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Giant Pulmonary Bullae Complicated by Hospital-Acquired Pneumonia in Chronic Obstructive Pulmonary Disease: A Multidisciplinary Approach to Successful Bullectomy

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A B S T R A C T

Giant pulmonary bullae (GPB), particularly in patients with chronic obstructive pulmonary disease (COPD), present significant management challenges due to compromised lung function. The development of hospital-acquired pneumonia (HAP) further complicates the clinical picture, increasing morbidity and mortality. This report details a case of GPB with HAP in a COPD patient. A 65-year-old male, a heavy former smoker with moderate COPD and a history of dust and wood smoke exposure, presented with progressive dyspnea and productive cough. Investigations revealed right-sided giant bullae occupying over 30% of the hemithorax, consolidation, and leukocytosis. Sputum culture grew Candida albicans, and intraoperative cultures later confirmed *Streptococcus viridans* HAP. Following initial medical stabilization with antibiotics, bronchodilators, and corticosteroids, he underwent an exploratory thoracotomy with bullectomy on day 14. In conclusion, the surgical intervention was successful, with no major complications. Post-operatively, the patient showed significant improvement in respiratory symptoms and lung expansion. This case highlights the efficacy of a multidisciplinary approach, combining intensive medical therapy with timely surgical bullectomy, for managing complex presentations of GPB, HAP, and COPD, leading to favorable outcomes.

1. Introduction

Giant pulmonary bullae (GPB), defined as air-filled spaces within the lung parenchyma greater than 1 cm in diameter, which in their "giant" form occupy at least 30% of a hemithorax, represent a severe manifestation of lung destruction. These are most commonly encountered in individuals with a significant smoking history and underlying chronic obstructive pulmonary disease (COPD), particularly the emphysematous type. The pathophysiology of bullae formation is rooted in the progressive destruction of alveolar walls, a process mediated by an imbalance between proteases (such as neutrophil elastase and matrix metalloproteinases) and anti-proteases within the lung microenvironment, often triggered and perpetuated by chronic inflammation from cigarette smoke and other inhaled irritants. This destruction leads to a loss of elastic recoil, progressive air trapping, and the formation of these large, non-functional air spaces. While the exact prevalence of GPB is difficult to ascertain, it is a recognized complication of advanced COPD, contributing significantly to patient morbidity.[1,2]

Clinically, patients with GPB may initially be asymptomatic or present with progressively worsening dyspnea due to the compression of adjacent, healthier lung tissue, and the creation of physiological dead

space, leading to profound ventilation-perfusion (V/Q) mismatch and hypoxemia. Complications of GPB are not uncommon and include spontaneous pneumothorax due to bulla rupture, infection of the bulla (leading to an empyema-like picture or abscess formation), and hemoptysis. The presence of GPB in a patient with COPD inherently signifies advanced disease and often correlates with poorer lung function, reduced exercise tolerance, and diminished quality of life.[3,4]

COPD itself is a major global health problem, characterized by persistent respiratory symptoms and airflow limitation due to airway and/or alveolar abnormalities, usually caused by significant exposure to noxious particles1 or gases. Exacerbations of COPD, often triggered by respiratory infections, are key events in the natural history of the disease, leading to acute worsening of symptoms, further decline in lung function, hospitalizations, and increased mortality. Hospital-acquired pneumonia (HAP), defined as pneumonia developing 48 hours or more after hospital admission that was not incubating at the time of admission, poses a serious threat, especially to hospitalized patients with underlying respiratory conditions like COPD. These patients often have impaired host defenses, including compromised mucociliary clearance and cough reflex, making them susceptible to aspiration of oropharyngeal or gastric contents colonized with pathogenic microorganisms. The presence of structural abnormalities like bullae can further increase the risk of HAP by causing stasis of secretions and obstructing airways. Common pathogens in HAP can include a range of Gram-negative bacilli and Gram-positive cocci, and the development of HAP in a COPD patient invariably worsens their clinical status, prolongs hospital stay, and significantly increases the risk of mortality.[5,6]

The concurrence of GPB, active HAP, and an acute exacerbation of moderate COPD, as presented in this case, constitutes a highly complex clinical scenario. Each condition individually contributes to respiratory compromise, and their synergistic effects can lead to rapid deterioration. Managing such patients requires a nuanced understanding of the pathophysiology and a carefully orchestrated therapeutic strategy. Treatment options for GPB range from conservative management for asymptomatic or minimally symptomatic patients to surgical intervention, typically bullectomy, for those with significant symptoms or complications. Bullectomy aims to remove the non-functional bullae, thereby allowing re-expansion of compressed lung tissue, improving respiratory mechanics, reducing dead space, and alleviating dyspnea. Surgical approaches include traditional thoracotomy and minimally invasive video-assisted thoracic surgery (VATS), with the choice often depending on the size, location, and number of bullae, as well as patient factors and surgeon expertise. However, undertaking major thoracic surgery in a patient with active HAP and acutely exacerbated COPD carries substantial risks.[7,8]

