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Cardiac Trauma: An Unusual Cause of Dysrhythmias and Electrocardiographic Changes during Laparoscopic Nissen Fundoplication

Christopher E. Swide, M.D., * Paul F. Nyberg, M.D.,†

LAPAROSCOPIC surgery is undergoing a rapid expansion of applications from its long-time use in gynecologic surgery. Surgeons are using laparoscopic techniques for cholecystectomies, appendectomies, hernia repairs, exploratory abdominal procedures, varicocelectomies, nephrectomies, colostomy closures, and colectomies. The advantages of laparoscopic surgery include shorter hospital stays, greater patient acceptance, and decreased morbidity.1−4 However, laparoscopic procedures have unique risks, including organ damage, vascular injury, pneumothorax, and carbon dioxide embolism.4,5 The majority of these injuries result from

References


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traumatic placement of the laparoscopic instruments or from the pneumoperitoneum required to perform these procedures. Anesthesiologists need to be familiar with the unique challenges these procedures present and prepare to manage potential perioperative complications. We present a case of direct cardiac trauma in a patient undergoing laparoscopic Nissen fundoplication.

Case Report

A 55-year-old woman with a history of severe gastroesophageal reflux was scheduled for laparoscopic-assisted Nissen fundoplication. Her medical history was significant only for paroxysmal atrial tachycardia, which was controlled with digoxin. She had no history of ischemic heart disease, angina, or significant cardiac risk factors. The remainder of her medical history was unremarkable. Her physical examination revealed no significant abnormalities. Her preoperative electrocardiogram (ECG) revealed a normal sinus rhythm with a rate of 72 beats/min without evidence of ischemia (fig. 1). She had negative cardiac stress test and normal cardiac echocardiogram results before surgery. Her medications on admission were digoxin, omeprazole, Premarin (Wyeth-Ayerst Laboratories, Philadelphia, PA), Donnatal (AH Robbins, Richmond, VA), and diphenhydramine.

The patient received her regular medications on the morning of surgery and was brought to the operating room after premedication with 1 mg intravenous midazolam. Her preinduction blood pressure was 168/70 mmHg, with a heart rate of 100 beats/min in normal sinus rhythm. Her cardiac rhythm was monitored with a five-lead ECG with ST-T wave analysis. Anesthesia was induced with thiopental and fentanyl, and tracheal intubation was facilitated with succinylcholine. Anesthesia was maintained with isoflurane in air and oxygen, supplemented with intravenous fentanyl. Muscle relaxation was maintained with vecuronium.

The procedure was uneventful; the surgeons had freed the stomach and were preparing to wrap the fundus around the posterior esophagus. At this time, the posterior fundal wall slipped from a Babcock clamp before a stitch could be placed. Several attempts were made to wrap the fundus when the loaded needle driver was lost from camera view. A wide complex rhythm was noted, which lasted only a few seconds. The pulse oximeter remained functional with an SpO2 of 100%. End-tidal carbon dioxide was 36 mmHg and unchanged from its previous reading. Blood pressure remained stable at 110/75 mmHg. The surgeons were informed of the dysrhythmia and were asked whether surgical manipulation of the patient’s heart was occurring as they were working close to the diaphragm at the time of the initial dysrhythmia. It was noted that the needle driver had traveled through the esophageal hiatus into the mediastinum. The surgeon attempted to withdraw the needle driver but noted moderate resistance, and a wide complex cardiac rhythm was noted simultaneously on the cardiac monitor. This rhythm was brief and resolved without specific treatment. The surgeon slowly and carefully rotated the needle driver and was able to withdraw the instrument from the mediastinum. A sinus rhythm with ST segment depression and inverted T waves in lead II was noted. The patient remained normotensive.

The surgery was completed as an open Nissen fundoplication because of continued difficulty obtaining surgical exposure through the laparoscope. The patient was awakened, the trachea was extubated, and she was transferred to the postanesthesia care unit in stable condition. A 12-lead ECG obtained in the postanesthesia care unit revealed 1-mm ST depression in leads V3–V5 and T wave inversion in leads II and III, AVF, and leads V4–V6 (fig. 2). A chest radiograph revealed a small left apical pneumothorax, which did not require a thoracostomy tube.

