Non Cardiogenic Pulmonary Edema as Consequence of Upper Airway Obstruction

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Non cardiogenic pulmonary edema is a rare complication of upper airway obstruction. Its etiology is controversial, but probably can be explaired by the Starling's law, when the large negative intrathoracic pressure generated excedes the intravascular and interstitial pressures, shifting fluids from capillaries to interstitium and alveoli. In addition, alteration of capillary permeability potentiates fluid migration.

We present herein, a case of non cardiogenic pulmonary edema following relief of upper airway obstruction in a 14 years old girl underwent surgical repair of cleft palate.

Cardiogenic pulmonary edema could be excluded by a normal CVP, wedge pressure and four chamber echocardiography. The edema fluid: plasma protein ratio greater than 0.7 can indicate an increased capillary permeability. Mendelson's syndrome could be ruled out by the rapid improvement seen and the soft clinical course. (Key words: plumonary edema, airway obstruction)

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Pulmonary edema is an eventual complication of acute or chronic upper airway obstruction. This hazard is far more frequent in children, but sometimes can be observed in adolescents and adults.

In this article, we deal with a case of non cardiogenic pulmonary edema (NCPE) ocurred after relief of upper airway obstruction in a 14 years old girl submitted to a repair of cleft palate under general endotracheal anesthesia.

Case Report

A 14 years old arab origin girl weighing 34 kg was admitted to the ENT department for repair of congenital cleft palate. The clinical history did not reveal any previous heart or pulmonary disease, the physical examination was completely normal, as well as the preoperative chest x-Ray, the hematological and biochemical profiles.

ANESTHETIC TECHNIQUE: After a premedication of IM meperidine 25 mg, prometazine 25 mg and atropine 0.25 mg administered 1 hour before the scheduled operation, the patient arrived to the surgery room responsive but sedated.

An IV infusion was started and 100 mg of sodium thiopentone followed by 30 mg of succinilcholine were administered. When muscular paralysis was obtained, a 6.5 cuffed vinyl orotracheal tube was uneventfully irserted.

The surgical procedure lasted about 3 hours and at the end of surgery, when the patient was breathing spontaneously, generating an occlusion inspiratory pressure deeper than -20 cm H_2O, responding to verbal solicitations and freely moving the 4 ex-

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tremities the endotracheal tube was removed. Few minutes later, she became agitated, cyanotic, with tachycardia (160 beats/min) and hypertension (systolic arterial pressure 180 mmHg), with profuse sweating, inspiratory stridor and increased respiratory effort. On chest auscultation air entry into the lungs was bilaterally decreased. We tried, during a short period of time (few minutes) to ventilate her through a face mask with 100% oxygen and positive pressure, but in face of the clinical deterioration we decided to reintroduce the endotracheal tube. On laryngoscopy extensive edema throughover the larynx was seen. Shortly after the intubation a large volume of a pinky and foamy fluid fluid came out from the lungs.

The patient was then transfered to our Intensive Care Unit mechanically ventilated with 100% oxygen. At admission, the arterial blood gases were: pH 7.20, P_{CO_2} 62 mmHg, and P_{O_2} 76 mmHg (with a $F_{I_{O_2}}$ of 1).

The superior vena cava was catheterized through the right subclavian vein using the seldinger technique with a Leader Cath (R) 18G (VYGON-FRANCE) and the measured CVP was 5 cm H_2O. A 6F Swan-Ganz catheter was inserted through the right internal jugular vein showing normal pressures (Right Ventricular Pressure 20/0 mmHg, Pulmonary Artery Pressure 20/5 mmHg and Wedge Pressure 6 mmHg).

A four chambers echocardiogram was performed, showing an uniform good contraction of both ventricles. The measured edema fluid: plasma protein ratio was 0.8.

Ventilatory support with non synchronous high flow low resistance IMV/CPAP system (fig. 1) coupled to a Bennet MA 1 ventilator and progressive tittered "ideal" PEEP of 8 cm H_2O was started. Paralelly furosemide 0.5 mg/kg body weight, 50 ml of 25% human albumin, crystalloids, dopamine 2 mcg/kg body weight/min and methylprednisolone 30 mg/kg body weight were given.

The patient's condition improved dramatically and 2 hours later, the arterial blood gases were: pH 7.38, P_{CO_2} 38 mmHg and P_{O_2} 150 mmHg on a $F_{I_{O_2}}$ of 0.4. She was breathing quietly, with a normal level of consciousness and thus, the mandatory rhythm could be gradually reduced untill its complete supression, keeping the patient breathing through CPAP with an inspiratory pressure of + 3 cm H_2O and 8 cm water of PEEP, without any further clinical or laboratory deterioration.

24 hours later, she was uneventfully extubated and the arterial blood gases during expontaneous breathing through an open mask with warmed, humidified, 30% O_2/air mixture, were: pH 7.40, P_{CO_2} 36 mmHg, P_{O_2} 83 mmHg. At the third admission day she was discharged to the ENT department without any late complication.

Discussion

Pulmonary edema can be seen after airway obstruction associated with enlarged adenoids[1] and tonsils[2], epiglottitis[3] and laryngospasm[4,5], although is seldom associated with anesthesia[6]. Though its highest incidence is among pediatric population[7], some reports of pulmonary edema following upper airway obstruction in adolescents[7,8] and adults[9,10] are found in the literature.