Therefore, a comprehensive, multidisciplinary approach involving pulmonologists, thoracic surgeons, intensivists, radiologists, and respiratory therapists is paramount for optimizing outcomes in these critically ill patients. This involves aggressive medical management to stabilize the HAP and COPD exacerbation, careful pre-operative assessment and optimization, meticulous surgical technique, and vigilant post-operative care.[9,10] This case report aims to illuminate a successful, integrated therapeutic pathway for managing the formidable challenge of concurrent giant pulmonary bullae, hospital-acquired pneumonia, and acute COPD exacerbation. By detailing the diagnostic intricacies, multidisciplinary decision-making, and phased medical-surgical interventions culminating in successful bullectomy, we seek to provide a practical framework and underscore the potential for favorable outcomes in similarly high-risk patients when aggressive, coordinated care is employed.

2. Case Presentation

Mr. S, a 65-year-old Indonesian male, was admitted to Dr. Moewardi Regional General Hospital with the primary complaint of progressively worsening

dyspnea over the preceding 14 days. He reported a history of intermittent dyspnea for the past 7 years, consistent with his later diagnosis of moderate COPD. His dyspnea had acutely worsened, making even minimal exertion difficult. Accompanying his shortness of breath was a productive cough for two weeks, yielding thick, green-colored sputum, without any hemoptysis. He also described a stabbing pain in the right side of his chest and an unintentional weight loss of approximately 5 kg over the previous two months. No high-grade fever was reported by the patient prior to admission (Table 1).

His past medical history was significant for a diagnosis of moderate COPD, for which he received intermittent treatment. He had a notable smoking history, having actively smoked 1.5 packs of cigarettes per day for 24 years, though he had reportedly quit 16 years prior to this presentation. His occupational history included work as a casual laborer and a brick craftsman, involving significant exposure to dust and wood smoke over many years. He also had a known cardiac history, for which he was on aspirin 80 mg, clopidogrel 75 mg, bisoprolol 2.5 mg, ramipril 2.5 mg, atorvastatin 40 mg, and spironolactone 25 mg daily, suggesting underlying ischemic heart disease and possibly heart failure.

Upon initial physical examination in the emergency department, Mr. S appeared moderately ill and in respiratory distress. His vital signs were: respiratory rate of 22 breaths per minute, and an oxygen saturation (SpO_2) of 91% on room air. Cardiovascular examination was largely unremarkable aside from tachycardia appropriate for his distress. Respiratory examination revealed diminished breath sounds on the right side, particularly at the base, with associated moist rales (crackles) and localized wheezing in the right lower lobe. The percussion note over the right lower hemithorax was hyperresonant.

Initial laboratory investigations on admission (Day 0) showed a leukocyte count of 10,000/µL. An arterial blood gas (ABG) analysis on room air revealed: pH 7.36, $PaCO_2$ 52 mmHg, PaO_2 62 mmHg, HCO_3^- 29 mEq/L, and SaO_2 91%. These ABG results were indicative of partially compensated respiratory acidosis with mild hypoxemia, consistent with an acute exacerbation of his known COPD. Other basic metabolic panel results were within normal limits. An initial chest X-ray (anteroposterior view) performed on admission demonstrated infiltrates in the right lung, suggestive of consolidation, and an area of hyperlucency with attenuated vascular markings in the right lower lobe, suspicious for bullous disease (Figure 1).

