Case Report
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Acute on Chronic Hypercapnic Respiratory Failure in a Patient with Childhood Tuberculosis: A Diagnostic and Management Case Study

Mayesha Ahmed[1] | Jasmine Cha[1*] | Michael Pierides[1] | Esha Kauravlla[1] | Roxana Lazarescu[2]

***Correspondence:** Jasmine Cha
Address: [1]Touro College of Osteopathic Medicine, New York, NY, USA; [2]Wyckoff Heights Medical Center, Department of Internal Medicine, NY, USA
E-mail ✉: jcha5@student.touro.edu
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ABSTRACT

A 61-year-old female with asthma and a history of childhood tuberculosis presented to an urgent care center with 3 days of worsening dyspnea and progressed to hypercapnic respiratory failure, prompting transfer to the emergency department and initiation of Bilevel Positive Airway Pressure (BiPAP) machine. Initial arterial blood gas on presentation demonstrated pH of 7.29, pCO_2 of 70 mmHg, and HCO_3 of 33.7 mEq/L with a pO_2 of 171 mmHg on BiPAP. Imaging revealed near-complete destruction of the left lung with mediastinal shift and compensatory hyperinflation of the right lung with severe emphysema. BiPAP was discontinued due to concern for dynamic hyperinflation and barotrauma. The patient was managed with careful oxygen titration targeting SpO2 88-92%, corticosteroids, and bronchodilators, with clinical improvement and discharge on home O2 as needed. This case highlights the individualized respiratory support required in a patient with functional single-lung physiology.

Keywords: *Post-Tuberculosis Lung Disease (PTLD), Unilateral Lung Destruction, Hypercapnic Respiratory Failure, Dynamic Hyperinflation, Single-lung Physiology, Barotrauma Risk, Non-invasive Ventilation (NIV), Obstructive Lung Disease.*

Introduction

Tuberculosis (TB) remains a leading infectious cause of morbidity and mortality worldwide (Berida and Lindsley, 2024). Post tuberculosis lung disease (PTLD) is increasingly recognized as a major contributor to chronic respiratory morbidity, affecting up to half of individuals following successful treatment (Yarbrough *et al*., 2024). One severe manifestation of PTLD is destroyed lung syndrome, characterized by extensive unilateral pulmonary destruction resulting from fibrosis, bronchiectasis, cavitation, and significant loss of lung volume (Gyabaah *et al*., 2024). In these patients, compensatory hyperinflation of the contralateral lung may develop to maintain ventilation (Gyabaah *et al*., 2024).

Clinical manifestations of destroyed lung syndrome include chronic cough, recurrent infections, and progressive respiratory impairment (Gyabaah *et al*., 2024). Patients with tuberculosis-destroyed lung may experience complications such as pneumonia, hemoptysis, pneumothorax, and acute respiratory failure requiring ventilatory support (Ryu *et al*., 2011).

Management of acute respiratory failure in patients with DLS presents unique challenges due to the marked asymmetry between the diseased and functional lungs. Conventional ventilatory strategies, including bilevel positive airway pressure (BiPAP), may risk overdistention of the hyperinflated contralateral lung while providing limited ventilation to the destroyed lung, potentially worsening ventilation-perfusion mismatch and gas exchange (Thomas and Bryce, 1998). In addition, patients with underlying structural lung disease may be at increased risk of barotrauma when exposed to positive pressure ventilation (Thachuthara-George, 2021).

We present a case of acute on chronic hypercapnic respiratory failure in a patient with childhood TB-related unilateral lung destruction, highlighting the diagnostic and management challenges associated with destroyed lung syndrome and single-lung physiology.

Case Presentation

A 61-year-old female with a history of asthma, managed with Symbicort and Flovent without prior need for intubation or home oxygen, and hypertension presented to the emergency department after being referred from an urgent care center for hypoxia. She reported a 2–3-day history of progressively worsening shortness of breath accompanied by new chest and throat tightness. At urgent care, her SpO_2 was reportedly 77%, and she received two doses of albuterol and dexamethasone of unknown dose. EMS administered a DuoNeb during transport. She stated that this episode was significantly more severe than any prior asthma exacerbation and that her home inhalers provided no relief. She denied fever, chills, cough, chest pain, nausea, vomiting, or lower extremity pain. Her history was notable for a recent 14-hour flight and a remote childhood history of tuberculosis in Nepal, for which she was treated twice. She reported that she had been informed during a previous hospitalization that she “had only one lung.”

