Case Report

Anaesthetic management of a patient of post chemotherapy for carcinoma ovary with hypertrophic cardiomyopathy

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Abstract

Cancer is treatable if detected early. Cancer is the one of the leading cause of death in India. cancer patients deserve special anaesthetic considerations. It requires a very close cooperation among surgeon, anaesthesiologist and referring physician to assure the conduct of surgical procedures on the patient cancer with maximal saftey.
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INTRODUCTION

Hypertrophic cardiomyopathy is the disease of myocardium in which a portion of myocardium is hypertrophied. The incidence of HOCM is about 0.2% to 0.5% of general population. Anesthetic technique and the perioperative management of these patients must aim to maintain hemodynamic stability, maintaining adequate preload and after load. Intraoperative goals include maintaining sinus rhythm, minimal stressful stimuli and minimize or prevent dynamic LV outflow tract obstruction.

CASE REPORT

A 48 year old female patient with pain abdomen, with distention and vomiting since two months diagnosed as carcinoma ovary who had received 3 cycles of chemotherapy was posted for interval cytoreductive surgery. Her chemotherapeutic drugs were cispaltin, methotrexate and 5 fluorouracil. Past history revealed hypertension since 10 years and dyspnea on exertion since 4 years. Patient is on T Amlodipine 5mg OD with T chlorthiazide 2.5mg and T metaprolol 50mg OD. There was no family history of cardiac illness or sudden death. Airway examination is MPC-1. On physical examination pulsr rate-90/min, BP 130/96mmhg. Systemic examination of cardiovascular examination revealed systolic murmur of grade 3. Respiratory system normal. Routine investigations were within normal limits. Chest x-ray showed cardiomegaly with clear lung fields. ECG showed HR-98/min with LVH.2D Echo showed concentric LVH, dilated lt atrium, systolic anterior motion of mitral valve, systolic flutter of aortic valve, hypertrophic cardiomyopathy with LVOT gradient of 124mmhg with MR grade-1, LVEF-35%. Pt was advised to be take T. amlodipine, T chlorthiazide 2.5mg and T metaprolol 50mg on the morning of surgery. Cardiology opinion was taken. After securing iv line pre-induction parameters like HR, NIBP, SPO2, ECG were monitored. under strict asepsis epidural catheter was placed in L2-3 space. Premedicated with iv midazolam 0.03mg/kg, inj fentanyl 2mcg/kg. right internal jugular vein cannulated and cvp monitored. Induction done with IV titrated dose of inj etomidate 0.3mg/kg, inj vecuronium 0.1mg/kg intubation done with 7.5mm ET tube. Intraoperatively maintained with 02+N20 60:40, inj vecuronium 0.1mg/kg/hr with inj fentanyl 2mcg/kg/hr. Intraop ECG, pulseoximetry, CVP, Etco2, NIBP, monitored. IV fluids given according to CVP. Intraop period was uneventful.

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Patient was reversed with Neostigmine 0.05mg/kg with glycopyrrolate 8mcg/kg and extubated. Pt shifted to anaesthesia care unit, where pt was monitored for 24 hrs. postop period was uneventful. Postop fluid given according to CVP and urine output. Analgesia maintained with epidural fentanyl infusion.

DISCUSSION

There are two important aspects with regards to this patient.

1. Chemotherapy and anaesthesia concerns
2. Hypertrophic cardiomyopathy and chemotheraptic drug interactions.

Chemotherapy forms an important aspect of cancer treatment. With an increased number of patients surviving for a longer period of time, a number of patients, who have received chemotherapy, may be subjected to elective and emergency surgery, therefore it is essential to know the effects of the chemotherapeutic agents on normal organ systems. The toxicity of cancer. chemotherapy drugs and their relevance to perioperative anaesthesia management relates to the specific agents used, their cumulative dosage, and drug toxicity etc. The most common toxicities to chemotherapeutic agents include cardiac, pulmonary, hematologic, bone marrow, and gastrointestinal effects. Coagulopathies, thrombocytopenia, and anaemia with ulceration and bleeding of the gastrointestinal tract may often occur[1].

Table 1: Common complications associated with cancer chemotherapy agents

System toxicity	Chemotherapeutic agents2
Cardiac toxicity	Busulphan, cisplatin, cyclophosphamide, daunorubucin, 5-fluorouracil
Pulmonary toxicity	Methotrexate, bleomycin, busulphan, cyclophosphamide cytarabine,carmustine
Renal toxicity	Methotrexate, L-asparginase, carboplatin, ifosfamide,mitomycin-C
Hepatic toxicity	ActinomycinD,methotrexate, androgens, L-asparginase, busulphan, cisplatinum, azathiopine
CNS toxicity	Methotrexate, cisplatin, interferon, hydroxyurea, procarbazine, vincristine
SIADH secretion	Cyclophosphamide, vincristine

Anaesthetic considerations for patients after chemotherapy

The interaction between anaesthesiologist and a cancer patient starts with a preoperative visit for a surgical procedure. The goals of such a preoperative visit would be as follows:-

- To optimize patient's physical status.
- To assess effects of cancer and cancer therapies (chemotherapy radiotherapy, and surgery) on patient.

Some of the important features and care before planning anaesthesia in such a chemotherapy pt. Routine preanaesthetic check up and investigations have to be done. In pts receiving chemotherapy, special investigations might be required according to class of drugs and their side effects.

