## CASE REPORT

# Anesthetic management of a patient with dilated cardiomyopathy and low ejection fraction

Pravin Ubale, MD*, Ashish Mali, MD**, Pinakin Gujjar, MD***

**Associate Professor; **Assistant Professor; ***Professor & Head*
*Dept. Of Anaesthesiology, TNMC & BYL Nair Hospital, Mumbai.*

**Correspondence**: Dr. Pravin Ubale, Anand Bhavan , B–16, TNMC & BYL Nair Hospital., Mumbai Central, Mumbai (India); Tel: 9322211472; E-mail: drpravinubale@gmail.com

## ABSTRACT

We report a case of 54 year male, a case of dilated cardiomyopathy with low ejection fraction who underwent Functional Endoscopic Sinus Surgery (FESS) under general anesthesia. Anesthetic management of such patients is always requires the highest level of expertise as they are usually complicated by progressive congestive cardiac failure (CHF). The uneventful course of the anesthesia in the presented case was related to the thorough systemic evaluation and careful anesthetic strategy.

**Key words:** Dilated cardiomyopathy; Anesthesia, General; Operative Time

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## INTRODUCTION

Dilated cardiomyopathy (DCM) is one of the group of diseases that affects primarily the myocardium. In DCM a portion of the myocardium is dilated often without any obvious cause. Left or right ventricular systolic pump function of the heart is impaired leading to progressive cardiac enlargement and hypertrophy, a process called remodeling. DCM is the most common form of non-ischemic cardiomyopathy. The incidence of DCM is reported to be 5 to 8 cases per 100,000 population per year.[1] It occurs more frequently in men than women, and is most common between 20 and 60 yrs of age. Anesthetic management of patients with cardiomyopathy with reduced systolic function is challenging and may be associated with high mortality.[2] Maintaining cardiovascular stability with optimal hemodynamic parameters during the anesthetic management of patient with DCM can be extremely challenging. We report successful management of a patient with DCM with low ejection fraction (EF) who underwent Functional Endoscopic Sinus Surgery (FESS) under general anesthesia.

## CASE REPORT

A 54 year male patient was referred to our institute for FESS due to frontal mucocele. He was a diagnosed case of DCM and hypertension. He had a history of dyspnea (NYHA functional class II), but no history of nocturnal dyspnea, orthopnea and palpitations. On physical examination there were no signs of congestive cardiac failure e.g. raised JVP, ankle edema or hepatomegaly. His heart rate was 76/min and blood pressure was 130/86 mmHg. On auscultation no signs of rhonchi or crepitations were present. His recent 2-D Echo showed DCM, depressed LV systolic function (EF 20%), Mild MR, Mild AR, Mild PH, and PASP by TR jet 35 mmHg. X-ray chest showed cardiomegaly. There was sinus rhythm on ECG. His hemoglobin was 12 gm/dl and all biochemical parameters were within normal limits. He was on treatment, tab amlodipine 5 mg BID, Tab furosemide 40 mg OD, tab atorvastatin 20 mg OD. Cardiologist consultation was requested for patient's management, who advised to continue the above mentioned drugs. Patient and his relatives were explained about anesthetic risk and a high risk consent was obtained.

General anesthesia was planned for removal of frontal mucocele by endoscopic approach. All emergency drugs and defibrillation were kept ready. Standard monitors (ECG, NIBP and $SpO_2$) were attached. With all aseptic precaution arterial line and triple lumen internal jugular venous catheterization

was achieved under local anesthesia. Patient was premedicated with inj. glycopyrrolate 0.2 mg, inj. midazolam 1 mg, inj. fentanly 100 $\mu$g and inj. hydrocortisone 100 mg. For gastric acid prophylaxis inj. ondensetron 8 mg and inj. ranitidine 50 mg given prior to induction. Anesthesia was induced with double diluted inj. thiopentone sodium 250 mg slowly given till loss of eye reflex and vecuronium bromide 8 mg was given after mask ventilation was confirmed. Patient's trachea was intubated with 8.5 mm ID cuffed endotracheal tube. Throat pack was inserted. Patient was maintained with sevoflurane in $O_2/N_2O$ and intermittent dose of vecuronium bromide. Intraoperatively $SpO_2$ was maintained between 98 to 99%, HR 70 to 80 beats/min, systolic BP was maintained between 110-130 mmHg, and diastolic BP was maintained between 70-80 mmHg with continuous use of invasive monitoring. Central venous pressure was kept at 8-10 $cmH_2O$. Blood loss was minimal and patient received one litre of crystalloids over a period of 2 hrs with urine output of 200 ml. Patient remained hemodynamically stable throughout the procedure. Throat pack was removed and adequate suctioning was done at the end of surgery. Neuromuscular blockage was reversed with inj. glycopyrrolate 0.4 mg and inj. neostigmine 2.5 mg and patient was extubated and shifted to intensive care unit for postoperative monitoring. After observing 24 hrs in the intensive care unit, patient was shifted to ward.

## DISCUSSION

Dilated cardiomyopathy is a primary myocardial disease of varied causes. It is characterized by left ventricular or biventricular dilatation and impaired ventricular contractility.[3] Several types of treatment modalities for dilated cardiomyopathy are available to improve systolic function. Patients should initially be managed medically. Biventricular pacing, cardioplasty or cardiac transplant may also be required to improve cardiac function.[4] Arrhythmias are managed with amiodarone and/or an automatic implantable cardioverter defibrillator (ICD). Medical management to improve systolic function includes administration of diuretics, beta-blockers, angiotensin converting enzyme (ACE) inhibitors.