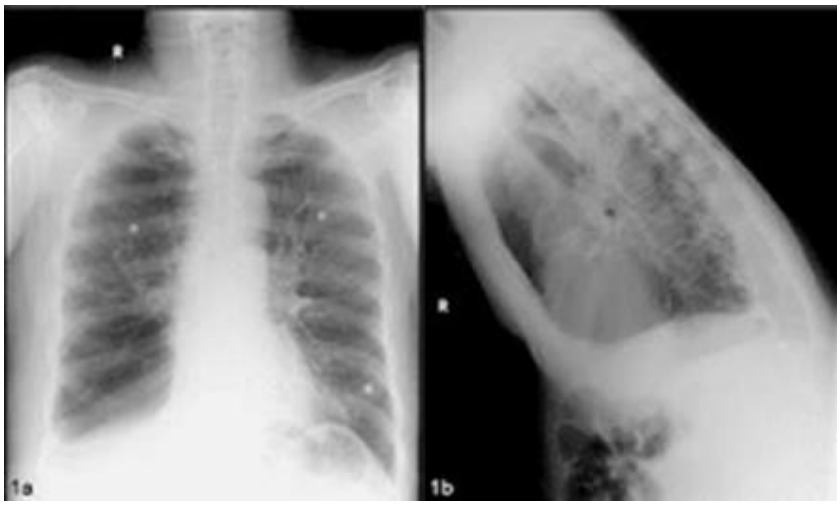

Figure 1. Preoperative (a,b) chest X-rays.

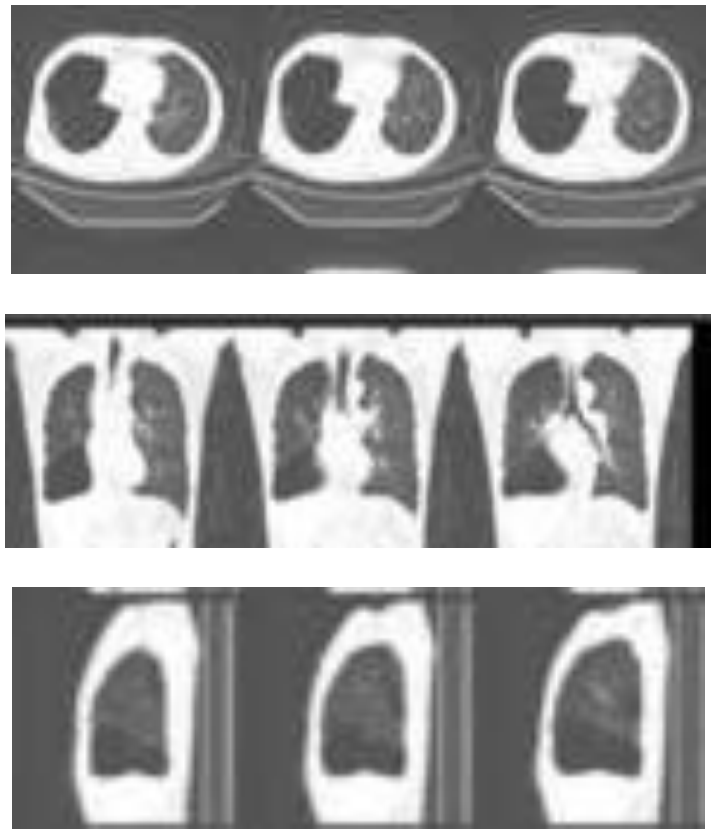

Figure 2. Preoperative thoracic chest CT-scan.

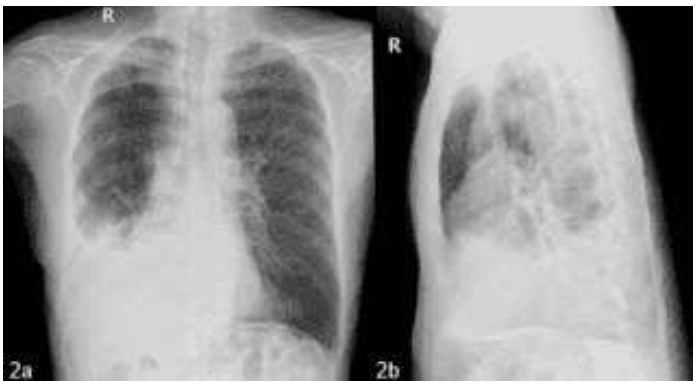

Figure 3. Postoperative (a,b) chest X-rays.

The patient was admitted to the general ward with a diagnosis of acute exacerbation of COPD and suspected community-acquired pneumonia. He was started on supplemental oxygen via nasal cannula at 3 L/min, nebulized ipratropium bromide 0.5 mg combined with salbutamol 2.5 mg every 6 hours, inhaled corticosteroids (budesonide/formoterol), intravenous 0.9% NaCl infusion for hydration, a high-calorie diet (1500 kcal/day), oral levofloxacin 750 mg daily, intravenous methylprednisolone 62.5 mg daily, and N-acetylcysteine 200 mg three times daily orally. His cardiac medications were continued as prescribed. Despite initial treatment, the patient's respiratory status showed only modest improvement over the first

few days. By hospital day 5, his leukocyte count had risen significantly to 17,000/μL, with a neutrophil predominance, raising concerns for worsening or inadequately treated infection, or the development of a new, hospital-acquired infection. His dyspnea persisted, and he continued to produce purulent green sputum.

