

Cardiomyopathies and anaesthesia

Address for correspondence:

Dr. Rajiv Juneja,
Department of Cardiac
Anaesthesia, Medanta - The
Medicity, Gurgaon,
Haryana, India.
E-mail: juneja@hotmail.com

Rajiv Juneja, Prajeesh M Nambiar

Department of Cardiac Anaesthesia, Medanta - The Medicity, Gurgaon, Haryana, India

ABSTRACT

Cardiomyopathy is considered as a heart muscle disease of multiple aetiologies, unlike other cardiac diseases related to a definitive pathophysiology. With more and more research and with the advent of genetic analysis pin pointing the disease causing mutations, causative factors have been defined and classifications and definitions have changed over time. Patients with these conditions present to anaesthesiologists in elective and emergency situations, placement of automated internal cardioverter defibrillator (AICD) devices or biventricular pacing but may also be diagnosed at anaesthetic pre-assessment. We describe cardiomyopathies such as dilated cardiomyopathy, hypertrophic cardiomyopathy, post-partum cardiomyopathy and Takotsubo cardiomyopathy in brief and their anaesthetic management.

Key words: Anaesthesiologist, cardiomyopathy, hypertrophic cardiomyopathy

| |
|---|
| Access this article online |
| Website: www.ijaweb.org |
| DOI: 10.4103/ija.IJA_385_17 |
| Quick response code |
|  |

INTRODUCTION

Cardiomyopathy is a disorder of the heart muscle in which the myocardium is structurally and functionally abnormal in the absence of coronary artery disease (CAD), hypertension, valvular disease or congenital heart disease.^[1] The American Heart Association in 2006 classified cardiomyopathies as primary or secondary. Primary are those exclusively confined to heart muscle and are genetic, acquired or of mixed origin. Secondary cardiomyopathies are due to the pathophysiologic involvement of the heart as part of a generalised systemic disorder [Table 1].

However, there may be considerable overlap as some primary cardiomyopathies can have extracardiac components, and secondary cardiomyopathies can affect the heart exclusively. In 2008, the European Society of Cardiology introduced a classification^[2] that includes five specific types of cardiomyopathies with their genetic involvement: hypertrophic, dilated, arrhythmogenic, restrictive and unclassified. These are further classified into familial/genetic or non-familial/non-genetic. The non-familial group can be idiopathic or part of a generalised disorder not specific to cardiac muscle only.

Recently, the MOGE(S) classification system based on phenotype and genotype has been proposed which

incorporates information on structural and functional abnormalities (M), organ involvement (O), genetics (G), etiology (E) and disease severity (S). However, it does not include certain cardiomyopathies as post-partum cardiomyopathy (PPCM) or risk of sudden death and is complex to use.^[3]

DILATED CARDIOMYOPATHY

Dilated cardiomyopathy (DCM) is a primary myocardial disease which reduces global myocardial contractility, leading to left ventricular (LV) or biventricular dysfunction. DCM presents with decrease in LV ejection fraction (LVEF), congestive heart failure (CHF) and ventricular arrhythmias. Initially, the ventricle dilates to increase the force of contraction and stroke volume (Frank–Starling relationship); however, these compensatory mechanisms gradually fail, progressive ventricular failure ensues and cardiac output (CO) decreases [Figure 1].^[4,5]

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Juneja R, Nambiar PM. Cardiomyopathies and anaesthesia. *Indian J Anaesth* 2017;61:728-35.

The condition is more common in males, has a prevalence of 92/100,000 population, and is common in Afro-Caribbean population. It is the most common cardiomyopathy, third most common cause of CHF and most common indication for heart transplantation. It can be idiopathic, post-viral or hereditary (30%–40%, autosomal dominant). Secondary cardiomyopathies causing DCM include alcohol and cocaine abuse, hypothyroidism, chemotherapy and muscular dystrophy. Peripartum cardiomyopathy (PPCM) is a form of DCM.^[5-7]