Anesthetic management of patients with severe cardiomyopathies is associated with high morbidity and mortality, so requires proper planning, preparation and meticulous monitoring. The goals for anesthetic management consist of 1) avoidance of drug induced myocardial depression, 2) maintenance of normovolemia, and 3) prevention of increased ventricular afterload. Patients of DCM may deteriorate from induction of anesthesia till extubation and also in the postoperative period, so early recognition and immediate intervention of hemodynamic instability with appropriate vasoactive or inotropic medications is required to prevent catastrophic events. Life threatening ventricular arrhythmias may also occurs during the intraoperative period, so all emergency drugs such as lignocaine and amiodarone should be ready in the operating room.

As patients are on diuretics from preoperative period, they tends to be dehydrated, a further cause for hypotension during the perioperative period. However this dehydration is generally beneficial for these patients as it improves limited cardiac function. Preloading may not be possible in these patients as it may lead to CCF. So fluid management is an important task in these patients. In our patient fluid therapy was guided by monitoring blood pressure, CVP and urine output. Arrhythmias may also occur due to diuretics when potassium or magnesium levels are decreased. So one must have a close watch on electrolyte imbalance for correction as early as possible.[5]

Most of the anesthetic drugs tend to depress myocardium, slow the heart rate and dilate the blood vessels. Therefore, selection of drugs that have minimal myocardial depressant effect is very essential in these patients. Induction agents like propofol and thiopentone sodium have depressant effect on heart. Etomidate is the ideal induction agent in these patients. As Etomidate was not available in our institute, we use thiopentone sodium as induction agent.

The predictors of poor prognosis in our patient were, depressed LV systolic function (EF 20%), mild MR, mild AR, and mild PH. Also patient was a known case of hypertension. For these reasons, condition was explained to the patient as well as relatives and high risk consent was obtained. Patient's hemodynamic status was carefully observed and fluid management was guided by CVP. We monitored this patient in the intensive care unit, because, the postoperative management also requires intensive monitoring, similar to the intraoperative period until the patient is stabilized.

In conclusion careful and intense hemodynamic monitoring and slow and judicious titration of anesthetic drugs and fluids is important in patient of DCM with low ejection fraction.

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# My Most Memorable Patient

## Tigecycline induced bradycardia


Vikas Saini MD*, Sanjeev Palta MD **

**Assistant Professor, Department of Anaesthesia , PGIMER, Chandigarh*

***Associate Professor, Department of Anaesthesia & Intensive Care, GMCH, Chandigarh*


The increased use of antibiotics is not absolutely free of complications or side effects. We report an unusual cause of bradycardia secondary to use of tigecycline, an addition to the armamentarium of ever expanding list of antibiotics. A 35 year old male patient who underwent pancreatic necrosectomy was weaned off gradually from ventilator to T piece. During the course of his stay in ICU for 37 days, he developed fever with leucocytosis (WBC- 14,500) and was started on tigecycline day 10 according to the culture sensitivity from trachea and the wound. While the fever subsided and patient showed signs of improvement, tigecycline was continued for 21 days as patient was resistant to all the other drugs. During the stay in ICU, the patient started having episodes of after bradycardia from day 22 that was associated with sweating and complaints of heaviness in the chest. However, it responded to intravenous administration of 0.5mg of atropine. The frequency of episodes increased over a few days and patient responded very well to atropine every time. All the possible causes for bradycardia were ruled out. The biochemical parameters were unremarkable. The cardiac evaluation was also found to be normal. On reviewing the treatment, no drug that had been prescribed was known to cause bradycardia. Since the patient had already received tigecycline for 21 days and the parameters were normal, it was decided to withdraw the drug. The patient had a single episode of bradycardia after discontinuation of the drug and no episode was reported after 24 hours of discontinuation, thus implicating tigecycline to be the causative agent of bradycardia retrospectively. The patient was transferred to high dependency unit on day 37 of the ICU admission and later discharged to ward. Tigecycline is the first commercially available member of the glycylcyclines, derivative of the tetracycline antibiotics, with structural modifications that allow for potent gram-positive, gram-negative, and anaerobic activity, including resistant gram-positive organisms such as penicillin-resistant Streptococcus pneumoniae, methicillin-resistant Staphylococcus aureus (MRSA) and Staphylococcus epidermidis (MRSE), and vancomycin-resistant Enterococcus (VRE) species.[1] It was approved in June 2005 for the treatment of complicated skin and skin structure infections (SSSIs) and complicated intra abdominal infections. The most common side effects of the drug include nausea (29.5%), vomiting (19.7%) and diarrhea (12.7%), the causation of which is attributed the irritant effects of tigecycline on gastric mucosa.[2] Bradycardia with the use of tigecycline has not been reported before. The exact mechanism of the same cannot be underlined but, at the same time, it is imperative to watch the patient for this complication as this may be a preceding event to a fatal catastrophe. In 2010 FDA has issued a statutory warning about limiting the use of tigecycline in specific conditions as there is increased risk of mortality and death with the use of tigecycline when compared to other antibiotics used for similar infection. Therefore, Tigecycline should only be used for complicated skin infections, complicated intra abdominal infections and community acquired pneumonia. We were fortunate to have unknowingly discontinued the drug, thus probably averting a situation that could have possibly harmed the patient.

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