Given the timeframe (beyond 48 hours of hospitalization) and the new inflammatory surge, a diagnosis of hospital-acquired pneumonia (HAP) was strongly considered. Sputum samples were collected for further microbiological analysis. GenXpert MTB assay for Mycobacterium tuberculosis was negative. Fungal culture of the sputum isolated Candida albicans, which was reported to be sensitive to fluconazole. This was considered likely colonization in the context of his COPD and recent antibiotic/corticosteroid use rather than primary fungal pneumonia. To better delineate the underlying lung pathology and assess for complications, a non-contrast thoracic computed tomography (CT) scan was performed (Figure 2). The CT scan confirmed the presence of giant bullae in the right lower lobe, with the largest bulla measuring approximately 16 cm in maximal diameter and occupying an estimated 40-50% of the right hemithorax. These bullae caused significant compression of the adjacent normal lung parenchyma in the right middle and lower lobes, with some atelectatic changes noted. There was no evidence of a tumor mass. Patchy areas of consolidation consistent with pneumonia were also visualized in the peribullous regions of the right lung.

To further investigate the airway and obtain better microbiological samples, a flexible bronchoscopy was performed on hospital day 8. The bronchoscopy revealed evidence of external compression leading to stenosis of the B2 segment of the right upper lobe. Abundant purulent hypersecretion was observed throughout the bilateral tracheobronchial tree, more pronounced on the right side. Bronchial washings were collected for cytology and microbiology. Cytology was negative for malignant cells. Cultures from bronchial washings were pending at the time of surgical decision-making but later, intraoperative tissue cultures would grow *Streptococcus viridans*. Based on the clinical progression, laboratory findings (leukocytosis), imaging (CT findings of bullae and consolidation), and bronchoscopic findings (purulent secretions, compression), the patient was diagnosed with hospital-acquired pneumonia, moderate COPD with an acute exacerbation and right-sided giant pulmonary bullae.

A multidisciplinary team meeting was convened, involving the primary admitting physician, pulmonologists, thoracic and cardiovascular surgeons, intensivists, and radiologists. The consensus was that while medical management for HAP and COPD exacerbation was crucial and ongoing, the underlying giant bullae were a significant contributing factor to his current presentation and posed a high risk for recurrent infections, prolonged respiratory compromise, and potential further complications like pneumothorax. The compression of functional lung tissue by the bullae was deemed a major impediment to his respiratory recovery. Given his persistent dyspnea despite medical therapy and the large size of the bullae, surgical intervention (bullectomy) was considered the most definitive treatment option to improve his long-term respiratory status. The patient and his family were counseled regarding the risks and benefits of surgery, especially in the setting of active HAP and COPD, and they provided informed consent.

Intensive medical therapy was continued to optimize the patient's condition prior to surgery. This included a full course of intravenous antibiotics based on expected HAP pathogens, ongoing bronchodilator therapy, systemic corticosteroids, mucolytics, aggressive chest physiotherapy, and nutritional support. His cardiac status was carefully monitored and medications adjusted as needed by the cardiology team. Efforts were made to ensure adequate oxygenation, manage secretions, and improve his overall strength.

On 14 days after admission, the patient underwent an exploratory right thoracotomy and bullectomy. The

surgery was performed under general anesthesia with single-lung ventilation using a double-lumen endotracheal tube. A standard posterolateral thoracotomy incision was made, and the pleural space was entered through the 5th intercostal space. Upon entry, multiple large, thin-walled bullae were immediately apparent, predominantly involving the right lower lobe. The bullae were described intraoperatively as resembling "giant verrucae." There were some adhesions between the bullous lung and the chest wall, which were carefully lysed. The surgical team performed a limited resection of the right lower lobe, encompassing the giant bullae (bullectomy). This was achieved using surgical stapling devices, ensuring an airtight seal along the resection margins. Bovine pericardial strips were used to reinforce the staple lines to minimize the risk of post-operative air leak. The resected bullous tissue was sent for histopathological examination. After resection, the remaining right lung parenchyma, particularly the right upper and middle lobes, was observed to re-expand well to fill the hemithorax. The pleural cavity was irrigated, and hemostasis was confirmed. Two chest tubes (one apical and one basal) were placed for drainage and to monitor for air leaks. The estimated blood loss during the procedure was approximately 200 cc. The surgery proceeded without any immediate complications.

