Perioperative Crisis Management: Negative Pressure Pulmonary Edema After Violent Hiccups At Emergence And Endotracheal Tube Occlusion Caused By Biting.

P SC, K JA, J SR, P SS

Citation

P SC, K JA, J SR, P SS. *Perioperative Crisis Management: Negative Pressure Pulmonary Edema After Violent Hiccups At Emergence And Endotracheal Tube Occlusion Caused By Biting.*. The Internet Journal of Anesthesiology. 2009 Volume 25 Number 1.

Abstract

A 28 year-old male enrolled for functional endoscopic sinus surgery (FESS) and had violent hiccups at emergence prior to extubation. The patient had paradoxical chest wall motion, desaturation, and agitation as he obstructed the endotracheal tube by biting on it, compounding to airway obstruction leading to negative pressure pulmonary edema. Pulmonary edema subsided in 8 hours after controlled positive pressure ventilation and diuretics. The patient was extubated without any further complications.

INTRODUCTION

Negative pressure pulmonary oedema (NPPE) is a non cardiogenic pulmonary edema caused by an upper airway obstruction (UPO). It has been reported in a variety of clinical situations. The frequency of its occurrence is however difficult to ascertain from cases reported thus far, but in adult age group laryngospasm during induction or emergence accounts for over 50% of the documented cases of airway obstruction leading to pulmonary edema.[1] Markedly negative intrapleural pressures generated against an obstructed airway is the dominant mechanism involved in the genesis of pulmonary oedema.

There are three cases reported so far of pulmonary edema due to biting over an endotracheal tube [2, 3, 4] and one case reported so far of hiccup induced pulmonary edema in a child at induction of anesthesia.[5] We report a case of a young male enrolled for FESS under general anesthesia who developed negative pressure pulmonary edema at emergence from anesthesia after forceful inspiratory efforts and violent hiccups against an occluded endotracheal tube which he was biting on. Hiccups are usually not harmful and are not associated with significant morbidity. Hiccups however can generate strong negative intrathoracic pressures as much as four times the normal inspiration cycle.[6] Apart from discussing the possible causes of negative pressure pulmonary oedema at emergence in this case, we also talk about how best we could have prevented such a crisis at extubation.

CASE REPORT

A 28 year-old, 72 kg, male with no previous medical or surgical history, known drug (cocaine) usage or known drug allergy was scheduled for functional endoscopic sinus surgery under general anaesthesia. The physical examination and laboratory investigations were within normal limits. Preoperative chest radiogram was normal. After confirmation of consent for procedure and an overnight starvation patient was taken in Operation Theater. Electrocardiogram leads, pulse oximeter probe and non invasive blood pressure (NIBP) cuff were attached to patient. He had normal cardiovascular and respiratory system examination on auscultation. His heart rate (HR) -98/min, non invasive blood pressure (NIBP)-120/80mmhg, pulse oximeter oxygen saturation (SPo2)-100% and RR -14/min. The patient had received 0.6 mg atropine intramuscular as premedication 1 hour prior to procedure.

The patient was sedated with midazolam2 mg diluted given slowly intravenously. General anesthesia was induced with 250 mg Thiopentone and 100 mg succinyl choline was given for muscle relaxation. 9.0 mm internal diameter poly vinyl chloride (PVC) portex endotracheal tube was placed under direct vision after laryngoscopy. Laryngoscopy and intubation were smooth and without any trauma. After confirming air entry equal and clear bilaterally, the cuff was

inflated with air; the tube was fixed and secured with tube tape. Under direct vision the throat was packed with saline soaked throat pack, sealing the space around the tube. Anesthesia was maintained with isoflurane (end tidal 0.8-1%) in nitrous oxide 66% and oxygen, fentanyl 100 µg and vecuronium for neuro muscular blockade. Surgery was uneventful. The procedure ended in 90 minutes and a total of 1000 ml of isotonic fluids of ringer lactate and dextrose in 0.9% normal saline were infused.

At the end of surgery isoflurane was shut off. Laryngoscopy was done and the throat pack soaked in secretions and blood was removed followed by oropharyngeal suction. There was no evidence of trauma, postnasal bleeding or displacement of endotracheal tube. After around 30 minutes of the last vecuronium dose, the patient started reacting to endotracheal tube in the form of vigorous hiccups involving strong diaphragmatic motion. Neuromuscular blockade was reversed with neostigmine 3.0 mg and atropine 1.2 mg. The patient opened his eyes spontaneously. Soon the hiccups became more frequent and were accompanied with tracheal tugging and never ceased for around ten minutes.

