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Use of Airway Blocks in Patients with Dilated Cardiomyopathy

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Abstract: *Dilated Cardiomyopathy is characterised by an enlarged left ventricle(left ventricular diastolic dimension >60mm) with decreased systolic function(LV ejection fraction <30%). We report the use of Airway Blocks in anaesthetic management of patient with dilated cardiomyopathy.*
Key Words: *Dilated Cardiomyopathy, Airway Blocks.*

I. Introduction

Various cardiovascular responses like hypertension, tachycardia, and dysrhythmias are seen during laryngoscopy and endotracheal intubation. These changes may be associated with myocardial ischemia, heart failure or cerebral haemorrhage in severely ill.[1]
Using Airway Blocks is one such technique to attenuate these unwanted hemodynamic responses.

II. Case Report

A 28 years old female, weighing 45 kg was posted for stoma closure. She was a known case of idiopathic dilated cardiomyopathy since 4 years and was diagnosed with ileocecal tuberculosis for which she had undergone resection with stoma formation under general anaesthesia, 6 months ago details of which were not available.

On pre- anaesthetic evaluation her heart rate was 84/min and regular. The blood pressure was 96/56 mm hg, with respiratory rate of 15/min. On auscultation there were no rales, heart sounds were normal, no signs of congestive failure.

Patient's symptoms were well controlled on Furosemide 20mg BD, Digoxin 0.25mg OD, Spironolactone 25mg OD, Ramipril 2.5mg HS, Carvedilol 3.125mg OD. Patient was also taking anti tuberculosis treatment since 6 months.

III. Investigations

Preoperative 12 lead ECG showed poor progression or R wave in leads V1-V4, T wave inversion
In lead V5-V6.

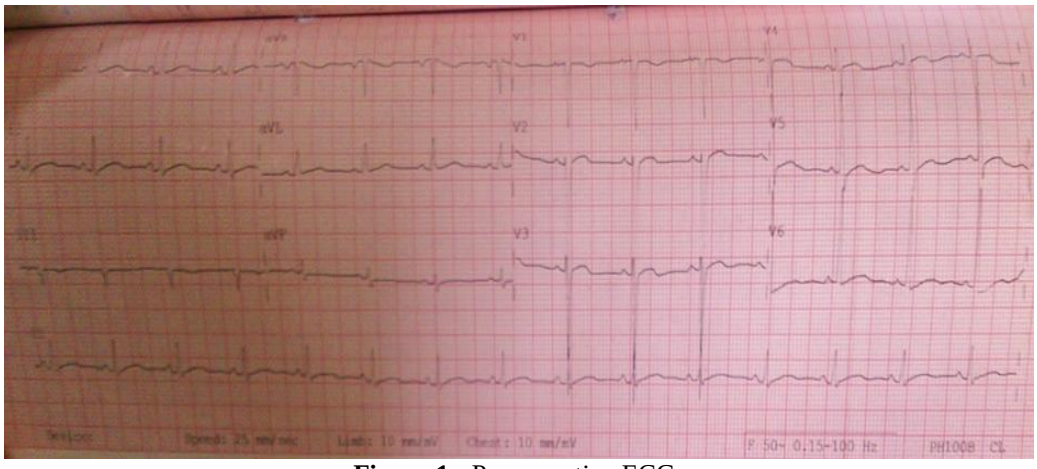

Figure 1 - Pre-operative ECG.

Chest X-Ray showed Cardiomegaly, lung fields were clear.

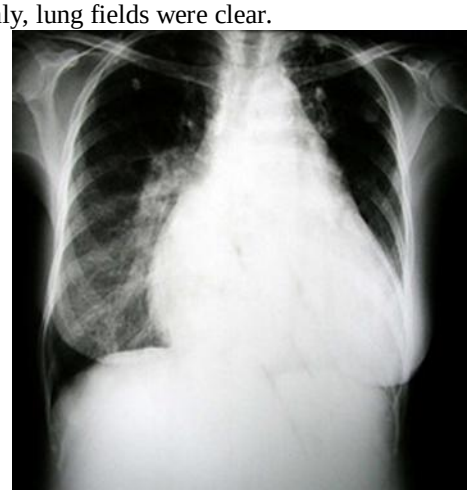

Figure 2 – Pre-operative Chest X-ray.

Echocardiography showed global LV hypokinesia, severe MR, compromised LV function EF of 20-25%, e/o pericardial effusion.All investigations were within normal limits except liver enzymes which were slightly on the higher side.

A high risk consent was obtained and general anaesthesia was planned .No premedication was advised.

Preoperative, patient Blood Pressure (BP) was 94/60 mm Hg, Heart Rate (HR) was 76/min and oxygen saturation (SaO2) was 96% on room air. A left arterial line and right internal jugularcentral catheter was placed under local anaesthesia prior to the induction of anaesthesia. Parameters monitored were ECG, End tidal CO_2, SpO_2, Invasive blood pressure monitoring, central venous pressure was 10-12 cm H20, oxygen was supplemented with Hudson mask at 6L/min. Sedation was given withIV Midazolam 0.5mg, IV Fentanyl 50 µg & IV Ketamine 20 mg. Posterior pharyngeal wall was anesthetised using 10% Lignocaine spray. Bilateral superior laryngeal nerves were blocked, using 2cc of 1% Lignocaine, at the level of greater cornu of the hyoid bone. Transtracheal block was given using 2cc of 4% Lignocaine.

The patient was induced with IV Fentanyl 100 µg, IV Ketamine 30 mg & IV Atracurium 20 mg. Patient was intubated with cuffed endotracheal tube 7.0 mm ID.