Post-operatively, Mr. S was transferred to the Intensive Care Unit (ICU) for close monitoring and management for three days. He was extubated successfully in the ICU within 12 hours of surgery. His pain was managed with an epidural catheter initially, followed by patient-controlled analgesia and then oral analgesics. Chest physiotherapy was initiated early to promote lung expansion and clearance of secretions. The chest tubes were monitored for air leak and drainage; no prolonged air leaks were detected. Histopathology of the resected bullae confirmed the presence of emphysematous lung tissue with chronic inflammation and fibrosis; there was no evidence of malignancy. A follow-up chest X-ray on post-operative day 1 showed good expansion of the right lung with no residual air trapping or significant pneumothorax (Figure 3). After three days in the ICU, he was stable enough to be transferred to the thoracic surgery high care unit, and subsequently to the general ward. His dyspnea markedly improved compared to his pre-operative state, and his chest pain also significantly decreased. He was weaned off supplemental oxygen gradually. By post-operative day 10, Mr. S was ambulating independently, eating a regular diet, and maintaining normal oxygen saturation on room air. The chest tubes were removed on post-operative days 4 and 6, respectively. His leukocyte count normalized, and other inflammatory markers improved.

Pulmonary function tests (spirometry) were performed prior to discharge; Pre-operative (FEV1 1.1 L (42%), FVC 2.6 L (65%), FEV1/FVC ratio 42%. Post-operative (Day 12): FEV1 1.6 L (60%), FVC 3.0 L (75%), FEV1/FVC ratio 53%. This represented a significant improvement in his airflow parameters. The patient was discharged home on post-operative day 14 in a stable and improved condition. He was scheduled for follow-up in the thoracic surgery and pulmonology outpatient clinics and referred for a pulmonary rehabilitation program. At a 1-month follow-up visit, he reported sustained improvement in dyspnea, increased exercise tolerance, and an overall better quality of life. He remained oxygen-independent.

3. Discussion

This case report describes the complex and ultimately successful management of a 65-year-old male patient who presented with a challenging triad: right-sided giant pulmonary bullae (GPB), hospital-acquired pneumonia (HAP), and an acute exacerbation of moderate COPD. The favorable outcome achieved underscores the critical importance of a timely, well-coordinated multidisciplinary approach, integrating aggressive medical therapy with definitive surgical intervention. The patient's long history of heavy smoking and occupational exposure to dust and wood smoke are well-established primary risk factors for the development of both COPD and emphysematous bullae.[11,12]

Table 1. Summary of patient's clinical findings, management, and diagnosis.

Category	Details
Demographics	• **Age:** 65 years old • **Gender:** Male • **Occupation:** Casual laborer and brick craftsman (exposed to dust and wood smoke) • **Relevant Social History:** Heavy former smoker (1.5 packs/day for 24 years, ceased 16 years ago)
Anamnesis (History)	**Chief Complaint:** Dyspnea **History of Present Illness:** • **Dyspnea:**Intermittent for 7 years, worsened over 14 days prior to admission; progressive • **Cough:**Productive for 2 weeks with thick green sputum, no hemoptysis • **Chest Pain:** Right-sided stabbing pain • **Systemic:** 5 kg weight loss over 2 months; no high-grade fever reported **Past Medical History:** • **COPD:** Moderate• **Cardiac History:** Known (on aspirin 80mg, clopidogrel 75mg, bisoprolol 2.5mg, ramipril 2.5mg, atorvastatin 40mg, spironolactone 25mg)
Physical examination (On Admission)	**General:** • Moderate general weakness; **Vital Signs:** • **Respiratory Rate:** 22 breaths/minute • **SpO_2**: 91% on room air **Respiratory System:** • **Percussion:** Hyperresonance in the right lower lobe area • **Auscultation:** Moist rales in the right lung; localized wheezing in the right lower lobe
Laboratory findings	**Hematology:** • **Leukocyte Count (Day 0 - admission):** 10,000/uL • **Leukocyte Count (Day 5):** 17,000/uL (leukocytosis) **Microbiology:** • **Sputum (GenXpert MTB):** Negative • **Sputum (Fungal Culture):** *Candida albicans* isolated (fluconazole sensitive) • **Intraoperative Culture:** *Streptococcus viridans* positive (confirmed HAP diagnosis) **Histopathology (Resected Bullae):** • Chronic inflammation, no malignancy
Imaging findings	**Chest X-Ray (Initial):** Infiltrates in the right lung/consolidation infiltrates, hyperresonance in the right lower lobe area. **Non-Contrast CT Thorax:** Giant bullae in the right lower lobe, no evidence of tumor mass. **Bronchoscopy:** Compression stenosis of segment B2 in the right upper lobe • Purulent hypersecretion in the bilateral tracheobronchial tree **Chest X-Ray (Post-operative):** • Good expansion of the right lung • No residual air trapping
Clinical diagnosis (Final)	• **Hospital-acquired pneumonia** (intraoperative culture positive for *Streptococcus viridans*) • **Moderate COPD exacerbation improving** • **Right giant bullae**

Table 2. Procedure of treatment and follow-up.

