

Post-Anaesthetic Pulmonary Oedema Following Upper Airway Obstruction

Pages with reference to book, From 244 To 247

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Abstract

Twelve cases of post-anaesthetic pulmonary oedema (P0) secondary to upper airway obstruction (UAO) are reported. All were adult male patients undergoing uneventful elective surgical procedure under general anaesthesia. Post-anaesthetic laryngospasm was the single most important factor for the upper airway obstruction (UAO) in 5 (41.6%) patients. P0 secondary to partial UAO in drowsy patients was observed in 4 (33.3%) patients. UAO due to foreign body was responsible for P0 in two patients. A combination of negative intrathoracic pressure, hypoxia and associated hyperadrenergic state were the most likely causes of P0 in these patients with UAO. Early recognition, maintenance of patent airway and adequate oxygenation via face mask or endotracheal tube with mechanical ventilation resolved the syndrome within 6-36 hours in all of these patients. Invasive haemodynamic monitoring or aggressive drug therapy were not applied in any of the patients. A heightened awareness among anaesthesiologists of the varied causes of post-anaesthetic UAO leading to P0 may help reduce the occurrence and facilitate early management of the potential complications (JPMA 44:244,1994.).

Introduction

Since 1977 various case reports and larger series have described post-anaesthetic acute pulmonary oedema as a complication of upper airway obstruction (UAO). In majority of these patients post-extubation laryngospasm has been the cause of UAO¹⁻⁹. However, we have closely examined all cases of pulmonary oedema (P0) during the immediate post-anaesthetic period from 1987 to 1993 at our institution and have observed a wide range of conditions responsible for producing the UAO. We present our experience of 12 cases of post-anaesthetic UAO induced P0 in adults to highlight the need for early recognition and appropriate management of this syndrome.

Case Reviews

Twelve ASA I and II adult male patients (26.2SD±8.3year, range 16-45 year; 68SD±15.4 kg. range 45-95 kg) undergoing general anaesthesia for elective surgical procedures developed acute P0 in the post-anaesthetic period 1987 to 1993. Nine of these patients had undergone surgery of the head and neck region (Table).

Table. Demographic correlation with pulmonary oedema.

Case #	Age (yrs.)	Weight (Kg)	ASA grades	Associated condition	Nature of surgery	Initiation of UAO and onset of PO
1	25	50	I	-	Nasal septoplasty	Drowsy on arrival in PARR, staff noted obstruction, PO in PARR
2	22	71	I	-	Pilonidal sinus excision	Drowsy on arrival in PARR, increasing desaturation over 45 minutes, PO in PARR
3	16	45	I	-	Eye lid cyst excision	Drowsy on arrival in PARR, desaturated within 5 minutes, PO in PARR
4	16	58	I	-	Septorhinoplasty	Drowsy on arrival in PARR, desaturated within 5 minutes, PO in PARR
5	45	49	I	-	Tympanoplasty	On arrival in PARR patient noted to be apnoeic and cyanosed, PO in PARR
6	21	73	I	-	Forehead skin graft	Laryngospasm on arrival in PARR, PO in PARR
7	31	81	II	Obesity, smoker, mild cough	Tongue lesion excision	Throat pack inadvertently left but removed OR, PO noted in PARR 30 minutes later
8	33	76	I	-	Oromaxillary fistula repair	Obstruction in PARR by post-nasal Foley's balloon used for control of bleeding, PO immediately in PARR
9	24	82	I	-	Radial keratotomy	Post-extubation laryngospasm, PO in OR
10	25	95	II	Obesity, asthmatic	Nasal septoplasty	Post-extubation laryngospasm, PO in OR
11	23	75	I	-	Inguinal hernia repair	Post-extubation laryngospasm, PO in OR
12	34	61	I	-	Inguinal hernia repair	Post-extubation laryngospasm, PO in OR

ASA⁴¹ = American Society of Anaesthesiology classification of patients according to medical status

