DOI: 10.14260/jemds/2014/2238

CASE REPORT

ANESTHETIC MANAGEMENT OF A PATIENT WITH DILATED CARDIOMYOPATHY FOR PROXIMAL FEMUR NAILING

Ashwini H[1], Suresh S. B[2]

HOW TO CITE THIS ARTICLE:
Ashwini H, Suresh S. B. "Anesthetic Management of a Patient with Dilated Cardiomyopathy for Proximal Femur Nailing". Journal of Evolution of Medical and Dental Sciences 2014; Vol. 3, Issue 12, March 24; Page: 3020-3023, DOI: 10.14260/jemds/2014/2238

ABSTRACT: Dilated cardiomyopathy (DCM) is characterized by dilatation and impaired systolic function of one or both the ventricles. Malignant arrhythmias are the most common cause of death in DCM. Around 50% of cases of non-ischemic DCM are idiopathic. Anesthetic management of these patients is quite challenging. The anesthesiologist must have the knowledge of its pathophysiology, clinical features, diagnostic evaluation and treatment modalities. We report a successful anesthetic management of a patient with DCM who underwent proximal femur nailing (PFN).

INTRODUCTION: Cardiomyopathies are a heterogeneous group of diseases of the myocardium associated with mechanical and /or electrical dysfunction that usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilatation and are due to a variety of causes that are frequently genetic. Cardiomyopathies either are confined to the heart or are part of generalized systemic disorders; often leading to cardiovascular death or progressive heart failure related disability.[1] dilated cardiomyopathy (DCM) is a primary myocardial disease of varied etiology. Although it was formerly called as congestive cardiomyopathy, the term dilated cardiomyopathy is now preferred. Because the earlier abnormality usually is ventricular enlargement and systolic contractile dysfunction with the signs and symptoms of congestive cardiac failure often (but not invariably) developing later.[1,2] Prognosis of DCM is very bad with only 25%-40% patients surviving after 5 yrs. after the definitive diagnosis.[3] Anesthetic management of these patients is challenging as DCM is most commonly complicated by CHF and malignant arrhythmias (most common cause of death in DCM).[3] The anesthesiologist must have knowledge of its pathophysiology, clinical features diagnostic evaluation and the treatment modalities.

CASE REPORT: A 70yr old man sustained intertrochanteric fracture of head of left femur was scheduled for proximal femur nailing (PFN).

His previous medical history revealed stroke 5yrs ago. He was diagnosed to have ischemic heart disease 2 yrs. back. He gave history of smoking for 40 yrs. and had stopped since 2yrs. He used to take alcohol occasionally. He didn't have hypertension, diabetes, asthma or epilepsy. He didn't give history of chest pain, orthopnea or dyspnea on exertion. He was leading a sedentary life because of his age and previous health problems. He was not on any medications for his cardiac problems.

On examination, JVP was normal. Heart rate was 82/min regular and good volume. BP was 170/80mmHg. He had pallor. On auscultation, chest was clear and heart sounds were normal. Preoperative 12 lead ECG showed Q waves in lead II, III, a VF and T wave inversion in all chest leads (V1–V6). Cardiologist's opinion was sought. Echocardiography revealed regional wall motional abnormality, dilated left atrium and left ventricle, EF35%, left ventricular global hypokinesia. So

DOI: 10.14260/jemds/2014/2238

cardiologist had not given clearance for surgery in view of history of stroke, old age, poor cardiac function. They had advised Tab Telmisartan 40mg OD.

Preoperative Hb was 9.5gm%, random blood sugar was 138mg%, serum electrolytes were Na-136meq, K-4.1meq, Cl-100. Chest X-ray showed mild left sided pleural effusion. All other investigations were normal.

High risk consent was obtained. Regional anesthesia technique was explained to the patient. Tablet ranitidine 150 mg and t.telmisartan-40 was given orally 2hrs before the surgery. On table intravenous access was established with 18 G intravenous cannula and hetastarch was started. On table BP was 140/84mmHg. Room air saturation was 96% and with O2 mask it improved to 100%. ECG leads connected. Emergency drugs like amiodarone, furosemide, metaprolol, dopamine, dobutamine kept ready. Defibrillator checked and kept ready.

