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Case Report Open Access

A Different Type of Pulmonary Edema: Post Obstructive Pulmonary Edema

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Abstract

Post obstructive pulmonary edema (POPE) or negative pressure pulmonary edema is a unique clinical entity occurring in the context of airway obstruction. Two distinct subclasses of POPE have been described in literature: type I occurring in the setting of acute airway obstruction and type II occurring after relief of chronic partial airway obstruction. We report the case of a 40-year-old gentleman who developed POPE during rigid bronchoscopy soon after the central airway obstruction was relieved. Patient improved rapidly over the next 24 hours with positive pressure ventilation.

Keywords: Allergic bronchopulmonary aspergillosis; Bronchial asthma; Chest radiology

Introduction

POPE is a rare but serious complication associated with airway obstruction. POPE has been categorized into two types, type I and type II, based on the duration of airway obstruction. Type I POPE develops usually after acute upper airway obstruction or after manipulation of airway surgically. Type II POPE results after relief of longstanding airway obstruction [1]. In adults, POPE is most commonly seen in the setting of laryngospasm and airway tumors [2]. Cases of type II POPE are characterized by sudden onset of respiratory distress following relief of airway obstruction. Thus the occurrence of type II POPE in perioperative period necessitates acquiring knowledge regarding this entity especially among clinicians caring about patients with airway obstruction [3]. We present one such scenario of type II POPE in this case report.

Case report

A 40-year-old gentleman, recently diagnosed with carcinoma of the esophagus, presented to us with exertional breathlessness and stridor since 15 days. Patient had no prior co morbidities. On examination, he had a heart rate of 102 beats/min, respiratory rate of 24 breaths/min, blood pressure of 118/80 mmHg, oxygen saturation of 94% at room air and had stridor. Electrocardiogram was normal. Chest radiograph posteroanterior view revealed widened superior mediastinum with clear lung fields. On computed tomography of chest, a well-defined homogenous mass was seen arising from the upper esophagus infiltrating and obliterating the tracheal lumen [**Figure 1a**]. Flexible bronchoscopy revealed an exophytic growth arising from the posterior tracheal wall, about 4-5cm below the level of vocal cords almost completely occluding the lumen [**Figure 1b**]. We performed rigid bronchoscopy to relieve the critical airway narrowing. We debulked the tumor with the barrel of the rigid bronchoscope and restored 50 to 60% patency of the lumen. The patient was extubated and before intubation with a larger rigid bronchoscope (for stent placement); he developed worsening hypoxemia, along with pink frothy secretions from the oral cavity. We intubated the patient with a rigid bronchoscope, pink frothy secretions were visualized filling the airway. Secretions were continuously cleared using flexible bronchoscope inserted through the barrel of rigid bronchoscope, resulting in transient improvement in hypoxemia. A straight covered metallic stent of size 18x6 cm was deployed in the trachea and size 7.5 endotracheal tube was used to intubate the patient. Thin, frothy secretions continued to flood the airway throughout the procedure and was managed with suctioning and positive pressure ventilation. The immediate post-operative chest radiograph showed bilateral alveolar opacities consistent with pulmonary edema [**Figure 2a** and **Figure 2b**]. Intraoperative as well as post-operative electrocardiogram did not reveal any abnormalities. Bedside echocardiogram was also within normal limits. He was managed in the intensive care unit with invasive mechanical ventilation. After 24 hours of positive pressure ventilation respiratory failure improved and he was successfully extubated. Chest radiograph also showed significant improvement in alveolar opacities [**Figure 2c**]. Flexible bronchoscopy after extubation, did not show any secretions and the stent was in the desired position.

Discussion

POPE is an important entity to be remembered while managing patients with airway obstruction. Other common causes for respiratory distress like anaphylaxis, cardiogenic pulmonary edema, pulmonary embolism and volume overload should be excluded before diagnosing POPE [4].

POPE is divided into two types based on the time of onset of airway obstruction. The pathophysiology of the two entities also being different.

Type I POPE occurs in setting of acute airway obstruction as seen during laryngospasm, epiglottitis, croup, choking/foreign body, strangulation, hanging, endotracheal tube obstruction, laryngeal tumor, goiter, mononucleosis, postoperative vocal cord paralysis, and near drowning. Strong inspiratory efforts against acutely occluded airway leads to generation of intensely negative intrathoracic pressure to the tune of - 140cm H20 from a normal baseline of – 4cm H20. This causes increase in preload plus an increase in afterload of heart.