Presentation, investigations and medical management of DCM patients are shown in Table 2.^[4-6]

| Aetiology | Type of cardiomyopathy |
|------------------|--|
| Primary | |
| Genetic | Hypertrophic cardiomyopathy Left ventricular non-compaction Conduction abnormalities Brugada syndrome |
| Mixed | Dilated, restrictive |
| Acquired | Inflammatory - viral, Bacterial Takotsubo cardiomyopathy Peripartum |
| Secondary | Storage: Fabry's disease, hemochromatosis Infiltrative: Amyloidosis, Gauchers's disease Toxicity: Alcohol, mercury, lead, chemotherapy, radiotherapy Endocrine: Diabetes mellitus, acromegaly, pheochromocytoma |

| Clinical examination and investigations | Features |
|--|---|
| Signs and symptoms | Fatigue, weakness, dyspnoea, orthopnoea, raised JVP, pulmonary rales, ascites, ankle oedema, systemic embolization |
| Rhythm | Sinus tachycardia, atrial fibrillation, ventricular arrhythmias, sudden death |
| ECG | ST-T abnormalities, intraventricular conduction defects, atrial fibrillation, premature ventricular contractions |
| Chest radiography | Enlarged cardiac silhouette |
| Echocardiography | Dilated cardiac chambers, global hypokinesia, low EF/fractional shortening, mural thrombi, raised LVEDP, mitral or tricuspid regurgitation |
| Laboratory tests | Raised brain natriuretic peptide, coronary angiography-normal |
| Rhythm devices | AICD, CRT-D (biventricular pacing) |
| Medications | ACEI and angiotensin antagonists, beta blockers, diuretics, vasodilators, digoxin, statins, antiarrhythmics, anticoagulants, direct thrombin inhibitors |

ECG – Electrocardiogram; JVP – Jugular venous pressure; EF – Ejection fraction; LVEDP – Left ventricular end-diastolic pressure; AICD – Automated internal cardioverter defibrillator; CRT-D – Cardiac resynchronisation therapy device; ACEI – Angiotensin converting enzyme inhibitors

Pulmonary and systemic embolism can occur as blood stasis in dilated and hypocontractile cardiac chambers lead to activation of the coagulation cascade.^[7] Asymptomatic non-sustained ventricular tachycardia occurs commonly and is managed with antiarrhythmics, for example, amiodarone or AICD device which improves survival.^[4,8] Patients with LVEF <30% and an intraventricular conduction defect with wide QRS complex ≥ 130 ms may lack synchronised contraction of both ventricles. Resynchronisation of left and right ventricle with biventricular pacing through a cardiac resynchronisation therapy device (CRT-D) restores synchronous contraction of both ventricles, shortens QRS interval, decreases LV size and improves systolic function, stroke volume and survival.^[8,9] Treatment include drugs, cardiac resynchronisation therapy, intra-aortic balloon pump (IABP), LV assist devices (LVADs) as bridge to transplant or sustained recovery and cardiac transplant in drug refractory terminal heart failure.

Anaesthetic management
Pre-operative management

Anaesthetic management can be associated with morbidity and mortality, therefore, requires planning.^[4] Optimisation of CHF at least for a week before the planned surgery is advisable. In critically ill or high-risk procedures or those in whom CHF management is not fully optimised, IABP can be inserted preoperatively.^[9] The pre-operative preparation and optimisation have been explained in Table 3.^[4,9,10]

Premedication should be tailored and may include short acting anxiolytic/sedative like oral Alprazolam

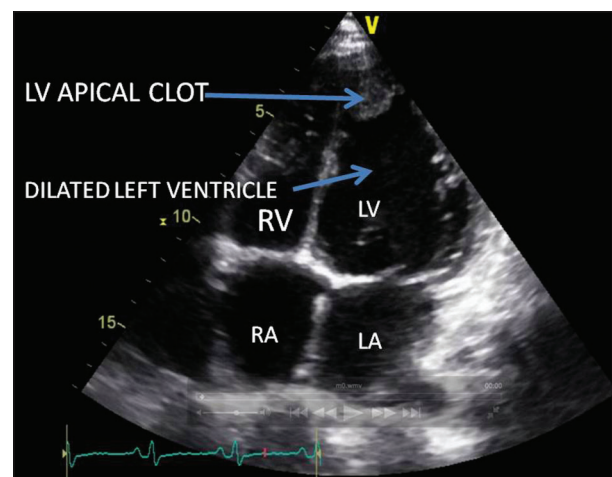


Figure 1: Dilated cardiomyopathy - Transthoracic echocardiography four-chamber view showing the dilated chamber with a left ventricular apical clot

or Midazolam. Regional anaesthesia or nerve blocks alone or in combination with general anaesthesia achieves the goals of anaesthesia and has minimal haemodynamic effects. American Society of Regional Anesthesia (ASRA) guidelines in the presence of anticoagulation must be adhered to.^[10,11]

Intraoperative management

Goals of anaesthesia are shown in Table 4.