While awakening, the patient bit on the endotracheal tube completely obstructing it. The chest movements were paradoxical and on auscultation of chest there were absent breath sounds bilaterally, SPO2 started to fall drastically and fell to 78%. Jaw thrust was applied to prevent biting of tube, nitrous oxide was shut off and 100% oxygen given. Intravenous hydrocortisone 100 mg and deriphylline 2ml (etophylline 169.4mg/2ml+ theophylline 23 mg/2ml) was given suspecting severe bronchospasm. The patient was agitated, had tachycardia, hypertension and had almost reached to self extubate. Intravenous Propofol 100 mg was given to calm the patient and a no 4 oropharyngeal airway inserted. After Propofol administration the patient was sedated but it was still difficult to control the hiccups and SPO2 dropped further to 60%, so succinylcholine 50 mg was given intravenous, soon hiccups stopped and laryngoscopy was done again to check for any other sources of airway obstruction. There were no signs of aspiration, or oropharyngeal, postnasal bleeding but mild supraglottic edema was seen. The reservoir bag felt smooth as ventilation was controlled with 100% oxygen and saturation picked up to 95%, but a gush of pink frothy secretions soon flooded the endotracheal tube. On auscultation the patient had fine crepitations bilaterally over mid and lower zones. The patient had florid negative pressure pulmonary edema as confirmed by chest radiogram taken subsequently. Arterial blood gas levels revealed PH-7.37, PCO2-45, and PO2-54. While on FIO2-0.1

Pulmonary edema responded to positive pressure ventilation with positive end expiratory pressures of up to 8 cm H2O and intravenous furosemide. The patient was extubated after spraying vocal cords with 10% lignocaine spray in the next 12 hours after stable saturation and hemodynamics and arterial blood gas analysis. His chest radiogram had improved in 12 hrs. The patient had a normal electro cardiogram and a cardiac Doppler study done postoperatively.

DISCUSSION

Negative Pressure pulmonary edema (NPPE) in our patient was a result of strong inspiratory efforts along with vigorous hiccups which may have had a contribution in the generation of excessive negative intrathoracic pressures while biting on the endotracheal tube. The rapidity of onset of pink frothy secretions and rapid recovery are characteristics of NPPE.[1]

The exact pathogenesis of NPPE is unknown. The predominant mechanism is probably related to transmission of markedly negative intrathoracic pressures to interstitium of lungs causing decreased perivascular pressures in pulmonary capillary bed, inducing hydrostatic transudation of fluid into interstitium of lung. Normal pleural inspiratory pressures of -2 to -5 cm H2O may drop to -50 cm H2O and as low as -100 cm H2O during severe episodes of obstructive sleep apnea.[7] Extreme low inspiratory pressures as much as four times the normal have been reported during hiccups in a study in cats.[6] Young muscular patients like ours are known to develop NPPE due to their strong inspiratory efforts generated against the closed airway.[4]

Furthermore the impaired left ventricular performance along with increasing pulmonary blood volume contributes to transudation of fluid into interstitium. It has also been postulated that hypoxic pulmonary vascular constriction and peripheral vascular constriction from central adrenergic responses impair the right and left ventricular performance.[8] Another potential cause is a capillary leak by capillary disruption from shear stretching forces of high intrathoracic pressures or as a result of extreme hypoxia, has been proposed.[9, 10] Finally, after sudden relief of obstruction, there is an abrupt change in airway pressure, which results in sudden increase in venous return and a redistribution of blood volume from peripheral to central compartment

leading to substantial increase in pulmonary blood volume and pulmonary hydrostatic pressures causing pulmonary edema.[11]

The incidence of NPPE as a complication of all anesthetic practices is 0.05-0.1%, as reported in a review article by McConkey, but was suggested that it occurs more frequently than is generally documented.[12] The morbidity and mortality of an under recognized event of NPPE however is as high as 40% [13] Recently the Australian incident monitoring study of 4000 incidences of laryngospasm during anesthesia reveals that NPPE is known to occur in up to 4% of all incident reports of laryngospasm and only in two cases among 4000, NPPE developed due to other etiology of acute airway obstruction.[10]

Laryngospasm is the most common reason of NPPE. [1, 7-10]Other less common causes include acute endotracheal tube obstruction,[14] laryngeal mask clench during emergence from anesthesia,[15] intensive hiccups during anesthesia,[5] aspiration, [10] bilateral vocal cord palsy,[16] biting of an endotracheal tube [2, 3, 4,] and by direct suctioning of the endotracheal tube adapter. [17]

There are more adverse incidents associated with extubation than intubation and are co-related with the pre-operative physical status, depth of anesthesia at extubation, increasing age and gender with a male preponderance.[18, 19] It is interesting to know more about the depth of anesthesia while planning extubation. In a postal survey conducted on extubation practice amongst anesthesiologist in United kingdom, it was seen that majority of the anesthetists performed extubation with patient either awake (31%) or while in light plane of anesthesia (22%), always deep (2%), and variable either awake if indicated (28%) and deep if indicated (17%). [20] Also the survey revealed that respiratory complications were more frequent when extubation was attempted in deep planes of anesthesia than while in awake. Monitoring the depth of anesthesia and neuro muscular blockade by nerve stimulator plays an important role in deciding the process of reversal and time of extubation.

