DOI: 10.14260/jemds/2015/1718

CASE REPORT

PERIOPERATIVE MANAGEMENT OF A PATIENT WITH DILATED CARDIOMYOPATHY UNDERGOING HEMICOLECTOMY

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HOW TO CITE THIS ARTICLE:
Abraq Asma, Shaheen Parveen, Malik Rameez, Mohamad Ommid, Rukhsana Najeeb. "Perioperative Management of a Patient with Dilated Cardiomyopathy Undergoing Hemicolectomy". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 68, August 24; Page: 11912-11917,
DOI: 10.14260/jemds/2015/1718

ABSTRACT: The anesthetic management of a patient with Dilated Cardiomyopathy (DCM) undergoing non-cardiac surgery is always a challenge to the anesthesiologist as DCM is most commonly complicated by progressive congestive heart failure and malignant arrhythmias. Idiopathic dilated cardiomyopathy is a primary myocardial disease of unknown etiology characterized by left ventricular or biventricular dilation and impaired contractility. Depending upon diagnostic criteria used, the reported annual incidence varies between 5 and 8 cases per 100,000 population. It is more common in men. Dilated cardiomyopathy is defined by the presence of: a) fractional myocardial shortening less than 25%(>2SD) and/or ejection fraction less than 45%(>2SD), and b) Left ventricular end diastolic diameter (LVEDD) greater than 117% excluding any known cause of myocardial disease. Such cases always prove a challenge to the anesthetist because of the complications associated with the condition. Here we report the successful perioperative management of a patient with severe dilated cardiomyopathy undergoing surgery for carcinoma colon.

KEYWORDS: Dilated cardiomyopathy, Congestion, Arrhythmia, Perioperative, Colon.

CASE REPORT: 60 years old, historically normotensive, non-diabetic, euthyroid female presented with chief complaints of severe abdominal pain and vomiting for 2 days. Patient was managed conservatively and subjected to further evaluation. Colonoscopy revealed polyps in anorectum, descending colon and transverse colon. Histopathological examination of polyp revealed villous adenoma with high grade dysplasia. CECT abdomen showed a descending colon growth with bilateral pleural effusion. While hospitalized, patient developed hepatomegaly, pedal oedema with CXR showing features of LVF and bilateral pleural effusion, more on right side. USG revealed congested hepatic veins with minimal (R) sided pleural effusion. ECG showed LBBB pattern. Patient was subjected to cardio logical evaluation and diagnosed as a case of severe dilated cardiomyopathy. ECHO revealed a dilated LV, severe global hypokinesia with ejection fraction of 15%, Grade iii diastolic dysfunction with moderate MR with severe PAH (Gradient 65mm Hg) with dilated LA. Patient was put on medical therapy (Tab. losartan, lasilactone and carvedilol) for 2 weeks by cardiologists.

Patient was reviewed by the cardiologist after 2 weeks of medical treatment and cleared for the surgery as a very high risk case. A detailed counselling of the patient and the attendants was done, explaining the risks involved, nature of the surgical procedure and outcome. After taking a high risk written and informed consent, patient was planned for elective (L) hemicolectomy. Pre op investigations were normal with KFT (57/1.1) Tablet losartan was stopped by the cardiologist 1 day prior to surgery while tablets lasilactone and carvedilol were continued on the morning of surgery. In OR, multichannel monitoring was attached.

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Pre op vitals were HR-62/min regular, BP-127/76mm Hg, respiratory rate 16/min and SpO2 - 95% on room air. There were no abnormal sounds on auscultation and heart sounds were normal. Jugular venous pressure was not raised and there was no hepatomegaly

Two i.v lines were secured with 18G cannula and preloading with 250ml of crystalloid (NS) was started. Ionotropes like dobutamine and dopamine and anti arrythmics like amidarone were kept prepared beforehand. Prior to induction, (R) radial artery was cannulated under LA and invasive monitoring of blood pressure started. An epidural catheter was placed in L1-L2 interspace using LOR technique to air. A test dose was administered with 3ml of 2% xylocard for ruling out intrathecal and intravascular injection. Patient was induced with O2 100%,Midazolam 2mg, fentanyl 50ug and STP 50mg and vecuronium 4mg.Immediately blood pressure decreased to 60/40mmhg. Dobutamine support (5ug/kg/min) was started immediately through the second 18G cannula. BP rose to 110/60 mmHg. Airway was secured with 7.5mm ID (Internal diameter) PVC cuffed endotracheal tube and anesthesia maintained with O2: N2O mixture, Isoflurane (MAC 0.2-1%) and vecuronium 1mg boluses.

