Delayed Resolution of Negative-Pressure Pulmonary Edema: A Case Report

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Negative-pressure pulmonary edema (NPPE) is a well-described complication of upper-airway obstruction, most commonly occurring amongst adults as a consequence of postanesthetic laryngospasm. With early diagnosis and prompt treatment, patients who sustain NPPE typically have a complete recovery within 24 hours of the condition’s first appearance. We herein describe the case of a patient who appeared to feature a delayed resolution of highly suspected NPPE possibly associated with perioperative crystalloid solution loading as a component of cervical spine surgery for a herniated intervertebral disc, and who was extubated when not fully awake post-surgery. Subsequent to reintubation and diuretic therapy, the patient recovered uneventfully.

Key words: pulmonary edema, upper-airway obstruction, delayed resolution, anesthesia

Introduction

Post-anesthetic laryngospasm is the leading cause of upper-airway obstruction-induced negative-pressure pulmonary edema (NPPE) amongst adults. It is usually possible to recognize the pulmonary edema resulting from acute airway obstruction within minutes of the relief of the initial airway obstruction, although the detection of such edema may be delayed for some hours subsequent to the initial insult. With proper diagnosis and appropriate treatment, this form of pulmonary edema usually shows a rather rapid resolution, typically within 24 hours of its initial appearance. We herein describe the case of a patient featuring delayed resolution of highly suspected NPPE possibly associated with perioperative crystalloid solution loading.

Case Report

A 66-year-old, American Society of Anesthesiologists (ASA) physical status II, nonsmoking male, featuring a weight of 66 kg and height of 1.62 m, underwent elective surgery for herniated 4th-7th cervical intervertebral discs. This patient revealed a medical history of hypertension and had been receiving regular anti-hypertensive medication for five years. There appeared to be no history of allergy or cardiac disease for this patient. Physical examination of the patient upon admission was unremarkable apart from apparent decreased muscle power for all four limbs. Preoperative laboratory tests provided results that all lay within normal limits. Electrocardiography (EKG) and chest X-ray (CXR) results proved to be normal. Microsurgical discectomy of the 4th-
7th cervical intervertebral discs with associated anterior plating with a “cage” was performed in the morning of the second day of hospitalization. General anesthesia was induced by use of a mask and the administration of oxygen, xylocaine, fentanyl and thiamylal. The patient was initially administered succinylcholine and pancuronium, and then successfully intubated with a 7.5-mm (I.D.) endotracheal tube on the first pass without apparent difficulty. Anesthesia was maintained with oxygen and sevoflurane. The surgical procedure lasted approximately four hours and appeared to proceed quite uneventfully. During surgery, the patient received 2,050 mL of an intravenously delivered saline solution, and he had a measured blood loss of 100 mL and a urine output of 850 mL. Upon completion of surgery, neuromuscular blockade was reversed with neostigmine and atropine. The endotracheal tube was removed subsequent to the patient resuming spontaneous respiration, although at that time the patient was not fully awake. Approximately five minutes subsequent to extubation, the patient developed progressive cyanosis. The oxygen saturation (SpO₂) level, as determined by means of pulse oxymetry, had fallen from 100% to 76%. Attempts at bag/mask ventilation proved unsuccessful. The patient underwent reintubation with a 6.5-mm (I.D.) endotracheal tube under the impression of postextubation laryngospasm, suctioning of the endotracheal tube yielding pink frothy fluid. The oropharyngolaryngeal examination while reintubated revealed no oropharyngolaryngeal injury. There was no evidence of any antecedent emesis. The patient’s arterial blood gas (ABG) levels when on 100% oxygen included: pH = 7.057, PO₂ 96 mmHg, PCO₂ 108.6 mmHg, HCO₃ 30.8 mmol/L and SaO₂ 92.4%. A portable CXR obtained 30 minutes after re-intubation revealed pulmonary edema (Figure 1). Consequent treatment consisted of the application of a mechanical ventilator and the administration of intravenous furosemide. The patient was later sent

Fig. 1 Chest film (A-P view), taken in the operating theatre with the patient in a supine position, showing extensive alveolar and interstitial edema, and normal heart size.
to the intensive-care unit, where he remained, with mechanical ventilation being maintained. Cardiac auscultation, EKG and cardiac enzyme assay all provided normal results. Echocardiography revealed normal cardiac chambers and normal left ventricular function. Under constant pulse oxymetry monitoring, the patient’s FiO₂ was progressively weaned from an initial level of 100% down to 45%, with SpO₂ at 99% saturation. A rather significant diuresis of 1,500 mL of urine within 10 hours of reintubation was noted. The repeat CXR conducted at 20 hours subsequent to reintubation revealed that the patient’s pulmonary edema had improved markedly but had not completely resolved (Figure 2). Forty hours subsequent to reintubation, a follow-up CXR revealed virtually complete clearing of pulmonary edema. He was then weaned from ventilatory support and extubated. He was transferred to the ward the next morning and discharged home on the 6th day postoperatively.