Monitoring

In addition to basic monitoring, central venous pressure (CVP) monitoring allows preload and central venous saturation (ScVO₂) assessment provides an access for administering inotropes and vasoconstrictors. Direct arterial pressure monitoring enables early identification of haemodynamic changes. Pulmonary artery pressure monitoring is useful in sick patients or high-risk surgery or those in whom large fluid shifts is anticipated. The role of non-invasive methods to estimate cardiac output as well as estimate the extravascular lung water, global end diastolic volumes and other indices are important for assessing cardiac function. Pulse Index Continuous Cardiac Output monitoring (PiCCO), and Lithium Dilution Cardiac Output (LiDCO) estimate CO, SV, stroke volume variation (SVV) etc.

Transesophageal echocardiography (TEE) is also useful as it identifies causes of hypotension, response to inotropes or fluid loading, estimates preload, diastolic dysfunction, CO, regional wall motion and valve function. Monitoring tools are similar to all types of cardiomyopathies (as mentioned above) and can be chosen depending on institutional availability, or provider comfort.

Anaesthetic technique

These patients may become haemodynamically unstable due to the depressant effect of anaesthetics, fluid shifts and blood loss, which add to poor myocardial function. Thiopentone, propofol and inhalational agents cause vasodilation and myocardial depression. Etomidate, ketamine and narcotics have minimal haemodynamic effects.^[12] Benzodiazepines and nitrous oxide may cause cardiovascular depression.^[13,14]

Narcotics provide haemodynamic stability, and a balanced anaesthetic technique should be used. Slow induction should be carried out. Response of induction agents may be delayed due to prolonged circulation time, and additional doses may not be required.^[9]

High-risk patients include those with LVEF <25%, pulmonary capillary wedge pressure >20 mmHg, cardiac index <2 L/min/m², pulmonary artery hypertension, raised CVP and mitral and tricuspid regurgitation. Inotropic support is commonly required, and inodilators such as dobutamine, phosphodiesterase inhibitors or calcium sensitisers such as levosimendan may be used. Vasoconstrictor agents such as phenylephrine or norepinephrine are used to counteract the vasodilatory effect of anaesthetics and inodilators to maintain mean arterial pressure >60 mmHg.

Arrhythmias can be managed with lidocaine, amiodarone, diltiazem or cardioversion/defibrillation may be required.^[11]

Guidelines for management of patients with AICD or CRT devices should be adhered to, when these devices are present.

Regional anaesthesia provides post-operative pain relief, reducing sympathetically mediated heart rate and afterload increase.^[13,14] Adequate pain management contributes to haemodynamic stability.

| Action | Drugs and equipment |
|--------------------|---|
| Stop | ACE/angiotensin inhibitors (Can cause hyperkalaemia, renal dysfunction, intraoperative hypotension) Diuretics (Can cause hypokalaemia, hypomagnesaemia, intraoperative hypotension) Anticoagulant |
| Continue | Beta-blockers, antiarrhythmics, statins |
| Reprogram | AICD, CRT devices, deactivate antitachycardia function |
| Magnet can be used | |
| Optimise | Haemoglobin ^[5] |
| Use IABP | Preoperatively if indicated |
| Standby | Inotropes, pacemaker, defibrillator pads, defibrillator |

ACE – Angiotensin-converting enzyme; AICD – Automated internal cardioverter defibrillator; CRT – Cardiac resynchronisation therapy; IABP – Intra-aortic balloon pump

| |
|---|
| Minimize negative inotropic effect of anaesthetic drugs |
| Maintain preload despite increased LVEDP |
| Prevent increase in afterload |
| Maintain perfusion, control arrhythmias, hypotension, tachycardia |
| LVEDP – Left ventricular end-diastolic pressure |

POST-PARTUM CARDIOMYOPATHY

Post-partum cardiomyopathy is a rare form of DCM that occurs from the third trimester of pregnancy until the first 5 months after delivery. Incidence is 1 in 3000–10,000 pregnancies.^[15] Risk factors include maternal age >30 years, multiparity, African descent, obesity, multiple pregnancy, hypertensive disorders and cocaine use. Possible causes include viral myocarditis, abnormal immune response or a maladaptive response to haemodynamic stress of pregnancy. Clinical features and investigations are similar to those of DCM and can be erroneously attributed to normal physiological changes of pregnancy or known congenital heart disease in the parturient, delaying the diagnosis. Tachyarrhythmias and thromboembolism can occur. Diagnosis requires four criteria as shown in Table 5.^[15,16]

Medical management is similar to that of DCM. Beta blockers, nitrates, hydralazine, digoxin and loop diuretics are safe during pregnancy. Angiotensin-converting enzyme (ACE) inhibitors are teratogenic and cause increased foetal loss when used in second and third trimesters, cause hypotension in infants of breastfeeding mothers, and are not administered. Calcium channel blockers have negative inotropic action, and aldosterone antagonists are not recommended during pregnancy.^[17] Supportive therapy may include non-invasive or invasive ventilation, IABP, LVAD or extracorporeal membrane oxygenation and cardiac transplant for those who do not improve. Maternal optimisation is the mainstay, and if pregnant, early delivery may be necessitated.