All patients underwent an uneventful general anaesthetic using nitrous oxide and oxygen with isoflurane or enflurane and intermittent fentanyl in appropriate dose. All patients were intubated and ventilation was controlled after relaxation with atracurium or vecuronium. At the end of the surgery, residual muscle paralysis was adequately reversed monitoring train-of-four ratio in each case. Patients were extubated after resumption of adequate spontaneous respiration. Patient # 1 to 6 (Table) were adequately breathing and saturating well (arterial oxygen saturation, SaO₂>95%) prior to transfer from operation room (OR) to post-anaesthesia recovery room (PARR). However, patients # 1 to 4 were recorded to be drowsy on arrival in PARR. Patient # 1 was noted to have obstructed breathing while patient # 5 was observed to be apneic and cyanosed on arrival in PARR. Patient #6 developed laryngospasm on arrival in PARR. All these 6 patients gradually desaturated (SaO₂<90%) and went on to develop signs and symptoms of acute PO over the next 5-45 minutes despite appropriate airway management and oxygen (O₂) administration with face mask. Use of furosemide, morphine and appropriate respiratory management quickly resolved the pulmonary oedema less than 24 hours in all patients. Patient # 7 (Table) underwent elective excision of a tongue lesion under general anaesthesia. A pharyngeal pack was placed by the attending anaesthesiologist. At the time of extubation the relieving anaesthesiologist, unaware of the pharyngeal pack, extubated the patient. The patient soon developed obstructed breathing and oxygen desaturation. Laryngoscopy within 5 minutes revealed the obstructing pack. Despite the relief of obstruction, patient developed bilateral chest crepitation 30 minutes later in the PARR. Patient responded well to conservative management without the need for intubation and positive pressure ventilation. Patient # 8 (Table) a 33 year old male with an oromaxillary fistula underwent uneventful repair under general anaesthesia. Towards the end of the surgery, a size 18 Foley catheter was passed through his right nostril and the balloon inflated in the post-nasal space to provide

tamponade to stop further bleeding. The free limb of the catheter was anchored to the right cheek with the aid of adhesive tape. Following uneventful extubation and spontaneous breathing for a few minutes, the patient was transferred to PARR. Within minutes of arrival, his oxygen saturation began to drop rapidly from the initial 100% despite oxygenation via bag mask. The patient became progressively cyanosed and bradycardic. Laryngoscopy revealed the balloon of the Foley catheter completely obstructing the larynx. Balloon was removed and the patient intubated. Florid PO was evidenced by the copious pink froth that issued from the endotracheal tube. He responded well to intra-venous morphine, diuretics and intermittent positive pressure ventilation (IPPV) with mild positive end expiratory pressure (PEEP). He was discharged from the ICU on the 2nd post-operative day. Patient #9 to 12 (Table) had uneventful intra-operative period. On extubation, each developed acute laryngospasm. Suxamethonium 25-50 mg was administered to relieve the spasm after initial failure to ventilate them with bag mask. All these patients rapidly desaturated ($SpO_2 < 90\%$) and were re-intubated in the OR. Each of these 4 patients demonstrated florid pulmonary oedema in the form of copious pink froth issuing via the endotracheal tube. After initiation of therapy with fentanyl, morphine and IPPV they were transferred to the ICU for continuing management of acute PO. All were extubated within 24-36 hours and discharged from the ICU in less than 48 hours with complete resolution of acute PO. Increasing hypoxia, as demonstrated by the pulse oximetry, was the first sign of impending PO in each case. This was further confirmed by arterial blood gas analysis. The clinical diagnosis of the PO varied from volume of fluid pouring up an endotracheal tube to coughing blood stained sputum. All patients had crepitations on auscultation of the chest. Diagnosis of PO was confirmed by chest x-ray in all patients. This was followed with serial chest x-rays which showed quick resolution. All patients had a final normal chest x-ray prior to discharge from the hospital.

Discussion

PO associated with UAO have been called negative pressure pulmonary oedema because it is largely related to the development of markedly negative intra-pleural pressure^{8,9}. A number of conditions can produce UAO. Willms and Shure⁷ analyzed 26 cases of PO due to UAO from the literature and observed that laryngospasm was the most common cause of UAO in 42.3% (11/26). 9 of these 11 patients (81.8%) had post-extubation laryngospasm. Our observations were nearly identical. UAO secondary to laryngospasm was observed in 41.6% (5/12) of our patients. Of these 80% (4/5) had post-extubation laryngospasm. Lorch and Sahn⁵ have identified 3 factors predisposing to UAO. These are: 1) Anatomically difficult intubation; 2) Nasal, Oral or Pharyngeal surgical site; 3) Obesity. One or more of these factors were present in 5 of our patients (41.6%). However, there are several case reports to demonstrate that this syndrome may also complicate UAO in patients without additional identifiable risk factors^{3,7,10,12}. The pathogenesis of PO associated with UAO is multifactorial⁵. However, the principal factor leading to PO following UAO appears to be the generation of markedly negative intra-thoracic pressure due to forceful inspiratory effort against a closed glottis. This results in a decrease in pulmonary interstitial pressure favouring transudation of oedema fluid from pulmonary capillaries¹³. It has been observed that markedly negative intra-thoracic pressure alone can explain the association of UAO and PO^{14,15}. A decrease in intra-thoracic pressure may also lead to augmented venous return which theoretically increases pulmonary blood volume and pulmonary arterial pressure. This fact also may contribute to the hydrostatic transudation of fluid. In addition, increased venous return is associated with right ventricular distention and a decrease in left ventricular compliance as a result of the leftward shift of the intra-ventricular septum (ventricular inter-dependence)^{16,17}. This leads to an increase in the left ventricular end-diastolic pressure thereby further increasing the pulmonary vascular pressure and hydrostatic transudation of fluid. All our patients were young males and ASA I or II. It has

