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Case Report

Rapid Bradycardic Arrest Upon Induction of Pneumoperitoneum During Laparoscopic Cholecystectomy

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Abstract

Bradycardia is a common occurrence during laparoscopic surgery, which can result from peritoneal distension with pneumoperitoneum leading to a vagal response. However, severe bradycardia leading to cardiac arrest is much rarer. While bradycardic arrest from pneumoperitoneum is documented in a small number of case reports, there remains no standardized guidelines in management after cardiac arrest. Here we present a case of a patient who experienced rapid onset bradycardia which progressed to cardiac arrest after abdominal insufflation during laparoscopic cholecystectomy. She had a Return of Spontaneous Circulation (ROSC) after Cardiopulmonary Resuscitation (CPR), and her initial operation was aborted. A few days later and after a cardiac workup, we proceeded with a modified perioperative and surgical plan which resulted in an uneventful laparoscopic cholecystectomy. Our case highlights considerations in developing a plan that can be implemented after pneumoperitoneum induced bradycardic arrest during laparoscopic cholecystectomy.

Introduction

Bradycardia is a known phenomenon during laparoscopic surgery, thought to be secondary to vagal response [1,2]. There is no clear estimate of the incidence of bradycardia during laparoscopy but multiple case series report a range of 14% - 30% [3]. Even rarer however, is bradycardia leading to hemodynamically significant cardiac arrest, for which the incidence and mortality rates are unknown [2]. Broadly speaking, the incidence of mortality for a standard laparoscopic cholecystectomy is reported to be 0.1% - 0.7%, and low pressure compared to standard pressure pneumoperitoneum does not seem to affect those rates [4-6]. A review of the literature demonstrated that there are very few cases reported on bradycardic arrest in laparoscopic cholecystectomy. Amongst these cases, there was much variability in the post-cardiac arrest management which ranged from re-insufflation to complete cholecystectomy on the index operation, conversion to open, cancellation of the operation with interval cholecystectomy, to insertion of a pacemaker. We discuss a case of a patient without comorbidities who was scheduled for a routine laparoscopic cholecystectomy for acute cholecystitis. Intraoperatively, within a minute of peritoneal insufflation she had rapidly declining bradycardia which progressed to asystolic cardiac arrest within seconds. Brief CPR was performed with ROSC. While the bradycardia was suspected to be due to a hyperactive vagal response to pneumoperitoneum, the decision was made to abort the operation. The patient underwent a cardiac workup which demonstrated no cardiac abnormalities, subsequently underwent an uneventful interval laparoscopic cholecystectomy on the same admission, and was discharged home the next day.

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Case Presentation

A 47-year-old-female with no significant medical history and a surgical history of two Cesarean sections presented to the hospital with symptoms of postprandial, colicky epigastric pain. She was afebrile with Heart Rate (HR) in range of 60 to 100 Beats Per Minute (bpm) and had a Blood Pressure (BP) of 143/86 mm Hg, saturating 96% on room air. Exam was notable for minimal abdominal tenderness to the epigastrium and right upper abdominal quadrant without peritoneal signs. Labs were notable for total bilirubin of 0.9 mg/dL, direct bilirubinemia to 0.5 mg/dL and aspartate aminotransferase of 352 (U/L), alanine aminotransferase of 216 (U/L), alkaline phosphatase of 117 (U/L), and lipase of 39 (U/L). A right upper quadrant abdominal ultrasound showed cholelithiasis, borderline wall thickening and trace pericholecystic fluid. She had a working diagnosis of acute cholecystitis with possible choledocholithiasis given the transaminitis. A magnetic resonance cholangiopancreatography showed no evidence of choledocholithiasis and the decision was made to proceed with laparoscopic cholecystectomy for acute cholecystitis on hospital day one.

The patient was taken to the operative room and general anesthesia was induced with midazolam, fentanyl, propofol, lidocaine 2%, and rocuronium. Endotracheal intubation was performed with a 7 mm tube. The patient's vital signs in peri-induction period were noted to be HR of 60 bpm - 80 bpm, BP of 148/80 mmHg, pulse oximetry (SpO2) of 100%, end tidal CO_2 ($EtCO_2$) of 30 mmHg. The open Hasson technique was used to enter the peritoneal cavity and entry was obtained without difficulty. The initial intraperitoneal pressure was 6 mmHg and insufflation was continued with a flow rate of 20 L/min until a pressure of 15 mmHg was obtained at 17:16. A laparoscope was inserted and no injury was seen as a result of abdominal entry. The patient's vital signs were normal and stable upon initiation of

insufflation with BP 131/84 mmHg, HR of 68 bpm, SpO_2 100% and $EtCO_2$ 30 mmHg. After only one minute of insufflation with the insufflation pressure maintained at 15 mmHg, the patient suddenly became bradycardic to 39 bpm. Pneumoperitoneum was immediately evacuated; however, within only five seconds the patient rapidly progressed to asystole despite evacuation of pneumoperitoneum at 17:17. The patient became pulseless and CPR was initiated following ACLS protocol. The patient received CPR with one dose of epinephrine 1mg and ROSC was achieved at 17:18. The patient's vital signs at this time were noted to be BP of 162/125, HR of 150, SpO_2 100% and $EtCO_2$ 24 mmHg. The decision was made to abort the procedure. An arterial line was placed and sugammadex was administered. The patient remained normotensive without acute electrocardiographic or neurologic changes and was extubated 23 minutes after ROSC was achieved.