PPCM is a leading cause of maternal death, and prognosis is indicated by New York Heart Association (NYHA) class or TTE findings on presentation. Usually, 30–50% improve, CHF, arrhythmias or thromboembolism being the most common causes for mortality.^[16,17]

Anaesthetic considerations

Goals of anaesthesia, [Table 3] anaesthetic technique and monitoring are similar to DCM. Mode of delivery

Table 5: Diagnostic criteria of post-partum cardiomyopathy

Heart failure developing towards the end of pregnancy or up to 5 months post-partum

Absence of another identifiable cause of cardiac failure

Absence of cardiac symptoms or disease before late pregnancy

Left ventricular dysfunction - defined as an EF <45% or reduced fractional shortening of <30%^[13,14]

EF – Ejection fraction

and anaesthetic technique is influenced by NYHA class, urgency of delivery and haemodynamic status. Vaginal delivery is a viable option if the parameters allow.

Early labour epidural analgesia reduces the sympathetic response of labour contractions, decreases afterload and can be used for anaesthesia if operative intervention is required.^[4,17]

Oxytocin reduces SVR and ergometrine increases afterload and should be used carefully. After the procedure, CHF may occur due to increase in preload following uterine contraction. The patient should be monitored in the Intensive Care Unit (ICU). Neuraxial anaesthesia is beneficial but should be performed in accordance with ASRA guidelines if on anticoagulants. Significant reduction in SVR should be treated with optimal fluids and vasopressors to maintain coronary and other organ perfusion. The volume expansion of the central compartment following uterine contraction can lead to relative hypervolemia and result in heart failure.^[11,15]

HYPERTROPHIC CARDIOMYOPATHY

In hypertrophic obstructive cardiomyopathy, myocyte architecture is disorganised which leads to asymmetrical hypertrophy of the myocardium with myocardial scarring. It is a relatively common genetic disorder of the heart, with a prevalence of 1 in 500.^[18] LV hypertrophy develops mainly in the septum extending to the anterior free wall and develops in the absence of an identifiable cause such as aortic stenosis or hypertension.^[19]

Systolic function is normal or increased. Interstitial fibrosis and increased LV mass lead to a small LV cavity and end-diastolic volumes which reduce stroke volume despite a normal LVEF.

Myocardial stiffness is increased, reducing chamber compliance, leading to diastolic dysfunction, poor effort tolerance, atrial arrhythmias and diastolic failure. LVEF diminishes at a later stage contributing to poor outcome. Heart size on X-ray chest is enlarged due to increased thickness or may be normal.^[18,19]

Myocardial ischaemia occurs without CAD due to abnormal coronary arteries, mismatch between coronary size and ventricular mass, increased oxygen consumption due to hypertrophy and increased

left ventricular end-diastolic pressure (LVEDP) compromising coronary perfusion.

Supraventricular and ventricular arrhythmias occur in varying degree due to disorganised cellular architecture.^[19]

The basal septal hypertrophy encroaches into the LV outflow tract (LVOT) causing obstruction and increasing blood flow velocity through the LVOT [Figure 2]. An abnormally positioned mitral valve apparatus may result in drag of the anterior mitral valve leaflet which is pushed into the LVOT causing systolic anterior motion (SAM) of anterior mitral leaflet. This accentuates the LVOT obstruction (LVOTO) and causes mitral regurgitation (MR). Other cause of SAM may be a Venturi effect caused by accelerated blood flow through the LVOT which pulls the anterior mitral leaflet into the LVOT. LVOTO is dynamic and can be precipitated by episodes of decreased preload, SVR or increased heart rate and contractility of the LV.^[18,20] MR can also occur in hypertrophic cardiomyopathy (HOCM) independent of SAM due to intrinsic mitral valve disease in up to 20% cases. The clinical course varies widely [Table 6].^[18,19,21]

Medication includes beta blockers and calcium channel blockers which exert a negative inotropic effect, decrease heart rate and increase diastolic filling time improving coronary perfusion pressure and CO. They should be continued in the perioperative period.^[6]

Diuretics may be given for CHF but with caution, as a low preload may precipitate LVOTO. Arrhythmias

are treated with amiodarone. AICD is used to prevent sudden death. Atrial arrhythmias may be cardioverted and those in atrial fibrillation may be on anticoagulants.^[18,22]

Patients not responding to medical therapy may need surgery for LVOTO reduction or medical ablation by alcohol injection into septal arteries. Mitral valve replacement may be required in some. Mortality is 1%, but can increase to 5% in those with ventricular arrhythmias. LVOTO occurs in only 25% cases.^[23]

Anaesthetic considerations

Pre-operative assessment

Patients without symptoms, auscultatory finding, ECG changes or a family history of HOCM may go undiagnosed at pre-operative evaluation.^[24]

Pre-anaesthetic evaluation should include NYHA functional class stratification, cardiac and respiratory symptomatology, medications, arrhythmias, stroke or CHF and presence of AICD.

