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Pulmonary Oedema after general anaesthesia in a healthy child

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Abstract

Pulmonary oedema after general anaesthesia is a rare complication and it has been described as well in children as in adults. A case of a healthy child, who developed pulmonary oedema early after emergence from general anaesthesia is reported. Possible causes and management are discussed and a brief review of the literature is referred.

Case Report

A 3.5 years old male kid, weighting 15kgs, was scheduled for an urgent appendicectomy. He was transferred from a paediatric clinic, where he was treated for pain in the abdomen (right lower quadrant) associated with elevated WBC count, fever, cough, clear secretions running from the nostrils. Pure upper respiratory tract infection was excluded from the differential diagnosis on the basis of clinical examination and the child was transferred to the operating room. Anaesthesia was induced with midazolam 0.5mg, fentanyl 50μg, propofol 30mg, vecuronium 15mg and was maintained with a mixture of nitrous oxide 50% in oxygen and sevoflurane 1-2%. Trachea was easily intubated with a 5.5 uncuffed endotracheal tube, although engorged tonsils were observed during laryngoscopy. The operation was uneventful with duration just over 1 hour. During the procedure SpO_2 never fell below 99%, $EtCO_2$ ranged from 4.5 to 5.0 kPa, HR fluctuated between 80 and 90 beats per min, and blood pressure was stable around 95/45 mmHg approximately. Concerning fluid administration, we continued the already existing Ringer's Lactated drip. A total amount of 200 ml was administered during the procedure.

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Following extubation we were confronted with difficulty in maintaining the airway totally unobstructed due to laryngospasm. An existing small degree of micrognathia made manipulation of the airway more difficult. SpO2 fell abruptly as thoracoabdominal discoordination and inward movement of the suprasternal notch was more than obvious. With the administration of midazolam 2mg IV, insertion of an oropharyngeal tube, jaw thrust and tight fit of the face mask (FiO2 1.0), the SpO2 rose steeply as the pattern of breathing was completely normalized. From time to time we assisted the child's spontaneous ventilation by manually giving a deeper breath (manual bag assist). After 15 min the child was awake and fully oriented. Suddenly he began coughing expectorating pink frothy sputum, the SpO2 began falling again and the level of consciousness deteriorated. Midazolam 1mg plus 1,5mg of morphine IV made manipulation of the airway easy-tight fit of the face mask with 100% O_2 to create CPAP (the intrathoracic pressure at the spirometer was fluctuating between 5 and 15mmHg). These simple noninvasive measures gave rise to SpO_2 (96-97%). Negative pressure pulmonary edema, due to the forceful inspiratoty efforts, was the first thought. The chest x-ray was typical of pulmonary oedema. It showed centralized bilateral alveolar infiltrates:

centrally and bilaterally symmetric pattern of the oedema on the chest radiograph argue against any significant aspiration of gastric contents. Moreover there were no obvious sign of frank regurgitation. After 2 hours the child regains consciousness and fully oriented, but SpO_2 did not rise above 92% with an oxygen face mask (FiO_2≈40%). With enrichment from a second O_2 source SpO_2 was 96-98%. Trying to keep the only available, at that time, bed in the ICU free, we decide to keep the child under supervision waiting for further improvement. Unfortunately very soon the child deteriorated and after an episode of severe dyspnoea, tachypnoea, arterial desaturation he was reintubated. Bolus frusemide was given and a drip of dopamine began as the small patient was transferred to the intensive care unit. After 36hrs the child was extubated and 72 hrs from the initial event he was discharged from ICU to the surgical ward. Vital signs, temperature, clinical examination, chest radiograph, arterial blood gases and level of consciousness were normal.

Discussion:

Pulmonary oedema after general anaesthesia is a rare complication as well in children as in adults. It is usually subsequent to acute airway obstruction and has been well described in otherwise healthy children [1,2,3].

The pathogenesis of this type of pulmonary edema seems to be multifactorial, but as the terminology "negative pressure pulmonary oedema" reveals, markedly negative intrapleural pressure created by vigorous inspiratory effort against a partial or totally obstructed glottis (or generally upper airway) is the primary mechanism involved. Negative pressures as high as the level of 100 cm H_2O have been reported during severe episodes of obstructive sleep apnoea [4]. Pathophysiologic mechanisms such as increased venous return to the right heart, interventricular septal shift, reduced left ventricular compliance, hyperadrenergic state due to hypoxia, and increased afterload, are suggested. This haemodynamic changes in combination with the high negative hydrostatic pressure in the pulmonary interstitium (the result of the excess negative intrapleural pressure created by the forceful Müller maneuver) changes the Starling forces of the pulmonary circulation favoring the transudation of fluid to the pulmonary interstitium and then into the alveoli.

