

IMMEDIATE PULMONARY EDEMA FOLLOWING CAROTID ENDARTERECTOMY

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Usually noncardiogenic pulmonary edema is caused by upper airway obstruction due to laryngospasm after general endotracheal anesthesia. For obvious reasons this syndrome of "negative pressure pulmonary edema" (NPPE) is well documented in clinical anesthesia. We report an immediate NPPE following carotid endarterectomy (CEA) surgery.

Case Report

A 68-year-old male patient presented with a history of transient ischemic attacks (TIA) for the last year. Carotid artery stenosis was diagnosed using Doppler scan of the carotid arteries. Right-side internal carotid artery was found to be 30% occluded, while the left side was 70% occluded. The patient was scheduled for left CEA under general anesthesia. He was apparently free from other medical conditions. Clinical examination revealed a normotensive patient with normal chest and heart. His ECG and chest x-ray were normal. Echocardiography revealed normal study. His laboratory results were also normal. He was receiving aspirin 80 mg orally once/day. Premedication consisted of 2 mg lorazepam orally 2 hours preoperatively. During surgery, the following parameters were continuously monitored: noninvasive blood pressure (Dinamap, Critikon, USA), ECG, tissue oxygen saturation (SpO₂) (Ohmeda, USA), end-tidal CO₂ (Capnography, Datex, Finland) and body temperature (rectal probe). Right radial artery was cannulated for continuous measurement of the blood pressure as well as comparing the mean blood pressure with the carotid stump pressure values intraoperatively. Ipsilateral cerebral oxygen saturation was also monitored (INVOS 3100, Somanetics). After preoxygenation induction of anesthesia was achieved with i.v. sufentanil 10 ug, propofol 200 mg and endotracheal intubation was facilitated with vecuronium 8 mg. Due to unpredicted anterior larynx, difficult endotracheal intubation was encountered. Using the reinforced tube with the stylet in place resulted in a successful second attempt.

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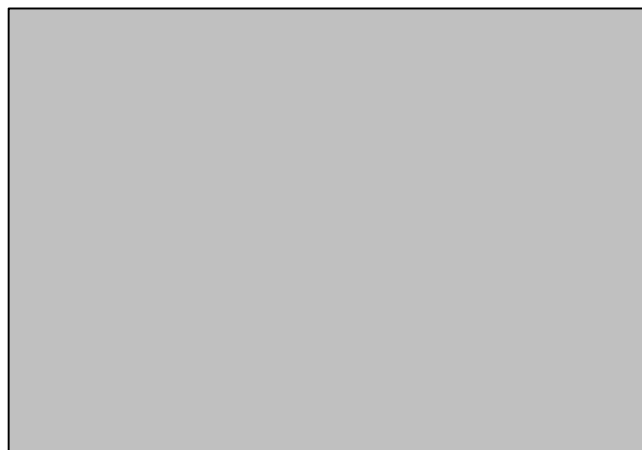


FIGURE 1. Bilateral pulmonary edema, more on the right side.

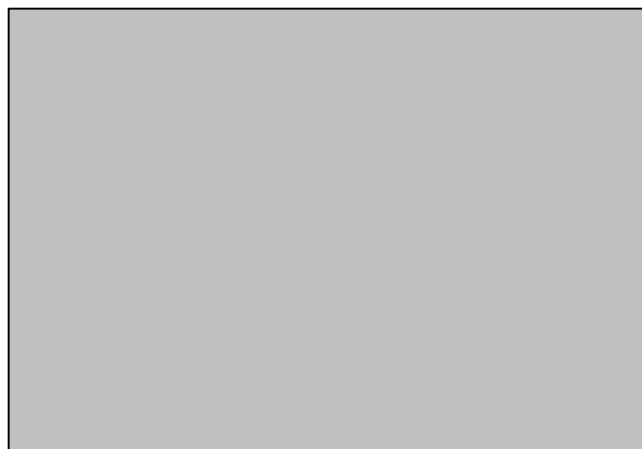


FIGURE 2. Normal chest-x-ray with complete resolution.