been shown that these young healthy patients may be at increased risk for laryngospasm-induced P0 because they can generate large negative intra-thoracic pressure¹⁸. In our series patients #2 to 5 (Table D) who were drowsy on arrival in the PARR, exhibited semi-obstructed airway possibly due to the approximation of the tongue to the posterior pharyngeal wall. Nevertheless they went on to develop clinical picture of P0 despite appropriate airway management. Nearly identical mechanism of UAO was observed by Warner et al¹⁹. We postulate that UAO need not be absolute like laryngospasm for the P0 to develop. Even mild to moderately obstructed airway can lead to negative intrathoracic pressure, hypoxia and subsequent P0. Although negative intra-thoracic pressure is the primary pathological event in the development of P0 associated with UAO, hypoxia and hyperadrenergic state both contribute to its development. Hypoxia can alter capillary integrity and produce a hyperadrenergic state^{5,20}. This hyperadrenergic state may be associated with a redistribution of blood from the periphery to the pulmonary circuit²¹ and to increased pulmonary vascular resistance^{22,23}. Furthermore, hypoxia alters peri-capillary pulmonary vascular resistance in a non-uniform fashion²⁴⁻²⁶ leading to a generalized increase in pulmonary vascular pressure. Finally, hypoxia and metabolic acidosis may produce direct myocardial depression²⁷ and may potentiate other factors known to enhance formation. Peri-operative administration of naloxone has been implicated in triggering P0^{28,29}. However none of our patients received peri-anaesthetic narcotic antagonists. In all our patients with post-exubation laryngospasm (Case # 9-12) and in the patient with Foley balloon obstruction, florid P0 was evidenced only after the relief of UAO by intubation. It is postulated that UAO creates more positive pressure during expiration which serves as a form of “auto-PEEP” to oppose transudation until the obstruction is removed^{5,7,30,31}. In all these patients, the subsequent course of events was nearly identical to those previously reported^{6,18,32,34}. Lang et al³⁵ reviewed 77 cases of P0 associated with UAO and observed that 85% required tracheal intubation for a short period, 50% needed mechanical ventilation and about 50% required continuous positive airway pressure or positive end-expiratory pressure. This was in contrast to 66.6% of our patients (8/12) who had to be intubated. All these patients were mechanically ventilated from 6 to 36 hours. Furosemide was administered to all our patients. Herrick, Mahenderan and Penny³⁶ reviewing 19 cases in literature of post-obstructive P0 found diuretic administration in 13 cases (68.4%). The use of diuretic has been questioned by some in the light of normal pulmonary capillary wedge pressure (PCWP)³⁷. However, it is recommended by others³⁰. Invasive haemodynamic measurements were not performed in any of our patients as data from previous studies suggests a normal haemodynamic picture (central venous pressure, pulmonary artery pressure and PCWP) in these patients^{3,10,38,39}. This is a characteristic finding into following UAO. Invasive haemodynamic monitoring is important in situations where the diagnosis is not clear especially in patients with uncertain iatrogenic volume overload and/or cardiogenic aetiologies. In few of our cases radiological differential diagnosis suggested aspiration pneumonia/P0 despite a complete lack of history of regurgitation and aspiration. However, the management of aspiration pneumonia is identical to that of P0 associated with UAO unless an infectious complication ensues⁴⁰. A rapid resolution of the radiological findings in less than 36 hours in all our cases pointed more towards P0 secondary to UAO than to aspiration pneumonia. We have observed a variety of causes of UAO which all led to a similar syndrome of rapid onset of P0 followed by quick resolution with appropriate therapy. We should be vigilant therefore not only for post-anaesthetic laryngospasm but also for the partially obstructed airway as may be seen in the drowsy patients and rare iatrogenic causes of airway obstruction. Aggressive haemodynamic monitoring, mechanical ventilation, or drug therapy are not mandatory. Maintenance of adequate oxygenation and a patent airway are the main stays of its treatment.

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