A Case of Negative Pressure Pulmonary Edema After Breast Implant Surgery

B Langenderfer

Citation

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Abstract

This case report concerns a 50 year old woman with no prior history of lung disease who exhibited respiratory distress, dyspnea and low oxygen saturations in the recovery room following breast implant surgery. The diagnosis of negative pressure pulmonary edema was made based on immediate history, radiographic exams, and testing that ruled out myocardial infarction and pulmonary embolus. High resolution CT scans revealed multiple non-calcified pulmonary nodules along with moderate-to-severe emphysema from a concealed smoking history.

INTRODUCTION

Negative pressure pulmonary edema (NPPE) is an uncommon but recognized complication of upper airway obstruction. It was first described in children in 1973 ([1]). This form of non-cardiogenic pulmonary edema occurs when the patient struggles to inhale against a partially or completely occluded upper airway. In a vigorous adult the inspiratory muscles can generate a negative intrapleural pressure in the range of -50 to -100 cm H2O ([2]). The high negative pressure gradient causes fluid to extravasate from the pulmonary capillaries into the interstitial and alveolar spaces ([3]). This fluid leak represents non-cardiogenic pulmonary edema, and it may result in immediate or delayed hypoxemia. Even after an upper airway obstruction is alleviated, pulmonary edema can develop immediately or up to six hours later ([4]). Upper airway compromise may be due to occlusion of an artificial airway, as when a patient bites down on an endotracheal tube. Or it may come from internal or external obstruction of the natural upper airway, as might happen in epiglottitis or strangulation.

In the case presented below, the radiographic exam was complicated by the recent placement of bilateral breast implants. An under-reported smoking history and unrecognized moderate to severe emphysema were contributing factors.

CASE REPORT

A 50 year old woman status post bilateral breast implant surgery exhibited dyspnea, wheezing, chest pressure, and low oxygen saturations after extubation in the post-anesthesia recovery room. Before extubation she was noted to be in respiratory distress with severe retractions.

A pulmonary consult several hours later described her as pleasant, cooperative, alert and oriented, and speaking in full sentences with only mild dyspnea and some chest pressure. Physical exam at that time revealed clear lungs without wheeze or crackles and an SpO2 of 89% on 2 L/min oxygen by nasal cannula. Cardiac exam noted regular rate and rhythm without chest pain or palpitations. The patient had a history of rheumatoid arthritis treated with methotrexate and gold salts, but no history of pulmonary disease, hemoptysis or pleurisy. Family history was positive for coronary artery disease in both mother and father. Before surgery she had informed the anesthesiologist that she smoked one pack of cigarettes per week, but after the episode of hypoxemia she admitted to smoking more than a pack per day.

The differential diagnosis included reaction to anesthesia, excessive IV fluids, myocardial infarction, pulmonary emboli, and negative pressure pulmonary edema.

A-P portable chest X-ray (Figure 1, chest X-ray of 6/26) revealed bilateral diffuse perihilar infiltrates and right lower lobe atelectasis, but the presence of breast implants complicated the radiographic analysis of both lower lung fields. High resolution chest CT scans with contrast were used to rule out pulmonary emboli. Over a hundred sequential scans revealed no evidence of pulmonary emboli or pneumothorax, but did show pulmonary edema

superimposed on moderate-to-marked emphysema with multiple non-calcified small pulmonary nodules (Figure 3, Chest CT #21, and Figure 4, chest CT #31). Subcutaneous emphysema and bilateral breast prostheses in the soft tissue of the anterior chest were consistent with the surgical history.

Figure 1

Figure 1: Chest X-ray of 6/26 reveals bilateral diffuse perihilar infiltrates and right lower lobe atelectasis, but the presence of breast implants complicates the radiographic analysis of both lower lung fields.

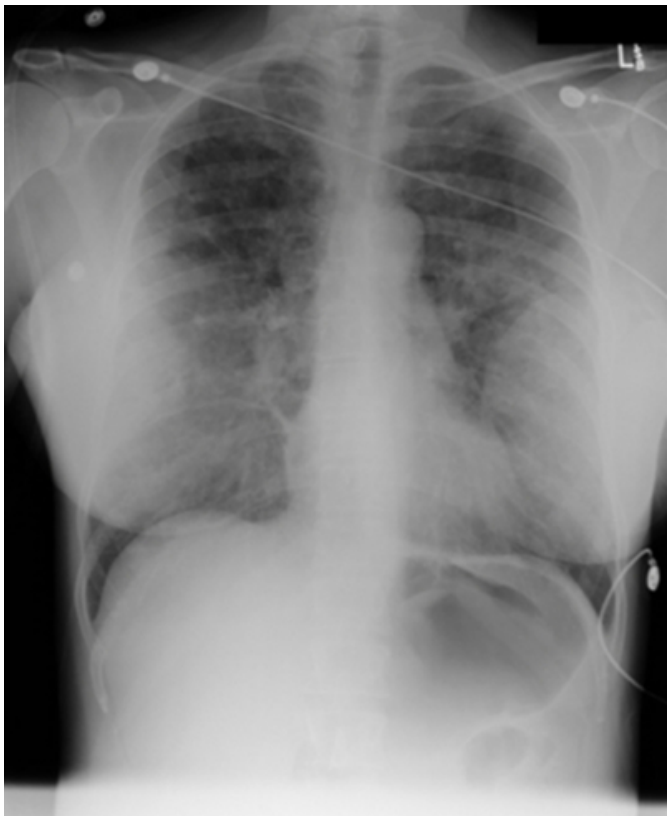


Figure 2

Figure 2: Chest X-ray of 6/27 shows almost complete resolution of the right and left perihilar infiltrates.