Anesthesia was maintained with N₂O/O₂ with 0.4-0.6 vol% isoflurane. Incremental dosages of sufentanil and vecuronium were given when required. Intraoperative blood loss was negligible. The operation lasted 3 hours and the patient received 500 mL 6% hydroxyethylstarch (HES) and 500 mL Ringer lactate. The intraoperative course was uneventful. Upon completion of surgery, neostigmine and atropine were given and after full recovery and response to verbal commands the trachea was extubated. Immediately after, the patient developed severe laryngospasm, and frothy blood-stained secretions from the mouth were

noticed. The SpO₂ dropped to 80% and chest x-ray showed bilateral pulmonary edema, more on the right side (Figure 1).

The diagnosis of negative pressure pulmonary edema (NPPE) was made, and the trachea was again reintubated, facilitated with i.v. morphine (10 mg), propofol (100 mg) and suxamethonium (100 mg). Upon laryngoscopy the epiglottis was found to be moderately edematous. Positive pressure-controlled ventilation was established with positive-end expiratory pressure (PEEP) of 10 mm Hg. Frusemide 80 mg i.v. was given and the right internal jugular vein was cannulated. The patient was then transferred to the surgical intensive care unit for further management. The SpO₂ improved gradually to 99% on FIO₂ 0.4. After 12 hours the chest improved clinically and chest x-ray became normal (Figure 2). The trachea was then extubated and the patient has made an uneventful recovery.

Discussion

Several cases have been reported in children and adults of NPPE following extubation laryngospasm. The mechanism of it is marked negative intrathoracic pressure secondary to forced inspiration against closed glottis, which results in transudation of fluid from the pulmonary capillaries and hence pulmonary edema. It was also assumed that the pathogenesis of NPPE is related to alveolar and capillary damage induced by the same mechanism.¹ In pediatrics NPPE is well documented, with an incidence of 12% in patients with epiglottitis requiring endotracheal intubation.^{2,3} In adults, 25 cases of NPPE have been reported, where postanesthetic laryngospasm was the most common cause.⁴⁻⁶ Scattered cases of unilateral pulmonary edema have also been reported. The explanation given suggests that if airway obstruction occurs in the lateral position, development of NPPE in the dependent

lung is favored by hydrostatic forces and possibly the elevated resting position of the dependent hemidiaphragm.⁷ An unusual cause of NPPE was recently reported following biting of the laryngeal mask airway with subsequent airway obstruction.⁸ Usually the edema clears rapidly with supportive care. Aggressive diagnostic and therapeutic interventions may be avoided if mild symptoms exist. Maintenance of oxygenation and a patent airway are the mainstays of treatment of NPPE, although the spectrum of treatment is quite large. It depends upon the severity of upper airway obstruction and the degree of desaturation. We believe that in severe cases endotracheal intubation and controlled ventilation with PEEP is an ideal strategy. In the present case report, we think that due to the difficult intubation, moderate epiglottic edema could have been the main contributing factor of NPPE. We believe that immediate recognition of NPPE and treatment are important for safe outcome.

References

1. Oswalt CE, Gates GA, Holmstrom MG. Pulmonary edema as a complication of acute airway obstruction. *JAMA* 1977;238:1833-5.
2. Kanter RK, Watchko JF. Pulmonary edema associated with upper airway obstruction. *Am J Dis Child* 1984;138:356-8.
3. Fouron JC. Pulmonary edema with upper airway obstruction. *Am J Dis Child* 1985;139:331-4.
4. Tami TA, Chu F, Wildes TO, Kaplan M. Pulmonary edema due to upper airway obstruction in adults. *Chest* 1990;97:255-6.
5. Cochran M, De Battista C, Schmiesing C, Brock-Utne JG. Negative-pressure pulmonary edema: a potential hazard in patients undergoing ECT (letter). *JECT* 1999;15:168-70.
6. Effros RM, Jacobs ER, Schapira RM, Lin W, Presberg K. Increasing airway pressures can promote transvascular edema reabsorption. *Chest* 1999;116(1 Suppl):30S-31S.
7. Sullivan M. Unilateral negative pressure pulmonary edema during anaesthesia with a laryngeal mask airway. *Can J Anaesth* 1999;46:1053-6.
8. Devys JM. Biting the laryngeal mask: an unusual cause of negative pressure pulmonary edema. *Can J Anaesth* 2000;47:176-8.