BMH MEDICAL JOURNAL

BMH Medical Journal 2015;2(3):71-73

Brief Review

Safely Navigating Patients With Hypertrophic Obstructive Cardiomyopathy (HOCM) During Anaesthesia

MC Rajesh DA, MD, MBA

Baby Memorial Hospital, Kozhikode, Kerala, India. PIN: 673004

Address for Correspondence: Dr. MC Rajesh DA, MD, MBA, Senior Consultant Anaesthesiologist, Baby Memorial Hospital, Kozhikode, Kerala, India. PIN: 673004. E- mail: rithraj2@yahoo.co.in

Keywords: Hypertrophic Obstructive Cardiomyopathy, HOCM, anaesthesia

In order to have an uneventful peri-operative period it is imperative for anaesthesiologist to maintain patients in perfect haemodynamics. But patient's underlying cardiac status has lot to do with the patient's peri-operative course. Hypertrophic obstructive cardiomyopathy (HOCM) is one such condition which is relatively common with an incidence of 1 in 500 [1]. In order to safely cruise patients during anaesthesia, it is important for anaesthesiologists to have a basic understanding of the pathophysiology, haemodynamic changes and anaesthetic implications of disease. If adequate anticipation and peri-operative care are not taken, anaesthesia and surgery can complicate the peri-operative outcome [2]. Intraoperative factors like increase in myocardial contractility, tachycardia, hypotension or vasodilation can precipitate an exacerbation of the obstruction [2].

HOCM is a genetically inherited condition with basic histological change as abnormal myocytes. There is unusual myocardial hypertrophy developing without pressure or volume overload [3]. In the initial stages of disease, hypertrophy may be confined to septum. But as the disease advances, it can extend to the free wall of the left ventricle, and finally can result in concentric hypertrophy of left ventricle. This can result in a pressure gradient between apical left ventricular chamber (LV) and left ventricular outflow tract (LVOT). LVOT obstruction thus created can result in an increase of LV pressure further leading to hypertrophy, reduced compliance and worsening LVOT obstruction [4]. Twenty to thirty percent of hypertrophic cardiomyopathy patients develop significant septal hypertrophy that can even obstruct the aortic outflow, resulting in sudden unexplained death [5]. Systolic anterior motion of mitral valve can lead to mitral regurgitation [6]. Stiff and hypertrophied ventricle with impaired compliance results in diastolic dysfunction [6].

Pre-operative echocardiogram is important to reveal left ventricular function. This could be normal to supranormal with high ejection fraction [7]. Abnormalities of ventricular relaxation, rhythm disturbances, diastolic dysfunction, moderate mitral regurgitation and septal motion abnormalities are all reflected on the echocardiogram [8]. Electrocardiographic (ECG) abnormalities noted are increased precordial voltage and non specific ST-T changes [9]. Asymmetrical septal hypertrophy produces deep narrow Q waves in lateral and leads. This can be confused with ECG finding in myocardial infarction. Left ventricular diastolic dysfunction may result in compensatory left atrial

hypertrophy, with signs of left atrial enlargement. Atrial fibrillation, ventricular dysrhythmias and supraventricular

Medical management of HOCM includes treatment with beta adrenergic receptor antagonists, which should be continued in the peri-operative period [10]. Many of the symptomatic patients for surgery come with an Automatic implantable cardioverter defibrillator (AICD) in situ. In fact AICD has become more popular and the equipment has evolved over a time to be very sophisticated. Understanding the functions of the device is essential for optimal care of the patient and equipment. Septal ablation with ethanol is a relatively new modality and successful ablation can result in immediate decline of LVOT gradient [11].

Intraoperative monitoring include pulse oximetry, ECG, non invasive BP, invasive BP, automated ST segment analysis, central venous pressure (CVP), end-tidal carbon dioxide (ETCO2), temperature and urine output monitoring. Use of trans-oesophageal echocardiogram is strongly advocated.