**Discussion**

Pulmonary edema has been described as having arisen following hanging, strangulation, the presence of a tumor obstructing major airways, foreign-body inhalation, goiter and laryngospasm. The exact pathophysiological mechanism by which pulmonary edema occurs subsequent to upper-airway obstruction is most likely a multifactorial, hypoxic, neurogenic phenomenon, and the impact of hydrostatic forces have been reported to have been implicated. A patient who attempts inspiration against an obstructed airway can generate a substantial negative intrathoracic pressure. Such high negative intrapleural pressures augment venous return, pulmonary blood volume and pulmonary capillary hydrostatic pressure, while decreasing perivascular interstitial hydrostatic pressure. As a consequence, water tends to accumulate in the interstitial spaces, leading to

Fig. 2 Chest film (A-P view), taken with the patient in a supine position 20 hours subsequent to reintubation, revealing that the patient’s pulmonary edema had improved somewhat but had not completely resolved.
pulmonary edema following relief of the upper airway obstruction\(^8\).

The etiology of postoperative pulmonary edema can be cardiogenic, noncardiogenic or a combination of both\(^9\). Before diagnosis of NPPE is settled, other types of acute pulmonary edema, such as cardiogenic, volume overload, multiple blood transfusions, smoke inhalation, aspiration, pancreatitis, drug-induced, infectious or neurogenic, must be carefully excluded based on clinical, laboratory, radiographic and echocardiogram findings\(^10\). In our patient’s case, no evidence of cardiac failure, aspiration of gastric contents to the lungs or any drug hypersensitivity reaction was apparent. By logically reasoning from exclusion, we concluded that this patient’s pulmonary edema was due to partial upper-airway obstruction, which may have contributed to ineffective bag/mask ventilation before reintubation. Our patient did not demonstrate stridor immediately upon extubation. Post-anesthetic laryngospasm may not have been the only probable cause of airway obstruction for the patient described in this report. Since the patient was not completely awake when he was extubated, central nervous system depression and upper-airway muscle relaxation associated with anesthesia could have precipitated soft tissue obstruction following extubation\(^7\). Furthermore, any surgical procedure with some level of traction to the trachea should be considered to be able to induce laryngeal edema, which may also contribute to upper-airway obstruction subsequent to extubation.

What remained to be judged was whether fluid loading had contributed, directly or in combination with upper-airway obstruction, to the development of the pulmonary edema. Our patient received isotonic saline 2050 mL within 4 hours. The total intraoperative fluid requirement is composed of compensative intravascular volume expansion, deficit replacement, maintenance fluids, restoration of losses and substitution for fluid redistribution. There was no fluid overload based on the principle of routine intraoperative fluid administration\(^11\). In addition, the amount of intravenous fluid is the same as the saline suppression test used for evaluating aldosterone-secreting tumors, in which isotonic saline is infused intravenously at a rate of 500 mL/hour for 4 hours\(^12\). The quantity of fluid would not induce pulmonary edema unless patients were elderly or had cardiovascular, hepatic or renal disorders. This proves that the administration of crystalloid solutions to our patient during anesthesia may not have contributed directly to the development of acute pulmonary edema. However, the quantity of fluid necessary to induce pulmonary edema varies according to such factors as age, weight, tissue turgor, or cardiovascular, pulmonary and renal function, plasma vasopressin levels, plasma proteins and the volume of the “third space”\(^13\). Aggressive hemodynamic monitoring should be considered when there is diagnostic uncertainty.

Our patient recovered uneventfully subsequent to reintubation and diuretic therapy. The appropriate therapeutic regimen for such a condition includes oxygen, diuretics, reintubation and/or positive-pressure ventilation. The re-establishment of a physically normal airway and the administration of supplemental oxygen appear to be the two most important factors ensuring rapid recovery from pulmonary edema\(^2,7,14-16\). ABG monitoring and CXR image should be undertaken regularly and continually subsequent to diagnosis, and the patient progressively weaned from ventilatory support as the edema resolves. For our patient, cardiogenic pulmonary edema was unlikely based on the echocardiographic findings and aggressive hemodynamic monitoring was not necessary. Measurement of pulmonary wedge pressure or central venous pressure should be considered as a guide for treatment and differentiation of cardiogenic pulmonary edema when there is diagnostic uncertainty\(^3,10\).
This type of pulmonary edema commonly shows rapid resolution with significant clinical and radiographic improvement within 24 hours\(^5\). However, our patient failed to have rapid resolution despite treatment. By reviewing the literature, we think that the crystalloid solution loading the patient received during anesthesia might have contributed to the delayed resolution\(^2,17\).

Upper-airway obstruction-induced pulmonary edema needs to be recognized early and treated aggressively. We suggest that anesthesia personnel should ensure that the surgery-exiting patient is fully awake prior to extubation, since we believe that this will minimize the likelihood of the occurrence of laryngospasm and/or soft-tissue obstruction, and possibly secondary pulmonary edema. Furthermore, for patients suspected of suffering from NPPE, perioperative airway and fluid management should be carefully handled in order to prevent the occurrence of pulmonary edema, no matter what the cause.

References

延遲改善之負壓肺水腫：病例報告

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上呼吸道阻塞可能併發急性肺水腫，此症於成人常因麻醉後引起咽喉痙攣而造成。儘早診斷並給予適當治療後，病患可於24小時內恢復。在此我們提出一位接受頭椎手術，術後在病患尚未完全恢復意識時拔除氣管內管，因而造成延遲改善的急性肺水腫之病例。該病患於重新插管與給予利尿劑治療後完全恢復。術中適當的輸液處理與確認病患即將完全清醒再拔管可以避免急性肺水腫的發生。

關鍵詞：肺水腫，上呼吸道阻塞，延遲改善，麻醉