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CASE REPORT

Anesthetic management for laparoscopic cholecystectomy in a patient with severe mitral regurgitation and severely depressed cardiac function

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ABSTRACT

Severe mitral valve disease in an elderly patient, may exhibit serious hemodynamic disturbance and pose a great challenge to the anesthesiologist. During laparoscopy, positive pressure pneumoperitonium with carbon dioxide insufflation has additional deleterious effects on hemodynamic stability. This case report describes successful management of a patient with severe mitral regurgitation scheduled to undergo laparoscopic cholecystectomy. It highlights the importance of adequate optimization, perioperative monitoring and use of preemptive regional anesthesia.

Key words: Laparoscopic cholecystectomy; Rheumatic heart disease; Mitral regurgitation

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INTRODUCTION

Severe valvular disease is a high risk clinical predictor for anesthesia and/or surgery and identified in 2007 ACA/AHA guidelines for perioperative cardiovascular evaluation for noncardiac surgery.[1] Laparoscopic surgery in patients with severe valvular disease with compromised cardiac function may lead to high probability of intraoperative cardiac failure.[2] Therefore, standard literatures often consider patients with cardiac dysfunction a relative contraindication for laparoscopic surgery. We successfully managed anesthesia for a patient with severe mitral regurgitation scheduled to undergo laparoscopic cholecystectomy.

CASE REPORT

A 65 year old male with symptomatic gall bladder polyp was scheduled for laparoscopic cholecystectomy. He was a diagnosed case of hypertension and rheumatic heart disease (RHD) with mild mitral stenosis (MS) but severe mitral regurgitation (MR). He presented with dyspnea of NYHA grade III. Radial pulse was irregularly irregular with a holosystolic murmur on auscultation. Echocardiography showed RHD, mild MS with severe MR, moderate tricuspid regurgitation (TR) and pulmonary hypertension (PHT), dilated left atrium (LA) and left ventricle (LV) with severe hypokinesia, and LVEF of 20-25%. He was on diuretics, telmisarton, diltiazem, digoxin, warfarin / ecosprine. Preoperative lab reports showed Hb-7.1, PT-54.2 and INR-3.2. He was transfused with 2 units of PCV and 6 units of FFP. This corrected the PT to 14.7 and INR to 1.15. He was posted for laparoscopic cholecystectomy with ASA grade III risk. Informed consent was obtained.

Inside operating room, standard monitors were connected, emergency drugs and defibrillator were kept ready. He showed atrial fibrillation (AF) with a basal heart rate of 106/min, BP 130/74 mmHg, and SpO_2 95% on room air. Right radial artery and right internal jugular vein were cannulated. After 3 min of preoxygenation, he was induced with etomidate

10 mg, fentanyl 50 μg and vecuronium 4 mg. 2% Lignocaine 3ml was administered IV. Within 3 min the heart rate rose to 130/min (AF), with BP 102/66 mmHg, yet satisfactory levels of SpO_2 100% (FiO_2 - 50%) and $EtCO_2$ 45 mmHg. Anesthesia was maintained with O_2 and air and 1% sevoflurane. USG guided right sided subcostal TAP block using 20 ml of 0.2% ropivacaine was given just after induction.

Pneumoperitoneum was created slowly with maximum pressure of 10 mmHg. However, 4-5 min after pneumoperitoneum, AF worsened with HR 150-160/min with BP of 130/88 mmHg. Immediately, inj amiodarone150 mg bolus was administered. Since heart rate couldn't be controlled, Diltiazem bolus 0.25 mg/kg was given. Within 3-4 min, the HR and BP returned to 86/min and 112/67 mmHg respectively with $EtCO_2$ 35 mmHg. Diltiazem infusion started at 5 mg/h to maintain the HR. Further course of 75 min was uneventful. Patient was reversed with neostigmine and glycopyrrolate and extubated.

After extubation, he became dyspneic and could not maintain oxygen saturation. ABG done at this point showed CO_2 retention with $PaCO_2$ of 80mmHg and pH of 7.18. He was put on noninvasive ventilation for 6 hours and ABG corrected to $PaCO_2$ 42 mmHg and pH 7.37. He was observed in ICU for one day and was discharged on 3rd post operative day.

