Case Report

Anesthetic Management of a Patient with Severe Dilated Cardiomyopathy: Case Report

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Abstract

Anesthetic management of patients with dilated cardiomyopathy (DCM) is a challenge to the anesthesiologist, due to poor left systolic function, ventricular enlargement, risk of malignant arrhythmias and sudden cardiac death. Therefore, preoperative assessment and appropriate anesthetic management are important in patients with DCM. Five to eight people per 100,000 develop this disorder each year. Malignant arrhythmias are the most common cause of death in DCM.[1] Around 50% of cases of nonischaemic dilated cardiomyopathy is idiopathic. Other causes are familial, infectious, infiltrative and connective tissue diseases. This is a report of successful anesthetic management of a patient with severe DCM undergoing laparoscopic cholecystectomy using general anesthesia (GA).

Keywords: *Anaesthetic management, dilated cardiomyopathy, laparoscopic cholecystectomy.*

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Introduction:

Dilated Cardiomyopathy (DCM) is characterized by dilatation and impaired systolic function of one or both ventricles. Cardiomyopathies are diseases of the heart muscle and may present with cardiac dysfunction. DCM is defined by the presence of: (a) Fractional myocardial shortening <25% and/ or left ventricular ejection fraction (LVEF) <45%; and (b) LV end diastolic diameter >117% excluding any known cause of myocardial disease.[2] DCM is the most common type of non-ischemic cardiomyopathy, the third most common cause of heart failure, and the most common indication for cardiac transplantation. World Health Organization reclassification in 1995 was expanded to include all known causes and is based upon anatomical and physiological features. Within this classification, the three main identified types of cardiomyopathy are dilated, hypertrophic, and restrictive. The cause of DCM is unknown, although it may be associated with myocarditis, neuromuscular disorders, familial disease, idiopathic causes and other possible diseases. Previously, it was thought that the largest proportion of DCM was idiopathic (66%).[3] Increasing evidence has shown that DCM has a familial basis.[4] Over 30 genes have been confirmed to be related to DCM[5], and sudden cardiac death in DCM was found to be associated with the long arm of chromosome 10. Approximately, 50% of cases of non-ischemic DCM are idiopathic. Here, we report a case of DCM with low EF posted for laparoscopic cholecystectomy surgery under general anesthesia

Case report

A 32 years old female, weighting 52kg having Cholelithiasis is scheduled for laparoscopic cholecystectomy. She was a known case of idiopathic dilated Cardiomyopathy. On preanesthetic examination her heart rate was 68/ min and regular. The systolic and diastolic blood pressures were 112mmHg and 60mmHg respectively.

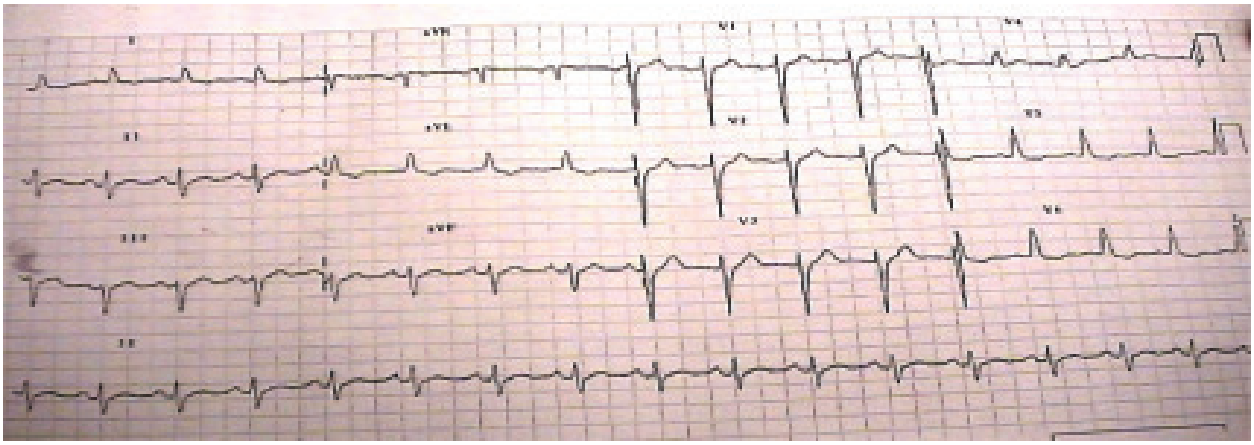

Fig 1 *ECG showing left bundle branch block and poor progression of R wave in lead V1 – V5*

The respiratory rate was 16/min .There was no ronchi or rales on auscultation and heart sounds were normal. Jugular venous pressure was not raised and there was no hepatomegaly. Preoperative 12 lead electrocardiograph (ECG) [Fig-1] showed Left Bundle Branch Block (LBBB) and poor progression of R wave in leads V1-V5. X-Ray chest [Fig-2] revealed cardiomegaly.

