


# Clinical Triage of Massive Pulmonary Embolism Masked by Giant Bullous Disease: The Diagnostic Value of Silent Hypoxia and Physical Examination

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**Background:** Giant bullous disease (GBD) frequently complicates the diagnosis of acute pulmonary embolism (PE) by creating radiographic ambiguity that mimics pneumothorax. In settings where advanced imaging is not immediately available, clinical examination is the primary tool for risk stratification.

**Case Presentation:** A 65-year-old female presented with subjective dyspnea. Despite a profound oxygen saturation of 80%, she exhibited a normal respiratory rate and minimal distress, a state known as silent hypoxia. Initial chest X-rays showed a hyperlucent left hemithorax. While this suggested a pneumothorax, the lack of hyper-resonance on percussion pointed toward GBD. Bedside echocardiography confirmed right ventricular strain and a thrombus. Due to the lack of on-site resources, the patient was transferred to a private facility for CT pulmonary angiography (CTPA), confirming a massive PE and a 15-cm giant bulla.

**Discussion:** This case illustrates how silent hypoxia serves as a physiological red flag for vascular obstruction. By using percussion to rule out pneumothorax, we avoided a potentially fatal chest tube insertion into the bulla. Bedside ultrasound provided the clinical justification for a costly, off-site transfer.

**Conclusion:** Clinical acumen remains the most effective safeguard against iatrogenic injury and diagnostic delay in complex cardiopulmonary cases.

**Keywords:** happy hypoxia, pulmonary embolism, giant bullous disease, resource-limited setting, Echocardiography, case report

## Introduction

The diagnosis of pulmonary embolism (PE) relies on the integration of clinical pre-test probability, typically assessed via the Wells Score<sup>1</sup> or the Pulmonary Embolism Rule-out Criteria (PERC),<sup>2</sup> followed by definitive imaging as per international guidelines.<sup>3</sup> While a chest X-ray (CXR) is not a diagnostic tool for PE, it is a vital initial screening step to exclude other causes of acute dyspnea. However, in patients with giant bullous disease (GBD)—formally defined as air spaces occupying over one-third of the hemithorax<sup>4</sup>—initial screening is fraught with difficulty. GBD creates a significant radiographic trap by mimicking a tension pneumothorax on CXR.<sup>5</sup> This challenge is amplified in the Somali context, where the rising burden of chronic non-communicable diseases often presents amidst limited specialized diagnostic modalities, leading to late or incidental diagnoses of life-threatening conditions.<sup>6</sup> This mimicry does not affect the technical accuracy of definitive imaging like CT pulmonary angiography (CTPA), but it significantly complicates the initial clinical suspicion and triage.

This case highlights how clinical vigilance is paramount when incongruous findings emerge, particularly in resource-limited environments like Borama, where cardiovascular and respiratory emergencies must be managed with a high index

of suspicion due to the lack of immediate specialized care.<sup>7</sup> The phenomenon of silent hypoxia describes a physiological state where profound arterial hypoxemia occurs without a commensurate increase in objective respiratory work, such as severe tachypnea or accessory muscle use.<sup>8</sup> In our patient, the clinical picture was highly discrepant: she failed the PERC criteria due to her profound hypoxemia (80%), yet her Wells Score was only moderately suggestive of PE due to the absence of tachycardia (HR 88 bpm).<sup>1,2</sup> Such unexplained hypoxia—which far exceeds the chronic hypoxemia typically associated with baseline GBD or emphysema—serves as a vital marker of severe ventilation-perfusion (V/Q) mismatch.<sup>9–11</sup> In environments where diagnostic resources are limited, recognizing these incongruous objective signs is the primary safeguard against diagnostic error and inappropriate intervention.<sup>3</sup>

## Case Presentation

A 65-year-old female (BMI 27.4 kg/m<sup>2</sup>) presented to the emergency department with acute-onset dyspnea. She was not a smoker herself, but she had a 30-pack-year history of significant second-hand smoke exposure from her husband. Her medical history was significant for chronic cough, though no formal diagnosis of COPD or emphysema had been established. Before the last month, her exercise tolerance was preserved; she was able to walk approximately 500 meters and climb one flight of stairs without significant distress. However, she had been sedentary for the past four weeks due to malaise, which served as the provocative factor for her unprovoked PE. She had no history of surgery or prior use of long-term inhalers.