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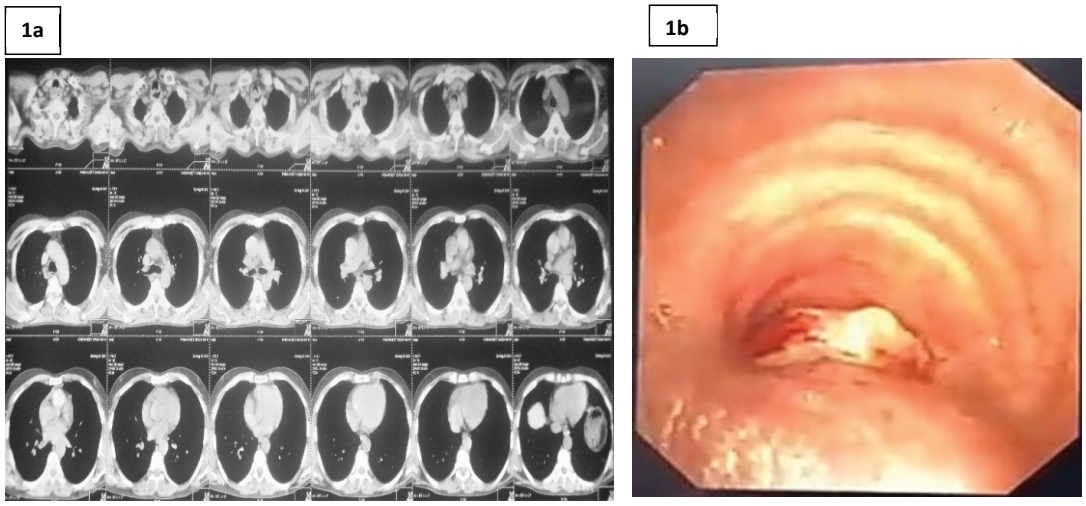

Figure: (1a) Contrast enhanced computed tomography of chest axial cut shows mass circumferentially encasing upper thoracic esophagus. The mass is seen projecting into posterior wall of the trachea and compromising the lumen of trachea. **(1b)** Flexible bronchoscopy showing near total occlusion of tracheal lumen by growth extending from posterior tracheal wall.

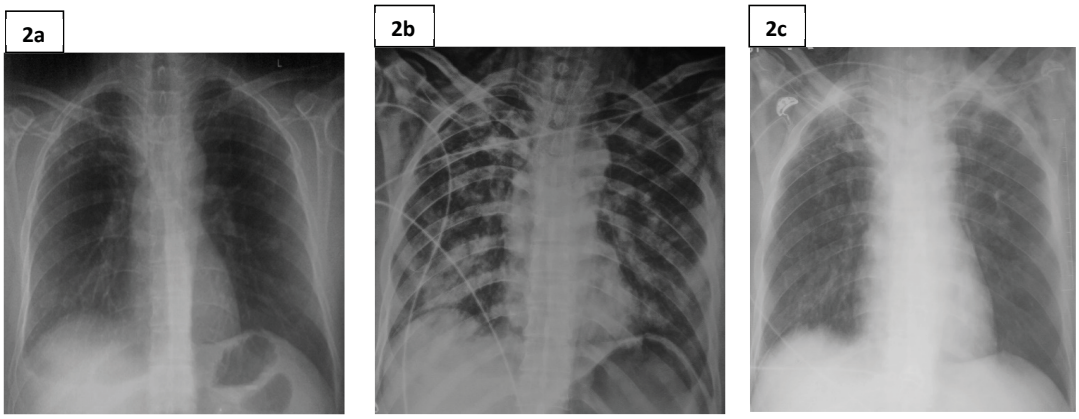

Figure: (2a) Pre operative chest radiograph posteroanterior view shows clear lung fields with widened superior mediastinum. **(2b)** chest radiograph anteroposterior view done post rigid bronchoscopy shows bilateral alveolar opacities consistent with pulmonary edema. Straight metallic stent and endotracheal tube are also seen. **(2c)** chest radiograph anteroposterior view showing resolution of alveolar opacities within 24 hours of positive pressure ventilation.

The increase in preload causes elevation in hydrostatic pressures and subsequently resulting in pulmonary edema [2].