The mechanisms of pulmonary edema following upper airway obstruction formation are not fully understood One of the theories states that the alveolar flooding is a consequence of the gradient between the extremely negative intraalveolar pressure (produced by inspiration against a closed glottis) and the intravascular pressure, which promotes fluid migration from the capillaries into the alveoli and interstitium[11].

Another possibility is that this pulmonary edema can be caused by an hypoxia induced increased capillary permeability superimposed to the pulmonary hypertension and consequent increased hydrostatic pressure, dislocating fluid from the capillaries into interstitium and alveoli[12].

In the case described herein, the high (more than 0.7) alveolar fluid: plasma protein ratio seems to indicate that increased capillary permeability plays an important role in genesis of the pulmonary edema[13,14]. The normal CVP and wedge pressure, in association with a normal cardiac echogra-

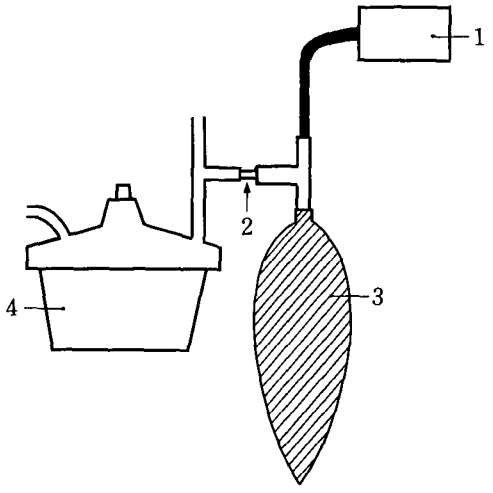

Fig. 1. The IMV external attachment
1. O_2/air mixer 3. breathing bag
2. one way valve 4. cascade humidifier

phy rule out cardiogenic pulmonary edema. Mendelson's syndrome can be excluded, according to Price[15], by the rapid improvement seen in this case.

The treatment of non cardiogenic pulmonary edema as consequence of upper airway obstruction seems to be easy than in other forms of NCPE, like sepsis induced, or as consequence of lung contusion, pancreatitis, Mendelson's syndrome, etc. For this reason, partial respiratory support techniques as IMV, CPAP, or IMV/CPAP appears to be sufficient for ventilatory assistance in those patients.

We believe that the use of asynchronous low resistance high flow IMV systems is more reliable than the use of demand valve systems because with the formers the respiratory work and muscular oxygen consumption are lessened in comparison with the latters[16].

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References

1. Luke MJ, Mehrizi A, Folger GM jr, Rowe RD: Chronic nasopharyngeal obstruction as a cause of cardiomegaly, cor pulmonale and pulmonary edema. Pediatrics 37:762–768, 1966
2. Soliman MG, Richer P: Epiglottitis and pulmonary edema in children. Cor Anaesth Soc J 25:270–275, 1978
3. Travis KW Todres ID, Shannon DC: Pulmonary edema associated with croup and epiglottitis. Pediatrics 59:695–698, 1977
4. Jackson FN, Rowland V, Corssen G: Laryngospasm induced pulmonary edema. Chest 78:819–821, 1980
5. Oswalt CE, Gates GA, Homstrom FMG: Pulmonary edema as a complication of acute airway obstruction. JAMA 238:1833–1835, 1977
6. Lee KWT, Downes JJ: Pulmonary edema secondary to laryngospasm in children. Anesthesiology 59:347–349, 1983
7. Cozanitis DA, Leijala M, Pesonen E, Saki HA: Acute pulmonary oedema due to laryngeal spasm. Anaesthesia 37:1198–1199, 1982
8. Kamal RS, Agha S: Acute pulmonary oedema: A complication of upper pulmonary obstruction. Anaesthesia 39:464–467, 1984
9. Weissman C, Damask MC, Yang J: Non cardiogenic pulmonary edema following laryngeal obstruction. Anesthesiology 60:163–165, 1984
10. Melnick BM: Postlaryngospasm pulmonary edema in adults. Anesthesiology 60:516–517, 1984
11. Mellins RB, Levine OB, Shalak R, Fishman AP: Interstitial pressure of the lung. Circ Res 24:197–212, 1969
12. Mitzner W., Sylvester JT: Hypoxic vasoconstriction and fluid filtration in pig lungs. J Appl Physiol 51:1065–1071, 1981
13. Sprung CL, Rackow EC, Fein IA, Jacob AI, Isikoff SK: The spectrum of pulmonary edema: Differentiation of cardiogenic, intermediate and non cardiogenic forms of pulmonary edema. Am Rev Respir Dis 124:718–722, 1981
14. Sibbald WJ, Cunningham DR, Chin DN: Non cardiac pulmonary edema? A practical approach to clinical differentiation in critically ill patients. Chest 84:452–461, 1983
15. Price SL, Hecker BR: Pulmonary oedema following airway obstructon in a patient with Hodgkin's disease. Br J Anaesth 59:518–521, 1987
16. Gibney NRT, Wilson RS, Pontoppidan H: Comparison of work of breathing on high gas flow and demand valve continuous positive airway pressure systems. Chest 82:692–695, 1982