Physical examination revealed silent hypoxia; her SpO<sub>2</sub> was 80% on room air, yet her respiratory rate remained 16–18 breaths/minute. Arterial blood gas (ABG) analysis was unavailable at our facility due to resource constraints. However, there were no clinical signs of chronic hypercapnia, such as a flapping tremor or bounding pulse, and the sudden nature of her desaturation in the context of her previously active lifestyle pointed toward an acute V/Q mismatch rather than chronic respiratory failure.

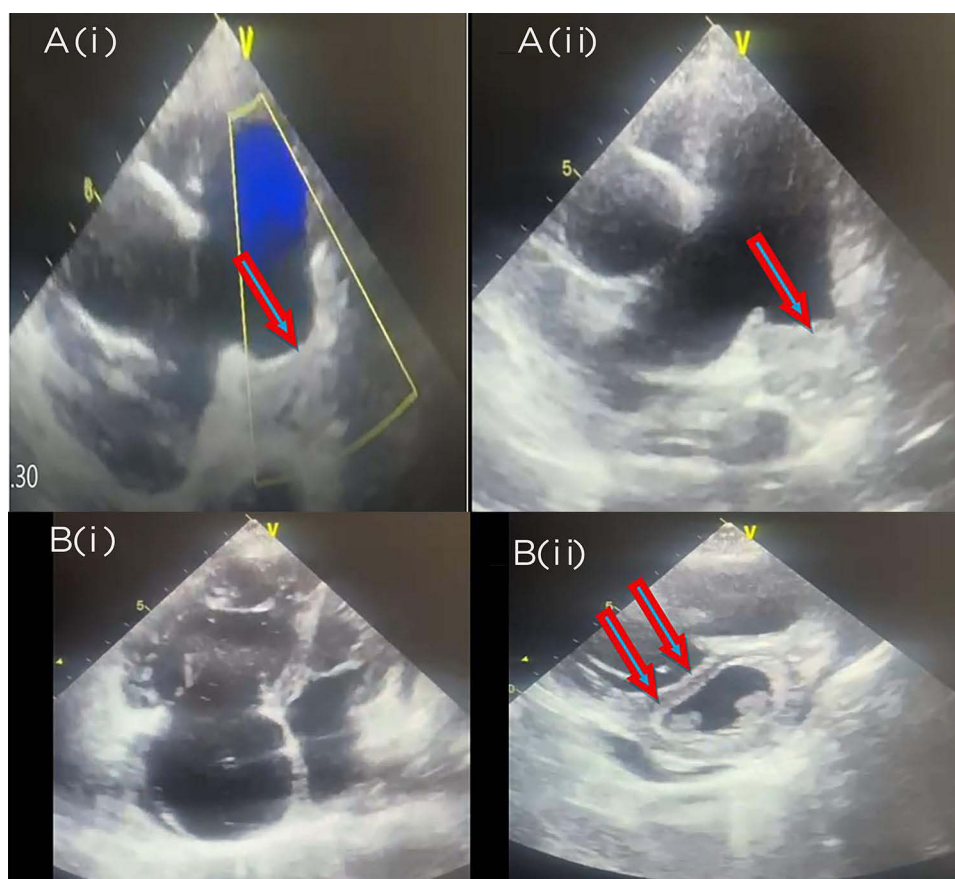
Initial X-ray showed a hyperlucent left hemithorax (Figure 1). Because our facility lacked an on-site CT scanner, we relied on clinical percussion (which showed no hyper-resonance) and bedside echocardiography (which showed RV strain and thrombus) (Figure 2) to justify the risk and cost of transferring the patient to a distant private facility for a CTPA. The CTPA confirmed a massive PE and a 15-cm giant bulla.

The CTPA revealed a massive left main pulmonary artery embolism (Figure 3) and confirmed a 15×10.2 x 6.0 cm giant bulla (Figure 4). The scan also showed cystic bronchiectasis, leading us to suspect prior tuberculosis (TB); however, sputum GeneXpert and AFB stains were negative, indicating inactive disease.