On arrival, the patient appeared tachypneic and hypoxic with diffuse wheezing and poor air movement, prompting initiation of BiPAP. Vital signs included a temperature of 36.7°C, heart rate of 101 bpm, blood pressure of 177/91 mmHg, and SpO_2 of 100% while on BiPAP. Physical examination showed increased work of breathing with diffuse expiratory wheezes; the remainder of the cardiopulmonary, abdominal, and musculoskeletal examinations was unremarkable. Her initial neurological examination was

intact, though she later developed blurred vision, dizziness, headache, memory impairment, and generalized weakness, consistent with hypercapnic encephalopathy. Pulmonology recommended discontinuation of BiPAP due to concerns for severe emphysema and risk of air trapping, and she was transitioned to nasal cannula oxygen.

Chest radiography revealed diffuse bilateral interstitial prominence, left basilar consolidation, and a large left pleural effusion (Fig. 1). A non-contrast CT of the chest demonstrated severe emphysema with marked hyperexpansion of the right lung, near-complete destruction and volume loss of the left lung with mediastinal shift, and multiple calcified granulomas (Fig. 2). A 1.2-cm spiculated right lower-lobe pulmonary nodule was identified. CT of the head showed no acute abnormalities. Laboratory testing revealed no leukocytosis, but troponin levels were elevated (83.7→69 ng/L), consistent with type 2 myocardial injury. Serum bicarbonate was elevated (33–39 mEq/L), reflecting chronic CO_2 retention. Her hemoglobin decreased from 12.1 to 10.0 g/dL during hospitalization, and creatinine rose slightly from 0.94 to 1.12 mg/dL. Infectious studies, including a respiratory viral panel, were negative.

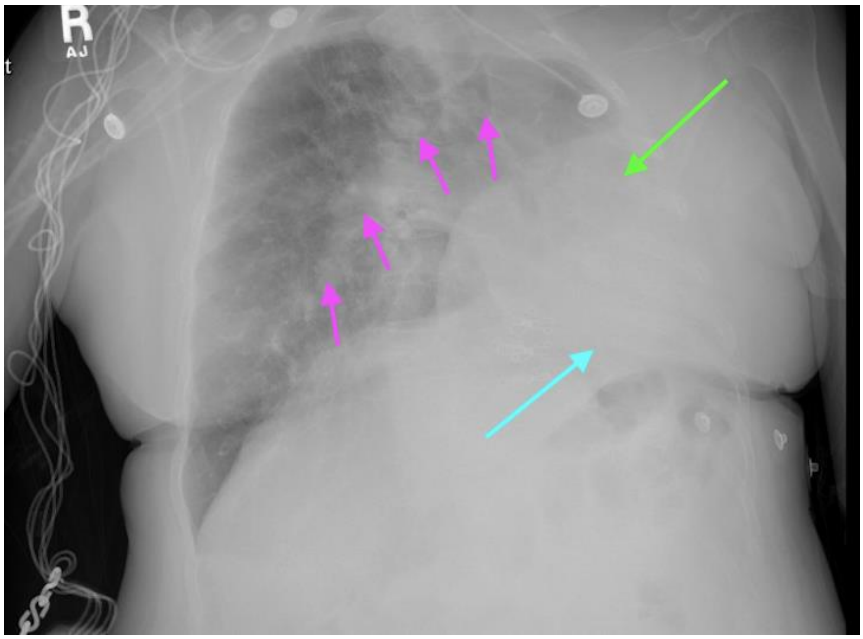

Figure 1: Xray Chest.

Arrows:

- Pink = Diffuse bilateral interstitial prominence
- Green = Large left pleural effusion
- Blue = Left basilar consolidation