DISCUSSION

We report a case of RHD with mild MS and severe MR in AF for Laparoscopic cholecystectomy which was successfully and uneventfully managed.

Patients with severe MR who have poor hemodynamic reserve are challenge for anesthesiologist. Although, many patients undergo safe intraoperative course of noncardiac surgery, their perioperative course is often complicated by tachyarrhythmias, AF which results in extraordinarily high morbidity and mortality especially in those with pre-existing AF and lower LVEF.The primary goal in such cases is to maintain a heart rate of 80 to 100 beats / min, reduce the afterload and to offer adequate depth of anesthesia. These patients require intensive care in the post-op period.[3,4]

This patient in addition to severe MR also had moderate TR with PHT. His long standing hypertension might have resulted in higher systolic driving pressures; which in turn causes exaggeration of regurgitation factor. The echocardiography revealed an LVEF of 20 to 25%. However, ejection fraction indices are poorly correlated with systolic functions. Hence, LVEF may be overestimated echocardiographically.

Role of laproscopic surgery in patients with cardiac dysfunction is controversial. Current evidence show that laparoscopic surgery can be successfully performed on NYHA grade III and IV patients.[5] This patient was subjected to laparoscopic procedure, which offered him all benefits including lesser pain, stress and morbidity.

This patient exhibited severe tachycardia of 160 / min 4-5 min after pneumoperitoneum. MR could become detrimental with marked increase in systemic vascular resistance and tachycardia associated with pneumoperitoneum.[6] Higher intraabdominal pressures of 8-10 mmHg along with CO_2 insufflation contributed to the acute episode of AF.

This was effectively and promptly controlled with diltiazem within a span of 3-4 min. Diltiazem prevents forward movement of calcium ions through slow channels in myocardial and vascular smooth muscle cells. It is a negative inotrope and it reduces vascular resistance.

Epidural analgesia would help in reduction in afterload but its role in presence of severely depressed LVEF is questionable and may lead to excessive hypotention. We chose Subcostal TAP block for perioperative pain. This reduced the requirement of anesthetics and opioids without causing much effect on hemodynemics. Also, use of invasive monitoring helped us to assess and judiciously control the intraoperative hemodynamic derangement and thus facilitated IV fluid management and appropriate use of ventilators.[7] TEE would be ideal in monitoring filling pressure but it was not available in our institute.

CONCLUSION

Laparoscopic surgeries can be safely done in patients with severe MR with severely depressed LVEF. However preoperative optimization of cardiac status, administration of balanced anaesthesia, proper intraoperative monitoring and low pressure pneumoperitoneum are essential steps to patient safety. The chances of life threatening complications are rare and can be easily managed in the hospital with adequate cardiac support.

Conflict of interest: Nil

Authors' Contribution:

PB, JK: Concept, conduction of study work

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ARTICLE RETRACTION

An ethical issue had been pointed out with a published case report in September 2016 issue of the journal entitled;

"Successful epidural anesthesia in a patient of Hurler syndrome for hernia repair"

by Sukhyanti Kerai, Lalit Sehrawat

Department of Anesthesiology & Critical Care, Maulana Azad Medical College & associated hospitals, New Delhi, (India).

The matter was referred to the ethical committee of the journal for scrutiny, and consequent to the committee's decision, we announce retraction of the under mentioned case report from publication;

Kerai S, Sehrawat L. Successful epidural anesthesia in a patient of Hurler syndrome for hernia repair. Anaesth, Pain & Intensive Care 2016;20(3):350-352.

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CASE REPORT

Accidental inferior turbinectomy: a complication of nasotracheal intubation

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ABSTRACT

Nasotracheal intubation is commonly performed for airway management during maxillofacial surgery. The semi-soft PVC tubes are known to cause upper airway injuries, including bleeding, avulsion of turbinates or nasal polyps. However, complete obstruction of the tube is a rare phenomenon. We report a case of an 18 years old male patient posted for fixation of fractured mandible in whom the inferior turbinate was avulsed while performing nasotracheal intubation. It occluded the endotracheal tube, resulting in ventilation failure which was successfully managed. We stress that if one encounters ventilation difficulty after nasal intubation, possibility of turbinate tissue occluding ETT lumen should be kept in mind and forceful ventilation must be avoided. "When in doubt, take it out."

