events but rather introduces acute, severe pain to already compromised patients, which leads to poor outcomes.

Naloxone has been administered many times in much higher doses with an impressive safety record.⁹ In another case, 9 patients were admitted to the Regional Poisoning Treatment Centre, Edinburgh, Scotland, with narcotic overdose and induced coma. Naloxone had been administered to the 9 unconscious patients in doses of 0.4 to 1.2 mg; results were therapeutic, and there were no adverse effects.¹⁰ In a case study of 2 infants with opioid poisoning, naloxone was titrated to effect in repeated doses as high as 0.4 to 0.5 mg, along with a continuous infusion of 0.4 to 0.5 mg/h, with positive results and no adverse effects.¹¹ In a case of a 2-year-old with diphenoxylate hydrochloride-atropine (Lomotil) poisoning, naloxone, 20 mg, was administered accidentally, and there were no adverse effects.¹²

In the present case, however, the naloxone dose of 0.04 mg was so small that it could not have fully reversed the opioid agonist effects of the morphine and induce severe pain. The patient's vital signs did not change after naloxone was administered. Also of importance, laparoscopic cholecystectomy surgery is not nearly as invasive and painful as the aforementioned open-heart procedures, and the naloxone was

administered after the patient developed true signs of distress. Therefore the naloxone, most logically, was not the cause of the pulmonary edema.

Inspiring against a closed glottis transmits negative intrathoracic pressure to the alveoli, causing fluid to shift from the pulmonary capillaries into the alveoli. The result is negative pressure (postobstructive) pulmonary edema. This was not a likely factor in the present case because there was no airway obstruction from a kinked or blocked endotracheal tube, no obvious airway swelling (stridor) after extubation, and no laryngospasm. The patient was extubated awake with full laryngeal reflexes present. Also of note, negative inspiratory pulmonary edema usually resolves without treatment within 24 hours.¹³ The results of the chest roentgenogram were consistent with cardiomegaly and CHF, which would not be the case if negative inspiratory pulmonary edema were the causative factor.

The patient in the present case had not received a previous diagnosis of hypertension or cardiac disease. Based on the patient's negative cardiac history, her elevated blood pressure (140/90 mm Hg) on the preoperative screening day and in the operating room before induction (180/98 mm Hg) probably was related to stress and anxiety ("white coat" syndrome); therefore, she required more than the average amount of preoperative benzodiazepines and narcotics. The patient reported vague symptoms of shortness of breath, which correlated with a long-standing history of obesity and COPD.

It was considered that the patient might have been developing cardiac symptoms that could have been masked by obesity and COPD. Women, compared with men, are known to develop different symptoms of cardiac disease that often are missed clinically. Women may have a higher incidence of noncardiac symptoms and/or atypical chest pain. For instance, women experience symptoms such as pain at rest and during sleep, neck pain, shoulder pain, abdominal pain, nausea, and vomiting.¹⁴ Diagnosis of cardiac disease in women also is clouded by the fact that, compared with men, women who develop coronary artery disease usually are older and have more comorbid factors.¹⁵ The woman in the present case was not diagnosed with coronary artery disease and did not have a family history of coronary artery disease, but did have risk factors such as obesity, smoking, and COPD. In this case, a myocardial infarction was ruled out, and cardiomegaly was not seen on the chest roentgenogram the next day. The echocardiogram showed an ejection fraction of 65% with no cardiac abnormalities. Also, 1 month postoperatively, the patient underwent an exercise tolerance test that was negative for ischemia.