Table -1 Hemodynamic Parameters.

	HR/min	SBP mm hg	DBP mm hg	MAP mm hg
Pre-operative	76	94	60	71
Before nerve block	76	106	68	80
Induction	70	98	62	74
Post intubation	78	104	64	78

Anaesthesia was maintained with O2/ N2O (60:40), Sevoflurane (MAC 1-1.5%) and intermittentAtracurium 5mg IV.

Surgery was completed in 2hrs 30 minutes. Intraoperatively vitals were maintained as follows MAP 60-70 mm Hg, HR 60-70 /min, SpO_2 99-100%, CVP 8-10 cm H2O, $EtCO_2$30-35 mm Hg.

Intraoperatively there was an episode of hypotension with MAP of 47 mm hg, CVP of 4-6 cm H2O, patient was given 100 cc colloid slowly, following which there was increase in MAP to 68 mm hg and CVP to 6-8 cm H2O.

For pain relief, 15 cc of local infiltration with 0.125% Bupivacaine was given at the incision site and IV Tramadol 50 mg.

Total fluid given was 200 ml crystalloid, 100 ml colloid. Blood loss was around 100 ml and urine output was around 100 ml.

At the end of surgery patient was reversed using IV Glycopyrolate 300 µg and IV Neostigmine 2.5 mg and extubated after adequate return of tone power and reflexes.

Patient was then shifted to the ICU for further observation. Post operatively vitals were stable and all investigations were within normal limits. Patient was shifted toward after 24 hrs observation in ICU and discharged after 7 days.

IV. Discussion

Dilated Cardiomyopathy (DCM) is a syndrome characterized bycardiac enlargement and impaired systolic function of one or bothventricles. Various etiologiesfor DCM include infectious mostly viral, toxic, metabolic, familial, idiopathic, peripartum.Clinical features range from asymptomatic to overt cardiac failure, usually left sided symptoms predominate.[2]

Patients are also prone to arrhythmias, systemic and pulmonary embolism.[3]

Management for DCM include drugs and pacemaker for those with incordinate chamber contraction.[4]

Goals of anaesthetic management include:-

- Avoid myocardial depression
- Maintain normovolemia
- Avoid increase in ventricular afterload

Drugs like ketamine,etomidate and narcotics have minimal depressing effect on cardiac function and are used frequently.[5] We used ketamine and fentanyl for induction.

Methods to improve cardiac output include

- Inotropes
- biventricular synchronized pacing
- intra-aortic balloon pump may be required.[6]

Skeletal muscle paralysis is to be provided by nondepolarizing muscle relaxant that lack significant cardiovascular effects.[6]Atracurium was selected as muscle relaxant as level of liver enzymes were on higher side.

Sensory innervation of upper airway, vocal cords and trachea is by superior laryngeal nerve and recurrent laryngeal nerve respectively.

Direct laryngoscopy and endotracheal intubation causes sympathetic stimulation and catecholamine release which can be deleterious to such patients.

Different drugs to prevent these responses include, calcium channel blockers[7], betablockers[8], intravenous lignocaine[7] and intravenous narcotics[8]. These drugs are associated with side effects like hypotension which is not desirable for patients with dilated cardiomyopathy.

Transtracheal lignocaine blocks the cardiovascular responses to endotrachealintubation. Airway Blocks can reduce the dose of narcotics required to block the cardiovascular responses to endotrachealintubation, and may be a useful strategy whencombined with other drugs to decrease the risk ofhypotension or delayed emergence.[9]

V. Conclusion

Airway blocks helps in reducing the sympathetic stimulation during direct laryngoscopy and endotracheal intubation, thus decreasing the incidence of cardiovascular responses like hypertension, tachycardia, and dysrhythmias. It also reduces the requirement of induction agents, thus decreasing the chances of post induction hypotension.

References

[1]. Fox EJ, Sklar GS, Hill CH, Villanueva R, King BD.Complications related to the pressor response to endotrachealintubation. Anaesthesiology 1977; 47: 524–5.
[2]. William G, Fuster V: Review Article- Idiopathic dilated Cardiomyopathy. New England Journal of Medicine; 331:1564-75, 1994.
[3]. Stoelting RK, Dierdorf SF. Cardiomyopathy. In: Stoelting RK (Ed).Anaesthesia and Coexisting Disease (3rd ed). New York: ChurchillLivingstone 1993; 97-102.
[4]. Molhoek SG, Bax JJ, Erven RV. Effectiveness of resynchronizationtherapy in patients with end stage heart failure. Am J Cardiol. 2002;90:379-83.
[5]. Schechter WS, Kim C, Martinez M. Anesthetic induction in a child withend-stage cardiomyopathy. Can J Anaesth. 1995; 42:404-08.
[6]. Kaur H, Khetarpal R, Aggarwal S. Dilated cardiomyopathy: an anaesthetic challenge. Journal of Clinical and Diagnostic Research: JCDR. Jun 2013; 7(6): 1174-1176.
[7]. Fujii Y, Saitoh Y, Takahashi S, Toyooka H. Diltiazem lidocaine combination for the attenuation of cardiovascularresponses to tracheal intubation in hypertensivepatients. Can J Anaesth 1998; 45: 933–7.
[8]. Korpinen R, Saarnivaara L, Siren K, Sarna S.Modification of the haemodynamic responses to inductionof anaesthesia and tracheal intubation with alfentanil,esmolol and their combination. Can J Anaesth1995; 42: 298–304.
[9]. Takita K, Morimoto Y, Kemmotsu O: Tracheal lidocaine attenuates the cardiovascular response to endotracheal intubation. Can J Anaesth 2001; 48:732-736.