Phase / Aspect	Details
Pre-operative medical management & HAP treatment	**Goal:** Stabilize HAP, optimize COPD, improve overall condition for surgery. **Oxygen Therapy:** Nasal cannula oxygen at 3 L/min. **Pharmacotherapy:** • **Bronchodilators:**Nebulized combination of ipratropium bromide 0.5 mg and salbutamol 2.5 mg every 6 hours; Inhaled corticosteroids budesonide/formoterol. • **Systemic Corticosteroids:** Intravenous methylprednisolone (62.5 mg/day). • **Antibiotics (HAP):** Oral levofloxacin 750 mg daily. • **Mucolytics:** N-acetylcysteine 3x200 mg orally. • **Cardiovascular Medications:** Aspirin 80 mg, clopidogrel 75 mg, bisoprolol 2.5 mg, ramipril 2.5 mg, atorvastatin 40 mg, spironolactone 25 mg administered according to cardiac history. **Nutritional Support:** High-calorie diet (1500 kcal); Intravenous 0.9% NaCl infusion. **Monitoring:** • Clinical: Dyspnea and chest pain monitored. Productive cough with thick green sputum monitored. • Laboratory: Leukocyte count (initial 10,000/µL on admission day, rising to 17,000/µL by day 5). • Physiological: SpO_2(91% on room air initially).
Surgical intervention (Bullectomy)	**Date:** On day 14. **Procedure:** Exploratory thoracotomy; Limited resection of the right lower lobe (bullectomy). **Anesthesia:** General anesthesia. **Intraoperative Findings:** • Multiple large bullae removed. • Intraoperative appearance resembling giant verrucae. **Surgical Technique:** • Multiple large bullae removed. • Tissue taken for histopathology. **Chest Drainage:** Chest tube placed. **Blood Loss:** ~200 cc. **Immediate Complications:** The procedure was uneventful.
Post-operative management	**Intensive Care Unit (ICU):** • **Duration:** Treated in the ICU for three days postoperatively. • **Transfer:** Transferred to non-critical intensive care (high care unit). **Ward Care & Recovery:** • **Chest Tube Management:** No prolonged air leaks were detected. • **Mobilization & Diet:** On postoperative day 10, the patient was eating and ambulating independently without oxygen. • **Symptom Improvement:** Dyspnea markedly improved; Chest pain decreased after surgery. • **Histopathology Results:** Confirmed chronic inflammation without malignancy. • **Imaging:** Postoperative chest X-ray demonstrated adequate right lung expansion without residual air trapping.
Discharge planning & follow-up	**Condition at Discharge:** • On further monitoring, the patient's condition remained stable with normal oxygen saturation without supplemental oxygen. **Follow-up:** • Postoperative pulmonary rehabilitation. • Regular spirometric monitoring. • Follow-up chest X-ray showed good expansion of the right lung. • Spirometry before and after surgery showed increased FEV1.

In COPD, chronic inflammation within the airways and lung parenchyma leads to progressive airflow limitation and structural lung damage. The specific emphysematous component involves the destruction of alveolar walls and the loss of pulmonary elastic recoil, leading to the formation of abnormally enlarged air spaces. When these spaces become exceptionally large, occupying more than 30% of the hemithorax, they are termed giant bullae. The pathophysiology of bullae formation involves an imbalance between proteolytic enzymes, such as neutrophil elastase and matrix metalloproteinases (MMP-9 and MMP-12), and their inhibitors (alpha-1 antitrypsin). Chronic exposure to irritants like cigarette smoke recruits inflammatory cells (neutrophils, macrophages, T lymphocytes) to the lungs, which release these enzymes, leading to the degradation of elastin and collagen, critical components of the alveolar matrix. This progressive loss of elastic tissue results in increased lung compliance, overinflation, and the coalescence of damaged alveoli into bullae.[13,14]