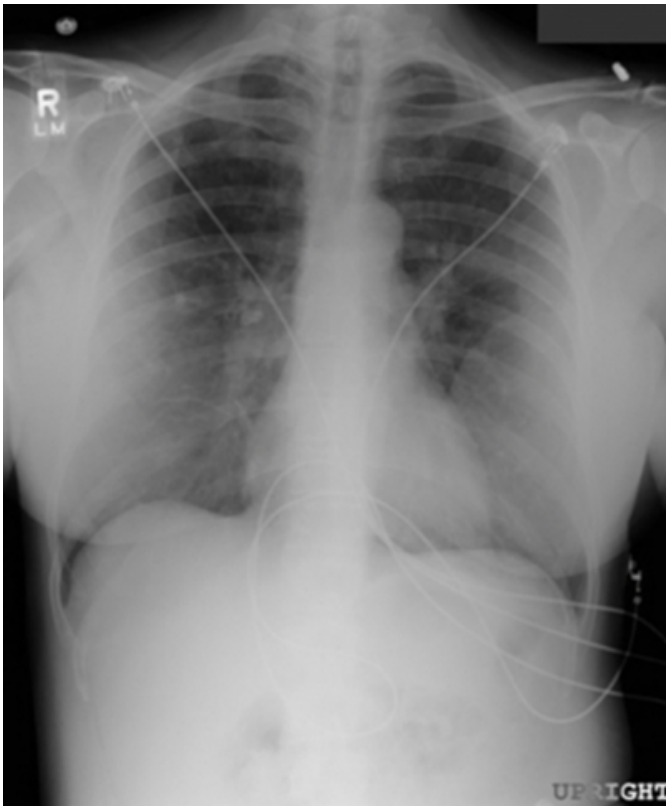


Figure 3

Figure 3: Chest CT image #21 shows pulmonary edema superimposed on moderate-to-marked emphysema but no evidence of pulmonary emboli.

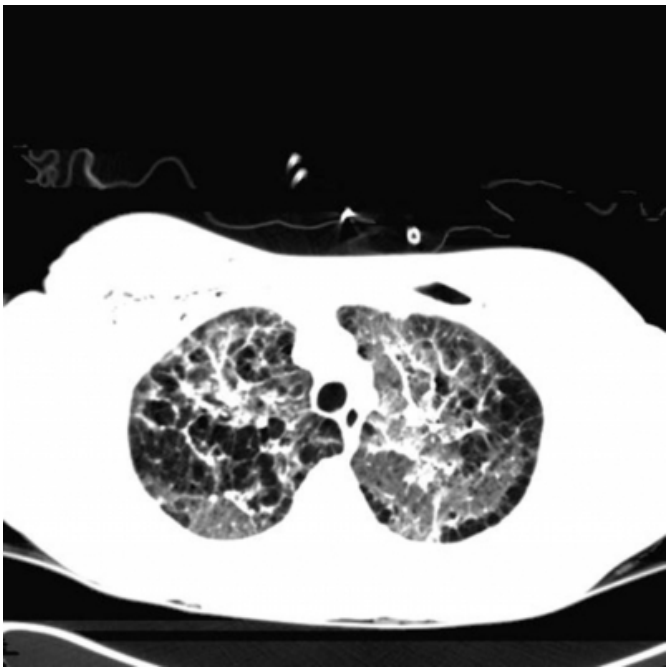
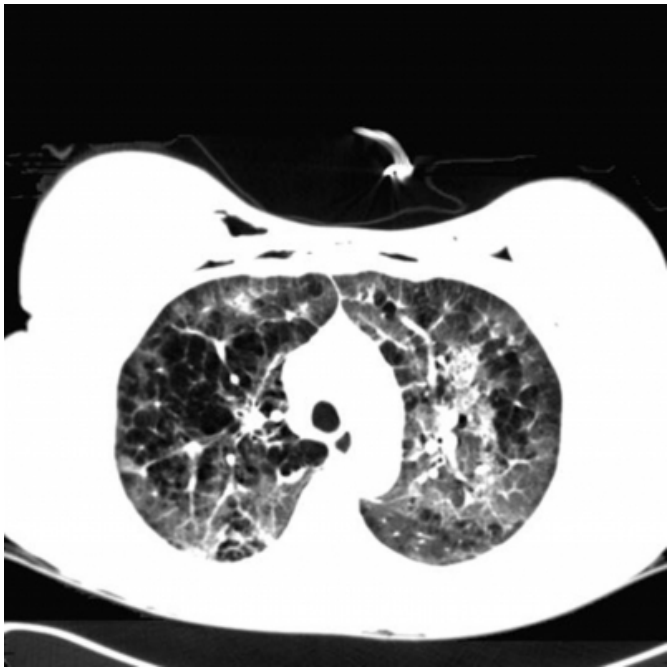


Figure 4

Figure 4: Chest CT image #31 shows pulmonary edema superimposed on moderate-to-marked emphysema but no evidence of pulmonary emboli. Subcutaneous emphysema and bilateral breast prostheses in the soft tissue of the anterior chest are consistent with the surgical history.



