

Anaesthetic Management of A Case of Long QT Syndrome Undergoing Laparoscopic Cholecystectomy

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Citation

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Abstract

The anaesthetic management of a patient with Long QT Syndrome (LQTS) is always a challenge to the anaesthesiologist as these patients carry a very high risk of intraoperative malignant ventricular arrhythmias, which may be refractory to treatment. We report such a case of undiagnosed, asymptomatic, 51 years old female posted for laparoscopic cholecystectomy.

INTRODUCTION

The long QT syndrome (LQTS) is a disorder of cardiac ion channel producing prolonged ventricular repolarization; usually diagnosed by prolonged QT interval on electrocardiograph (ECG). LQTS may be congenital (resulting from mutation involving genes encoding cardiac ion channel) or acquired (caused by metabolic abnormalities or drugs). Congenital LQTS with associated deafness is known as Jervell and Lange-Nielsen syndrome.¹ The absence of deafness with prolonged QT interval is called Romano Ward syndrome.^{2,3} Both varieties have familial tendency with very high mortality and contribute to high incidence of sudden infant death syndrome.⁴ Congenital LQTS may be sporadic, non-familial in occurrence.⁵ A patient with LQTS is at risk of ventricular tachyarrhythmias, especially torsades de pointes. Usually an episode of torsades de pointes is self-terminating and results in a syncopal episode due to decrease in cerebral perfusion, but it may lead to sudden death when it deteriorates to ventricular fibrillation.

The patients, whether treated or untreated, remain at risk of life threatening episodes of torsades de pointes in the perioperative period. The practical considerations of anaesthesia for patients with LQTS therefore include immediate management of torsades de pointes, and, in known cases, avoidance of factors that may increase the risk of precipitating arrhythmias (torsades de pointes). We describe such a case of undiagnosed, asymptomatic 51 years old female posted for laparoscopic cholecystectomy.

CASE HISTORY

A 51 years old female of 45 kilograms was admitted with the diagnosis of cholelithiasis and was scheduled to undergo laparoscopic cholecystectomy. She was normotensive, non-diabetic with no other comorbidities. All vital parameters, including, hearing were normal. Abdominal examination revealed tenderness in the right hypochondrium. Biochemical examination revealed anaemia with increased serum alkaline phosphatase (Hb-8.6gm/dl, alkaline phosphatase-314 IUnits). Ultrasonography (USG) abdomen revealed splenomegaly with cholelithiasis. The chest X-ray was normal. On ECG, long QT waves of 450msec duration, as corrected QT (QTc), were present. Echocardiography revealed mild MR, mild pericardial effusion with no LV regional wall motion abnormality. The diagnosis of cholelithiasis with long QT interval was made. A cardiologist was consulted about the patient's general condition. It was decided that cardiologist and pacemaking devices would be present at the time of surgical procedure.

General anaesthesia was planned and she was premedicated with tab diazepam 10 mg and ranitidine 150mg at night before surgery and on the morning of surgery. Pretreatment with IV fentanyl 100mcg was given to attenuate sympathetic response. Anaesthesia was induced with thiopentone sodium 250 mg and tracheal intubation was facilitated with vecuronium bromide 8 mg. Patient's trachea was intubated with cuffed endotracheal tube (7.5mmID). Anaesthesia was maintained with O₂/N₂O in isoflurane (MAC of 1-1.5%). For laparoscopy, pneumoperitoneum was created using low flow of CO₂ while keeping the intraabdominal pressure (IAP) in range of 7-12mmHg. The surgery lasted 50 minutes. During

deflation of pneumoperitoneum, patient had tachyarrhythmias which were controlled with metoprolol 1mg and lidocaine 84mg. The tachyarrhythmias were ventricular bigeminy and supraventricular tachycardia. Neuromuscular blockade was reversed with neostigmine 2.5mg and glycopyrrolate 0.4 mg at the end of the procedure and the trachea was extubated. Immediately after extubation, patient was conscious and comfortable. She was shifted to postanesthesia care unit (PACU) for close observation. After 3-4 hours, she was shifted to Coronary Care Unit (CCU) for further management.