Adequate premedication allays anxiety and decreases sympathetic stimulation. Pre-operative volume expansion can reduce the incidence of hypotension on induction of anaesthesia and minimise the effects of positive pressure ventilation.

Beta blockers or calcium channel blockers and hydration should be continued preoperatively. If an AICD is present, its reprogramming and deactivation of antitachycardia capability should be carried out. Post-operative intensive care is required depending

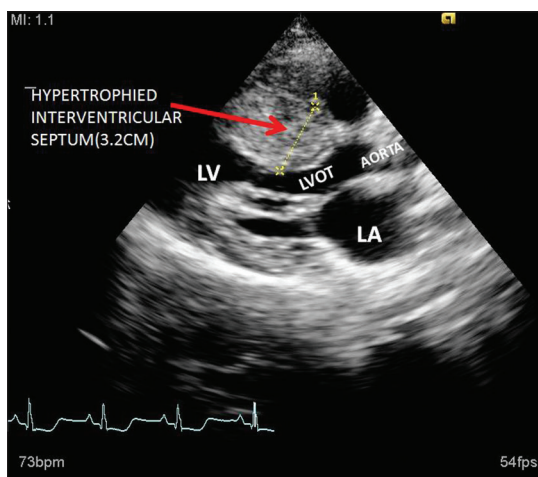


Figure 2: Hypertrophic cardiomyopathy - Transthoracic echocardiography parasternal long axis view showing the hypertrophied interventricular septum (3.2 cm)

Table 6: Presentation of hypertrophic cardiomyopathy patients

| Clinical examination and investigations | Features |
|---|---|
| Signs and symptoms | Fatigue, weakness, dyspnoea, syncope, sudden death |
| ECG | ST-T abnormalities, Q waves, atrial arrhythmias, premature ventricular contractions |
| Echocardiography | Concentric LV hypertrophy, raised LVEDP, raised LVOT gradient, mitral regurgitation, SAM, dagger shaped Doppler velocity envelope across aortic valve |
| Rhythm devices | AICD |
| Medications | beta blockers, antiarrhythmics, anticoagulants |
| Interventions | Medical therapy, surgical LVOT resection, Medical alcohol septal ablation, Mitral valve replacement |

ECG – Electrocardiogram; EF – Ejection fraction; LV- left ventricle; LVEDP – Left ventricular end-diastolic pressure; AICD – Automated internal cardioverter defibrillator; LVOT- left ventricular outflow tract; SAM- systolic anterior motion

on severity of disease, as up to 60% patients may experience myocardial ischemia, arrhythmias and CHF.^[6]

There are no guidelines for anaesthetic management and cohort studies and case reports guide management.

Intraoperative management

Diastolic dysfunction makes the heart sensitive to changes in volume, SVR and contractility.^[19]

Sympathetic stimulation following anxiety, intubation, surgical incision, decrease in preload, afterload due to anaesthetic agents, blood loss and post-operative pain can cause haemodynamic compromise. Sinus rhythm is important to maintain CO. Sudden atrial fibrillation may require cardioversion. Haemodynamic goals are similar to aortic stenosis patients: a slow heart rate, sinus rhythm and preload and afterload maintenance.^[6,18]

Extent of monitoring depends on the extent of surgery, potential for fluid shifts, degree of LVOTO, the presence of CHF and other comorbidities, and may include direct arterial pressure monitoring and preload estimation. A central venous catheter is useful for ScVO₂ estimation and delivering vasoactive medication while dynamic parameters of preload are more reliable for fluid responsiveness. However, CVP and PCWP may not reflect LVEDP accurately in the presence of diastolic dysfunction and use of trends would be more reliable. The most useful monitor is TEE which can determine the cause of haemodynamic disturbance such as hypovolemia, LVOTO or SAM.^[18,19,21]

