

Anaesthetic Management Of A Patient With Ischemic Dilated Cardiomyopathy And Biventricular Pacing With Resynchronization

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Citation

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Abstract

Despite advances in pharmacologic therapy, prognosis of patients with ischemic dilated cardiomyopathy remains poor as many of the patients ultimately land in congestive heart failure, cardiac conduction abnormalities such as left bundle branch block or interventricular conduction delays that can lead to ventricular dyssynchrony.

Biventricular pacing is an alternative, recent non pharmacologic therapy under active investigation. Use of this has been associated with improvement in ejection fraction¹, pressure gradient (dP/dT)², stroke work, cardiac index, myocardial energetics and functional class ultimately decreasing morbidity and mortality. Such patients with low ejection fraction have increased anesthetic risk for implanting such combo device.

We report the successful anaesthetic management of a case of ischemic dilated cardiomyopathy with severe left ventricular dysfunction with ejection fraction 15% undergoing biventricular pacing with resynchronization perioperatively.

CASE REPORT

A 65 year old non smoker, non hypertensive, diabetic male with past history of ischemic heart disease and one episode of unstable angina one year back presented with symptoms of class IV dyspnoea with history of orthopnea. One year back coronary angiography had revealed double vessel disease (mid left anterior descending vessel – 70% diffuse stenosis, left circumflex – diffuse insignificant plaque, right coronary artery – mid portion 95% greatest stenosis). He was on medical management with I.V. diuretics, dobutamine and ACE inhibitor presented with class III NYHA dyspnea for past 3 months.

Significant findings on examination were raised JVP- 15cm H₂O with prominent 'v' wave and rapid 'y' descent. Chest auscultation revealed bilateral basal crepitations and Chest x-ray showed cardiothoracic ratio of 0.6 with left ventricular contour of the apex with evidence of early pulmonary edema. Investigations revealed ECG: Sinus rhythm, LAD and LBBB with QRS width of 150m seconds 2-D echo showed dilated left ventricle with LVEF of 15% with RWMA. Colour Doppler findings were severe MR, mild TR and pulmonary artery systolic pressure 60mmHg. Coronary angiography repeated 3 months back showed LAD 70%, left circumflex 50% and RCA 95% stenosis. Echocardiography

showed significant increase in LV diastolic and systolic volume with decreased ejection fraction as compared to one year back. Investigation for LV dyssynchrony showed inter and intraventricular dyssynchrony.

Dobutamine stress test showed no evidence of viability in affected segment. All the above features suggested highly in favour of left ventricular dysfunction of ischemic etiology. Hence the decision was taken in favour of combo device implantation.

ANAESTHETIC MANAGEMENT

Under general anesthesia, patient was at high risk to develop congestive heart failure with various arrhythmias. So the implantation of combo device was decided to be done under conscious sedation.

Management of such a case was a challenge to anesthesiologists due to ejection fraction of 15% with cardiac conduction abnormality and severe LV dysfunction.

Patient was premedicated with tab. Diazepam 5mg at bed time in previous night. After securing I.V. line, monitoring was done with pulse oximeter and cardioscope. Conscious sedation was given with bolus midazolam 0.015mg/kg and fentanyl 1mcg/kg. It was maintained with O₂:N₂O(50:50)

given through nasopharyngeal airway as Hudson's mask could have interfered with fluoroscopy. BP dropped during this procedure to 70/50mmHg for which dopamine infusion was started at 5 /kg/min. After implantation, the position and functioning of the combo device was checked by giving shocks. Low dose ketamine was given for proper amnesia and analgesia for this maneuver.

All vitals were monitored continuously throughout the procedure to tackle any untoward hemodynamic change. Defibrillator was kept standby for any sudden arrhythmia. 2% lignocaine was infiltrated prior to pacemaker implantation. Successful implantation of the combo device was done with transvenous leads placed in right atrium, right ventricle and left ventricle via coronary sinus. After the procedure, blood pressure increased to 110/80mm Hg and dopamine infusion was tapered and then stopped. Vitals of the patient were stable after operation and echocardiography done on the following day revealed an increase in ejection fraction to 50%. After 48 hrs of uneventful observation, patient was discharged from anesthesia care.

DISCUSSION

Heart failure is one of the most prevalent diseases in most countries. Despite major advances in medical therapy, mortality and morbidity remains high.³

DCM represents a large subset of congestive heart failure cases characterized by depressed systolic function or systolic pump failure, cardiomegaly with ventricular dilatation. Reduced left ventricular contractile force leads to decreased cardiac output resulting in increased residual volume in end systole.

Low cardiac output causes upregulation of sympathetic nervous system and renin angiotensin system causing volume expansion, which induces vasoconstriction and further decrease in cardiac output. Treatment of this disorder attempts to breakdown this vicious cycle.

Clinical surveys indicate that up to 30% of DCM cases have a familial distribution. Only known disease gene dystrophin causes x-linked DCM.⁴ Cardiomyopathies have multiple etiologies. In ischemic cardiomyopathy, both systolic and diastolic dysfunction, result in decreased cardiac output and increased pulmonary venous pressure. Anatomically heart has a greater left ventricular cavity. Progressive dilatation can lead to MR and TR which further diminish cardiac output and increased end systolic volume and ventricular

wall stress.

Diagnosis rests mainly on clinical features, lab. Findings, echo, colour Doppler and coronary angiography.

Pharmacology therapy is aimed at decongestion with heart transplantation in suitable cases. But despite major advances in medicine, biventricular pacing with combo device remains a novel and goal oriented approach approved by FDA in 2001. The 2002 joint guidelines of ACC/AHA/NASPE endorse the use of CRT in patients with medically refractory, symptomatic, NYHA class III or IV disease and a QRS interval of at least 130 msec, a left ventricular end-diastolic diameter of at least 55 mm, and an LVEF of 30 percent or less.⁵ Similar recommendations have been made by the Canadian Cardiovascular Society,⁶ and the European Society of Cardiology.⁷

These guidelines were refined by an April 2005 American Heart Association Science Advisory,⁸ which stated that optimal candidates for CRT have a dilated cardiomyopathy on an ischemic or nonischemic basis, an LVEF 0.35, a QRS complex 120 ms, and sinus rhythm, and are NYHA functional class III or IV despite maximal medical therapy for heart failure.

Among inhalational and IV anaesthetic agents, low dose ketamine has least direct negative inotropic action on myocardium in patient with ischemic DCM with low EF. IV midazolam and fentanyl can cause hypotension but titrated low dose has minimal effect. Propofol and thiopentone have unacceptably high risk of causing hypotension in patient with DCM. For that reason we used drugs in titrated manner for appropriate level of conscious sedation and kept defibrillator and dopamine infusion ready for any accidental event.

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