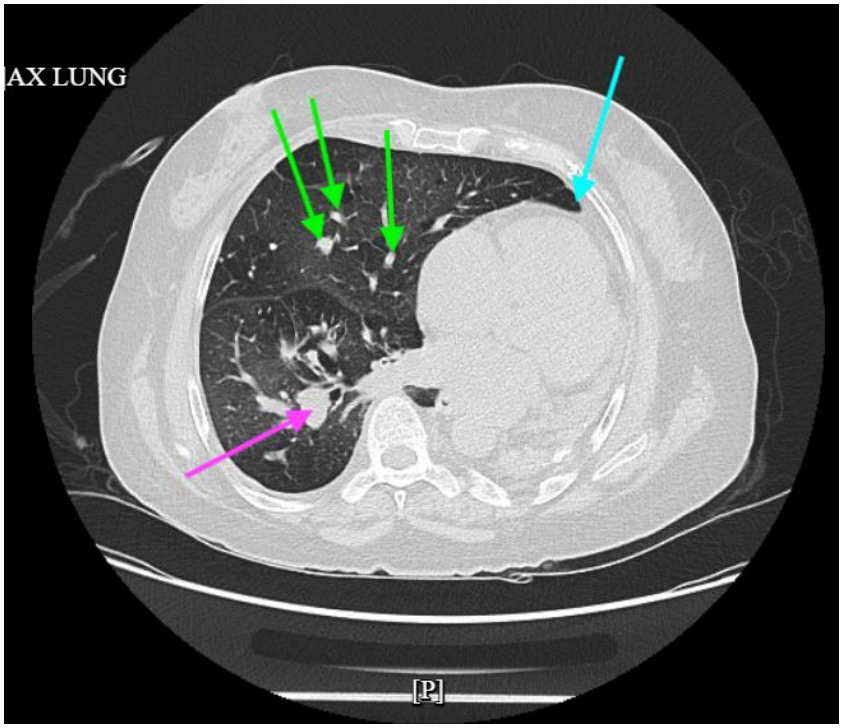

Figure 2: Non-contrast CT chest.

Arrows:

- Green = multiple calcified nodules
- Blue = volume loss of the left lung with mediastinal shift
- Pink = 1.2-cm speculated right lower-lobe pulmonary nodule

Initial arterial blood gas demonstrated a pH of 7.29, pCO_2 of 70 mmHg, and HCO_3 of 33.7 mEq/L with a pO_2 of 171 mmHg on BiPAP, consistent with acute on chronic hypercapnic respiratory failure. Repeat ABGs showed persistent but compensated hypercapnia (pH 7.34, pCO_2 73 mmHg, HCO_3 51 mEq/L).

The patient was treated with intravenous methylprednisolone, scheduled and PRN bronchodilator nebulizations, nebulized budesonide, and empiric ceftriaxone and doxycycline. She received DVT prophylaxis, PPI therapy, and PT/OT support with aspiration and fall precautions. She was closely monitored with telemetry, serial troponins, and serial ABGs. Over her ICU course, her mental status and work of breathing gradually improved as CO_2 levels decreased, and she avoided the need for invasive mechanical ventilation. Her bicarbonate trended downward, and troponins declined without EKG changes. No infectious source was identified. She developed transient hypercapnic encephalopathy and mild anemia but remained hemodynamically stable.

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The patient was ultimately stabilized on nasal cannula oxygen with improved respiratory status. Plans for outpatient follow-up included evaluation for asthma–COPD overlap syndrome, long-term management of chronic unilateral lung destruction and emphysema, and repeat chest imaging or PET/CT in three months to assess the incidentally discovered 1.2-cm spiculated pulmonary nodule.

Discussion

This case presents a diagnostically complex presentation of acute-on-chronic hypercapnic respiratory failure in a 61-year-old woman with a history of childhood TB, unilateral lung destruction, severe compensatory emphysema, and probable asthma-COPD overlap syndrome (ACO). TB remains one of the leading infectious causes of chronic obstructive pulmonary disease worldwide, particularly in patients from endemic regions (Meghji *et al*., 2025). Post-tuberculosis lung disease (PTLD) encompasses a spectrum of structural and functional abnormalities including bronchiectasis, fibrosis, cavitation, and lobar or total lung destruction that can persist long after successful treatment (Malefane and Maarman, 2024). Our patient, originally from Nepal with two treated childhood TB episodes, presented with near-complete destruction of the left lung and mediastinal shift consistent with advanced PTLD. She was unaware of the full extent of her disease, having only been told she had "one lung" during a prior hospitalization. This underscores the importance of detailed immigrant health history, as patients with PTLD from resource-limited settings may carry severe structural disease without formal workup or diagnostic clarity.

The near-complete destruction of this patient's left lung imposed a physiologic state analogous to that of a post-pneumonectomy patient, with the entire burden of gas exchange falling on a single, structurally compromised right lung. Pulmonary reserve is severely diminished in this context, meaning that acute insults a patient with two healthy lungs might tolerate, such as bronchospasm or mild infection, can precipitate rapid respiratory failure. This likely explains why a moderate asthma exacerbation resulted in an SpO_2 of 77% at urgent care. Pulmonary vascular resistance is also chronically elevated, as the loss of the left lung's vascular bed concentrates the entire cardiac output through the remaining right-sided pulmonary circulation, placing a sustained pressure burden on the right ventricle and reducing tolerance for further increases in vascular resistance from hypoxia or hypercarbia (Lan *et al*., 2010). Ventilation-perfusion mismatch is an inherent consequence of this physiology, worsened here by superimposed emphysematous changes and bronchospasm in the remaining right lung. The compensatory hyperinflation of the right lung, while adaptive over time, also flattens the diaphragm and reduces its mechanical efficiency, further limiting the patient's ability to augment minute ventilation during periods of increased demand (Deslauriers *et al*., 2011). Collectively, these physiologic vulnerabilities explain both the severity

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of this patient's presentation and the narrow margin between stability and decompensation that characterized her hospital course.

