The Greek E-Journal of Perioperative Medicine 2003; 1:69-73 (ISSN 1109-6888) www.anesthesia.gr/ejournal
Ελληνικό Περιοδικό Περιεγχειρητικής Ιατρικής 2003; 1:69-73 (ISSN 1109-6888) www.anesthesia.gr/ejournal

Negative pressure pulmonary oedema in a patient ventilated with laryngeal mask.

A Petrou[1], K Valmas[1], E Svarna[2], G Chortaria[1], A Giamarelou[1], G Papadopoulos[1]

Abstract

We present a case of severe postoperative negative pressure pulmonary edema (NPPE) in a young male patient scheduled for elective surgery. During recovery from anesthesia the patient got agitated, bit the laryngeal mask persistently and made forceful inspiratory efforts. He soon presented severe arterial hemoglobin oxygen desaturation, later followed by expectoration of pink frothy sputum. After transient improvement in oxygenation, the patient was extubated and treated with supplemental O_2, fluid restriction and diuresis. X - ray examination revealed bilateral pulmonary edema. The edema resolved within 24 hours. The episode was attributed to complete obstruction of the laryngeal mask airway during awakening from anesthesia and strenuous inspiratory efforts that presumably produced excessive negative intrathoracic pressures.

Introduction

We report a case of severe postoperative pulmonary edema, due to negative inspiratory pressures, in a young male patient, scheduled for a minor elective surgery (vasectomy).

Case report

He was a 29 years old semi-professional football player. His medical history did not reveal any pathologic signs. Premedication was administered 45 minutes before operation and consisted of ranitidine 150 mg PO, metoclopramide 10 mg PO and temazepam 20 mg PO.

He was introduced to anesthesia with propofol 200 mg and Fentanyl 150 μg. When an acceptable depth of anesthesia was achieved, a laryngeal mask (LM) no 4 was placed in situ (no bite block was used) and the patient was ventilated manually with a mixture of N_2O in O_2 50%. The patient soon regained spontaneous ventilation and anesthesia was maintained with Sevoflurane 2.5% end tidal concentration during the whole procedure. The operation lasted 55 minutes and 600 ml of Ringers Lactate solution was infused to the patient.

When the operation ended, Sevoflurane was discontinued and 5 minutes later the patient woke up. At that time he was agitated and was coughing vigorously. He soon clenched the LM, producing thus complete obstruction of the artificial airway. Oxygen saturation decreased quickly to about 60% while the anesthesiologist attempted to regain control of ventilation with manual bag assist. As oxygenation deteriorated further (SpO_2 55%), the

1=Department of Anaesthesia
2=Department of Radiology

University Hospital of Ioannina, Greece

	Rec. Unit	PACU-1h	PACU-5h	PACU-8h	PACU-15h	PACU-24h
FiO_2	0,6	0,6	0,6	0,3	0,3	0,21
pH	7,35	7,37	7,44	7,45	7,42	7,43
PaO_2 (mmHg)	58	65	298	120	161	110
$PaCO_2$ (mmHg)	44	42	38	39	43	39
SaO_2 (%)	84	92	100	98	99	98
HCO_3^- (mEq L^{-1})	22	24	25,8	27	27,4	29,1
BE (mEq L^{-1})	-2	0,5	2	3,3	3,3	4,9

Table 1. Arterial blood gas analysis in Recovery Unit (Rec. Unit) and Post-Anaesthesia Care Unit (PACU) at 1, 5, 8, 15 and 24 hours (PACU-1h, PACU-5h, etc) after admission.

anesthesiologist put him again to sleep with another 100 mg of Propofol. The LM was withdrawn and the patient ventilated manually with face mask and an FiO_2 of 100%. SpO_2 increased again to 96% and a few minutes later the patient was awake, fully oriented with SpO_2 value of 90-94% while breathing O_2 enriched air (approximately 60% O_2).

During the operation, cardiovascular status was stable (HR approximately 60 bpm, AP: 110/60 mmHg) while during the desaturation episode an increase in heart rate was observed (approximately 104 bpm) followed by elevation of blood pressure (to 167/94 mmHg). Auscultation revealed no galloping rhythm and breathing sounds were normal with no dyspnoea and normal expansion of the thoracic cavity.

