

Anaesthetic Management of A Case of Dilated Cardiomyopathy With Global Hypokinesia

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Citation

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Abstract

We report the anesthetic management of a 75 yr. old patient with dilated cardiomyopathy, scheduled for surgery for total hip replacement. The risks involved and the potential benefits of the use of regional and general anaesthesia are discussed.

INTRODUCTION

Dilated Cardiomyopathy is a primary myocardial disease of varied causes¹. 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 function, with the sign and symptoms of congestive heart failure often (but not invariably) developing later. Anesthetic management of these patients is quite challenging. The anesthesiologist must have the knowledge of its pathophysiology, clinical features, diagnostic evaluations and the treatment modalities.

CASE REPORT

A 75-year-old man sustained fracture of the head of femur and was scheduled for hip replacement surgery. His previous medical records revealed that he was previously admitted to the Coronary Care Unit for episodes of congestive cardiac failure nearly 2 years back. He had a past history of alcohol abuse for 25 years. The heart rate was 64/min and regular. The blood pressure was 114/74 mmHg. There were no ronchi or rales on auscultation. Heart sounds were normal. Preoperative 12 lead EKG showed LBBB and poor progression of R wave in V₁-V₄. Echocardiography report demonstrated, global hypokinesia with poor systolic function, ejection fraction of 25%; mitral regurgitation, and type 1 diastolic dysfunction.

His symptoms were well controlled with Tab betaloc 50mg od, Tab lasix 40 mg od, Tab amlovas 10mg od, and Tab atorvastatin 20 mg od for the last 2 years. No abnormalities were noted in the laboratory investigations. Preoperative hemoglobin was 11.4 gm%.

A high-risk consent was obtained. Regional (epidural) anesthesia technique was explained to the patient. No premedication was advised. Intravenous access was established with a 18 G i.v. cannula and patient preloaded with 500 ml of ringer lactate. Non-invasive blood pressure, arterial oxygen saturation (SpO₂) and lead II, V of the electrocardiogram were monitored throughout the surgery. CVP line was inserted peripherally through the right basilic vein.

After taking all aseptic and antiseptic precautions, an 18 G epidural catheter was introduced at L3-4 space. 2% xylocaine plain (without adrenaline) was injected slowly to attain a sensory and motor block up to T10 level. BP of 80 mmHg systolic was observed after 10 min. This was treated with intermittent bolus of inj. mephenteramine in doses of 3 to 6 mg i.v. with the aim to maintain the systolic BP of 90 mmHg. or more. Ventricular as well as junctional ectopics were seen on the EKG but not persistent enough to warrant any treatment. Intraoperative blood loss was about 500-600 ml. CVP was maintained at 9-10 cm of H₂O throughout the surgery with crystalloids and colloids. Surgery lasted for nearly 90-100 minutes. Postoperatively, there was a drop in the blood pressure to 70/40mmHg, CVP was 6 cm H₂O. Patient had no complaints of chest pain, sweating or difficulty in breathing. Fluid administration continued at the rate of 100ml/hr. Dobutamine infusion was started at the rate of 5-6 ug/kg/min. to maintain the systolic blood pressure to 90 mmHg. After 1 hour, ventricular ectopics were seen on ECG monitor, which were successfully treated by administering xylocard 80 mg i.v. Repeat Hb value was 7.8 gm%. One unit blood transfusion was given. The subsequent postoperative course was uneventful.

DISCUSSION

Dilated Cardiomyopathy (DCM) is a unique subset of primary myocardial disease of unknown cause characterized by left ventricular or biventricular dilatation and impaired myocardial contractility. Symptoms of left sided heart failure usually predominate². The key hemodynamic features of the DCM are elevated filling pressures, failure of myocardial contractile strength, and a marked inverse relationship between after-load and stroke volume. Clinical picture of DCM may vary from asymptomatic with only cardiomegaly to severe CHF. Apart from CHF, dysrhythmias and embolism (systemic or pulmonary) are also common features of DCM patients. The true natural history of the disease onset is difficult to determine, since asymptomatic cardiomegaly may be present for months or years.

The predictors of poor prognosis¹ in our patient were an ejection fraction of 0.25, a hypokinetic heart, the presence of mitral regurgitation, systolic and diastolic dysfunction. For these reasons a high risk consent was obtained. It is recommended that fluid therapy and pharmacological management be guided by the use of pulmonary artery catheterization and the determination of cardiac filling pressures³; but this was not available in our O.T.

The goals for anesthetic management consists³ of 1) avoidance of drug induced myocardial depression, 2) maintenance of normovolemia, 3) prevention of increased ventricular after load. However, these goals sometimes may be difficult during general anaesthesia. Epidural anaesthesia can be used safely and effectively as an alternative to general anaesthesia with carefully titrated dose of local anesthetics, and hemodynamic monitoring. The changes in preload and after-load produced by epidural anesthesia mimic the pharmacological goals³.

Epidural was selected in preference to subarachnoid block as the advantages of epidural over spinal block consists of; lower risk of post dural puncture headache, less hypotension and the ability to prolong the block via the indwelling

catheter⁴. Although this patient was successfully managed solely with regional technique, the anesthesiologist have to be prepared to administer sedatives or general anesthesia if or when the effect of regional technique is unsatisfactory or it fails. However one must remember that during general anaesthesia, opioids with benzodiazepines or N₂O can cause cardiovascular depression³. Intravenous infusions should be guided by determining the cardiac filling pressures if possible³.

Postoperatively this patient had hypotension possibly due to due to intraoperative blood loss. This was treated with intravenous infusion of crystalloids and inotropic support with dobutamine, as dopamine can cause unwanted tachycardia. Blood was transfused to raise the Hb level. Ventricular ectopics could possibly be secondary to hypotension and hypoxemia due to a fall in the hemoglobin level. Xylocard (preservative free lignocaine) was used successfully to treat ventricular ectopics.

In summary, the factors which ultimately favored the good outcome of this high-risk patient were a thorough preoperative assessment, formulating the anaesthetic plans, postoperative monitoring, prompt diagnosis and management of the complications. Anaesthetic management of such patients is quite challenging. 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.

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