These giant bullae are largely non-functional; they do not participate effectively in gas exchange and act as a ventilation dead space. They compress adjacent, healthier lung tissue, further impairing ventilation and perfusion matching, which exacerbates hypoxemia and hypercapnia. The increased dead space and mechanical disadvantage imposed on the respiratory muscles significantly increase the work of breathing, leading to the progressive dyspnea experienced by this patient. Furthermore, structural changes in the small airways (terminal bronchioles) due to fibrosis and inflammation contribute to increased airway resistance, further decreasing FEV1 and vital capacity. The development of HAP in this patient significantly complicated his clinical course. Hospitalized COPD patients are inherently at higher risk for HAP due to factors such as impaired mucociliary clearance, frequent microaspirations, potential need for invasive devices, and alterations in oropharyngeal flora. The presence of giant bullae can further predispose to infection by causing stasis of secretions and obstructing bronchial drainage. In this case, the HAP was caused by *Streptococcus viridans*, identified from intraoperative cultures. While often considered normal oral flora, *Streptococcus viridans* group bacteria can become pathogenic, particularly in immunocompromised individuals or when aspirated into the lower respiratory tract, leading to pneumonia, and are sometimes associated with instrumentation or dental procedures, though no specific source was identified here beyond likely microaspiration. The inflammatory response triggered by HAP, with recruitment of neutrophils and release of pro-inflammatory cytokines (IL-1, IL-6, TNF-α), leads to alveolar exudate accumulation, further reducing oxygen diffusion and worsening hypoxemia, especially in a patient with already limited ventilatory reserve due to COPD and GPB. The initial finding of *Candida albicans* in the sputum was appropriately interpreted as colonization, a common finding in COPD patients on corticosteroids or broad-spectrum antibiotics, rather than invasive candidal pneumonia, which is rare in immunocompetent individuals.[15,16]

Diagnosing HAP in a patient with an acute COPD exacerbation and underlying GPB can be challenging, as symptoms like cough, sputum production, and dyspnea are common to all three conditions. The rise in leukocyte count to 17,000/μL by day 5 was a key indicator of a new or worsening infective process, prompting the classification as HAP. Chest radiography, while useful for initial assessment, often has limitations in clearly delineating bullae and superimposed infiltrates. The thoracic CT scan was invaluable in this case, confirming the extent of the giant bullae, their compressive effect on the adjacent lung, and the presence of associated consolidations. CT imaging is crucial for surgical planning in bullectomy candidates.[17]

Bronchoscopy served multiple purposes: it allowed direct visualization of the airways, confirmed purulent hypersecretion indicative of infection, identified compression stenosis of a segmental bronchus (B2 RUL) likely due to the bullae, and enabled collection of lower airway samples for microbiology. Although pre-operative bronchial washings did not immediately

yield the causative pathogen for HAP in this instance, intraoperative cultures did. This highlights the importance of obtaining deep tissue or fluid samples when HAP is suspected and surgery is performed.[18]

The successful outcome in this high-risk patient hinged on a robust multidisciplinary approach. The initial phase focused on stabilizing the patient's HAP and COPD exacerbation. This involved appropriate broad-spectrum empirical antibiotic therapy with levofloxacin, which has good activity against many common respiratory pathogens, including atypical organisms and many strains of *Streptococcus pneumoniae* and some Gram-negatives. Subsequent confirmation of S. viridans (typically sensitive to fluoroquinolones or beta-lactams) affirmed the appropriateness of this choice or would have guided de-escalation if a narrower spectrum agent was suitable. Systemic corticosteroids (methylprednisolone) were vital for managing the inflammatory component of the COPD exacerbation and, to some extent, the HAP-related inflammation, helping to reduce airway edema and bronchospasm. Aggressive bronchodilator therapy (nebulized salbutamol/ipratropium and inhaled budesonide/formoterol) aimed to maximize airway patency. Oxygen therapy corrected hypoxemia, and N-acetylcysteine potentially aided in mucus clearance. Nutritional support with a high-calorie diet was essential to counteract catabolism associated with chronic illness, infection, and increased work of breathing. The cardiology team's involvement ensured that his pre-existing cardiac conditions were optimally managed throughout this stressful period.[19]

Once the acute infection showed signs of control and the patient was deemed optimized from a medical standpoint, surgical intervention was pursued. The indications for bullectomy in this patient were clear: symptomatic giant bullae occupying a significant portion of the hemithorax (estimated >30-40%), causing compression of functional lung tissue, and contributing to severe dyspnea and likely recurrent infections. While VATS bullectomy is often preferred for its minimally invasive nature, leading to potentially less pain and faster recovery, thoracotomy (as performed here) remains a valid and sometimes necessary approach, especially for very large bullae, multiple bullae, or anticipated significant adhesions, or based on surgeon preference and experience. The goal of bullectomy is to resect the non-functional bullous lung, allowing the compressed, healthier lung to re-expand, thereby improving V/Q matching, reducing physiological dead space, and enhancing overall respiratory mechanics. The intraoperative finding of multiple large bullae resembling "giant verrucae" underscores the extensive nature of the disease. The use of stapling devices, potentially with buttressing material, is standard for ensuring an airtight closure and minimizing post-operative air leaks, which are a common complication after surgery on emphysematous lungs.

