NEGATIVE PRESSURE PULMONARY EDEMA

- Five Case Reports -

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Introduction

Negative Pressure Pulmonary Edema (NPPE) was first demonstrated in 1927 by RL Moore[1] in spontaneously breathing dogs exposed to resistive load. The first description of the pathophysiological correlation between creation of negative pressure and the development of pulmonary edema was in 1942 by MF Warren et al[2]. In 1973, the relationship between pulmonary edema and upper airway obstruction in two children, who had croup and epiglotitis was reported by MA Capitanio et al[3]. In 1977 the report by CE Oswalt et al[4] was the first showing the clinical significance of this phenomenon in three adult patients, who experienced the onset of pulmonary edema minutes to hours after severe acute upper airway obstruction.

From 1984 until 2001 several reports appeared in the literature[5-19] discussing the phenomenon of acute, non cardiogenic pulmonary edema in response to acute or chronic upper airway obstruction.

Negative Pressure Pulmonary Edema (NPPE) is a syndrome caused mainly due to negative intrathoracic pressure generated during spontaneous respiration against obstructed upper airway. The pathophysiological changes are complex, which result in damage of pulmonary capillaries with transcapillary volume shift to the interstitium

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of the lungs, and hemodynamic changes that increase the transpulmonary blood volume. This syndrome is noticed more frequently in young, healthy and usually athletic patients.

We report five cases of NPPE proved clinically and by investigations. The management was either by using CPAP mask or IPPV plus PEEP in addition to diuretic administration. The edema resolved completely in 2-12 hours after initiation of treatment in the ICU.

Patients and Method

Five healthy young patients were diagnosed as having NPPE postoperatively after extubation in Jordan University Hospital over a period of three years (1999-2001). The onset time of pulmonary edema varied from 5 to 45 minutes. All patients were severely hypoxic in the immediate postoperative period with striking inspiratory stridor, which directed the attention of the anesthetist to the possibility of development of pulmonary edema. This was confirmed by auscultation of the chest and chest X-ray.

The method used to treat the NPPE in these patients was by using Furosemide as a diuretic and application of CPAP in one patient and ETT, IPPV plus PEEP in four patients in the ICU.

Results

The pulmonary edema resolved completely in response to the treatment and the patients were discharged from the ICU free of any residues.

The Summary of the five cases is shown in the following table.

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Case	Age	Sex	BW	Operation & Duration	Drugs Used	Onset Time of NPPE	Management in ICU	Time for Resol. Of NPPE	Cardio-lgist Evalua-tion	Length Of Stay In ICU	Outcome
I	16	M	53	Tonsillectomy 35 minutes	Thiopentone Fentanyl Atracurium Isoflurane Neostigmine Atropine	20 min.	CPAP-(Mask) Furos. Hydro-Cortis-one	3 hours	Normal	1 day	Disch-arged Well
2	19	M	68	Septo-Rhino-Plasty 150 minutes	Thiopentone Fentanyl Atracurium Isoflurane Neostigmine Atropine Na-Nitropr-usside Naloxone	45 min	ETT IPPV PEEP Furos.	10 hours	Normal	1 day	Disch-arged Well
3	21	M	62	Septoplasty	Thiopentone Fentanyl Atracurium Isoflurane Neostigmine Atropine Na-Nitropr-usside	5 min	ETT IPPV PEEP Furos.	8 hours	Normal	1 days	Disch-arged Well
4	19	M	72	Appendicec-tomy	Thiopentone Fentanyl Atracurium Isoflurane Neostigmine Atropine Succinylch.	15 min	ETT IPPV PEEP Furos.	12 hours	Normal	2 days	Disch-arged Well
5	26	F	55	Diagnostic Laparascopy	Thiopentone Fentanyl Atracurium Isoflurane Neostigmine Atropine Succinylch.	20 min	ETT IPPV PEEP Furos.	2 hours	Normal	1 day	Disch-arged Well

The cardiologist`s evaluation based on ECG and Cardiac Echo revealed complete freedom from any cardiac origin of the edema.