Under aseptic precaution right internal jugular vein was cannulated and connected to manometer for CVP monitoring. CVP was 7Cm H20. Patient preloaded with 500ml 0f hetastarch. CVP raised to 10Cm H2O. Next one ringer lactate (RL) started.

It was decided to give combined spinal epidural anesthesia (CSEA). Under aseptic precautions, 18G epidural catheter was introduced at L3-L4 interspace. 3ml of 2% plane lignocaine was given as test dose. Subarachnoid block was given at L4-L5 space with 26G quincke babcock's needle using 1.5ml of 0.5% bupivacaine(heavy) and 0.5ml of preservative free fentanyl 25µgm. Epidural catheter was fixed and patient made supine immediately. Patient had good pain relief and the level of block (LOB) was L1. After 5 min, LOB was T11. Surgeons were allowed to put the patient into operating position. Meanwhile the LOB was brought up by slow epidural injection of 5ml of 2%plane lignocaine and 5 ml of 0.25% of bupivacaine. After 10 min (15 min since SAB), the LOB was T8. Patient had first drop in BP at this point (80mmHg). CVP was 10 CmH2O. 3 mg mephentermine was given and 100ml of RL was given as bolus. BP came up to 90mmHg systolic. RL was kept at a rate of 100ml/hr. Patient needed two more doses of mephenteramine intraop to maintain the BP above 90mmHg systolic. CVP maintained 9-10Cm H20. One unit of packed cells started intraoperatively when the estimated blood loss was 400ml. O2 by mask was given throughout the surgery. All other hemodynamics remained stable throughout the procedure. Patient didn't have any complaints intraoperatively. Surgery lasted for 90min. total blood lost was 500-600ml. Intra operative fluids given were 500ml 0f hetastarch, 200ml RL. Patient shifted to post-operative ward. Blood transfusion completed in post op ward.

In the post-operative ward, on admission, patient received a bolus of 10 ml of 0.125% bupivacaine with 100µgm of buprenorphine. All the vitals remained stable. Patient received same epidural bolus drug at 8th hour interval for 48 hours and then patient was shifted toward after the removal of epidural catheter. 7thpost operative day patient was discharged home with the advice of static quadriceps exercises and non-weight bearing mobilization with walker.

DISCUSSION: DCM is a unique sub set of primary myocardial diseases of unknown cause characterized by left ventricular or biventricular dilatation and impaired myocardial contractility.[1] Symptoms of left sided heart failure usually predominate. Right heart catheterization reveals high pulmonary capillary wedge pressure, high systemic vascular resistance and a low cardiac output.[1] Clinical picture may vary from asymptomatic with only cardiomegaly to severe congestive heart failure (CHF).[3] Apart from CHF, dysrhythmias and embolism (systemic or pulmonary) are also

DOI: 10.14260/jemds/2014/2238

common features of DCM. The true natural history of the disease onset is difficult to determine, since asymptomatic cardiomegaly may be present for months or years.[1] Anesthetic management of patients with cardiomyopathy is associated with high morbidity and mortality and therefore requires careful planning.

Management of cardiomyopathy is to improve systolic function and to prevent sudden death due to ventricular arrhythmias. Management includes, bed rest, salt and fluid restriction.[1] Medical management to improve systolic function includes administration of diuretics, beta blockers, angiotensin converting enzyme (ACE) inhibitors or angiotensin receptor blockers. Biventricular pacing is often used in patients with cardiomyopathy to improve systolic function. Cardiac transplant may be recommended for end stage cardiomyopathy. For arrythmias amiadarone is recommended. Implanted cardioverter defibrillator is implanted to treat ventricular tachyarrythmias.[2]

The predictors of poor prognosis in our patient were EF 35%, a hypokinetic heart, systolic dysfunction. For these reasons high risk consent was taken. Patients with EF >45% usually do not require any change in anesthesia technique.[2]

Preoperative preparation of these patients must be meticulous as they have minimal cardiac reserve. Any arrhythmias to be treated appropriately. Any decrease in myocardial contractility, heart rate or vasodilatation can cause profound hypotension. Preoperatively patient tends to be dehydrated due to the use of diuretics. This dehydration is beneficial for the patient. Preoperative hydration to prevent hypotension during anesthesia technique is not recommended as this can cause congestive cardiac failure. Fluid management is critical. To err on the hypovolemic patient is prudent. Therefore vasopressors to mitigate against the vasodilating effect of anesthetic is a rational approach.[2] It is recommended that the fluid therapy and pharmacological management of these patients should be guided by the use of pulmonary artery catheterization and determination of cardiac filling pressures[4]. This was not available in our O.T. so we used only CVP to guide the fluid management.