Therapeutic anticoagulation was initiated. The patient's oxygen saturation improved steadily during her hospital stay. At the three-month follow-up, her SpO<sub>2</sub> was 94% on room air. This significant recovery from an initial 80% saturation—coupled with her previously preserved exercise capacity—confirmed that the profound hypoxemia was primarily an acute vascular event rather than a chronic baseline state caused by the giant bullous disease.



**Figure 1** Anteroposterior chest X-ray demonstrating a hyperlucent left hemithorax with an absence of lung markings. The label L indicates Legends.

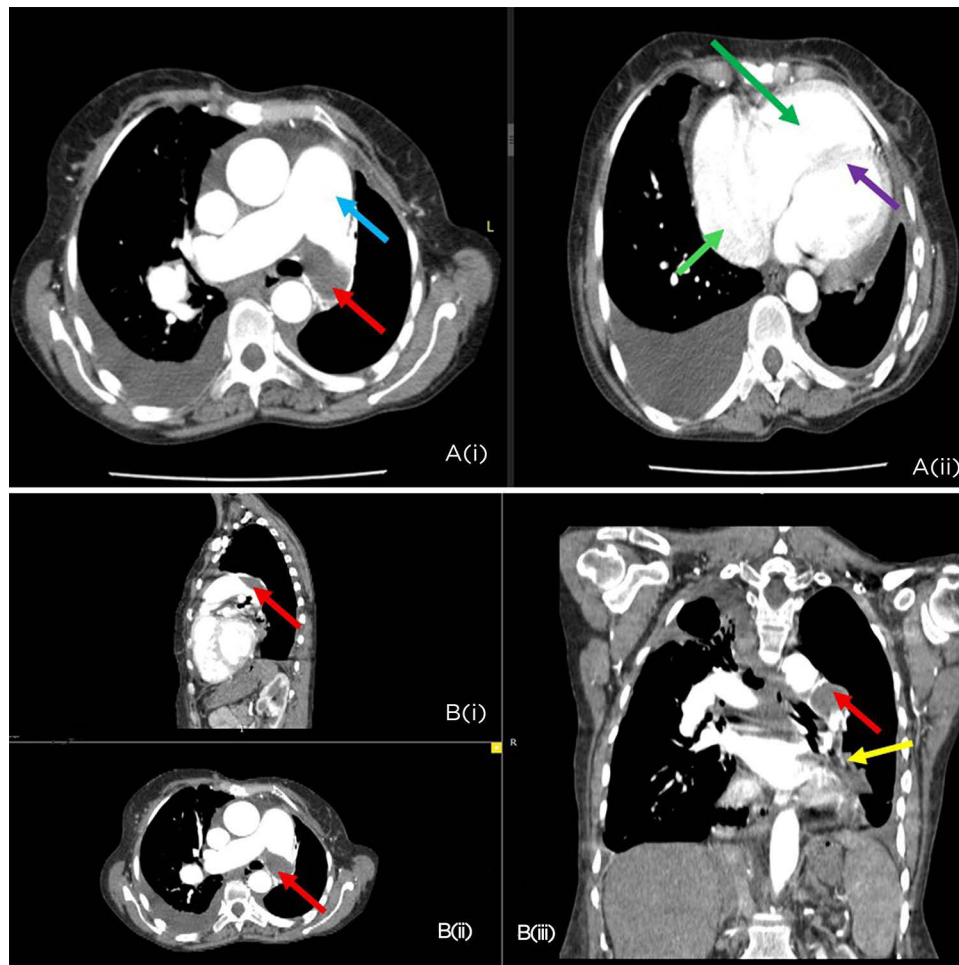


**Figure 2** (A) PSA view shows dilated main pulmonary artery with echogenic thrombus filling the left main pulmonary artery (red arrows). (B) Apical 4 chamber view shows dilated right atrium and right ventricle. Parasternal short-axis echocardiogram view showing septal flattening (red arrows) creating D-shaped LV configuration indicating RV pressure overload.

