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Case Report

Anaesthesia management of a difficult airway patient with severe aortic stenosis for major oncosurgery

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ABSTRACT

Over the past few decades, oncosurgical procedures are increasing in number, require considerable expertise and training for anaesthetising such patients. Aortic Stenosis itself poses great challenge, causes significant increase in morbidity and mortality in the perioperative period. Head, neck oncosurgical procedures with difficult airway requiring awake fiberoptic intubation in such patients adds to the challenge. We describe once such case of previously operated Carcinoma of oral cavity with new growth involving mandible for excision of tumour with neck dissection and mandibular reconstruction with a free Fibula flap. This patient now presented with anticipated difficult airway with restricted mouth opening and a recent diagnosis of severe Aortic stenosis with mean gradient across aortic valve of 52mmHg and valve area 0.8 cm2.

Such patient requires multidisciplinary team approach by cardiologist, anaesthesiologist, surgeon and intensivist to prevent perioperative morbidity and facilitate early recovery.

1. Introduction

Over the past few decades, oncosurgery procedures are increasing in number, require considerable expertise and training for anaesthetising such patients. Severe Aortic Stenosis and difficult airway both add to this difficulty and pose major anaesthetic challenge. Aortic stenosis increases perioperative morbidity and mortality.[1]

We describe once such case of recurrent carcinoma of oral cavity including mandible, posted for excision of tumour with neck dissection and mandibular reconstruction with free Fibula flap. He was operated in the past for the same and now had minimal mouth opening and an anticipated difficult airway. A patient with difficult airway requiring awake fiberoptic intubation, recently diagnosed with severe aortic stenosis and prolonged surgery.

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2. Case Report

A 54 years old, male patient, 74 kgs presented with growth on routine Positron emission tomography (PET) scan detected extensive local disease progression with development of metabolically active recurrent disease involving a large full thickness gingivolabial ulcerative lesion on the posterior under surface of the left mandible at the angle, right posterosuperior alveolar margin with erosive bone destruction.

He was a case of Squamous cell carcinoma of right mandible who underwent right composite resection with segmental mandibulectomy along with mandibular reconstruction with free Fibula flap, 1 year back. He was diagnosed with Hypertension 4 months back and started on Tab. Cilnidipine 10 mg twice a day and Tab. Metoprolol 25mg once a day.

Routine pre-operative cardiac evaluation, done 2 months prior, revealed Echocardiography findings of Severe aortic

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stenosis. Moderately thickened and calcific trileaflet aortic valve leaflets. Peak/mean gradient across aortic valve: 73/40mmHg (Peak velocity 427 cm/sec). Aortic valve area (AVA) by continuity equation was 1.0 cm2. This progressed over 2 months and Echocardiography done 2 days prior to surgery showed very severe aortic stenosis. Moderately thickened and calcified aortic valve leaflets with restricted opening. Peak / Mean gradient across aortic valve 95/52 mmHg with mild AR. Aortic valve area (AVA) by continuity equation: 0.8 cm2. Mild pulmonary hypertension with Right ventricular systolic pressure of 40mmHg. Normal sized LV cavity with mild concentric LV hypertrophy and good contractility. No LV regional wall motion abnormality at rest. Overall LVEF = 60%. Mild LV diastolic dysfunction with raised LV filling pressures and Dilated LA (41 mm). Electrocardiogram showed ST depression in lateral leads. All biochemical and haematological value were within normal limits.

Airway examination revealed mouth opening less than 1 finger with limited neck extension. Mento-hyiod distance of 4cms and thyromental distance of 6cms.

Patient was scheduled for Wide Local excision with segmental mandibulectomy with neck dissection along with mandibular reconstruction with free Fibula flap. He was at High risk for anaesthesia in view of severe aortic stenosis. However due to malignant nature of the disease and hence semi-urgent indication for surgery, joint decision was taken that surgery was needed and could not be delayed for cardiac intervention. High risk consent was taken and patient as well as relatives were counselled regarding possible fatal outcome perioperatively. Patient was explained about awake fiberoptoic intubation to reduce anxiety and stress response.

Arterial Line was secured on Left hand with 20G cannula under Local Anaesthesia. Central Line was secured in Right Internal Jugular vein with 7 French triple lumen catheter under Local Anaesthesia and USG guidance. Intraoperative monitoring done with Electrocardiogram with ST trend, SO2, Invasive BP, Central Venous Pressure, Core Temperature (Rectal), multi gas analyser and Urine output monitoring.

Patient was sedated with IV Dexmedetomidine infusion at 0.5mcg/kg over 10 mins followed by infusion at 0.2-0.4 mcg/kg/hr.

Awake Fiberoptic bronchoscope guided intubation was planned. Airway was prepared with 3ml of 4% lignocaine nebulisation. Xylometazoline drops were instilled in right nostril as well as packed with pledgets soaked in 2% lignocaine with 1 in 2,00,000 adrenaline for 10 minutes. Transtracheal 2cc of 2% Lignocaine was given. Fiberoptic bronchoscope loaded with RAE (Ring-Adair-Elwyn) Nasal North Pole No 7 was passed through the right nostril. Spray-as-you Go (SAYGO) technique was used and 2cc 2% lignocaine was sprayed over vocal cords and fiberoptic bronchoscope was passed till carina was visible. Endotracheal (ET) tube was then threaded over the Fiberoptic bronchoscope. ETT position confirmed by auscultation and EtCO2 confirmed along with tidal volume.