Induction of anaesthesia reduces venous tone and preload that can increase LVOTO. Anaesthetic agents should be selected to minimise SVR reduction and prevent tachycardia. Propofol can be avoided, narcotics are safe and induction should be titrated slowly. Vecuronium is preferred as it does not have haemodynamic or histamine-releasing effects and isoflurane or sevoflurane have minimal negative inotropic effect.^[19] Small tidal volumes and rapid respiratory rate should be used to minimise reduction in venous return. Hypertension should be treated with beta blockade and increasing depth of anaesthesia; vasodilator agents should be avoided. Hypotension is treated with change in position, volume expansion and vasopressors that do not increase heart rate or contractility, for example, norepinephrine and vasopressin.^[18]

Both general and neuraxial anaesthesia can be used as long as pathophysiology is understood and adequate monitoring is utilised.^[19] A slow, controlled titration of medication through an epidural catheter is preferred over spinal anaesthesia with a goal of maintaining preload, afterload, avoiding sympathetic stimulation. Vasoconstrictors may be required.

Cardiac arrest in these patients may be difficult to treat, and fluids and rhythm management are the mainstay.

The parturient

Due to increased blood and red cell volume, pregnancy is usually well tolerated even though there is decreased SVR and impaired venous return due to vena cava compression. However labour-induced contractions cause sympathetic stimulation and bearing down mimics the Valsalva manoeuvre which can reduce CO. Graded epidural analgesia is well tolerated and general analgesia is required only for obstetric indications. Oxytocin should be used carefully as it drops SVR and causes tachycardia. Pulmonary oedema may occur after delivery due to uterine blood adding to the maternal circulation in the presence of diastolic dysfunction. Vasoconstrictors and beta blockers are used. Diuretics are used carefully.

Postoperatively, they should be monitored in the ICU. Shivering, pain, hypoxia, hypercarbia and anxiety should be avoided and euvolemia maintained.^[18,21]

TAKOTSUBO CARDIOMYOPATHY

Takotsubo cardiomyopathy is a rare condition of reversible severe LV dysfunction and characterised by chest pain, dyspnoea, ST-T changes in ECG, elevated cardiac enzymes, regional wall motion abnormalities on echocardiography, haemodynamic instability, ventricular arrhythmias, pulmonary oedema, cardiogenic shock or cardiac arrest without angiographic evidence of CAD.^[25]

It mostly follows emotional or physiologic stressful situations such as general anaesthesia, road traffic accidents, cerebral events, pheochromocytoma, anaphylactic shock, antidepressant overdose and attempted suicidal hanging and has a predilection for menopausal females, with an incidence of 0.00006%.^[25]

ECG changes may include prolonged QTc interval which resolve in 1–2 days, ST-T changes, Q waves, resolve by discharge from hospital and T inversion resolves slowly.^[27]

This condition is also known as apical ballooning syndrome and broken heart syndrome [Figure 1]. Echocardiography shows akinesia of apical or midventricular segments leading to systolic dysfunction. The normal basal segments become hypercontractile, giving a ballooned out appearance of the apical or mid-cavity segments [Figure 3]. Ballooning may lead to altered spatial relationships between mitral leaflets and subvalvular apparatus, which may result in MR and dynamic LVOTO causing SAM.^[25,26]

Reversible myocardial ischaemia is seen on myocardial perfusion imaging, and positron emission tomography and magnetic resonance imaging confirm LV dysfunction. Biopsy shows lymphocytic infiltrates. Plasma levels of brain natriuretic peptide, catecholamines, cardiac enzymes and metanephrine are found to be elevated.^[27]

Occurrence of an adrenergic storm is the suggested cause.^[28] Possible mechanisms are:

- Coronary artery spasm
- Catecholamine-mediated myocyte injury causing myocardial stunning, in patients with a possible genetic predilection. Catecholamine release may be endogenous or following administration (epinephrine, dobutamine)
- A neuropsychiatry link is also suggested.^[27]

Optimal therapy is yet to be defined. Beta-blockers, diuretics and ACE inhibitor and vasodilators have been used.^[28] Adrenergic agonists and antiadrenergic therapy (beta adrenergic blockers or alpha 2 agonists) and QT prolonging medications are to be avoided.