## Discussion

The presentation of silent hypoxia in our patient—profound arterial desaturation (80%) without commensurate tachypnea (16–18 breaths/minute)—is an incongruous phenomenon increasingly recognized in vascular lung pathologies. Identifying this discrepancy between her subjective dyspnea and her relatively normal objective respiratory effort was the first indication that the pathology was vascular rather than parenchymal.<sup>8,10</sup> Unlike the happy hypoxia often described in asymptomatic COVID-19 cohorts, our patient was symptomatic; however, her lung compliance likely remained high enough to prevent an increased work of breathing despite the severe ventilation-perfusion (V/Q) mismatch caused by the massive vascular obstruction.<sup>8,11</sup> Furthermore, in the early stages of a massive PE, the absence of significant hypercapnia prevents the activation of the central respiratory drive, resulting in the discrepant objective findings observed here.<sup>8,12</sup>

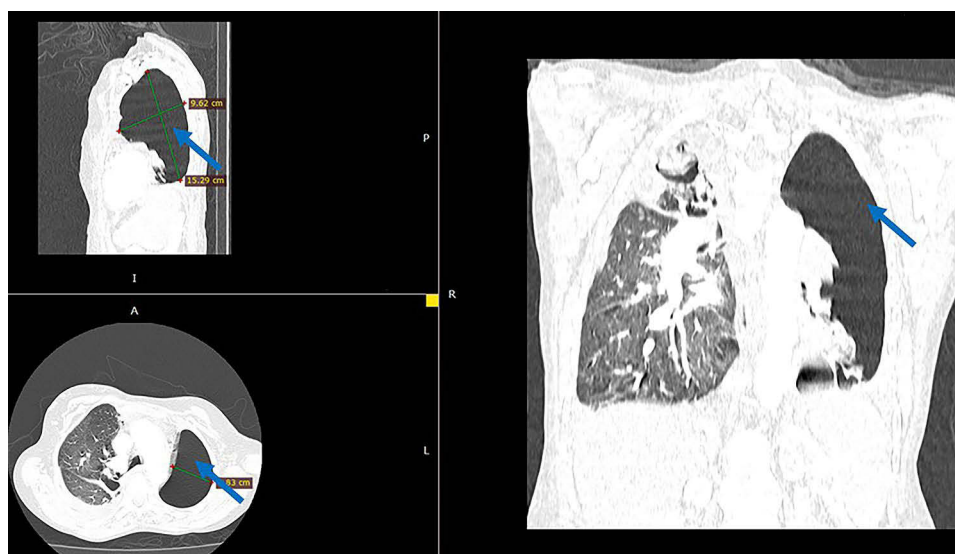
This case is primarily important because it illustrates the need for clinicians to rigorously navigate such incongruent findings, particularly to overcome diagnostic distraction. This phenomenon, where a prominent clinical or radiographic finding (such as a giant bulla) captures the clinician's attention and potentially delays the identification of underlying life-threatening pathologies, is a known challenge in complex cases within the Somali region.<sup>13</sup> Standard diagnostic pathways like the Wells Score<sup>1</sup> and the Pulmonary Embolism Rule-out Criteria (PERC)<sup>2</sup> were instrumental but required careful interpretation. The patient failed the PERC criteria due to her profound hypoxemia; however, her Wells Score was only moderately suggestive because she lacked tachycardia (HR 88 bpm)—a common clinical pitfall where stable vital signs lead to an underestimation of PE risk.<sup>1</sup> Identifying the discrepancy between her preserved pre-morbid exercise tolerance (climbing stairs and walking 500 meters) and this sudden, unexplained hypoxia was the critical factor in prioritizing PE. While giant bullae can cause chronic hypoxia, her pre-morbid history and the normalization of her SpO<sub>2</sub> to 94% post-treatment confirm that the initial 80% saturation was an acute vascular event.<sup>3</sup>



**Figure 3** (A) (axial Chest CT) shows massive left main pulmonary artery lobar and segmental hypodense filling defect dilating the vessels (Red arrows in **A(i)** and **B(iii)**, yellow in **B(iii)**). The enlarged main pulmonary artery measures 3.76cm larger than the adjacent aorta (blue arrow in **A(i)**). Dilation of both right atrium and right ventricle (green arrows in **A(ii)**) with bulge of the interventricular septum with RVD:LVD ratio >1 (Violet arrow in **B(iii)**). (B) (sagittal, axial and coronal image) showing left main pulmonary artery filling defect (red arrows **B(i-iii)**) with extension into lobar and segmental branches segmental arteries (yellow arrow **B(iii)**).