A central diagnostic challenge in this case was distinguishing between an acute asthma exacerbation and an exacerbation of underlying COPD or ACO. The patient carried a longstanding diagnosis of asthma and presented with diffuse wheezing and bronchospasm, yet the CT findings of severe emphysema and the ABG pattern of chronic CO_2 retention with metabolic compensation, with bicarbonate as high as 51 mEq/L, indicate a degree of physiologic impairment far exceeding what asthma alone would typically produce. ACO is characterized by persistent airflow limitation with features of both asthma and COPD and is associated with worse outcomes than either condition alone, including more frequent exacerbations, accelerated lung function decline, and greater comorbid burden (Calverley and Walker, 2021). Formal spirometry was not obtainable during this acute admission, but the clinical and radiographic findings strongly support ACO as the underlying diagnosis. This distinction carries important therapeutic implications, as patients with ACO and underlying emphysema require careful oxygen titration targeting SpO_2 88-92% to avoid blunting hypoxic respiratory drive, and heightened vigilance for hypercapnic decompensation (Lius and Syafaah, 2022).

Ventilatory management in this case required careful reassessment as new diagnostic information emerged. Initial BiPAP use was appropriate given the severity of bronchospasm and hypercapnic respiratory failure on presentation13, but pulmonology subsequently recommended discontinuation due to the risk of worsening air trapping and dynamic hyperinflation in the setting of severe emphysema14. Positive pressure ventilation, including noninvasive modalities, can increase intrinsic PEEP and precipitate hemodynamic compromise or barotrauma in this population, making the transition to low-flow nasal cannula oxygen both necessary and appropriate (Liu *et al*., 2016). Beyond ventilation mode, several additional risk mitigation principles guided successful management. Careful fluid management was essential, as volume overload exacerbates V/Q mismatch and can precipitate pulmonary edema in a system with no contralateral reserve14. Avoidance of dynamic hyperinflation through extended expiratory times and conservative respiratory rates would similarly have been critical had invasive ventilation been required (Leatherman, 2015). The type 2 myocardial injury observed, with troponin peaking at 83.7 before trending downward, was attributed to demand ischemia in the setting of severe hypoxia, tachycardia, and chronically elevated pulmonary vascular resistance, and appropriately resolved with treatment of the underlying respiratory failure rather than a primary acute coronary syndrome intervention (Thygesen *et al*., 2018).

Finally, the incidentally discovered 1.2 cm spiculated right lower lobe nodule introduces an important and time-sensitive concern (MacMahon *et al*., 2017). Spiculated morphology is associated with malignancy in a substantial proportion of cases, and this patient carries multiple compounding risk factors including a history of TB, which is independently associated with elevated lung cancer risk likely due to chronic inflammation and fibrosis, immigration from a TB-endemic region, and age over 60 (Hwang *et al*., 2022). While the calcified granulomas seen on CT are consistent with her prior TB history and suggest prior granulomatous disease, they do not exclude a concurrent malignant process (Yedgarian *et al*., 2025). Current guidelines support either repeat CT in three months or PET/CT depending on clinical suspicion (Yedgarian *et al*., 2025). Of particular importance in this patient is that any malignancy arising in the only functional lung would carry profoundly limited therapeutic options, making early detection especially consequential. This case ultimately illustrates the intersection of post-infectious structural lung disease, functionally single-lung physiology, and ACO, a combination requiring individualized ventilatory management, careful diagnostic reassessment, and coordinated subspecialty care. Clinicians encountering similar presentations should maintain a broad differential, avoid anchoring on a single diagnosis, and remain alert to the cumulative consequences of structural, obstructive, and infectious lung disease acting in concert.

Conclusion

This case illustrates the complexity of obstructive airway disease with an underlying post-tuberculosis destroyed lung and more broadly, functional single-lung physiology. Patients with unilateral lung destruction have significantly reduced pulmonary reserves, rendering them highly vulnerable to rapid decompensation from even moderate respiratory insults. This case highlights the diagnostic challenge of distinguishing between asthma exacerbation and asthma-COPD overlap without pulmonary function testing. Furthermore, standard ventilatory approaches, including noninvasive positive pressure ventilation, may be detrimental in patients with one lung due to heightened risks with hyperinflation and barotrauma in the remaining lung. Early imaging, recognition of hypercapnia, and careful reassessment of respiratory support were crucial to avoid invasive ventilation in stabilizing this patient. Broadly, this case emphasizes the importance of recognizing underlying structural lung disease and adopting an individualized approach to respiratory management.

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