Discussion

The pathophysiology of NPPE is attributed to three major mechanisms:

(1) Creation of marked intrathoracic negative pressure of -50 to -100 Cm H_2O results in a sudden increase of venous return of blood to the heart, which will expose the left ventricle to an after load stress and an increase in both end diastolic and end systolic ventricular volumes[12,23,24]. Because of the interdependent effect of both ventricles, the stress on the left ventricle will be excessive, leading to an increase of LVEDP. The sudden increase of pulmonary micro vascular pressure, due to very low intrapulmonary pressure, in the face of the high LVEDP and low left ventricular compliance, will favour the formation of pulmonary edema.

(2) The hypoxemia that results as a complication of upper airway obstruction, will increase pre and post capillary pulmonary vascular resistance in a non-uniform fashion[25,26] increasing the pulmonary vascular resistance and capillary pressure and integrity precipitating a hyper adrenergic state, mimicking neurogenic pulmonary edema[27]. Hypoxemia also redistributes blood from the systemic veins to the pulmonary circulation, increasing by that the pulmonary capillary resistance.

(3) In chronic upper airway obstruction there is a modest level of Auto PEEP with an increase of end expiratory lung volume. When this chronic obstruction is relieved acutely, the Auto Peep will disappear, the lung volumes and pressure return to normal, creating a negative intrapulmonary pressure, and if it is severe enough it will result in transudation of fluids in the lung interstitium and alveoli. This type of edema is called Type II NPPE[11].

Reports of NPPE revealed that the overall incidence is rare making less than 0.1% of all surgeries performed under general anesthesia[10,17]. Nevertheless the incidence of developing Type I NPPE associated with

acute postoperative upper airway obstruction is 9.6-12%[11,13], while the incidence of developing Type II NPPE is 44%[11].

We believe that because NPPE may still occur few hours after extubation (up to 30 hours)[11,12,28] and because they are mild in character they may pass undiagnosed, so we have missed them from reporting. A big study including the follow up of all patients at risk should be performed in order to get more accurate incidence of this syndrome.

All of our patients who developed Type I NPPE were young, and this correlated well with the previous studies[10,17]. Type II NPPE occurs in extremes of ages[11].

Reported cases showed male predominance. This was evident in our study also (four out of five).

Type I NPPE develops usually after upper airway acute obstruction, some times after manipulation of the airway surgically, which made some authors to call it Laryngeal Spasm Induced Pulmonary Edema[15,16,17]. Other factors may increase the risk of Type I NPPE such as hanging, strangulation, upper airway tumors, foreign bodies, epiglottitis, croup, chocking, migration of Folly`s catheter balloon used to tamponade the nose in epistaxis, near drowning, endotracheal tube obstruction, goiter and mononucleosis.

Type II NPPE can result after relief of upper airway obstruction caused by big tonsils, hypertrophic adenoids, redundant uvula etc.

Patient No. 2 in our study received 200 ug of Naloxone at the end of surgery because of suspected narcotic overdose. It is known that Naloxone can cause pulmonary edema[29], and this may have contributed to the development of NPPE in this patient.

The resolution of pulmonary edema in our patients occurred after 3-12 hours with a completely normal chest x-ray and excellent clinical condition, which correlated well with other authors findings[9,12,14,15,16,17,18]. JD Goldenberg et al reported one mortality among 6 otolaryngology patients, who developed NPPE postoperatively, in spite of aggressive treatment.

Most authors recommend non-aggressive therapy and non-invasive hemodynamic monitoring. More than 50% of the patients will benefit from application of CPAP masks. Less than 50% need IPPV and PEEP through an ETT[9-29].

Furosemide was used in all our patients as advocated by most authors[10,11,12].

Conclusion

Negative Pressure Pulmonary Edema (NPPE) Types I & II are not very uncommon in anesthetic practice, especially in patients developing acute upper airway obstruction after extubation and/or those patients operated on for relief of chronic upper airway obstruction.

Early diagnosis, awareness of the anesthetist, careful surgical manipulation of the upper airway, vigilance of the nurses in the recovery room and in the wards contribute to the successful management of this syndrome.

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