General anaesthesia was induced with IV Etomidate 0.2 mg/kg, IV Fentanyl 2mcg/kg and IV Succinyl choline 1mg/kg followed by IV Atracurium bolus. Patient was ventilated with Volume controlled mode to maintain normocapnia. Intraoperatively, IV Fentanyl and IV Atracurium was used to maintain depth of anaesthesia. Also all measures were used to maintain temperature in between 35 to 37 degree Centigrade.

Intraoperatively, during primary excision of tumour, MAP was maintained above 75 mmHg using Noradrenaline infusion. Maximum dose of noradrenaline infusion required was 0.1mcg/kg/min. IV Phenylepherine boluses 50-100mcg were also used as required for maintaining BP. CVP was maintained between 8-12 cmH2O during surgery. Total surgical duration was 9 hours. Total blood loss was estimated at 500ml and urine output was 1000ml. Intraoperative input was 3000ml of crystalloids. Towards end of surgery, noradrenaline infusion was tapered and omitted.

Patient was shifted to ICU on IPPV, sedated with infusion of IV Fentanyl 20mcg/hr and IV Midazolam 2mg/hr. Patient was weaned off ventilator and extubated on Post Operative Day (POD) 1. Shifted to ward the following day and had uneventful course in hospital prior to discharge on POD 7 with Tab. Metoprolol 25mg twice a day.

3. Discussion

Patients with aortic stenosis are at an increased risk of perioperative cardiac events.[2] Risk is higher in patient with severe aortic stenosis, hence it is essential that condition is known in advance and appropriate monitoring and management is used during the surgery.

Aortic stenosis increases perioperative morbidity and mortality. Preoperative echocardiographic assessment of stenosis severity and left ventricular function is highly desirable. Perioperative invasive monitoring is advised for patients with an aortic valve area <1.0 cm2 or a mean aortic valve gradient >30 mm Hg. Invasive arterial line for continuous BP monitoring is essential to know early BP changes. Central venous access is also essential as such patients may need vasopressor medications and for CVP monitoring.[1]

In our patient, mean gradient across aortic valve was 40mmHg and valve area was 0.8 cm2. Hence after securing intravenous access, we secured arterial line for BP monitoring and central venous access for administering vasopressor under Local Anaesthesia.

Difficult airway adds to this risk for anaesthetist. Patient with limited or no mouth opening, there are few choices for securing the airway- blind nasal, retrograde intubation of awake fiberoptic intubation. Awake fiberoptic intubation

is considered as gold standard is such patients.[3]

For Topical anaesthesia for awake intubation there are various options for delivering local anaesthetic on airway such as nebulisation of local anaesthetic, use of local anaesthetic spray, use of local anaesthetic viscous (gargling), use of local anaesthetic jelly, instillation of trans-tracheal local anaesthetic, airway blocks or Spray-as-you Go (SAYGO) technique.[4]

Psychological preparation of patient for awake fiberoptic intubation also plays important role in patient cooperation during procedure. Procedure must be described in detail with all caveats explained to patient and also its importance for safety of the patient and answer any queries that the patient or relatives may have.[4]

The use of Sedation allows for a more cooperative patient for Awake Fiberoptic. Choices include small intravenous boluses, infusion or target-controlled infusions of various drugs such as Benzodiazepines, propofol, opioids, alpha2-adrenoceptor agonists, and ketamine. The difficulty with sedation is in achieving an appropriate level of sedation without compromising the airway.[5]

Higher dose of sedation can drop in BP, which was unwarranted in our case with severe aortic stenosis. And lack of sedation can lead to increase in BP and tachycardia during handling of airway, which could compromise our patient with severe aortic stenosis.

In our case we had counselled the patient, few days in advance. Also we managed to anaesthetize the airway well. Hence we managed to do awake fiberoptic intubation with infusion of IV Dexmedetomidine.

There are very few case reports of patients having both aortic stenosis and difficult airway. One reported case they managed to successfully do awake Fiberoptic intubation with Clonidine sedation in patient with Aortic stenosis.[6]

Intraoperative management of patient with Aortic stenosis it is essential to avoid systemic hypotension. The aim is to maintain blood pressure at normal pre-anaesthetic values. Also, heart rate and rhythm has to be maintained to ensure ventricular filling.[1]

Hypotension can lead to decrease in coronary perfusion leading to myocardial dysfunction further decreasing contractility. Hence decrease in Systemic vascular resistance has to be avoided at all cost. Aggressive management of fall in Systemic vascular resistance with Phenyl ephedrine or Noradrenaline followed by management of the cause. Like during episodes of blood loss, use of blood or intravenous fluids as replacement. Administration of vasoconstrictors by infusion, rather than boluses, facilitates cardiovascular stability.

Arrhythmias must be treated promptly. New onset atrial fibrillation may require cardioversion, if there is hemodynamic instability. Tachycardia can also be detrimental as it reduces the diastolic time for myocardial perfusion and can casue myocardial dysfunction.

In our patient we used baseline Noradrenaline infusion to avoid any drop in BP and Phenyl ephedrine boluses to manage episode of sudden drop in BP. And at same time to treat cause like blood loss with intravenous fluids.

4. Conclusion

Oncosurgery procedures requires considerable expertise and training for anaesthetising such patients. Severe Aortic Stenosis and difficult airway both add to this difficulty and pose major anaesthetic challenge. Also duration of surgery adds to challenge of maintaining hemodynamics for prolonged duration in such patients to avoid complications.

Improvements in understanding has helped to carry out the surgery more safely. For anaesthesiologist, it is essential to know various complications that could arise and ways to deal with it. Oncosurgery procedures are semi-urgent requiring suitable anaesthesia, along with good team work for relief of symptoms and disease free life.

5. Source of Funding

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6. Conflict of Interest

The authors declare no conflict of interest.

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