Key words: Inferior turbinectomy; Ventilation failure; Nasotracheal intubation; Complication; Foreign Bodies/etiology; Humans; Trachea

Citation: Pravin Ubale, Swati Chhatrapati, Shweta Jaju, Supriya Pabalkar, Ashwinkumar Karre. Anaesth Pain & Intensive Care 2018;22(2):234-237

INTRODUCTION

Nasotracheal intubation is commonly performed during maxillofacial and oral surgeries as it has the advantage of giving easy accessibility and an optimal working environment in the oral cavity for the surgeon. Although nasotracheal intubation is generally safe, it still poses risk of complications, the most common being nasopharyngeal bleeding with an incidence of 18–66%.[1]

Various other complications reported in the literature include sinusitis due to obstruction to sinus drainage, transient bacteremia, endotracheal tube (ETT) obstruction, dislodgement of adenoids from mechanical trauma, turbinate avulsion, retropharyngeal perforation, mucosal laceration, cribriform plate fracture, olfactory nerve injury and perforation of pyriform fossa.[2-5]

The nasal turbinates contribute to the nasal function of humidification, warming, and filtration of inspired air.[6] The avulsion of turbinate during nasotracheal intubation is not very common. We report successful management of the avulsed inferior turbinate that caused ETT occlusion resulting in ventilatory failure.

CASE REPORT

An 18 years old male patient was referred to our institution for fixation of fracture of right ramus of the mandible. Patient had history of road traffic accident a month back with loss of consciousness and one episode of convulsions. Patient was intubated and required ventilatory support for three days He was extubated on the fourth day in private hospital. Patient was started on tab phenytoin sodium 200 mg TDS. CT scan revealed linear un-displaced fracture of right frontal bone extending up to the roof and right ethmoid sinus, anterior clinoid process and lateral pterygoid plate, fracture of medial and lateral

Figure 1: Inferior turbinate stuck at the tip of ETT

wall of the right orbit, and fracture of right ramus of the mandible. Visualized intracranial structures appeared normal.

On general examination, his pulse rate was 88/min, and BP, 120/80 mmHg. His cardiovascular and respiratory system were within normal limits. Upper airway examination revealed Mallampatti score of grade I. All biochemical investigations were within normal limits. Both nostrils were examined and the right nostril was found to be roomier with good blast of air.

On the day of surgery, written informed consent was obtained and patient was transferred to operation theatre where standard monitors were placed. 30 minutes prior to surgery, xylometazoline nasal drops were put in both nostrils. Patient was premedicated with Inj. glycopyrrolate 0.004 mg/kg, Inj. fentanyl 2 μg/kg, Inj. midazolam 0.03mg/kg, and Inj. Ondansetron 8mg. Anesthesia was induced with graded dose of Thiopentone sodium 3-5 mg/kg till loss of eyelash reflex. Inj. vecuronium 0.1 mg/kg was administered after confirmation of mask ventilation. After 3 min