Acute and Subacute cardiotoxicity

It can occur immediately after a single dose or a course of anthracycline therapy. Acute toxicity commonly (40%) takes the form of ECG changes such as nonspecific ST-Tchanges, decreased QRS voltage, and QT prolongation. Decreased R wave amplitude has been thought by some to signal development of chronic cardiomyopathy later, though it is not proved. Sinus tachycardia is the most common rhythm disturbance but a variety of arrhythmias, including ventricular, supraventricular, and junctional tachycardia, have been reported. Atrioventricular and bundle-branch block have also been seen. These changes occur at all dose intervals and except for decreased QRS voltage, resolve 1 to 2 months after cessation of the therapy. Sudden death may also occur, due to ventricular fibrillation. Rare cases of sub-acute cardiotoxicity resulting in acute failure of the left ventricle, pericarditis or a fatal pericarditis-myocarditis syndrome, particularly in children, have been reported4. If these patients recover they should not receive further treatment with anthracyclines. In elderly patients with preexisting heart disease, congestive heart failure can occur, which is generally transient and responds to normal medical management[3].

Problems with anaesthesia management

An appropriate anaesthetic plan including the invasive monitoring techniques hinges on thorough preoperative assessment. Invasive arterial blood pressure recordings and a pulmonary artery catheterization may be necessary if significant myocardial impairment is present. Anthracycline treated patients under anaesthesia can develop acute intraoperative left ventricular failure refractory to beta- adrenergic receptor agonists. Amrinone and sulmazole are the new class of cardiotonics with inotropic drugs useful in such conditions.

Hypertrophic cardiomyopathy

Cariomyopathy is an ongoing disease process that damages the muscle wall of the lower chambers of heart[13,14]. HOCM is a form of cardiomyopathy in which the walls of heart chambers thicken abnomally. HOCM is the result of abnormal growth of the heart muscle cell. It is an autosomal dominant trait, more than 140 mutations

on 9 different genes coding for scromere proteins and 2 genes coding for non sacromere proteins involved in cardiac metabolism are responsible with it. Patho-physiology is mainly related to dynamic LVOT (LEFT VENTRICULAR OUTFLOW TRACT) obstruction, diastolic dysfunction, myocardial infarction and arrthymias. Dynamic LVOT obstruction is usually due to SAM (SYSTOLIC ANTERIOR MOTION) of the anterior leaflet of the mitral valve leading to its apposition with hypertrophied septum. SAM of the mitral valve was initially thought to be due to the septal subaortic bulge, narrowing of the outflow tract causing high velocity flow and venture effect. Recent echocardiographic evidence indicates that drag, the pushing force of flow is the dominant hydrodynamic force on the mitral leaflet.[5,6] Factors influencing left ventricular tract outflow obstruction in pts with HOCM7,[8,9]

Events that increase outflow obstruction

Increased myocardial contractility

- Beta –adrenergic stimulation(catecholamines)
- Digitalis

Decreased preload

- Hypovolemia
- Vasodilators
- Tachycardia
- Positive pressure ventilation

Decreased afterload

- Hypotension
- Vasodilators

Events that decrease outflow obstruction

Decreased myocardial contractility

- Beta-adrenergic blockade
- Volatile anaesthetics
- Calcium channel blockers

Increased preload

- Hypervolemia
- Bradycardia

Increased afterload

- Hypertension
- Alpha- adrenergic stimulation

Signs and symptoms

The clinical course of HOCM varies widely with most patients remaining asymptomatic throughout life. Some, however have symptoms of severe heart failure and others die suddenly. The principal symptoms of HOCM are angina pectoris, fatigue or syncope (may represent aborted sudden death), double apical impulse and cardiac murmurs which increases in intensity with valsalva maneuver.

Diagnosis

ECG depicts LVH. ECHO demonstrates myocardial hypertrophy, presence of SAM (systemic anterior motion) of mitral valve and diastolic dysfunction. Colour Doppler reveals presence of LVOT obstruction and pressure gradient across the LVOT.

Treatment

Medical therapy with beta blockers, calcium channel blockers, diuretics. Surgical therapy includes septal myomectomy, dual chamber cardiac pacing and alcohol septal ablation[10].

Anaesthetic management

Management of anaesthesia in pts with HOCM centres on fluid and pharmacological interventions directed towards minimizing LVOT obstruction and at the same time, lessen the severity of mitral insufficiency. Optimizing preload and ventricular filling is appropriate in managing a pt with outflow tract obstruction, after load reduction should be avoided because it worsens the obstruction. Obstruction is exacerbated by hypercontractile states and elevtions in HR; therefore reducing heart rate by beta blockers is considered. Intra-operative complications in HOCM pts like congestive cardiac failure, diastolic dysfunction, MI, dysrthmia and hypotension have been reported. Maintainenace of sinus rythm is most important. Acute hypotension requires prompt volume replacement and administration of phenylephrine. Major complications like cardiac arrest and refractory shock have been reported[11,12]. In our patient smooth induction and intubation was done, by avoiding stress response and maintaining hemodynamic parameters within acceptable range.

CONCLUSION

The cancer patient like any other high risk patients requiring anaesthesia deserves a special care and considerations. A growing number of patients undergoing surgical procedures with general anaesthesia soon after receiving chemotherapy; occasionally such treatment can be given during surgery. Therefore, it is worthwhile and prudent to understand the pathophysiology of cancer and consider the pharmacological interactions between anticancer and anaesthetic drugs. Anti-cancer chemotherapeutic drugs may cause generalized and specific organ toxicities and may also give rise to various unpredictable or life-threatening peri-operative complications, rendering a detailed pre-operative assessment of patients with previous chemotherapy mandatory. Thus, special consideration and understanding of the cancer patient's anaesthesia-related needs will result in superior patient care and outcomes. Management of HOCM includes through understanding of pathophysiologic mechanisms that may trigger or accentuate dynamic LVOT obstruction to prevent intraoperative complications.

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