The goals of anesthetic management in these patients are: [5]

1. Avoidance of drug induced myocardial depression.
2. Maintenance of normovolemia.
3. Prevention of increased after load.
4. Avoidance of sudden hypotension especially where regional techniques are used.

These goals are difficult to achieve during general anesthesia. Regional techniques can be used safely and effectively as an alternative to general anesthesia with careful titrated doses of local anesthetics and hemodynamic monitoring. We selected CSEA because it has lower failure rate than epidural anesthesia alone. Epidural route can be used to provide post-operative analgesia which is very beneficial to patients with DCM. In addition vasodilatation produced by regional anesthesia is beneficial in patients with isolated left ventricular dysfunction.[6] Small dose of local anesthetic given intrathecally to avoid sudden drop in blood pressure due to high level of blockade. Preloading was done carefully, monitoring the CVP. Since SAB in elderly patients with fracture femur is known to result in hypotension, we decided to preload. This would have been hazardous if general anesthesia would have been planned. Titrated doses of epidural local anesthetics given to achieve the desired level of block slowly. Over loading with fluids to manage hypotension is risky in elderly patients

DOI: 10.14260/jemds/2014/2238

especially with borderline cardiac function like the one our patient had. So intraoperatively hypotension was treated with mephentermine and blood loss was replaced with blood. Although this patient was successfully managed solely with regional technique, anesthesiologists must be prepared to induce general anesthesia. However one must remember that, during general anesthesia, inhalational agents, opioids, benzodiazepines, N2O all can cause myocardial depression. Positive pressure ventilation can further decrease cardiac output. Intravenous infusions must be guided by determining cardiac filling pressures[7].

In summary, the factors which favored the good out come in this patient were thorough preoperative assessment, formulating the anesthetic plan, careful perioperative fluid management and monitoring of CVP and blood pressure. Anesthetic management of such patients is quite challenging especially with no invasive monitoring facilities. The anesthesiologist must have good knowledge of its pathophysiology, clinical features, diagnostic evaluation and treatment modalities. This is to be followed by careful planning for the provision of safe anesthesia for that particular patient.

REFERENCES:

1. Hines RL, Marschall KE, Paul AK. Heart failure and Cardiomyopathies. In: Stoelting's. Anesthesia and Coexisting Disease (5th ed). Saunders Elsevier 2010; 128-29.
2. Prashan HT, Thiagarajah S, Elizabeth AMF. Anaesthetic considerations in patients with cardiomyopathies. A review - MEJ. Anaesth.2009; 20(3): 347-354.
3. William G, Valentin Fuster. Review Article- Idiopathic dilated Cardiomyopathy. New England Journal of Medicine 1994; 331:1564-75.
4. Christopher M bernards. Epidural And Spinal Anesthesia. In: Clinical Anesthesia, Lippincot Raven 3rd Edition.1996; 26:665.
5. Surabhi M, Bharat S. Anaesthetic management of a case of severe dilated cardiomyopathy with splenic abscess for splenectomy. The Indian Anaesthetist's Forum – July 2011.
6. Sharrock NE, Bading B, Minco R, Blumenfeld JD. Deliberate hypotensive epidural anaesthesia for patients with normal and low cardiac output. Anesth Analg 1994; 79:899-904.
7. Sangeet Narang. Anaesthetic management of dilated cardiomyopathy M.E.J. Anaesthesia. 2007; 19(1):243-249.

AUTHORS:

1. Ashwini H.
2. Suresh S. B.

PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of Anaesthesia and Critical Care and Pain Management, Sapthagiri Institute of Medical Science, Bangalore.
2. Associate Professor, Department of Anaesthesia and Critical Care and Pain Management, Sapthagiri Institute of Medical Science, Bangalore.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Ashwini H,
#29, Sanmati, Udupa Gardens,
S. M. Road, Jalahalli Cross,
Bangalore - 15.
E-mail: drashwinih@rediffmail.com

Date of Submission: 19/02/2014.
Date of Peer Review: 20/02/2014.
Date of Acceptance: 03/03/2014.
Date of Publishing: 19/03/2014.