Factors that could have augmented the airway obstruction in our patient were the residual effects of sedatives, neuro muscular blocking drugs and volatile anesthetics used. Neuromuscular reversal after 30 minutes of last dose of vecuronium could have been premature in our patient, as we failed to use nerve stimulator to assess the degree of blockade and decide the right time of reversal. The presence of hiccups, swallowing and breathing attempts does not necessarily indicate consciousness. Early attempt to extubate the patient who did not fully awaken led to biting over the endotracheal tube. Biting of tube in lighter planes of anesthesia while having strong inspiratory efforts and vigorous hiccups led to NPPE in our patient. Perhaps use of nerve stimulator and monitors for depth of anesthesia would have avoided the early reversal and subsequent complications.

Modified bite block to completely or partially surround the tube and securely fasten the tube to the bite block so as to accommodate a range of tube sizes without a risk of slippage of narrow tubes within the block is advisable while extubating patients in lighter planes and or awake. Bite blocks used while gastro-endoscopy procedures are found to be more effective in preventing occlusion and damage to endoscopes but are expensive specially if used as disposable ones. Use of an oropharyngeal airway throughout the procedure has been associated with sore throat post-operatively [21] and thus be used with caution specially when used only for bite block purposes.

Our patient also had hiccups while awakening for 10 minutes period. Hiccups started before neuromuscular reversal and disappeared only after administration of succinyl choline chloride. Hiccups are described as brief, powerful inspiratory efforts arising from spasmodic contractions of diaphragm, causing sudden intake of breath which is involuntarily cut off by closure of glottis, thus producing the characteristic sound.[6] Hiccups arise as a result of various causes, most commonly gastric distension, central and peripheral nervous system disorders, metabolic disturbances, nerve irritation particularly vagus , glossopharyngeal, phrenic. Studies in cats have also confirmed the reflex arc of hiccup is mediated by pharyngeal branch of glossopharyngeal nerve from their observation of eliciting hiccup like response by mechanical stimulation of epipharynx.[6,22] The presence of blood and secretions in the pharynx could have elicited the hiccups in our patient. It is also worth noting that hiccup could generate strong negative inspiratory pressures as low as four times the normal inspiratory pressures.[6] Though it is difficult to say that the NPPE in our patient was a result of vigorous hiccups alone but hiccups definitely compounded the strong inspiratory efforts made by patient against the endotracheal tube which he was biting on.

Apart from the biting of endotracheal tube there are

numerous other more common causes of intraoperative ventillatory failure like bronchospasm, an endotracheal lumen obstruction from kinking, foreign body , blood clot, secretions, Endo- bronchial intubation, puemothorax, pulmonary embolism, pulmonary edema, each of which can simulate each other. It becomes crucial to quickly identify the cause of ventillatory failure in a patient.

In our patient we prematurely treated the patient for bronchospasm with pharmacotherapy, when we could have relieved the agitation by deepening anesthesia with Propofol. That would have allowed an earlier prevention of biting and subsequent complications. Also an earlier administration of succinyl choline would allow laryngoscopy to rule out other causes of tube obstruction under direct vision and treat hiccups earlier in the course of events.

A simple mishap like a bite on the endotracheal tube could prove fatal in a healthy young patient. Extubation in lighter planes of anesthesia could have been avoided by a proper judgment of depth of anesthesia and status of neuromuscular blockade. Monitoring depth of anesthesia with bispectral index (BIS) helps us adjust the anesthetic doses particularly when using multiple drugs, volatile anesthetics and muscle relaxants which in turn improves recovery.[23] Ideally, the information from BIS monitor should be integrated with information from other clinical monitors and patient assessment. The routine use of BIS monitors in all anesthetic settings is difficult though, due to cost issues.

We advocate the routine use of bite blocks after endotracheal intubation, particularly when extubation in awake patients. Also nerve stimulators should be used as standard monitoring throughout when muscle relaxants are a part of anesthesia.