Type II POPE usually results following relief of chronic airway obstruction. Expiration against an obstructed airway produces a modest level of positive end expiratory pressure [5] and increases end expiratory lung volume. The resulting positive pleural and airway pressures lead to decrease in right as well as left ventricular preload. Sudden removal of this obstruction returns lung volumes and pressures to normal. The sudden drop in PEEP leads to increase in venous return, however left ventricle is unable to match the necessary output resulting from increase in venous return. This leads to alteration in starlings forces, that is elevated hydrostatic pressure in pulmonary circuit, thereby resulting in interstitial fluid transudation and pulmonary edema [6].

In the paucity of large studies, data from individual case reports suggest anatomically difficult intubation, obesity with obstructive apnea, short neck, vocal cord palsy, saber sheath trachea and premature extubation to be risk factors for developing POPE in perioperative setting [5,7-9].

Common clinical picture of POPE includes immediate onset of respiratory distress after relief of an airway obstruction. Although symptoms of POPE usually develop immediately post extubation , in some cases delayed presentation of up to 24 hours has been seen [4]. The presence of rales, frothy pink secretions and progressive oxygen desaturation suggests the diagnosis of POPE in appropriate setting [10]. Chest radiograph findings of rapid onset bilateral opacities consistent with pulmonary edema support the diagnosis[11]. Rapid onset as well as resolution of symptoms and radiological changes occurs within 24 hours in POPE [4]. Similar findings were seen in our case described above following relief of airway obstruction.

Anaphylaxis, cardiogenic or neurogenic pulmonary edema, pulmonary embolism and aspiration pneumonitis are important differentials which should be ruled out apart from POPE in the setting of acute postoperative respiratory distress [4]. Presence of rales, pink frothy secretions, bilateral infiltrates on chest radiograph, absence of significant cardiac ailment or physical examination findings can help in differentiating POPE from above causes of respiratory distress in post-operative setting [2]. Rapid onset as well as resolution of symptoms and radiological findings with positive pressure ventilation is also a pointer

Table 1: Cases of POPE described in the peer reviewed literature encountered during bronchoscopic management of central airway obstruction.

References	Age (Years)	Sex	Duration of airway obstruction (days)	Etiology of central airway obstruction	Procedure precipitating POPE	Time duration between relief of obstruction and development of POPE (minutes)	Managed with	Outcome
Bashir et al. [14]	1	Male	<1	Aspirated foreign body	Rigid bronchoscopy guided removal of foreign body	45	Invasive mechanical ventilation	Improved
Hou et al. [15]	75	Male	Not available	Esophageal mass	Bronchoscopy guided tracheal stent deployment	10	Noninvasive ventilation	Improved
Sato et al. [16]	39	Male	Not available	Malfunctioning insitu dumon stent	Rigid bronchoscopy guided replacement of malfunctioning dumon stent	<5	Noninvasive ventilation	Improved
Qin li et al. [17]	2	Male	30	Aspirated foreign body	Fiberoptic bronchoscopy guided removal of foreign body	<5	Noninvasive ventilation	Improved
Morales-Estrella et al. [18]	82	Female	Not available	Tracheal stenosis	Flexible bronchoscopy aided balloon bronchoplasty	2	Intravenous dexamethasone	Improved
Index case	40	Male	15	Esophageal malignancy	Rigid bronchoscopy guided coring	<5	Invasive mechanical ventilation	Improved

towards POPE as a cause [3].

Effective airway management and correction of hypoxemia are cornerstones of management of POPE. Artificial airway and ventilation with appropriate levels of positive end expiratory pressure may be required in severe cases [12]. Rapid resolution of clinical and radiological changes occurs with addition of PEEP. Our patient was required intubation for management of hypoxemia. Role of diuretics is uncertain as they may precipitate hypovolemia and hypoperfusion in post-operative setting [13].

A total of 5 cases of POPE have been reported in literature encountered during bronchoscopic management of central airway obstruction [14-18] [**Table 1**]. Four of these patients improved with positive pressure ventilation.

Thus, POPE is an important differential for respiratory distress in post-operative setting following relief of airway obstruction. Anticipating the complication along with early recognition and management may help in reducing morbidity and mortality associated with this condition.

Conclusion

POPE is a catastrophic clinical scenario in postoperative period presenting as rapid onset respiratory distress. With prompt diagnosis and therapeutic action, POPE resolves generally within 24 hours. A high index of suspicion must be maintained for POPE in patient who experience respiratory distress post relief of airway obstruction. Early recognition of this entity is crucial to decrease morbidity and mortality in these patients.

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