The patient was transferred to the coronary care unit for subsequent care because of the postoperative ECG changes and intraoperative dysrhythmias. Cardiac enzyme measurements revealed a creatinine phosphokinase concentration of 285 mg/dl and an MB isoenzyme fraction of 2.2%. To differentiate between an infarction and myocardial trauma from the needle driver, an echocardiogram and a technetium-99m pyrophosphate scan were performed on postoperative day 1. The echocardiogram revealed normal wall motion, minimal mitral regurgitation, and moderate tricuspid regurgitation. The technetium-99m pyrophosphate scan revealed a small punctate lesion consistent with cardiac contusion in the inferior aspect of the left anterior descending coronary artery (fig. 3). In review of the non–stereotactic myocardial infarction, positive exercise test, and intraoperative observation, it was believed that myocardial injury from the trauma was the etiology of the ECG changes and myocardial infarction.

Fig. 2. Twelve-lead electrocardiogram taken on arrival in the postanesthesia care unit. Arrows point to ST segment depression in leads V3–V5 and T wave inversion in leads II, III, AVF, and V4–V6.

Discussion

Our case illustrates a potentially catastrophic complication of injury secondary to trauma from an intraoperative needle driver. Early recognition of an ECG change and diagnosis of myocardial infarction in a patient with a history of dysrhythmia was critical for patient survival. The differentiation of myocardial infarction from trauma was made by a combination of the patient’s history, the ECG changes, and a technetium-99m pyrophosphate scan. The patient was found to have no evidence of myocardial injury.

Fig. 3. Technetium-99m pyrophosphate scan revealed focal area of increased tracer uptake in the inferior aspect of the left ventricle.
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ventricle (fig. 3). In view of the normal wall motion study, small cardiac isoenzyme elevation, positive technetium$^{99m}$ pyrophosphate scan, and intraoperative observation of likely myocardial trauma, it was believed that myocardial injury from the laparoscopic instrument was the etiology of her ECG changes. The patient had an uneventful recovery. An ECG on postoperative day 2 had returned to baseline, and cardiac stress test results on postoperative day 7 were normal. A thallium stress test obtained 3 months postoperatively revealed a small, fixed, inferior defect without reversible ischemia, consistent with the diagnosis of intraoperative myocardial trauma from the needle driver.

Discussion

Our case illustrates a previously unreported and potentially catastrophic complication of direct myocardial injury secondary to trauma from a laparoscopic instrument. Early recognition of the etiology of the patient's electrocardiographic changes prevented a mistaken diagnosis of myocardial infarction and its resultant effect on assumed risk for future surgery.

The differentiation of myocardial trauma from perioperative infarction was made in this case from a combination of the patient's history, observed clinical course, and selected objective studies. The time course of the initial dysrhythmia with the needle driver passing into the mediastinum followed by the initiation of a second dysrhythmia during removal of the needle driver made myocardial trauma a likely event. The ST-T wave changes were observed immediately after the second arrhythmia and were confirmed on the postoperative 12-lead ECG. The postoperative echocardiogram showed no evidence of wall motion abnormality, such as might occur in an infarction, and the cardiac isoenzyme studies were only slightly elevated. A technetium$^{99m}$ pyrophosphate scan was chosen to attempt to document the anatomic distribution of the injury. Technetium$^{99m}$ pyrophosphate is an isotope taken up by injured myocardium. It is most sensitive for myocardial trauma in the first 24 h, whereas uptake in myocardial infarction usually peaks in 3–5 days.$^6$ In this case, it showed a clear point lesion in the area of the heart exposed to the needle driver on postoperative day 1. This result, in a patient with no evidence of ischemic heart disease, observed intraoperative ECG changes during removal of a surgical instrument from the mediastinum, equivocal cardiac isoenzymes, and rapid resolution of the ECG changes made the diagnosis of intraoperative trauma the likely etiology of her perioperative findings.

Although our patient had no clinical bleeding or significant hemodynamic compromise, the potential for profound hemorrhage or cardiac tamponade in this type of injury is apparent. This was avoided in our case by careful removal of the surgical instrument from the heart.

This case illustrates that patients scheduled for laparoscopic procedures are at risk for unique laparoscopic complications in addition to the risks for any patient undergoing anesthesia and surgery. Adequate preoperative preparation, careful intraoperative care, and adequate postoperative followup are essential for good patient outcomes.

References


Fig. 3. Technetium$^{99m}$ pyrophosphate scan. Arrows point to focal area of increased tracer uptake seen in the inferior wall of the left ventricle.

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