After securing the airway, RIJV was cannulated using standard seldinger's technique and CVP monitoring started. Dobutamine support (5-10ug/kg/min) was continued through RIJV. In order to supplement analgesia, 10ml of 0.25% bupvaicaine and 10ug of fentanyl was given in incremental doses through the epidural catheter. Surgery was commenced and the hemicolectomy proceeded smoothly with minimal blood loss. While mobilizing the gut, there was one episode of bradycardia that was managed with 0.6mg of atropine. Rest of the intraop course was uneventful. Intra op ABG was unremarkable. Intraop fluid management was done according to CVP and a target of 7 was achieved with 500ml of colloid and 250ml of crystalloid. And a urine output of 300ml. Estimated blood loss was 200ml. Towards, the end of surgery, patient was on minimal dobutamine support. Procedure was completed within one and a half hour following which patient was extubated and shifted to ICU for monitoring and further management.

In ICU, invasive monitoring was continued and patient's haemodynamic status closed watched. Patient received top up analgesia with 5ml of 0.25% and 5ug of fentanyl through epidural catheter. 4hrs post-surgery, patient developed decreased urine output with raised post op creatinine (2.1). Cardiology and nephrology consultation was sought who advised fluid management by revising the target CVP to 10mmHg. By 1ohrs post-surgery, patient was off dobutamine support and forming adequate urine out. Repeat creatinine after 48hrs was 1.6. After 48hrs, epidural catheter was removed and patient shifted out of the ICU to the high dependency unit. After 6 days of uneventful stay at HDU, Patient was discharged from the hospital with the advice to attend cardiology and general surgery OPD for follow up.

DISCUSSION: Dilated cardiomyopathy is a syndrome characterized by cardiac enlargement and impaired systolic function of one or both the ventricles. Although it was formerly called congestive cardiomyopathy, the term dilated cardiomyopathy is now preferred because the earliest abnormality usually is ventricular enlargement and systolic contractile dysfunction, with the sign and symptoms of congestive heart failure often (But not invariably) developing later.[1] Most patients are first seen between the ages of 20 and 50 Years, but it may affect children and the elderly. The most common initial manifestation as seen in this patient is heart failure, which occurs in 75 to 85 percent of patients. Symptoms of left sided heart failure predominate.[2] The true natural history of the disease onset is difficult to determine, since asymptomatic cardiomegaly may be present for months or years. Majority of the cases are idiopathic.

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Rest are associated with ischemia, valve dysfunction and post viral infection. DCM can also be found in association with sickle cell disease, muscular dystrophy, excess alcohol, hypothyroidism and some chemotherapy agents or during peripartum period.

Clinical picture of dilated cardiomyopathy may vary from only cardiomegaly to severe CHF.[3] Apart from CHF, dysarrythmias and embolism (Systemic and Pulmonary) are also common.[4] Prognosis of DCM patient is poor with only 25% to 40% of patients surviving 5 years after the definitive diagnosis. Early studies reported poor survival rates, but recent observation suggest better survival, reflecting the earlier detection and better treatment.[2]

The management of a patient with DCM undergoing non-cardiac surgery is always a challenge to the anesthesiologist as DCM is most commonly complicated by progressive congestive heart failure (CHF), the cause of death in 75 % of these patients.[4] The key hemodynamic features of the DCM are elevated filling pressures, failure of myocardial contractile strength, and a marked inverse relationship between afterload and stroke volume.[5]

Recent management includes medical therapy with drugs for example, vasodilator, diuretics, or beta blockers and atrio-ventricular pacemakers for patients with in coordinate movements of heart chambers.[6] It is difficult to decide the optimal time for surgery but the medical control of heart failure for >1week is desirable. In our case, patient was put on medical therapy for 2 weeks prior to surgery.

Goals of anaesthetic management consist of i) Myocardial depression should be avoided ii) normovolemia should be maintained iii) Avoid overdose of drugs during induction as circulation time is slow. iv) Ventricular after load is avoided v) avoid sudden hypotension when regional anaesthesia is a choice.[4]

GA carries a high risk as these patients may develop CHF or arrhythmias during intraoperative period. Regional anaesthesia may be an alternative to general anaesthesia in selected patients with DCM. Yamaguchi et al.[7] reported a case of total prostectomy under continuous epidural anaesthesia and total intravenous venous anaesthesia (TIVA) using ketamine and propofol in a patient of DCM. They demonstrated it a useful combination. Nerve blocks are a rational approach for appropriate surgery as they have minimal hemodynamic abrasion.[8] Epidural anaesthesia produces changes in the preload and after load that mimic pharmacological goals in the treatment of this disease.[4]