The diagnostic logic followed a stepwise approach to rule out other causes of acute dyspnea. Acute left-sided heart failure was considered unlikely due to the absence of orthopnea or crackles, and the echocardiogram showed acute right ventricular (RV) pressure overload rather than chronic left-heart dysfunction.<sup>14</sup> Pneumonia was ruled out as the patient was afebrile, and the subsequent sputum GeneXpert results were negative, identifying the cystic bronchiectasis as inactive post-tuberculous sequelae. Malignancy was less likely given the lack of constitutional symptoms such as weight loss. The patient's specific risk factors of a 30-pack-year history of second-hand smoke exposure from her husband, a BMI of 27.4 kg/m<sup>2</sup>, and four weeks of sedentary behavior due to malaise—provided the classic pro-thrombotic environment for unprovoked PE.<sup>3,9</sup>

The second critical importance of this case lies in the radiographic trap presented by GBD. While GBD does not affect the definitive diagnosis of PE via CTPA, its presence on an initial screening chest X-ray creates a high risk for the misdiagnosis of tension pneumothorax.<sup>5</sup> Classic radiographic markers of PE, such as the Westermark sign or Palla's sign, were obscured by the 15-cm bulla (occupying >33% of the hemithorax).<sup>4</sup> The structural distortion and vascular destruction hide the subtle clues of vascular obstruction, a common barrier when diagnosing PE in patients with established chronic lung disease.<sup>15</sup> In many emergency settings, such hyperlucency leads to the immediate, and sometimes blind, insertion of a chest tube. If a tube had been inserted into this patient's giant bulla, the resulting iatrogenic injury would have been catastrophic.<sup>5</sup> Here, clinical percussion remained the most reliable low-tech method to prevent iatrogenic harm.



**Figure 4** Large hyperlucent zone with thin imperceptible wall filling most of the left lung measuring 15cm × 6.0cm × 10.2cm suggesting Giant bullae (blue arrows).

Ultimately, this case offers a framework for managing high-risk patients when advanced tools are unavailable off-site. Establishing a diagnosis of venous thromboembolism (VTE) in the Somali context requires overcoming significant system challenges, as previously observed in other rare thrombotic presentations in the region.<sup>16</sup> Regarding the concerns about resource availability, we emphasize that a CTPA scanner was not available at the presenting hospital. In resource-limited settings, the incidental discovery of critical conditions during the management of an acute crisis is often the only pathway to definitive care.<sup>17</sup> By using bedside echocardiography as the diagnostic gatekeeper to identify indirect signs of vascular obstruction—specifically RV pressure overload and a visible thrombus—we were able to provide the necessary objective evidence to mandate an urgent and hazardous referral for a definitive CTPA.<sup>12,14</sup>

## Conclusion

This case underscores the importance of a comprehensive diagnostic approach that prioritizes navigating incongruous clinical findings to resolve complex cardiopulmonary pathology. The diagnostic process followed a rigorous stepwise logic: identifying the discrepancy between profound hypoxemia and normal respiratory effort (silent hypoxia), utilizing physical percussion to differentiate giant bullae from tension pneumothorax, and employing bedside echocardiography as a pivotal diagnostic bridge. The primary implication of this report is that in resource-limited settings where advanced imaging is not readily available, bedside ultrasound must be used to justify the risks and costs associated with off-site CTPA. Furthermore, recognizing silent hypoxia and discrepant objective signs can alert the clinician to vascular obstruction even when classic radiographic markers are obscured by pre-existing disease, ensuring timely management and preventing catastrophic iatrogenic errors.

## Data Sharing Statement

Data supporting the conclusions of this report are contained within the report. Additional non-relevant patient data are protected under patient privacy regulations and policies.

## Ethical Considerations

The case details were reviewed and approved for publication by the Somaliland Medical Association Institutional Review Board (SMA-IRB). Institutional approval for publication of the case details was required prior to dissemination of the anonymized information. Written informed consent was obtained from the patient before publication.

## Informed Consent

Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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## Disclosure

The authors report no conflicts of interest in this work.

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