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Noncardiogenic Pulmonary Edema Following Laryngeal Obstruction

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Pulmonary edema is a recognized complication of acute upper airway obstruction, especially in the pediatric population.^{1,2} Interestingly, when pulmonary edema occurs, it usually follows relief of the obstruction. We present a case of noncardiogenic pulmonary edema that occurred in an adult patient after the treatment of upper airway obstruction following extubation of the trachea caused by a combination of laryngospasm and laryngeal edema.

REPORT OF A CASE

A 46-year-old man was admitted for an elective total hip arthroplasty. He had been in good health except for degenerative arthritis of both hips. Preoperatively, he had a hematocrit of 46%, total protein of 7.2

mg/dl, serum albumin of 4.1 mg/dl and a pH_a 7.43, PaCO_2 35 mmHg, and PaO_2 87 mmHg, with a FI_{O_2} of 0.2. Anesthesia was induced with diazepam 5 mg, thiopental 275 mg, and succinylcholine 120 mg, iv. The trachea was intubated easily with an 8-mm cuffed portex tube. Anesthesia was maintained with 70% nitrous oxide and 1.0% isoflurane along with 16 mg metocurine iv. Cephalosporin 1.0 g was administered iv at the beginning of the 4-hour surgery. Blood loss was estimated at 600 ml; the patient received 4 l lactated Ringer's solution iv. At the end of the procedure, the paralysis was reversed with 2.5 mg of neostigmine and 0.5 mg glycopyrrolate iv. The trachea was extubated uneventfully with an end-tidal PCO_2 of 38 mmHg. On admission to the recovery room, stridor, marked abdominal excursions, and intercostal retractions were evident with little movement of air. Ventilatory assistance was initiated with mask and Ambu-bag. Physical examination revealed no evidence of neuromuscular blockade and nerve stimulation revealed no posttetanic facilitation or fade in response to a tetanic stimulus. Because ventilation remained difficult to achieve with the mask and Ambu-bag, 40 mg of succinylcholine was administered iv with a partial resolution of the apparent upper airway obstruction, pH_a was 7.15, PaCO_2 61 mmHg, and PaO_2 160 mmHg. After the return of muscle function, audible grunting and stridor again were noted, along with a rocking respiratory motion, pH_a was then 7.17, PaO_2 57 mmHg, and PaO_2 114 mmHg (with a FI_{O_2} of 0.50-80). We decided to reintubate the trachea. During direct laryngoscopy following the administration of a second dose (40 mg) of succinylcholine iv, the vocal cords were closed, reddened, and edematous. Attempts to insert

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TABLE 1. Respiratory and Hemodynamic Parameters

Time	Vent	FI _{O₂}	PEEP (cmH ₂ O)	V _T (ml)	f (bpm)	pH _a	PaCO ₂ (mmHg)	PaO ₂ (mmHg)	CVP (mmHg)	PAP (mmHg)	PCWP (mmHg)
Pre-op	Spont	Ra (0.21)				7.43	35	87			
12 Noon	Ambu ± Mask	0.50-0.80				7.15	61	160			
01:45 P.M.	IMV	1.0	3	800	15	7.26	48	74	10	30/17	7
02:30 P.M.	IMV	1.0	10	900	15	7.28	45	280			
03:10 P.M.	IMV	0.7	10	900	15	7.32	37	126	9	28/16	7
04:20 P.M.	IMV	0.6	10	900	15	7.32	43	99			
06:30 P.M.	IMV	0.6	10	900	15	7.34	44	103	10	22/15	8
10:30 P.M.	IMV	0.4	12	900	10	7.36	41	122	9	18/11	
03:00 A.M.	IMV	0.4	12	900	10	7.42	37	152	9-10	18/11	9-10
05:00 A.M.	IMV	0.4	6	900	10	7.42	37	138	4	15/07	5
07:00 A.M.	IMV	0.4	3	900	18	7.36	46	112	3	12/08	5
10:00 A.M.	FM	1.0				7.37	45	187	2	14/06	5
11:00 A.M.	FM	0.5				7.35	50	146	2	15/07	4-5

RA = room air; FM = face mask; V_T = tidal volume; CV = controlled ventilation; f = frequency; IMV = intermittent mandatory ventilation.

first an 8-mm and then a 7-mm endotracheal tube with a stylet past the vocal cords were initially unsuccessful. An additional 80 mg of succinylcholine was administered iv and an 8-mm endotracheal tube then was inserted successfully into the trachea. Ventilation then was controlled using a respirator with an FI_{O₂} of 1.0, a tidal volume (V_T) of 800 ml, rate of 10 breaths·min⁻¹, and a PEEP of 3-5 cmH₂O. Hydrocortisone 200 mg iv was administered to reduce the laryngeal edema. Thirty minutes after reintubation of the trachea, the PaO₂ was 74 mmHg (FI_{O₂} = 1.0). At this point, frothy pink fluid emerged from the endotracheal tube. A chest roentgenogram showed proper location of the endotracheal tube and a diffuse homogenous alveolar infiltrate. A pulmonary artery catheter was inserted and revealed a central venous pressure (CVP) of 10 mmHg, pulmonary artery pressure (PAP) of 30/17 mmHg, and a pulmonary capillary wedge pressure (PCWP) of 7 mmHg. PEEP was increased to 10 cmH₂O, and V_T to 900 ml, PaO₂ increased to 280 mmHg (FI_{O₂} = 1.0) with a peak inspiratory pressure (proximal airway) of 30 cmH₂O. Over the next few hours, the FI_{O₂} was reduced slowly (table 1). Chest roentgenogram showed the pulmonary artery catheter tip in the right pulmonary artery, while the homogeneous alveolar infiltrate persisted. Serum albumin and total protein were 2.6 g/dl and 4.8 g/dl, respectively. The PEEP and FI_{O₂} both were decreased successfully (table 1). The PAP, PCWP, and CVP and the pulmonary artery diastolic pressure (PADP) pulmonary capillary wedge pressure gradient decreased slowly (table 1). A chest roentgenogram taken on the following morning showed that the alveolar infiltrate had cleared completely and arterial blood gases had improved (table 1). The trachea was extubated after markedly reduced laryngeal edema was observed. No further problems were encountered after extubation. He made an uneventful recovery and was discharged on the 16th hospital day.