At the recovery room his oxygenation deteriorated again (SpO_2 of 85% with 12 L/min O_2 via face mask). The patient was expectorating pink, frothy sputum. An X-ray exam revealed unilateral edema in the alveoli with diffuse bilateral alveolar infiltrates. A scattered shading (filling alveoli occupation) was observed, evenly distributed through the hilar and peripheral zones with the presence of airbronchograms, mainly on the middle and upper pneumonic fields. Cardiothoracic index was estimated as normal as well as blood vessel distribution and vascular pedicle width. There was no pleural effusion, no soft tissue thickness as well as peribronchial cuffing and Kerley lines (fig. 1). He was administered 100 mg of frusemide iv and a urine catheter was taken in place, in order to monitor urine output.

He was then transferred to the PACU for close monitoring. An arterial line was inserted for frequent arterial blood gas (ABG) analysis and a central venous catheter for CVP (central venous pressure) monitoring. He received almost 60% O_2 in air with face mask, while water intake was restricted. ABG revealed improvement of oxygenation after unloading the circulating blood volume by urinating 1600 ml of urine (Table 1). CVP gradually decreased from 1 mmHg in admission to –5 in the following two hours. WBC was within normal limits and distribution. BUN was slightly increased. No antibiotics were administered.

Indirect pharyngoscopy was performed, that revealed no trauma of the buccal cavity, the pharynx or the epiglottis.

During the following hours a series of X-rays revealed progressive resolution of the pulmonary edema. A few hours later, peripheral shadowing diminished, especially in the upper right pneumonic field. On the next day, the radiologic findings were normal (fig. 1).

The patient left the PACU 18 hours after admission and the edema resolved radiologically 24 hours later. The patient was expectorating frothy pink sputum during the next 30 hours.