Other causative factors of CHF could be related to the physiological changes from laparoscopic insufflation. There are several reported physiological changes from carbon dioxide insufflation related to increased IAP (>15 mm Hg). Pulmonary compliance and functional residual capacity decrease. Mean arterial pressure (MAP), systemic vascular resistance (SVR), and afterload increase. During insufflation, preload, coronary perfusion, and the cardiac index (CI) decrease. The cardiac output (CO) diminishes with an IAP of more than 15 mm Hg.¹² When the abdomen is desufflated, the preload is increased. Healthy patients often tolerate these physiological changes.¹⁶ Westerband et al,¹⁷ using cardiac radiography, reported a decreased CI and an increased SVR of 79% at an IAP of 15 mm Hg. The degree of decrease of CI was proportionate to the insufflation pressure. In a study of cardiac patients by Hein et al,¹⁸ the CI dropped 60% to 65% following carbon dioxide insufflation but returned to baseline when the abdomen was desufflated. Three of 17 cardiac patients in that study required nitroglycerin owing to increased MAP and SVR. One patient required dobutamine to maintain a CI greater than 1.5 L/min per square meter. Hein et al¹⁸ concluded that insufflation and head-up positioning significantly reduced the CI and increased MAP, SVR, and pulmonary artery occlusion pressure. Feig et al¹⁹ studied patients with cardiac and pulmonary disease who underwent laparoscopic surgery. Of 15 patients studied, 9 required intraoperative nitroglycerin, and 4 of 15 required the surgeon to decrease the insufflation pressure from 15 to 10 mm Hg. The CO decreased proportionately with the length of time insufflated. In a study of healthy patients, Joris et al²⁰ noted a 50% reduction in the CI and a 65% increase in SVR following carbon dioxide insufflation with a gradual return to normal 30 minutes after desufflation. Despite the hemodynamic alterations in all of the mentioned studies, there were no reported intraoperative or postoperative cardiorespiratory complications such as CHF. The results of these studies may be reflective of tight control of hemodynamic parameters by using invasive monitoring such as pulmonary artery catheters, the administration of nitroglycerine and dobutamine, and decreasing IAP as needed.

After summarizing the details of this case, it was determined through a process of exclusion that the primary cause of CHF was overinsufflation through the laparoscope with an IAP of greater than 20 mm Hg during the procedure. Studies on dogs by Ishizaki et al²¹ demonstrated a significant decrease in CO with a significant increase in SVR at an IAP of 16 mm Hg. The investigators reported no significant hemody-

dynamic changes at an IAP of 8 to 12 mm Hg. The findings of this study may correlate to humans undergoing laparoscopic procedures in clinical practice.²¹ The patient described in the present case study had an increase in systolic blood pressure of 40 points when desufflated and developed CHF with cardiomegaly. She may have developed ischemia and/or a stunned myocardium during insufflation, which further caused the SVR to increase and the CO to fall. The increased preload from desufflation would not be well tolerated if ventricular function were impaired.

This theory of a stunned myocardium from increased SVR supports the recent findings of a study by Rist et al,²² who evaluated the right and left end-systolic areas, end-diastolic areas, and ejection fractions with use of transesophageal echocardiography in healthy gynecological patients who were ASA physical status I or II. The left and right ventricular end-diastolic areas increased by 65% and 45%, respectively, and ejection fractions decreased by 18% in these healthy patients. Increases in afterload caused by a combination of humoral factors such as vasopressin release along with an increased IAP from pneumoperitoneum can contribute to global impairment of ventricular shape and function. Rist et al²² concluded that global increases in end-systolic dimensions could decrease biventricular function. Although reverse Trendelenburg position was shown to improve ventricular end-diastolic dimensions compared with Trendelenburg positioning, increasing IAP up to 15 mm Hg demonstrated significant decreases in ventricular function.

Anesthesia providers should be aware of these potential cardiovascular changes, which can occur with an increased IAP, and collaborate with surgeons if the patient's cardiac and/or respiratory status becomes compromised. To reduce the risk of CHF, surgeons should use lower insufflation pressures to achieve an IAP of 15 mm Hg or less. If the condition of the patient becomes compromised, desufflation to a lower IAP is an appropriate intervention. If the patient does not tolerate insufflation and visualization is a problem, changing to an open procedure is necessary. Invasive monitoring to provide interpretation of hemodynamic parameters along with arterial blood gas sampling may be useful during laparoscopic procedures in high-risk patients.

An indwelling urinary catheter should be placed for urine output measurement. Pharmacological treatment such as intravenous nitroglycerine, arterial vasodilators (nitroprusside and hydralazine), and inotropic agents (dobutamine, milrinone, and digoxin) may be helpful in high-risk patients. Anes-

thesia technique should consist of gentle awakening, with tight control of blood pressure and heart rate, especially in high-risk patients. Morphine may be beneficial for its venous dilatation effects, which decrease preload, thus minimizing CHF. In this particular case, the patient was extubated awake owing to a risk of aspiration because of GERD and obesity. Extubating patients when they are fully awake can make it more difficult to control blood pressure and heart rate.

Future research should expand on past studies by using larger samples and maintaining tighter control of variables. For example, a standardized IAP with blinded treatment protocols, control groups, and randomization of all patients (ASA physical status I-IV) undergoing laparoscopic cholecystectomy should be used. This may provide insight into the degree of hemodynamic changes and the efficacy of the aforementioned interventions.