The goal of anaesthesia is to ensure adequate ventricular filling with slow heart rate and modest expansion of intravascular volume [12]. Factors which can exacerbate the outflow tract obstruction like increase in myocardial contractility or a decrease in systemic vascular resistance has to be avoided meticulously [12]. So while anaesthetizing these patients, considerations should be given to maintain adequate intravascular volume and venous return, maintain systemic vascular resistance, slow heart rate, with sinus rhythm and aggressively treat atrial fibrillation and other tachyarrhythmias [12]. Due attention should be paid to post-op analgesia, since it can result in cathacholamine release and development of tachyarrythmias. Any potential sympathetic response should be minimized during the course of anaesthesia. Patients with HOCM are at risk for peri-operative development of of arrhythmias and development of ventricular arrhythmias is very common [13]. Routine infective endocarditis prophylaxis is no longer recommended [14]. Because of potential peripheral vasodilation, regional anaesthesia is better avoided [15]. But there are reports in literature, of caesarean delivery being successfully undertaken with regional anaesthesia [12, 16].

Managing a victim of cardiac arrest in HOCM is different and the pathophysiology of disease has to be kept in mind. Use of ionotropic agents is contraindicated if there is left ventricular outflow obsrtruction, as it can worsen the situation. Alpha agonists, IV fluids and rapid correction of arrhythmias are more justified in this situation. In fact, application of external defibrillator pads before inducing the patient is highly recommended [17].

Conclusion

The management HOCM includes through understanding of the pathophysiological process of disease. It is imperative to maintain specific haemodynamic goals to prevent the intraoperative complications associated with it.

References

1. Maron BJ, Gardin JM, Flack JM, Gidding SS, Kurosaki TT, Bild DE. Prevalence of hypertrophic cardiomyopathy in a general population of young adults: echocardiographic analysis of 4111 subjects in the CARDIA Study. Circulation 1995; 92:785-9.

2. Sahoo RK, Dash SK, Raut PS, Upasani CB. Peri-operative anaesthetic management of patients with hypertrophic cardiomyopathy for non cardiac surgery. A case series Ann Card Anaesth 2010;13:253-6.

3. Paul G Barash, Bruce F, Cullen, Robert K Stolting. Clinical Anaesthesia. Sixth edition, 2009;1080-81.

4. Robert R, Sigwart U: New concepts in hypertrophic Cardiomyopathies, part ll.Circulation 2001; 104:2249.

5. Wigle ED, Rakowski H, Kumball BP. Hypertrophic obstructive cardiomyopathy: Clinical spectrum and treatment. Circulation;1995,92:1680-92.

6. Konstadt S: Anesthesia for non-cardiac surgery in the patient with cardiac disease. Can J Anesth; 2005, 52:R1-R3.

7. Cregg N, Cheng DC, Kaiski JM et al. Morbidity outcome in patients with HOCM undergoing cardiac septal myomectamy:Early extubation anaesthesia versus high dose opioid anaesthesia technique. J Cardiothoracic Vasc Anaesthesia 1999;13:47-52.

8. Shah P M, Tylor RD, Wrong M. Abnormal mitral valve coaptation in hypertrophic cardiomyopathy. Am J Cardiol 1981;48:258-62.

9. Daniel D, Savage MD. Electrocardiographic finding in patients with Obstructive and Nonobstructive Hypertrophic cardiomyopathy. Circulation 1978;58:402-8.

10. Fairley CJ, Clark JT. Use of esmolol in a patient with hypertrophic obstructive cardiomyopathy. Br. J. Anaessth 1995;75:801-4.

11. Spencer WH,Roberts R. Alcohol septal ablation in Hypertrophic obstructive cardiomyopathy. The need for registry. Circulation 2000;102.

12. M C Rajesh, Ravi Varma, Lima P, E K Ramdas. Anaesthetic management of severe hypertrophic obstructive cardiomyopathy with bronchial asthma for emergency caesarean section. JIMA 2012;110:745-6.

13. Anderson KP, Stinson EB, Derby GC. Vulnerability of patients with obstructive hypertrophic cardiomyopathy to ventricular arrhythmia induction in the operating room. Analysis of 17 patients. Am J Cardiol 1983; 51:811.

14. Wilson W, Taubert K, Gewitz M. Prevention of Endocarditis. Guidelines from the American Heart Association. Circulation 2007;116:1736-54.

15. Thompson RC, Liberthson RR, Lowestein ER. Peri-operative risk of non-cardiac surgery in HOCM. JAMA 1985;254.

16. Paix B, Cyna A, Belperio P, Simmons S. Epidural analgesia for labour and delivery in a parturient with HOCM. Anaesth Intensive Care 1999;27:59-62.

17. Poliac LC, Barron ME, Maron BJ. Hypertrophic cardiomyopathy. Anaesthesiology 2006;104:183-92.