The decision to proceed with surgery in a patient with recent HAP and COPD requires careful risk-benefit analysis. Post-operative management in the ICU for three days allowed for intensive monitoring of respiratory and hemodynamic status, effective pain control (crucial for enabling deep breathing and coughing), and early institution of chest physiotherapy. The absence of prolonged air leak and other major surgical complications (empyema, bronchopleural fistula, major bleeding) in this case is a testament to meticulous surgical technique and good post-operative care. Histopathological confirmation of chronic inflammation without malignancy was reassuring.

Previous studies suggest that bullectomy in appropriately selected patients with GPB can lead to significant improvements in dyspnea, exercise capacity, and pulmonary function parameters such as FEV1 and FVC. Mortality rates for elective bullectomy are generally low, ranging from 0% to 6% in various series, but can be higher in patients with severe underlying lung disease or significant comorbidities. Postoperative complications, such as prolonged air leak (most common, >7 days), pneumonia, and arrhythmias, can occur in a proportion of patients. Another study reported postoperative pneumonia

occurring in approximately 14% of cases undergoing thoracoscopic giant bullaectomy. The successful outcome in our patient, with marked symptomatic improvement, good lung re-expansion on imaging, and an increase in FEV1, aligns with the positive results reported in the literature for well-selected cases. The fact that he did not develop further postoperative pneumonia or prolonged air leak despite his risk factors is noteworthy.

This case is particularly instructive because it involves the management of HAP concurrently with the decision-making and execution of bullectomy. Many surgical series would defer surgery until active infection is fully resolved. However, in some instances, the bulla itself may be a nidus for persistent infection, or the patient's respiratory compromise may be so severe that waiting is not feasible. The multidisciplinary team's decision to proceed after a period of intensive medical optimization proved to be correct in this instance.

While bullectomy for GPB in COPD is a well-established procedure, and management of HAP in COPD is a common clinical challenge, detailed reports of performing bullectomy in the acute setting of ongoing HAP are less frequent. Most guidelines would advocate for the complete resolution of pneumonia before elective surgery. However, if the bulla itself is perpetuating infection or if the compression is life-limiting, surgery may be considered with appropriate perioperative management. This case adds to the literature by demonstrating that with careful patient selection, intensive medical pre-optimization, skilled surgical intervention, and diligent postoperative care, a favorable outcome is achievable even in such high-risk scenarios.

As recommended, a comprehensive pulmonary rehabilitation program is essential post-discharge to consolidate and enhance the functional gains achieved from surgery, improving exercise tolerance, muscle strength, and health-related quality of life. Ongoing pharmacological therapy for COPD (bronchodilators, inhaled corticosteroids if indicated), smoking cessation reinforcement, and management of comorbidities are vital. Annual influenza vaccination and pneumococcal vaccination are strongly recommended to prevent future respiratory infections. Regular follow-up with spirometry and clinical assessment is necessary to monitor lung function and detect any late complications or disease progression. For hospitalized patients, especially those with COPD, adherence to hospital-wide HAP prevention bundles (head-of-bed elevation, oral hygiene, early mobilization, judicious use of sedation) is critical. This case also reinforces the need for early consideration of surgical consultation for patients with symptomatic giant bullae, as timely intervention can significantly alter the disease trajectory.

4. Conclusion

This case report details the successful management of a complex patient presenting with right-sided giant pulmonary bullae, complicated by an acute exacerbation of moderate COPD and concurrent hospital-acquired pneumonia. Giant bullae represent a severe manifestation of emphysematous lung disease, and their presence can significantly worsen respiratory function, particularly when compounded by acute infections like HAP. The cornerstone of success in this challenging scenario was a highly coordinated, patient-centered, multidisciplinary approach. This case underscores that even in high-risk patients with multiple, severe respiratory comorbidities, a combination of intensive medical therapy and judiciously timed surgical intervention can yield excellent outcomes. It emphasizes the critical role of collaboration between pulmonologists, thoracic surgeons, intensivists, and other allied healthcare professionals in navigating the complexities of such presentations. Further recommendations include robust postoperative pulmonary rehabilitation and consistent long-term follow-up to maintain and optimize lung health. This approach should be considered for similar patients to improve both survival and quality of life.

5. References

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