Laparoscopic surgery is a safe and effective surgical intervention with many advantages over open procedures. Careful surgical and anesthetic technique, thorough preoperative screening, and collaboration with primary care physicians to medically optimize the condition of patients before surgery are essential to positive postoperative outcomes.

REFERENCES

1. Rovina N, Bouros D, Tzanakis N. Effects of laparoscopic cholecystectomy on global respiratory muscle strength. *Am J Respir Crit Care Med.* 1996;153:458-461.
2. The Southern Surgeons Club. A prospective analysis of 1518 laparoscopic cholecystectomies. *N Engl J Med.* 1991;324:1073-1078.
3. Stoelting RK. Acute pulmonary edema during anesthesia and operation in a healthy young patient. *Anesthesiology.* 1974;33:366-369.
4. Desai S, Roaf E, Liu P. Acute pulmonary edema during laparoscopy. *Anesth Analg.* 1982;61:699-700.
5. Healzer JM, Nezhat C, Brodsky JB, Brock-Utne JG, Seidman DS. Pulmonary edema after absorbing crystalloid irrigating fluid during laparoscopy. *Anesth Analg.* 1994;78:1200-1207.
6. Prough DS, Roy R, Bumgarner J, Shannon G. Acute pulmonary edema in healthy teenagers following conservative doses of intravenous naloxone. *Anesthesiology.* 1984;60:485-486.
7. Michaelis LL, Hickey PR, Clark TA. Ventricular irritability associated with the use of naloxone hydrochloride. *Ann Thorac Surg.* 1974;18:608-614.
8. Pallasch TJ, Gill CJ. Naloxone-associated morbidity and mortality. *University of Southern California School of Dentistry.* 1981;52:602-603.
9. Chamberlain JM, Klein BL. A comprehensive review of naloxone for the emergency physician. *Am J Emerg Med.* 1994;12:650-657.
10. Evans LEJ, Swainson CP, Roscoe P, Prescott LF. Treatment of drug overdose with naloxone, a specific narcotic antagonist. *Lancet.* 1973;1:452-455.
11. Tenebein M. Continuous naloxone infusion for opiate poisoning in infancy. *J Pediatr.* 1984;105:645-648.
12. Rumack BH, Temple A. Lomotil poisoning. *Pediatrics.* 1974;53:495-500.
13. Gotta AW, Ferrari LR, Sullivan CA. Anesthesia for ENT surgery. In: Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia.* Philadelphia, Pa: Lippincott-Raven; 1997:931.

14. Goldberg RJ, O'Donnell C, Yarzebski J, Bigelow C, Savageau J, Gore JM. Sex differences in symptom presentation associated with acute myocardial infarction: a population-based perspective. *Am Heart J*. 1998;136:189-195.
15. Birdwell BG, Herbers JE, Kroenke K. Evaluating chest pain: the patient's presentation style alters the physician's diagnostic approach. *Arch Intern Med*. 1993;153:1991-1995.
16. Vierra MA, Howard SK. Laparoscopic general surgery. In: Jaffe RA, Samuels SI, eds. *Anesthesiologist's Manual of Surgical Procedures*. Philadelphia, Pa: Lippincott Williams & Wilkins; 1999:418-421.
17. Westerband A, Van De Water JM, Amzallag M. Cardiovascular changes during laparoscopic cholecystectomy. *Surg Gynecol Obstet*. 1992;175:535-538.
18. Hein HA, Joshi GP, Ramsay MA, et al. Hemodynamic changes during laparoscopic cholecystectomy in patients with severe cardiac disease. *J Clin Anesth*. 1997;9:261-265.
19. Feig BW, Berger DH, Dougherty TB. Pharmacologic intervention can reestablish baseline hemodynamic parameters during laparoscopy. *Surgery*. 1994;116:733-741.
20. Joris JL, Noirot DP, Legrand MJ, Jacquet NJ, Lamy ML. Hemodynamic changes during laparoscopic cholecystectomy. *Anesth Analg*. 1993;76:1067-1071.
21. Ishizaki Y, Bandai Y, Shimomura K, Abe H, Ohtomo Y, Idezuki Y. Safe intraabdominal pressure of carbon dioxide pneumoperitoneum during laparoscopic surgery. *Surgery*. 1993;114:549-554.
22. Rist M, Hemmerling TM, Rauh R, Siebzehnruhl E, Jacobi KE. Influence of pneumoperitoneum and patient positioning on preload and splanchnic blood volume in laparoscopic surgery of the lower abdomen. *J Clin Anesth*. 2001;13:244-249.

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