DISCUSSION

The QT interval normally varies with heart rate, lengthening with bradycardia and shortening at increased rates. The measured QT interval is therefore corrected for heart rate according to the formula of Bazette: $QTc = \text{Measured QT} / \sqrt{\text{RR interval}}$ (all measured in seconds).

A QTc interval of >440 ms is considered prolonged, although about 6% of patients with symptomatic LQTS have a normal QTc interval.⁶ As the QT interval on the ECG represents the total duration of both the depolarization and repolarization phases of the ventricular action potential, a lengthening of the QT interval occurring because of a prolongation in QRS complex duration does not constitute LQTS.

Anaesthesia in patients with untreated LQTS carries a very high risk of intra-operative malignant ventricular arrhythmias,⁷⁸⁹¹⁰¹¹¹²¹³¹⁴ which may prove refractory to treatment.¹⁵ Even in treated patients β -blockers are not completely protective and they remain at risk of life-threatening episodes of torsades de pointes in the perioperative period. The practical considerations of anaesthesia for patients with LQTS therefore include immediate management of torsades de pointes, and, in known cases, avoidance of factors that increase the risk of precipitating torsades de pointes.

Many episodes of torsades de pointes are short and self-limiting, but if prolonged with haemodynamic compromise, cardioversion is recommended. Intravenous magnesium is the agent of choice for the immediate treatment and prevention of torsades de pointes for both congenital and acquired LQTS.¹¹⁶¹⁷ If magnesium fails to prevent recurrence of torsades de pointes, then transvenous pacing at rate of 100-140bpm can be employed.¹¹⁶¹⁷¹⁸ These pacing rates prevent the pauses and shortening of the QT interval that could lead to torsades de pointes. Magnesium and

pacing are especially useful in congenital LQTS. Role of lidocaine and phenytoin is uncertain and as amiodarone prolongs the QT interval, its use is contraindicated. Maintenance of high normal concentration of serum potassium is important in LQTS since high serum potassium levels shorten the QTc and reduce QTc depression.

Long term treatment, includes β Blocker for LQTS, insertion of a permanent pacemaker or cardioverter defibrillator and rarely, left thoracic sympathectomy. The main aim is to reduce QTc interval and prevent recurrence of torsades.

The present case had two major problems, LQTS and its potential arrhythmogenic tendency faced during anaesthetic and surgical stimulation during laparoscopic surgery. Pneumoperitoneum required for the procedure produces increase in intraabdominal pressure (IAP) and leads to complex physiological changes affecting a number of homeostatic systems.¹⁹ Some studies demonstrate fewer adverse haemodynamic effects with low IAP. Therefore, we kept low IAP in the range of 7-12 mmHg.²⁰

A laparoscopic procedure may trigger sympathetic and parasympathetic stimulation leading to tachy and bradyarrhythmias. To prevent this stimulus, pretreatment with fentanyl was done during induction of anaesthesia. Lindgren et al²¹ advocated the use of pretreatment of fentanyl in different doses to prevent cardiac dysrhythmias during induction of anaesthesia and found all strengths useful. To minimize the sympathetic stimulation, adequate opioid balanced analgesia was given. To minimize postoperative pain, bupivacaine 0.25%, 20 ml was administered under the diaphragm at the end of surgery.

In the present case while the intraoperative course was uneventful, just after deflation of pneumoperitoneum, patient had tachyarrhythmias which were managed by lidocaine and metoprolol.

Patients with LQTS undergoing laparoscopic surgery always poses a challenge to the attending anaesthesiologist. The anaesthesiologist should aim to avoid the sudden release of catecholamines which may precipitate torsades de pointes. Perioperative anxiolysis may be beneficial.¹⁶ In healthy patients, induction by midazolam alone²² or in combination with fentanyl²³ neither prolongs the QT interval nor causes dysrhythmias. During laryngoscopy, tracheal intubation and extubation, catecholamine release can be reduced by administering additional beta blockers or opioids, or by topical anesthesia to the vocal cords or intravenous

lidocaine.²⁴ These patients can be very well managed by keeping stress factors under control by giving balanced anaesthesia and keeping assisted pacemaker devices readily available.

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