We planned a GA Supplemented by epidural analgesia for our patient. The predictors of poor prognosis,[2] in our patient were an ejection fraction of 0.15, a hypokinetic heart, the presence of moderate mitral regurgitation, Tricuspid regurgitation, severe PAH with systolic and diastolic dysfunction. For these reasons a high risk consent was obtained. The acceptable limit of decrease in blood pressure and heart rate for a patient depends upon underlying medical condition.[9] GA solely may increase the risk of CHF, myocardial ischemia or intraoperative arrhythmias. El-Dawlatly et al.[10] reported uneventful anesthetic management of a patient with DCM who underwent laparoscopic cholecystectomy (LC) under TEA. Aono et al.[11] compared three anesthetic techniques: GA, epidural analgesia (EA) and GA combined with EA for LC. They reported that, GA with sevoflurane/N2O could not suppress stress response of both hypothalamus-pituitary-adrenocorticalaxis and sympathy-oadrenal system while EA suppressed only the symathoadrenal responses. They concluded that TEA may be of advantage in patients with limited cardiac function undergoing abdominal surgery. Gramatica et al.[12] used EA as a sole technique for LC and recommended it for patients who are not good candidates for GA due to cardiorespiratory problems.

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The choice for anesthetic management in our case was fentanyl based EA with low concentration local anesthetic agent. This technique offers satisfactory analgesia with a relatively slow sympathetic blockade as well as decreased peripheral vascular resistance. In addition, induced reduction in afterload and preload benefits cardiac function.[13] Hashimoto et al.[14] reported that high dose epidural fentanyl anesthesia is an anesthetic method of choice for patients with DCM. The small dose of dobutamine used in this case was to support the circulation if hypotension occurred in light of the compromised cardiac status of the patient. Therefore, EA would not be satisfactory unless combined with relatively light GA to offer good analgesia with hemodynamic stability. Monitoring of CVP with the other vital signs was used to optimize the preload. Although cardiac output was not measured intraoperatively, it was believed that the circulatory effects of epidural block contributed to a relatively stable operative and postoperative course. Such patients could be well managed with preoperative optimized medical condition and well-planned anesthetic management.

It is recommended that fluid therapy and pharmacological management be guided by the use of pulmonary artery catheterization and the determination of cardiac filling pressures,[15] but this was not available in our settings. Continuous monitoring of preload by Trans esophageal Echocardiography (TEE) and of myocardial performance by cardiac output measurement (CCO) is also useful] but again this was not available in our hospital so, we relied on central venous pressure.

Anaesthetic management needs to be customized for those with left ventricular ejection fraction below 45%. Drugs like ketamine, etomidate and narcotics have minimal depressing effect on cardiac function and are used frequently.[16] Oxygen carrying capacity should be adequate. The main determinants of oxygen carrying capacity are cardiac output and hemoglobin. Therefore, hemoglobin should be maintained at higher level and 13-14gm/100ml has been recommended.[17] To improve cardiac output, inotropes, biventricular synchronized pacing or an intra-aortic balloon pump may be required. Skeletal muscle paralysis is to be provided by non-depolarizing muscle relaxant that lack significant cardiovascular effects.

Use of vasoactive or inotropic drugs may be required frequently to counteract the negative effects of the anaesthetics on cardiac function. Dopamine in appropriate dose range has positive inotropic, chronotropic and vasoconstrictive effects making it an ideal agent to negate adverse cardiovascular effects of anaesthetics. Anaesthesiologist should also be prepared to use lidocaine, Amidarone or defibrillation to treat the arrhythmias.[8] Arrhythmias occur when potassium and magnesium levels are decreased (As these patients are usually on diuretic therapy). These electrolytes should be assessed preoperatively and corrected as necessary.[18]

If the patient is having pacemaker then inhibition of pacemaker function may occur in presence of Electromechanical Interference (EMI) as produced by myogenic electrical activity associated with Muscle fasciculation induced by suuccinylcholine. Continuous invasive hemodynamic monitoring is essential as electrocardiography is not reliable in presence of EMI.[19] Cautery may lead to EMI. Bipolar cautery is less hazardous than unipolar, although EMI can still occur.

CONCLUSION: Patients with dilated cardiomyopathy are a challenge to the attending anesthesiologist. The anaesthesiologist should have the knowledge of its pathophysiology, clinical features, diagnostic evaluations and the treatment modalities. This is to be followed by careful planning for the provision of safe anaesthesia. These patients can be well managed by thorough preoperative assessment and medical management, formulating the good anesthetic plans and prompt diagnosis and management of complications.

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FINANCIAL OR OTHER COMPETING INTERESTS: None

Date of Submission: 07/08/2015.
Date of Peer Review: 08/08/2015.
Date of Acceptance: 18/08/2015.
Date of Publishing: 24/08/2015.