Laryngospasm [5], bilateral vocal cord paralysis [6], aspirated foreign body [7], epiglottitis, kinking or biting of the laryngeal mask [8] or endotracheal tube [9] at emergence from general anaesthesia, tumor, strangulation, interrupted hanging [10], premature administration of muscle relaxants [11], even vigorous hiccup [2], are causes reported up to day. As Lang SA in his excellent review stated, "…the frequency of the event is impossible to ascertain from the literature, but paediatric cases requiring airway intervention for croup or epiglottitis and adults requiring airway intervention for emergence laryngospasm or upper airway tumours account for over 50 per cent of the documented cases in each age group, respectively…" [12].

Many authors insist that hypoxia plays the major role in the loss of pulmonary capillary integrity (increasing capillary permeability) and the subsequent pulmonary edema rather than the negative intrapleural pressures, although recent publications indicate that this type of pulmonary oedema has a hydrostatic aetiology without increased alveolo-capillary membrane permeability [8,13]. These authors measured the ratio of total protein concentration between pulmonary oedema fluid and plasma, which is an established, accurate method for distinguishing hydrostatic from increased permeability pulmonary oedema. A ratio of less than 0.65 is characteristic of hydrostatic pulmonary edema, whereas patients with increased-permeability pulmonary oedema have a ratio between 0.75 and 1.0. Unfortunately, the protein concentration of the oedema fluid was not measured in our patient.

Most cases of negative pressure pulmonary oedema present within minutes after the relief of the obstruction [1,12]. Although there are

cases requiring fluid restriction, diuretics and inotropes, the majority of cases respond very well to conventional measures –maintenance of the airway unobstructed, supplemental O_2, PEEP (with invasive or non invasive modes of artificial ventilation) and the recovery is rapid (within hours).

Some colleagues pointed, that an excess of fluid infusion was administered in our case. In fact there was a mild fluid overload, but we believe (and most anaesthesiologists will argue) that to create acute pulmonary oedema to a non failing heart (and healthy lungs and kidneys), quite large volumes of crystalloids is needed to be infused, in order to produce a high hydrostatic pressure accompanied with a marked decrease in serum colloid osmotic pressure [14].

Conclusively, we faced a pulmonary oedema in a child early after general anaesthesia, which required management with diuretics, inotropes and mechanical ventilation with PEEP for 24 hours. In our opinion, the cause of this pulmonary oedema was mainly the strong negative inspiration pressure during emergence from anaesthesia and the moderate fluid overload during anaesthesia may have contributed.

References:

1) Willms D, Shure D: Pulmonary Edema Due To Upper Airway Obstruction in Adults. Chest 1988; 94:1090

2) Stuth EAE, Stucke AG, Berens RJ: Negative-pressure Pulmonary Edema in a Child with Hiccups during Induction. Anesthesiology 2000; 93:282-4

3) Cascade PN, Alexander GD, Mackie DS: Negative pressure pulmonary edema after endotracheal intubation. Radiology 1993; 186:671-5.

4) Timby J, Reed C, Zeilender S, Glauser FL: "Mechanical" causes of pulmonary edema. Chest 1990; 98:973-9

5) Jackson FN, Rowland V, Corsson G: Laryngospasm induced pulmonary edema. Chest 1980; 78:819-21

6) Dohi S, Okubo N, Kondo Y: Pulmonary edema after airway obstruction due to bilateral vocal cord paralysis. Can J Anaesth 1991; 38:492-5

7) Izsak E. Pulmonary edema due to acute upper airway obstruction from aspirated foreign body. Pediatr Emerg Care 1986; 2:235-7.

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; 47:176-8.

9) Warner LO, Beach TP, Martino JD: Negative pressure pulmonary oedema secondary to airway obstruction in an intubated infant. Can J Anaesth 1988; 35:507-10.

10) Oswalt CE, Gates GA, Holmstrom FM: Pulmonary edema as a complication of acute airway obstruction. JAMA 1977; 238:1833-5.

11) Warner LO, Martino JD, Davidson PJ, Beach TP: Negative pressure pulmonary oedema: a potential hazard of muscle relaxants in awake infants. Can J Anaesth 1990; 37:580-3.

12) Lang SA, Duncan PG, Shepard DA, Ha HC: Pulmonary oedema associated with airway obstruction. Can J Anaesth 1990; 37:210-8.

13) 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

14) Westcott JL, Rudick MG: Cardiopulmonary effects of intravenous fluid overload: radiologic manifestations. Radiology 1978; 129:577-85

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