References

1. Lang SA, Duncan PG, Shephard DA, Ha HC. Pulmonary oedema associated with airway obstruction. Canadian J Anaesthesia 1990 Mar; 37(2): 210-8.
2. Dicpinigaitis PV, Mehta DC. Post obstructive pulmonary edema induced by endotracheal tube occlusion. Intensive Care Medicine 1995; 21(12): 1048-1050.
3. Liu EHC, Yih PSW. Negative pressure pulmonary oedema caused by biting and endotracheal tube occlusion- A case for oropharyngeal airways. Singapore Medical journal 1999; 40(03): 174-5
4. Nishizawa M, Chaffee T.L, Goto H. Pulmonary edema induced by endotracheal tube obstruction: A case report. Respiratory care 1993; 38(5): 479-481.
5. Stuth, Eckehard A.E, Stucke, Astrid G, Berens, Richard J. Negative- pressure pulmonary edema in a child with hiccups during induction. Anesthesiology July 2000; vol93 (1): 282-284.
6. Oshima T, Sakamoto M, Arita H: Hiccuplike response elicited by mechanical stimulation of dorsal epipharynx of cats. J Applied physiology 1994; 76: 1888-95.
7. Timby J, Reed C, Zeilender S, Glauser FL. “Mechanical “ causes of pulmonary edema. Chest 1990 Oct; 98(4): 973-9.
8. Lorch DG, Sahn SA. Post extubation pulmonary edema following anesthesia induced by upper airway obstruction. Chest 1986; 90: 802-805.
9. Olsson GL, Hallen B. Laryngospasm during anaesthesia study in 136,929 patients. Acta Anaesthesiologica Scandinavica 1984; 28:567-7
10. Visvanathan T, Kluger MT, Webb RK, et al, Crisis management during anaesthesia: obstruction of the natural airway. Quality and safety in health care 2005; 14:e3.
11. Kamal RS, Agha S. Acute pulmonary oedema. A complication of upper airway obstruction. Anaesthesia 1984 May; 39(5): 464-7.
12. McConkey PP. Post obstructive pulmonary oedema-a case series and review. Anaesthesia Intensive care 2000; 28: 72-76.
13. Goldenberg JD, Portugal LG, Wenig BL, Weingarten RT. Negative pressure pulmonary edema in the otolaryngology patient. Otolaryngology Head Neck surgery 1997; 117: 62-66.
14. Changjoo P, Hyunjeong K, Kwangwon Y. Acute obstruction of an endotracheal tube: A case report. Anesthesia progress 2004; 51(2): 62-64.
15. Petrou A, Valmas K, Svarna G, Chortaria G, Giamarelou A, Papadopoulos G. Negative pressure pulmonary oedema in a patient ventilated with laryngeal mask. The Greek E-Journal of Perioperative medicine 2003; 1; 69-73.
16. Shuji D, Naomitsu O, Youichi K. Pulmonary oedema after airway obstruction due to bilateral vocal cord paralysis. Canadian journal of anesthesia 1991; 38(4): 492-5.
17. Pang WW, Chang DP, Lin CH, Huang MH. Negative pressure pulmonary oedema induced by direct suctioning of endotracheal tube adapter. Canadian journal of anesthesia 1998; 45: 758-8.
18. Beard K, Jick H, Walker AM. Adverse respiratory events occurring after general anesthesia. Anesthesiology 1986; 64; 269-72.
19. Miller KA, Harkin CP, Bailey PL. Postoperative tracheal extubation. Anesthesia and analgesia 1995; 80: 149-72.
20. Rassam S, Thomas MS, Vaughan RS, Hall JE. Airway management before, during and after extubation; a survey of practice in United Kingdom and Ireland. Anaesthesia 2005; 60(10): 995-1001.
21. Kallar SK, Jones GW. Post operative complications. In: White PF ed. Outpatient anesthesia New York; Churchill Livingston 1990; 397-415.
22. Kondo T, Toyooka H, Arita H. Hiccup reflex is mediated by pharyngeal branch of glossopharyngeal nerve in cats. Neuroscience research 2003 Nov; 47(3): 317-321.
23. Sebel PS, Rampil I, Cork R, White P, Smith NT, Brull S, Chamoun N. Bispectral analysis for monitoring anesthesia-A multicenter study. Anesthesiology 1992; 79: A178.

Author Information

Pawar SC, MD
Department of Anesthesiology, Padmashree Dr D.Y.Patil Medical College And Hospital Research Center

Kulkarni JA, MD
Department of Anesthesiology, Padmashree Dr D.Y.Patil Medical College And Hospital Research Center

Jagtap SR, MD, DA
Department of Anesthesiology, Padmashree Dr D.Y.Patil Medical College And Hospital Research Center

Patil SS, DNB, DA
Department of Anesthesiology, Padmashree Dr D.Y.Patil Medical College And Hospital Research Center