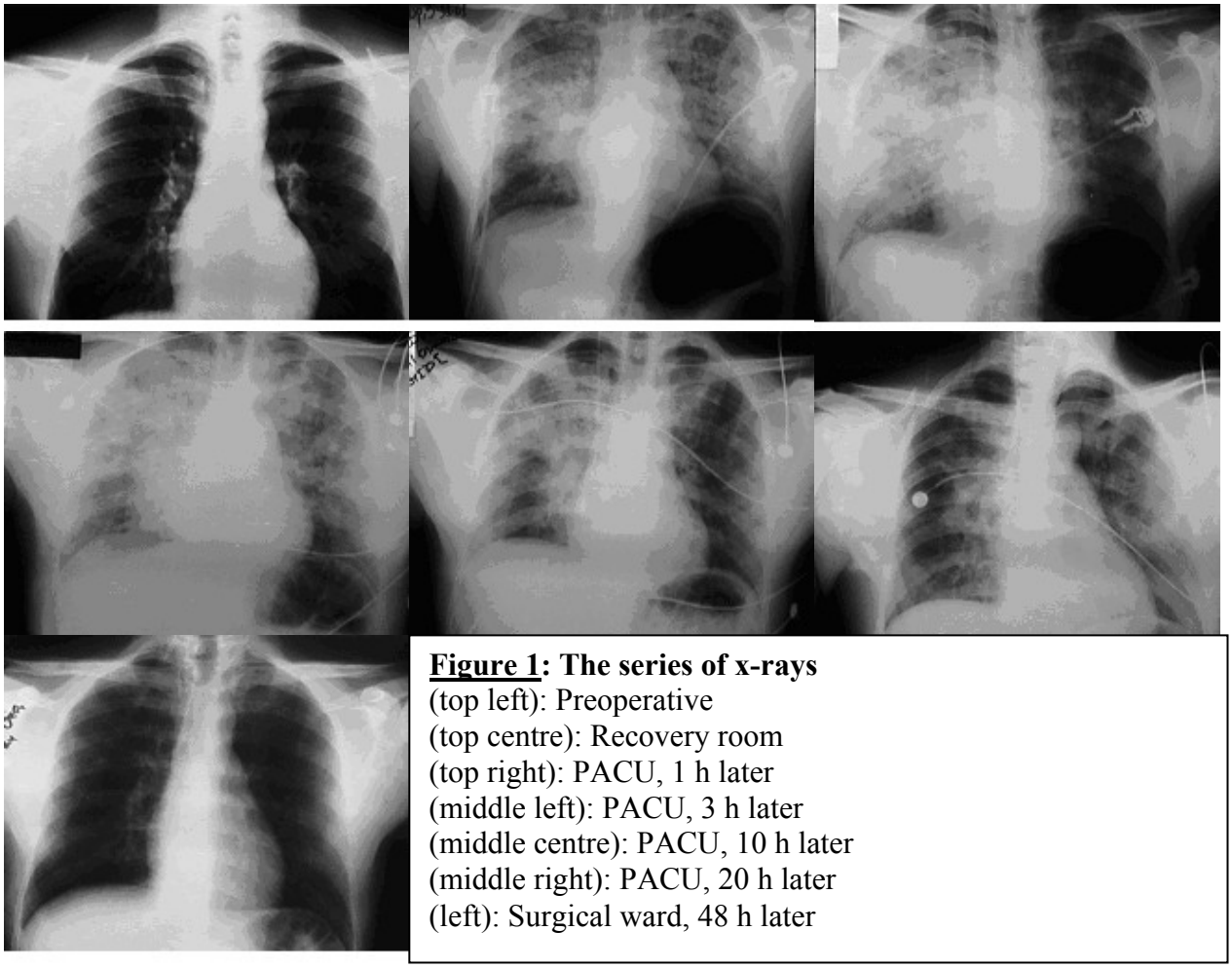

Figure 1: The series of x-rays
(top left): Preoperative
(top centre): Recovery room
(top right): PACU, 1 h later
(middle left): PACU, 3 h later
(middle centre): PACU, 10 h later
(middle right): PACU, 20 h later
(left): Surgical ward, 48 h later

Discussion

Immediately after the desaturation episode, differential diagnosis was established between negative pressure pulmonary edema (NPPE) due to artificial airway obstruction and aspiration of gastric contents. Hypersensitivity reaction was excluded, as we observed no cardiovascular instability, no bronchoconstrictive reaction as well as no dermatologic signs of allergic origin. Larygospasm could have been another possible causative or complicating factor. Even though we did not detect the characteristic inspiratory stridor during any phase of the episode, we cannot exclude the possibility of an evolving episode of larygospasm during the witnessed biting of the LMA.

Lang [1] noted that pulmonary edema associated with upper airway obstruction may be under recognized and supported this by quoting an 11–12% incidence of pulmonary edema in populations requiring active intervention (intubation or tracheostomy) for airway obstruction.

In a recent study [2], the incidence of aspiration pneumonia in surgical patients undergoing operations on the male genital system, is 0,3%. The diagnosis of aspiration was weighed down because at first, the aspiration of a small amount of mouth secretions, after the withdrawing of the LM, did not contain any visible gastric contents. Secondly the patient presented no increase in WBC count, no fever and no persisting infiltrates in x-ray on the following hours and days. Also, the quick reversal of clinical and radiologic presentation defended against chemical pneumonia secondary to silent aspiration.

We favored the diagnosis of severe pulmonary edema due to repetitive efforts of generating negative inspiratory pressure

against the obstructed LM, due to the biting of it. The patient's athletic masculature might have contributed to excessive negative inspiratory pressures during the period of artificial airway occlusion [2]. This enforces the recommendation to use a bite block during anesthesia with an LMA.

This type of edema has been reported in the literature and it's pathophysiologic interpretation has been extensively reviewed [3,4,5]. Negative intrathoracic pressure increases venous return to the right heart by decreasing right atrial pressure, caused by the transfer of negative intrathoracic pressure to the right atrial wall. It also increases the mean systemic pressure, due to the secretion of catecholamines, secondary to hypoxia, anxiety and hypercarbia [6].

This elevation of right ventricular volume produces displacement of the ventricular septum towards the left ventricle, thus reducing it's compliance. This reduced compliance elevates the wall tension of the left ventricle. Catecholamine secretion increases systemic vascular resistance. All these factors acting concomitantly finally decrease the ejection fraction of the left ventricle.