Post-operatively, a cardiology evaluation included an unremarkable echocardiogram, as well as a coronary computed tomography angiogram which showed no evidence of coronary stenosis or plaque. Given the patient's negative medical history, normal cardiac workup and uneventful postoperative course, we concluded that the etiology was a hypersensitive vagal reaction triggered by the peritoneal stretch during induction of pneumoperitoneum. Extensive discussions were held with the patient regarding the next steps, including the options of re-attempting laparoscopic cholecystectomy, proceeding directly to an open cholecystectomy, or pursuing non-operative management with or without percutaneous cholecystostomy. The patient had persistent pain and elected to proceed with another attempt at laparoscopic cholecystectomy.

To optimize the patient for a second attempt at laparoscopic cholecystectomy, steps were taken to minimize the risk and severity of a vasovagal response. Pre-operatively, the patient received maintenance Intravenous (IV) fluids for three days to ensure adequate hydration. The patient returned to the operating room on postoperative day four. Upon induction, glycopyrrolate 0.2 mcg was administered followed by a second dose five minutes later. The patient's heart rate remained in the 90 bpm - 100 bpm range throughout induction. Upon abdominal entry, insufflation was initiated and maintained at a flow rate of 4L/min (instead of the prior rate of 20L/min) with a set pressure limit reduced to 10 mmHg (rather than the previous pressure of 15 mmHg). The patient's vital signs were closely monitored throughout insufflation without evidence of hemodynamic changes or a vagal response. The operation proceeded uneventfully and a laparoscopic cholecystectomy was completed in the standard fashion without complications. The patient recovered well, was discharged home the next day and followed up in the clinic without any further issues.

Discussion

Cardiac arrest during laparoscopy is a known phenomenon [2,7]. While there are sparse case reports in the literature, a myriad of different factors which may lead to cardiac arrest in laparoscopy have been described. These include CO_2 embolization, vagal reaction secondary to insufflation or manipulation, decrease in cardiac index from decreased preload and increased afterload, reactions to anesthetic agents, anoxia or hypercarbia during anesthesia, and others. These should all be considered when managing a patient with sudden cardiac arrest during laparoscopy. Our patient did not demonstrate signs of CO_2 embolization (which would present with decreased $EtCO_2$ tachycardia and hypotension), or decreased preload (which would present with tachycardia). We suspect that our patient had a severe vagal response to the peritoneal stretch from insufflation. Insufflation is known to increase vagal tone and can result in bradyarrhythmias and asystole [8]. The incidence of bradyarrhythmias is reported to be 14% to 27% in young healthy individuals [3]. We identified eight other occurrences of asystole during laparoscopic cholecystectomy attributed to a vagal response [9-13]. There are known maneuvers to treat symptoms of a vagal response, these include cessation of gas insufflation, deflating the abdomen and administering atropine. However, these measures did not prevent our patient's rapid deterioration to asystole over just a few seconds.

The management of asystolic cardiac arrest associated with laparoscopy has been discussed in the literature. It includes atropine or epinephrine administration, CPR, rapid desufflation, and repositioning. Interestingly, in our review of the literature there were no documented reports of mortality secondary to vagal-mediated asystolic events during laparoscopy; however, with such few events, the risk of mortality is unknown [2]. While the management of the immediate arrest has been described, there is little reported on the post-arrest management of such patients. In our literature review, of the seven cases that experienced cardiac arrest during laparoscopic cholecystectomy four underwent completion laparoscopic cholecystectomy during the same case, two cases were canceled without reports of further management, one was converted to open cholecystectomy, and one underwent interval laparoscopic cholecystectomy with modifications, with the insertion of a temporary pacemaker and otherwise the same surgical approach [9-13].

For our patient, we decided to abort the initial procedure post-cardiac arrest for the patient to undergo further work up for any possible underlying cardiac pathology. Ultimately, her cardiac workup was negative, and we had a strong suspicion that intraoperative cardiac arrest was secondary to a hypersensitive response to peritoneal insufflation resulting in severe increased vagal tone. After extensive discussions with the patient, she wished to proceed with another attempt at laparoscopic cholecystectomy. We then proceeded to minimize her risk of another arrest by optimizing factors to prevent increased vagal tone or a severe vagal response. Pre-operatively she received continuous IV hydration to prevent any hypovolemia. Intraoperatively, she was treated with glycopyrrolate to block cardiac vagal inhibitory reflexes during induction of anesthesia and intubation. During the procedure, we used a lower insufflation flow rate of 4L/min with a lower set pressure of 10 mmHg. Once the patient reached goal insufflation pressure, we observed the patient for an additional 5 minutes to ensure continued hemodynamic stability prior to proceeding with the operation. The patient had a safe and successful second attempt at laparoscopic cholecystectomy without any further complications.

Conclusion

Cardiac arrest secondary to vagal response during laparoscopic procedures is a rare phenomenon. Currently, there are no clear guidelines on the management of these patients after successful CPR. We introduce a safe methodology which was successfully implemented in a patient with management including a cardiac workup to exclude underlying cardiac disease, IV hydration, administration of glycopyrrolate on induction, low flow peritoneal insufflation as well as lower goal set pressure, and consideration for bailout open cholecystectomy.

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