Figure 2: Excised inferior turbinate

of positive pressure ventilation, a size 7.5 RAE cuffed ETT (Ivory North Pole) was lubricated and inserted through right nostril. With minimal resistance, the tube was advanced into the oropharynx. Direct laryngoscopy was performed to negotiate the tube through vocal cords. Minimal bleeding from the nasal cavity was noted. After thorough suction, the tube was negotiated through the vocal cords with the help of Magill's forceps under vision. Cuff was inflated and breathing circuit was connected. On manual ventilation, there was resistance to ventilate. End tidal CO_2 ($ETCO_2$) tracing was absent with no chest movements on ventilation. Chest auscultation revealed complete absence of air entry bilaterally with no sound on abdominal area too. ETT position was rechecked by direct laryngoscopy and was found to be in correct position. Bronchospasm was suspected and 2cc of Inj. Etofylline and Theophylline, Inj. hydrocortisone 100mg, Inj. dexamethasone 8mg were given intravenously and two puffs of budesonide spray were given through ET tube. There was no response to the above-mentioned drugs, the bag was still tight and patient was not getting ventilated. Hence decision was taken to remove the ETT. On removal of the ETT, we found a soft tissue mass totally occluding the lumen of ETT which explained complete absence of air entry bilaterally. Patient was maintaining the oxygen saturation of 98%. Patient was mask-ventilated with 100% oxygen for 3 minutes. ENT surgeon was called for assessment. Nasal endoscopy was done by ENT surgeon and he diagnosed the tissue occluding the ETT as inferior turbinate. Active bleeding was not spotted. The avulsed turbinate was firmly stuck in the orifice at the end of the tube. As it was an oral surgery, it was decided to do nasal intubation through left nostril. Patient was successfully intubated with 7.5 number RAE cuffed ETT (Ivory North Pole) through left nostril, which was confirmed with auscultation and capnometry. Throat packing was done. At the end of the surgery, it was confirmed that there was no active bleeding from the nasal cavity. The patient was extubated after complete reversal of neuromuscular blocking drugs and was shifted to the PACU for postoperative monitoring.

DISCUSSION

During nasotracheal intubation, trauma to inferior turbinate is more common than middle turbinate because of its anatomic location and closer proximity to the endotracheal tube during nasal intubation.[7] However preexisting intranasal abnormalities, such as an enlarged inferior turbinate or a septal spur, by redirecting the nasotracheal tube

higher into the nasal cavity increase the risk of middle turbinate trauma.[8] Deviated nasal septum and nasal spurs are considered to be risk factors for trauma during intubation. With the left-sided bevel of the tube and the sharper tip on the right, the turbinates are more at risk of damage in the right nostril whilst the septum is more at risk in the left nostril.[9] Other causes of ETT obstruction during nasotracheal intubation are blood clots and foreign bodies. There is also a case report of nasotracheal tube obstruction by a central incisor.[10]

Moore has suggested a technique for nasotracheal intubation that prevents damage to the turbinates. The nose should be adequately decongested prior to intubation, and lubrication should be applied to the tube. Intubation is performed with cephalad traction on the tube and with the bevel directed laterally so that its leading edge is pointed away from the turbinates. As soon as the tip of the tube is visualized in the oropharynx, the part of the tube outside the nostril is returned to its normal curvature and advanced toward the larynx.[11]

Submental and Retromolar intubation are another option for fracture mandible, but in our case we proceed with nasal intubation. Experienced Anesthesiologists develop judgment to apply accurate threshold pressure to advance the tube into the trachea. However, if more than threshold pressure is required, it is advisable to do nasal intubation through another nostril.

It is prudent to check patency of the tube after passing it through the nostrils, but before pushing it through the glottis. If one encounters any degree of resistance to air flow, possibility of turbinate or mucosal tissue occluding the lumen of ETT should be kept in mind and forceful ventilation must be avoided. The best strategy is to follow the rule, "when in doubt, take it out". The tube can be removed and then intubation tried through the other nostril. If care is not taken, the tissue can be pushed into tracheobronchial tree and cause serious respiratory complications.

In our case, forceful ventilation could have dislodged the avulsed inferior turbinate in the trachea or bronchus. The exact cause for avulsion in our case is not known but excessive pressure while introducing the nasotracheal tube could be a reason.

Such complications can be prevented by selecting correct nostril and adequate preparation by local application of vasoconstrictive drugs. Lubrication and dilation of the nostril prior to intubation, softening the tube, using the smallest compatible tube and avoiding excessive pressure during intubation, can be helpful. A soft nasopharyngeal airway can be used as a pathfinder.

CONCLUSION

Accidental inferior turbinectomy is a rare and very dangerous complication during nasotracheal intubation. It is prudent to check patency of the tube after passing it through the nostrils, but before pushing it through the glottis. If one encounters ventilatory difficulty, possibility of turbinate tissue occluding the lumen of ETT should be kept in mind and forceful ventilation must be avoided. When in doubt, take it out.

Authors' Contribution:
PU: Conduction of study work
SC: Concept
SJ: Manuscript Editing
SP & AK: Searching literature + references

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