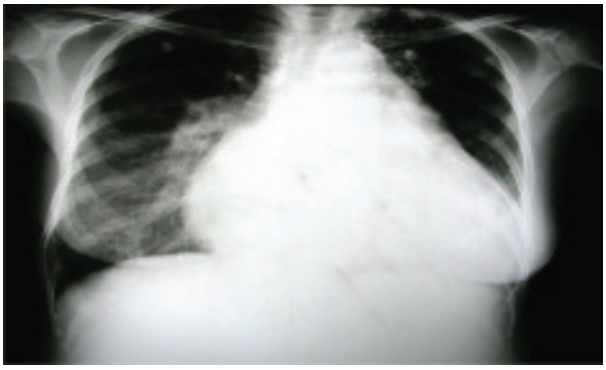

Fig 2 *Chest X-ray Showing dilated cardiomyopathy*

The lung fields were clear. Echocardiography reports demonstrated global hypokinesia of left ventricle, poor systolic function, ejection fraction of 20%, mitral regurgitation and left ventricular end diastolic dilatation. She was on Losartan 25 mg OD, Spirinolactone 50 mg OD and Digoxin 0.125 mg OD for the last 1 year. All investigations including serum sodium, potassium, calcium and magnesium were within normal limits. A high risk consent was obtained and general anaesthesia was planned .No premedication was advised. On the day of surgery, patient Blood Pressure (BP) was 120/80 mm Hg, Heart Rate (HR) was 72/min and oxygen saturation (SaO2) was 97% on room air.

Right internal jugular central catheter was placed under local anaesthesia prior to the induction of anaesthesia. Other parameters were monitored with continuous ECG, NIBP, end tidal carbon dioxide and oxygen saturation. Anaesthesia was induced slowly with iv fentanyl 100µgm, iv propofol 50 mg (stop when eyelash reflex diminished) and iv Atracurium 30mg. Lignocaine 50mg was given to blunt the hemodynamic response to intubation. The patient was intubated with cuffed endotracheal tube 7.0 mm ID (Internal Diameter). There was minimal response to intubation, her preintubation BP was 120/80 mmHg and HR was 72/min while post intubation BP was 126/82 mm Hg and HR was 76/min. Anaesthesia was maintained with $N_2O : O_2$ 50:50 and continuous infusion of propofol @ 15 – 20 ml/ hour. Pneumoperitoneum was made by CO2 insufflation. Intra abdominal pressure (IAP) was maintained 8 – 10 mm Hg. Surgery was completed in 40 minutes. Intraoperatively her mean arterial pressure was 66 -78 mm Hg, heart rate 60-70 / min, SPO2 was 99-100%, CVP was 8-10 cm H2O, end tidal carbon dioxide was 32-36 mm Hg and airway pressure was 15-20 cm H2O but ECG tracing showed sinus rhythm with infrequent PVC (< 4/min) without ischemic changes. Injection paracetamol 1 gm intraoperatively was given for postoperative pain management. At the end of surgery, patient was extubated smoothly and shifted to surgical ICU for better monitoring & management. On the 1st postoperative day patient

was discharged to surgical ward with stable hemodynamics.

Discussion:

Dilated cardiomyopathy is characterized by progressive cardiac dilatation and results in impaired ventricular function. It has a prevalence of 36 per 100, 000 population.[6] A large number of cases are idiopathic but within these there is a familial association. Clinical picture of dilated cardiomyopathy may vary from only cardiomegaly to severe CHF.[7]Apart from CHF, dysarrhythmias and embolism are (systemic or pulmonary) also common.[8] Recent management include medical therapy with drugs for example vasodilator, diuretics or beta blockers and atrio-ventricular pacemakers for patients with incoordinate movements of heart chambers.[9] It is difficult to decide the optimal time for surgery but the medical control of heart failure for >1 week is desirable.

