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# Negative Pressure Pulmonary Edema After Electroconvulsive Therapy

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A 22-year-old female was referred to electroconvulsive therapy (ECT) for a history of bipolar I with depressive episodes refractory to multiple medication trials. Additional medical history included obesity, well-controlled asthma and anemia. During her first treatment, there was concern for inadequate paralysis due to greater than expected movement. In recovery she had multiple episodes of emesis. Prior to her second treatment, the had clear breath sounds bilaterally, oxygen saturation 97%, and was normotensive and afebrile. She underwent right unilateral ECT at 30% charge, pulse width 0.3 milliseconds, frequency 40 Hz using Thymatron System IV integrated ECT device (Somatics LLC, IL, USA) with seizure duration of 99 seconds. She was agitated upon awakening, resisted ventilation, and desaturated to roughly 60% spO2 on room air. Despite bag-valve-mask ventilation and sedation with 2mg midazolam and 50mg propofol, oxygenation did not improve. She was intubated and spO2 increased to 95%. Phenylephrine was given for hypotension (86/44 mmHg) secondary to sedation. Chest X-ray (CXR) showed bilateral diffuse interstitial opacities compared to prior imaging (Figure 1a, 1b). She received 40mg IV Lasix, was extubated the next morning and subsequently weaned off supplemental oxygen. Repeat CXR showed a decrease in infiltrates consistent with resolving pulmonary edema (Figure 1c). She was normotensive, afebrile, and deemed stable for discharge on day two of hospitalization.

Pulmonary edema following ECT is a rare but serious adverse event. Mechanisms include neurogenic, cardiogenic, and negative pressure pulmonary edema (NPPE). Neurogenic pulmonary edema follows a central nervous system insult and is thought to be mediated by sympathetic discharge and vasoactive substances, and is suggested if other causes have been ruled out.[1] Cardiogenic pulmonary edema would be suggested by perihilar prominence on CXR and pulmonary venous congestion, as well as markedly abnormal vital signs.[2] In this case, cardiogenic pulmonary edema was thought to be unlikely based on normal EKG. NPPE is due to inspiration against an obstructed airway creating a large negative intrathoracic pressure resulting in transudation and injury to capillary endothelium.[2,3]

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In this case, NPPE was suspected due to the patient's age, history of reactive airway disease, acute agitation following anesthesia with rapid onset of respiratory distress, and attempted inspiration against mask airway.[2] Furosemide is a common treatment but debated due to lack of fluid overload in NPPE. Instead, alpha blockade has been suggested to modulate catecholamine effects on vascular permeability.[2,3] Finally, aspiration pneumonitis was considered in this case based on her emesis following the first treatment. However, given unremarkable exam findings preceding the second treatment and rapid resolution of opacities this is less likely. An important consideration in the future is the risks and benefits of continuing ECT in a patient who has undergone a serious adverse event. In the literature, ECT has been continued following pulmonary edema.[2] For this patient, early management of postictal agitation would be prioritized. Pulmonary edema is a serious complication of ECT that is likely underreported and warrants ongoing investigation regarding causes, incidence, and management.

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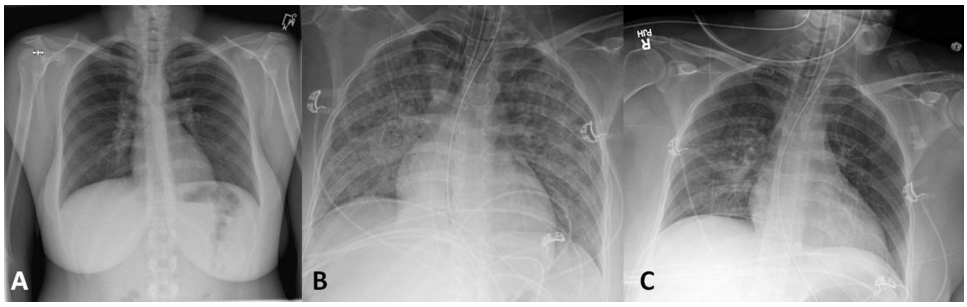


**Figure 1:**

A) Chest X-ray taken approximately 3 years prior to initiation of ECT. B) Chest X-ray taken upon admission to intensive care unit following second ECT treatment. C) Chest X-ray taken post-procedure day 1, showing interval resolution of pulmonary edema.