Serial troponin and creatinine phosphokinase levels along with echocardiography were used to rule out MI. Three successive troponin levels were all less than .35, and CK levels of 432, 497 and 716 reflected the surgical history. Echocardiography showed atria and ventricles normal in size and function, along with normal tricuspid, aortic and mitral valves.

Treatment was successful with oxygen therapy and diuresis. Figure 2, (chest X-ray of 6/27) shows almost complete resolution of the right and left perihilar infiltrates. The patient was discharged on the second postoperative day. Oximetry at rest and during ambulation three weeks later showed a normal saturation of 98% at rest, but during ambulation the SpO2 fell to 85%, reflecting the severity of the emphysema along with the side effects of treatment for rheumatoid arthritis.

The multiple pulmonary nodules seen on Chest X-ray and CT scan were most likely due to the gold and methotrexate used to treat her rheumatoid arthritis.

In this case the patient's lack of honesty before surgery with the anesthesiologist regarding the extent of her smoking contributed to her abrupt deterioration in the post-operative recovery room. Her change in condition was sudden and severe enough to cause significant concern between her surgeon and the anesthesiologist. The discovery of "moderate to severe emphysema" on the chest CT scan along with her admission of smoking more than a pack per day clarified the differential diagnosis.

DISCUSSION

NPPE is an uncommon problem, but it is common and serious enough to warrant quick recognition and effective treatment. NPPE has been estimated to occur in 11% of patients with clinically significant upper airway obstruction requiring treatment ([5]). In adults about 50% of NPPE occurrences are due to post-operative laryngospasm ([6]). Young physically fit athletes are at risk after surgery because they have the respiratory muscle power required to generate highly negative intrapleural pressures ([7]). Risk factors for NPPE include airway lesions, upper airway surgery, obesity and obstructive sleep apnea. Besides post-extubation laryngospasm, reported causes include foreign bodies, hanging, strangulation, croup, epiglottitis, obstructive sleep apnea, and artificial airway obstruction ([3]).

Several pathways for the development of NPPE have been proposed, but the exact mechanism has not been determined. In the usual explanation, NPPE begins with a significant upper airway obstruction. Strong inspiratory efforts to overcome the obstruction generate highly negative intrapleural and alveolar pressures, and the high pressure gradient cause fluid to move out of the pulmonary capillaries and into the interstitial and alveolar spaces ([3]).

Another explanation for NPPE holds that the highly negative intrathoracic pressures cause an increase in systemic venous return to the heart but a decrease in cardiac output, since pulmonary venous drainage to the left atrium is decreased. Pulmonary capillary pressures increase while alveolar pressures plunge, and alveolar cell junctions are disrupted. Fluid moves rapidly into interstitial and alveolar spaces, and the pulmonary edema fluid remains even after the airway obstruction is relieved ([4]). Both of these pathways along with other mechanisms contribute to the development of NPPE.

In clinical presentation, initial findings usually include decreased oxygen saturation, with pink frothy sputum and chest X-ray abnormalities ([2]). Manifestations of the acute airway obstruction include stridor, suprasternal and supraclavicular retractions, urgent use of accessory muscles of inspiration, and panic in the facial expression. As NPPE develops, auscultation usually reveals crackles and

occasionally wheezes. Pulmonary edema causes both impaired diffusion of oxygen and ventilation/perfusion mismatching, leading to sudden and possibly severe hypoxemia. The typical chest X-ray will show diffuse interstitial and alveolar infiltrates ($_{7}$).

The differential diagnosis would include acute respiratory distress syndrome, fluid overload, cardiac abnormalities, pulmonary embolism, and NPPE.

The first treatment priority is relief of the airway obstruction and correction of hypoxemia. The next step is to address the pulmonary edema with a diuretic unless the patient is hypovolemic. Effective airway management and immediate treatment with oxygen and diuretics is sufficient in most cases of NPPE. Persistent airway obstruction may necessitate an artificial airway, and acute respiratory failure would require artificial ventilation with oxygen and appropriate levels of PEEP. If the airway obstruction is due to the patient biting down on the endotracheal tube, a dose of succinylcholine (0.1-0.2 mg/kg) may be needed to relax the jaw muscles ($_{7}$).

CORRESPONDENCE TO

Bob Langenderfer MEd, RRT-NPS, Respiratory Care Program, School of Nursing and Health Professions, Northern Kentucky University, Nunn Drive HC-225C, Highland Heights, KY 41099. Email: langenderfer@NKU.edu

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Author Information

Bob Langenderfer, MEd, RRT-NPS
School of Nursing and Health Professions, Northern Kentucky University