Anaesthetic management of patients with Cardiomyopathy with reduced systolic function is challenging and may be associated with high mortality.[10] The goals of anaesthetic

management are: (i) avoiding myocardial depression by carefully titrating the anaesthetic drugs; (ii) maintaining normovolaemia; (iii) avoiding overdose of drugs during induction as the circulation time is slow; (iv) avoiding increase in ventricular afterload and (v) avoiding sudden hypotension where regional anaesthesia is the choice. Two key factors exist in the management of patients with Cardiomyopathies. These include: (i) to improve systolic function; and (ii) to prevent sudden death due to ventricular arrhythmias. To improve systolic function, patients should initially be managed medically with administration of diuretics, beta-blockers, angiotensin converting enzyme inhibitors or angiotensin receptor blockers. Our patient was being managed with these drugs. The preoperative preparation of these patients must be meticulous as they have minimal or no cardiac reserve. Preoperatively patients tend to be dehydrated as they would have been on diuretics leading to hypotension during anaesthesia. However, excessive preoperative hydration is not desirable as it may lead to congestive heart failure. As patients may develop ventricular arrhythmias in the perioperative period, antiarrhythmic medications should be continued. Arrhythmias occur commonly when potassium or magnesium levels are low. These electrolytes should be measured preoperatively and corrected as necessary.

Pneumoperitoneum should be started with reduced gas flow and intra-abdominal pressures should be kept as low as possible to minimize hemodynamic changes. Upto 10 mmHg of IAP, metabolic and haemodynamic parameters do not change significantly. With standard IAP of 12-15 mmHg, there is compression of capacitance vessels as opposed to collapse leading to increased venous return. The pressure threshold during Pneumoperitoneum, associated with minimal changes in the hemodynamics, is 12 mmHg.[11]

The acceptable limit of decrease in blood pressure and heart rate for a patient depends upon underlying medical condition. It is recommended that fluid therapy and pharmacological management be guided by the use of pulmonary artery catheterization and the determination of cardiac filling pressure.[12] Continuous monitoring of preload by Transesophageal Echocardiography (TEE) and of myocardial performance by cardiac output measurement (CCO) is also useful but this was not available in our hospital so, we relied on central venous pressure.

The optimum anesthetic technique for patients undergoing appendicectomy with dilated or other

forms of congestive cardiomyopathy is controversial and both general anesthesia[13,14] and regional anesthesia have been described.

Brown et al. described the use of general anesthesia because they feared catastrophic effects of reduction in systemic vascular resistance caused by epidural blockade.[13]

Regional anaesthesia used alone or in combination with general anaesthesia has the advantage of reducing after load which can improve cardiac output. However, hypotension must be prevented to avoid myocardial hypoperfusion.Treatment of arterial pressure changes should be considered if a 10% decrease in systolic pressures occurs.

For patients undergoing higher risk procedures under general anaesthesia or those in whom heart failure management is not optimized, invasive direct arterial pressure monitoring is indicated. Central venous pressure monitoring offers some

additional information on right ventricular preload but does not provide information on left heart pressures. Information from a pulmonary artery catheter can be useful, although may not improve outcome. Intraoperative transoesophageal echocardiography may also be useful for examining dynamic changes in cardiac performance and the response to inotropes and fluid loading. Where such technology is unavailable, then the use of oesophageal Doppler monitoring of aortic root velocities can provide information on cardiac performance. Inotropic support if required during and after surgery can be provided by the use of a variety of agents, including dobutamine, dopamine, phosphodiesterase inhibitors, and levosimendin. It may be necessary to counteract some of the peripheral vasodilator effects of general anaesthetic agents to assist coronary perfusion. This is achieved with the cautious use of norepinephrine which increases systemic vascular resistance and maintains mean arterial pressure.

Postoperative regional anaesthesia can be beneficial and good quality pain management avoids increases in systemic vascular resistance and heart rate. Cautious fluid management is important, and is best undertaken in an intensive care area.

Fluid management in patients with DCM is very critical. In our case intra-operative 500 ml of ringer lactate was given to prevent fluid overload. Over hydration may not be advisable as it may lead to CHF. Drop in BP was corrected with injection of ephedrine, a vasopressor which can neutralize the vasodilating effect of the anesthetics rather infusing intravenous fluids.

Conclusion

In conclusion, a patient of DCM poses many risks for anesthesiologist. Our patient was managed successfully under general anesthesia without any complications by a thorough preoperative assessment, optimized cardiac status, formulating good anesthetic plan & postoperative care.

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