The consequencial elevation of the blood volume into the pulmonary circulation increases the hydrostatic pressure within pulmonary capillaries. Simultaneously, the interstitial pressure in the lung decreases, as a result of the transmission of the negative intrathoracic pressures. This decrease may reach the level of –100 cm H_2O during a Mueller maneuver (inspiratory effort with closed glottis). The net effect is the production of intraalveolar transudative edema. Also the extremely negative intrathoracic pressures can result to rupture of pulmonary capillaries, leading to formation of interstitial edema.

The interesting but diverse radiologic characteristics of NPPE has been described in the literature [7]. Usually it is presented as bilateral centralized alveolar edema with increased vascular pedicle and a normal cardiothoracic ratio. Some times it has unilateral presentation or more peripheral than centralized patterns. It is considered that centralized edema is most probably due to the pathophysiologic mechanisms described above, whereas peripheral edema is most likely to be caused by capillary leak due to hypoxia [7]. There are also cases of NPPE that has been presented with interstitial edema, peribronchial cuffing and perihilar haze.

The expectoration of frothy pink sputum has been reported scarcely [4,6]. It seems that it is produced by disruption of the alveoli fine membrane and the bronchial mucosa vessels due to excessive negative pressure transferred from the pleural space to the interstitial space. Also, due to very complex pathophysiologic mechanisms involved in the production of this type of edema, the expectoration of frothy, pink secretions from the bronchial tree has been established as a hallmark of pulmonary edema.

In similar case reports [8,9] the authors measured the ratio of protein concentration of the edema fluid and plasma for distinguishing hydrostatic from increased permeability pulmonary edema. Our patient denied undertaking fiberoptic bronchoscopy in order to collect the sample, so we did not apply this useful diagnostic test.

Therapeutic measures taken up on these patients are mandated by the extend of hypoxaemia. Severe hypoxaemia has to be treated with increased FiO_2, mechanical ventilation (either invasive or not) and positive end-expiratory pressure. Diuretics help in reversing the osmotic gradient across the alveolar membrane [3,6].

Conclusion

We conclude that we faced an NPPE in a young male athlete, which was caused by repetitive and strenuous inspiratory efforts against an obstructed airway due to the biting of the LM, during an unease awakening from general anesthesia for minor elective surgery.

References

1. Lang SA, Duncan PG, Shephard DAE, Ha HC: Pulmonary oedema associated with

airway obstruction. Can J Anaesth 1990; 37: 210-8.

2. Kozlow JH, Berenholtz SM, Garret E, Dorman T, Pronovost PJ: Epidemiology and impact of aspiration pneumonia in patients undergoing surgery in Maryland. Crit Care Med 2003; 31(7): 1930-7.
3. Holmes JR, Hensinger RN, Wojtys EW: Post-operative pulmonary edema in young, athletic adults. Am J Sports Med 1991; 19(4): 365-71.
4. Koch SM, Abramson DC, Ford M, Peterson D, Katz J: Bronchoscopic findings in post-obstructive pulmonary oedema. Can J Anaesth 1996; 43(1): 73-6.
5. Sullivan M: Unilateral negative pressure pulmonary edema during anesthesia with a laryngeal mask airway. Can J Anaesth 1999; 46(11): 1053-6.
6. Schwartz DR, Maroo A, Malhotra A, Kesselman H: Negative Pressure Pulmonary Hemorrhage. Chest 1999; 115(4): 1194-7.
7. Cascade PN, Alexander GD, Mackie DS. Negative pressure pulmonary edema after endotracheal intubation. Radiology 1993; 186(3): 671-5.
8. Devys JM, Balleau C, Jayr C, Bourgain JL: Biting the laryngeal mask: an unusual cause of negative pressure pulmonary edema. Can J Anesth 2000; 47(2): 176-78.
9. Kallet RH, Daniel BM, Gropper M, Matthay MA: Acute pulmonary edema following upper airway obstruction: case reports and brief review. Respiratory Care 1998; 43: 476-80.

Correspondence :

Anastasios Petrou MD, PhD
Spyrou Loui 4, 452 21 - Ioannina, Greece

Tel: +30 26510 47970

e-mail: tasospetrou@in.gr

Key words: respiratory system - pulmonary oedema, intrathoracic pressure – negative, artificial airway - laryngeal mask, surgery – minor, X – ray examination – pulmonary