# NEGATIVE PRESSURE PULMONARY EDEMA DUE TO RIGOR AND CHILLS IN LIVER ABSCESS

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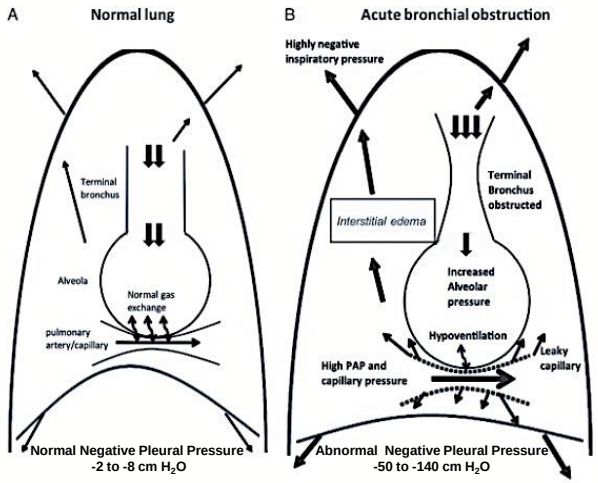
## Background

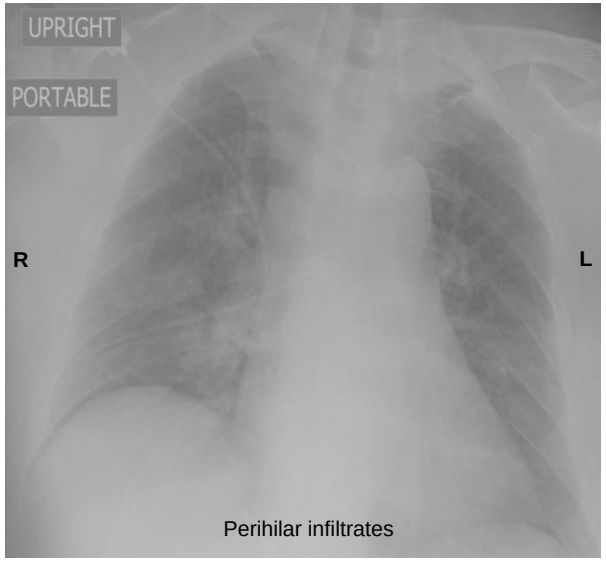
- Negative pressure pulmonary edema (NPPE) cause of acute respiratory failure that occurs after intense inspiratory effort against an obstructed airway, usually from upper airway infection, tumor, or laryngospasm.
- Commonly reported etiology of NPPE in adults is laryngospasm during intubation or in the postoperative period after anesthesia.
- Pathophysiology includes (1) generation of large negative intrathoracic pressure with subsequent increase in preload, (2) augmentation of systemic blood pressure with increase in afterload, and (3) stress failure of the alveolar capillary membrane. Pulmonary edema fluid has a low protein concentration, suggesting hydrostatic forces as the primary mechanism for the pathogenesis of NPPE.
- Clinical features include Dyspnea, Marked respiratory efforts, desaturation events and bilateral infiltrates on chest imaging.
- Treatment includes maintaining patent airway, oxygen supplementation, diuretics, noninvasive positive pressure ventilation (NIV) and mechanical ventilation.




## Case History

- We present case of NPPE occurring after episodes of rigors & chills associated with liver abscess.
- 61 years old male with PMHx of HTN, HLD, T2DM on Amlodipine, Aspirin, Atorvastatin, Hydrochlorothiazide, Lisinopril and Metformin
- He presented to the ED with progressive generalized weakness, myalgias, diaphoresis, fever, episodic chills & rigors that started 4 days ago.
- ED vital signs: T 99.6 F, HR 97, RR 20, BP 147/73, SpO2 92% room Air. investigations: CBC and CMP Unremarkable, lactic acidosis (3.3), AST 60, ALT 72, Glucose 323 UA: Glucose and trace ketones positive. CXR showed overlying curvilinear radiopacities. Abdominal CT revealed lesions in the liver & bilateral adrenals. He was started on empiric IV Piperacillin / Tazobactam for 7 days & admitted under diagnosis of gram-negative bacteremia/sepsis.
- Admission Day 1, after an episode of rigors, he developed dyspnea & acute hypoxic respiratory failure with inspiratory stridor on examination. CXR revealed a new, bilateral airspace disease. While on 100% FiO2 non-rebreather mask, he was transferred to ICU & given racemic Epinephrine, Solumedrol, Toradol, & Diphenhydramine under a presumptive diagnosis of foreign body aspiration or allergic reaction.
- **Neck X-ray demonstrated patent airway and excluded any large foreign body and retropharyngeal abscess. He continued to develop hypoxic respiratory failure with stridor and an incremental worsening of pulmonary edema on CXR, with each subsequent episode of rigor and chills.**
- **Attending Pulmonologist assessed that NPPE was secondary to intermittent vocal cord muscle spasm. He was diagnosed as NPPE likely caused by transient laryngeal dyskinesia induced by the increased work of breathing associated with rigors.**
- Blood culture exhibited Klebsiella bacteremia. The respiratory failure resolved after the course of antibiotics along with supplemental oxygen. He was gradually transitioned to room air and discharged 9 days later.

## Chest X-ray (Admission Day 1)




Perihilar infiltrates

## Discussion/Conclusion

- This patient developed NPPE secondary to transient laryngeal dyskinesia during the episodes of rigors and chills in the setting of Klebsiella bacteremia.
- He responded to racemic Epinephrine, Solumedrol, Toradol, Diphenhydramine, supplemental oxygen, IV antibiotics and supportive therapy.
- Patients with NPPE generate very negative airway pressures, which augment trans-vascular fluid filtration and precipitate interstitial and alveolar edema. These patients respond to supportive management.

## References

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