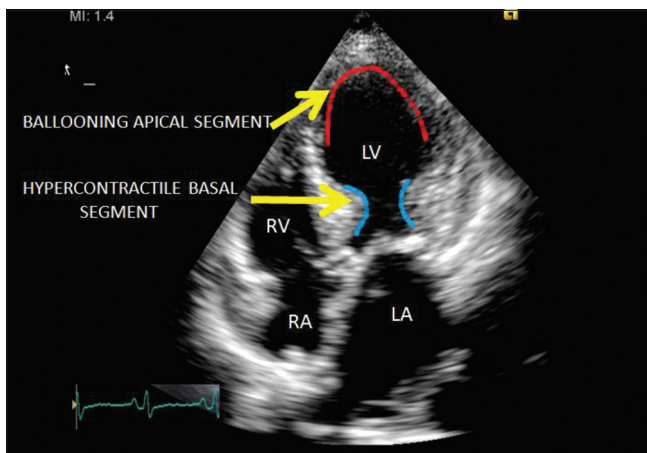


Figure 3: Takotsubo cardiomyopathy - Transthoracic four-chamber view showing the hypercontractile basal segment and the ballooning apical segment

Anaesthesiologists may confront this syndrome in patients who present with (a) acute coronary syndrome; (b) those admitted for other disease conditions such as acute intracranial disease, aneurysmal subarachnoid haemorrhage, pheochromocytoma and other critical illnesses, just before, during or after anaesthesia administration and (c) patients who have undergone the syndrome previously. There is no consensus regarding the anaesthetic management.^[25]

A principle anaesthetic goal is to avoid psychological and physical stress that could trigger acute cardiomyopathy in susceptible patients. Thorough patient counselling, effective premedication and pre-operative beta blocker therapy before transfer to operating room are advisable.^[28]

Laryngoscopy, intubation, extubation, emergence and inadequate post-operative pain control may increase catecholamine levels, and optimal anaesthesia/analgesia is required in these phases.

It is suggested that regional anaesthesia may be beneficial but there is no consensus.^[25]

It is unclear whether administration of inotropic drugs to treat systolic dysfunction is harmful. Inotrope of choice remains unclear, though Milrinone, a phosphodiesterase inhibitor and calcium sensitizer, levosimendan are suggested. Mechanical support of circulation with IABP or LVAD is an option to tide over periods of crisis.^[28] Beta blocker therapy may not be haemodynamically tolerated or could be potentially hazardous. Beta agonists should be avoided or used carefully, vasopressors may be used and supportive treatment for CHF should be instituted.

LV dysfunction resolves within 2–4 weeks. Most cases recover spontaneously with a mortality risk of 0%–8%. Recurrence occurs in 2%–5% cases.^[27]

CONCLUSION

Anaesthesia administration for patients with cardiomyopathy can lead to perioperative morbidity and mortality during elective and emergency surgery. Therefore, anaesthesia and post-operative care have to be planned and monitored, for which a thorough understanding of the type of cardiomyopathy and their pathophysiology is very much essential. Anaesthesiologists should expand their horizon from operating room to ICU with a thorough understanding