DISCUSSION

Pulmonary edema can follow the relief of an acute upper airway obstruction. This syndrome has appeared in young children (ages 1-5 years) with epiglottitis and laryngotracheobronchitis.^{1,2} These patients, like our patient, became hypoxemic and displayed the clinical and radiologic signs of pulmonary edema soon after relief of their airway obstruction. Four similar cases have been reported in adults.^{3,4} However, no attempt was made to characterize the pulmonary edema as either cardiogenic or noncardiogenic.

In our case, pulmonary edema followed a severe ep-

isode of extubation of the trachea for relief of upper airway obstruction. The obstruction appears to have been due to a combination of laryngeal edema (noted on direct laryngoscopy) and laryngospasm as evidenced by the patient's response to a dose of succinylcholine. This case is interesting because of the rapid onset and rapid resolution of the pulmonary edema. Within 16 to 18 hours, the radiographic abnormalities had resolved completely, while the pulmonary artery pressures decreased as did the PADP-PCWP gradient. Most importantly, oxygenation had improved sufficiently to allow extubation of the trachea.

We believe that this self-limited pulmonary edema probably was brought on by upper airway obstruction. Nonetheless, other possible causes of pulmonary edema were investigated. Since the patient had undergone major long bone surgery, fat embolism may have occurred. We found no evidence of urinary fat globules (although their absence does not exclude the possibility of fat emboli)⁶ or other symptoms of fat emboli such as petechiae, thrombocytopenia, mental disturbances, and unexplained anemia.^{7,8} Furthermore, the extremely short duration of the pulmonary edema is not typical of the fat emboli syndrome.^{6,7} An allergic reaction is another possible cause of pulmonary edema,⁹ but the usual signs of this diagnosis were not noted at any time during the perioperative period.

The hemodynamic data suggest that our patient had noncardiogenic pulmonary edema. A pulmonary artery occlusion pressure of 7 mmHg was measured while ventilation was controlled with a PEEP of 10 mmHg.¹⁰ This confirmed that the patient had low pulmonary vascular hydrostatic pressures. With the PCWP of 7 mmHg and an albumin of about 3 mg/dl, the edema was unlikely to be due to a decreased colloid osmotic pressure-pulmonary capillary wedge pressure gradient.

The mechanism by which an upper airway obstruction causes pulmonary edema is not known. Either hydrostatic forces or an increase in capillary permeability are likely

causes. The first theory assigns the development of the edema to negative transpulmonary pressure caused by inspiration against an obstruction.^{2,4} This causes the mean transpulmonary pressure to decrease further and results in the transudation of fluid from the pulmonary capillaries into the interstitial and alveolar spaces. Interstitial fluid accumulates so fast that the lymphatics are unable to clear the interstitial space of excess fluid.⁴ As the negative transpulmonary pressure returns to normal, the edema should resolve rapidly. Stalcup *et al.*¹¹ demonstrated in dogs that the amount of pulmonary fluid increases as the mean pleural pressure becomes more negative. The major difficulty with this theory is the timing of the obstruction and edema. The greatest degree of pulmonary dysfunction does not take place during the period of negative transthoracic pressure but rather after the obstruction is released,¹ *i.e.*, once there no longer is a negative pressure. Galvis *et al.*¹ suggested that, while the obstruction exists, expiration against it produces a PEEP-like effect, so that oxygenation is maintained. Once the obstruction is gone, this effect is no longer present and oxygenation deteriorates until the lymphatics have cleared the excess fluid. This mechanism would be consistent with our observations. Our patient's oxygenation deteriorated after the obstruction was relieved, but the deterioration lasted only a short time.

Perhaps the obstruction damaged the capillaries, resulting in increased permeability and the consequent exudation of fluid. A number of recent studies¹²⁻¹⁵ on re-expansion pulmonary edema (edema that occurs after a large quantity of air or fluid has been aspirated rapidly from a pneumothorax or pleural effusion) have revealed evidence of increased capillary permeability in both humans¹²⁻¹⁴ and animals,¹⁵ although this form of pulmonary edema was thought to be hydrostatic in nature, (*i.e.*, caused by the sudden negative intrapulmonary pressure due to the rapid evacuation of fluid or air).¹⁶⁻¹⁹ The mechanism of this increase in vascular permeability is not yet clear, but evidence from animal experiments suggests that it may be due to mechanical stresses applied to the lungs.¹⁵ In our patient the rapid swings in pressure may have caused similar mechanical stresses, and the pulmonary edema may have been due to increased capillary permeability. Another possible explanation for the pulmonary edema in our case was that it was secondary to hypoxia, a known cause of pulmonary edema of the increased permeability variety.²

The protein content of the pulmonary edema fluid was not determined¹⁹; as a result, it remains unclear whether the edema was due to a transudative hydrostatic process or an exudative one caused by increased permeability.

In summary, we described a case of pulmonary edema of short duration related to an episode of severe acute

airway obstruction. Hemodynamic data indicate that it was of the noncardiogenic variety. It is important to observe patients who have had upper airway obstruction for the development of this complication. If pulmonary edema does develop, it is likely to be of the noncardiogenic type.

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