of non-invasive and invasive monitoring methods and a basic knowledge of transthoracic echocardiography.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Maron BJ, Towbin JA, Thiene G, Antzelevitch C, Corrado D, Arnett D, *et al.* Contemporary definitions and classification of the cardiomyopathies: An American Heart Association Scientific Statement from the Council on Clinical Cardiology, Heart Failure and Transplantation Committee; Quality of Care and Outcomes Research and Functional Genomics and Translational Biology Interdisciplinary Working Groups; and Council on Epidemiology and Prevention. *Circulation* 2006;113:1807-16.
2. Elliott P, Andersson B, Arbustini E, Bilinska Z, Cecchi F, Charron P, *et al.* Classification of the cardiomyopathies: A position statement from the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J* 2008;29:270-6.
3. Arbustini E, Narula N, Dec GW, Reddy KS, Greenberg B, Kushwaha S, *et al.* The MOGE(S) classification for a phenotype-genotype nomenclature of cardiomyopathy: Endorsed by the world heart federation. *J Am Coll Cardiol* 2013;62:2046-72.
4. Oliver WC, Mauermann WJ, Nuttal G. Uncommon cardiac diseases. In: Kaplan J, Reich DL, Savino JS, editors. *Kaplan's Cardiac Anesthesia*. 6th ed., Ch. 22. Philadelphia: Elsevier Saunders; 2011. p. 675-736.
5. Falk RH, Hershberger RE. The dilated, restrictive, infiltrative cardiomyopathies. In: Mann DL, Zipes DP, Libby P, Bonow RO, editors. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 10th ed., Ch. 65. Philadelphia: Elsevier Saunders; 2015. p. 1551-74.
6. Popescu WM. Heart failure and cardiomyopathies. In: Hines R, Marschall K, editors. *Stoelting's Anesthesia and Coexisting Disease*. 6th ed., Ch. 6. Philadelphia: Elsevier Saunders; 2012. p. 120-43.
7. Davies MR, Cousins J. Cardiomyopathy and anaesthesia. *Continuing Education in Anaesthesia, Critical Care & Pain* 2009;9(6) 189-93.
8. Desai AS, Fang JC, Maisel WH, Baughman KL. Implantable defibrillators for the prevention of mortality in patients with nonischemic cardiomyopathy: A meta-analysis of randomized controlled trials. *JAMA* 2004;292:2874-9.
9. Thiagarajah PH, Thiagarajah S, Frost EA. Anesthetic considerations in patients with cardiomyopathies – A review. *Middle East J Anaesthesiol* 2009;20:347-54.
10. Narouze S, Benzon HT, Provenzano DA, Buvanendran A, De Andres J, Deer TR, *et al.* Interventional spine and pain procedures in patients on antiplatelet and anticoagulant medications: Guidelines from the American Society of Regional Anesthesia and Pain Medicine, the European Society of Regional Anesthesia and Pain Therapy, the American Academy of Pain Medicine, the International Neuromodulation Society, the North American Neuromodulation Society, and the World Institute of Pain. *Reg Anesth Pain Med* 2015;40:182-212.
11. Kaya C, Koksak E, Ustun YB, Semizoglu Y, Yilmaz N. Anesthetic management of hysterosalpingoophorectomy in a case with severe idiopathic dilated cardiomyopathy. *Med Arch* 2014;68:144-6.
12. Schechter WS, Kim C, Martinez M, Gleason BF, Lund DP, Burrows FA, *et al.* Anaesthetic induction in a child with end-stage cardiomyopathy. *Can J Anaesth* 1995;42:404-8.
13. Narang S. Anesthetic management of dilated cardiomyopathy – A case report. *Middle East J Anaesthesiol* 2007;19:243-9.
14. De Hert SG, Van der Linden PJ, ten Broecke PW, Vermeylen KT, Rodrigus IE, Stockman BA, *et al.* Effects of desflurane and sevoflurane on length-dependent regulation of myocardial function in coronary surgery patients. *Anesthesiology* 2001;95:357-63.
15. Thompson L, Hartsilver E. Peripartum cardiomyopathy; 2015. Available from: <http://www.wfsahq.org/resources/update-in-anaesthesia>.
16. Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E, *et al.* Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: A position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. *Eur J Heart Fail* 2010;12:767-78.
17. Johnson-Coyle L, Jensen L, Sobey A, American College of Cardiology Foundation. American Heart Association. Peripartum cardiomyopathy: Review and practice guidelines. *Am J Crit Care* 2012;21:89-98.
18. Hensley N, Dietrich J, Nyhan D, Mitter N, Yee MS, Brady M, *et al.* Hypertrophic cardiomyopathy: A review. *Anesth Analg* 2015;120:554-69.
19. Maron PJ, Olivetto I. Hypertrophic cardiomyopathy. In: Mann DL, Zipes DP, Libby P, Bonow RO, editors. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 10th ed., Ch. 66. Philadelphia: Elsevier Saunders; 2015. p. 1574-88.
20. Maron BJ. Hypertrophic cardiomyopathy: A systematic review. *JAMA* 2002;287:1308-20.
21. Melacini P, Maron BJ, Bobbo F, Basso C, Tokajuk B, Zucchetto M, *et al.* Evidence that pharmacological strategies lack efficacy for the prevention of sudden death in hypertrophic cardiomyopathy. *Heart* 2007;93:708-10.
22. Poliac LC, Barron ME, Maron BJ. Hypertrophic cardiomyopathy. *Anesthesiology* 2006;104:183-92.
23. Franz WM, Müller OJ, Katus HA. Cardiomyopathies: From genetics to the prospect of treatment. *Lancet* 2001;358:1627-37.
24. Hessel EA 2nd, London MJ. Takotsubo (stress) cardiomyopathy and the anesthesiologist: Enough case reports. Let's try to answer some specific questions! *Anesth Analg* 2010;110:674-9.
25. Banerjee S. Takotsubo cardiomyopathy: A review. *JSM Atheroscler* 2016;1:1011.
26. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-tsubo or stress cardiomyopathy): A mimic of acute myocardial infarction. *Am Heart J* 2008;155:408-17.
27. Littlejohn FC, Syed O, Ornstein E, Connolly ES, Heyer EJ. Takotsubo cardiomyopathy associated with anesthesia: Three case reports. *Cases J* 2008;1:227.
28. Hinkelbein J, Mey C, Brinker G, Pfister R, Böttiger BW. Case report of tako-tsubo cardiomyopathy associated with repetitive anaesthesia in a female patient with tako-tsubo cardiomyopathy. *BMC Anesthesiol* 2015;15:39.