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ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE 43 YEARS AFTER LEFT PNEUMONECTOMY: A CASE REPORT

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ABSTRACT

Background: Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) after pneumonectomy is clinically hazardous because the remaining lung must meet the entire ventilatory demand. **Case Presentation:** A 55-year-old man, 43 years after left pneumonectomy, presented with one week of progressive dyspnoea, cough and increased sputum production. Oxygen saturation was 84% on room air. Examination showed expected left-sided post-pneumonectomy findings and diffuse wheeze over the right lung. Arterial blood gas analysis showed pH 7.32, $PaCO_2$ 50 mmHg, PaO_2 89 mmHg and HCO_3^- 26 mmol/L; the oxygen-delivery conditions at sampling were not documented. Chest radiography and computed tomography demonstrated complete left hemithoracic opacification/volume loss with ipsilateral mediastinal shift and compensatory right-lung hyperinflation, without a new right-sided infiltrate. The patient received controlled oxygen, nebulized bronchodilators, systemic corticosteroids, empirical antibiotics, cautious intravenous fluids and bilevel non-invasive ventilation. Respiratory distress improved by day 3, and he was subsequently discharged on optimized inhaled therapy with vaccination, smoking-cessation and pulmonary-rehabilitation advice. **Conclusion:** AECOPD in a patient with a single lung requires prompt assessment, controlled oxygen delivery, early ventilatory support when indicated, and careful avoidance of iatrogenic injury to the remaining lung.

INTRODUCTION

Pneumonectomy permanently reduces total lung volume and vascular bed, while the remaining lung undergoes compensatory hyperinflation and cardiorespiratory adaptation. Although many long-term survivors achieve useful functional capacity, their reserve for acute respiratory stress remains limited.[1,2]

AECOPD can cause acute worsening of airflow limitation, gas exchange and respiratory muscle load. Standard management includes short-acting bronchodilators, systemic corticosteroids, antibiotics when clinically indicated, controlled oxygen therapy and ventilatory support according to the severity of respiratory failure.[3,4] Evidence describing these principles in patients decades after pneumonectomy is sparse. This report highlights the practical challenges of treating AECOPD when only one lung is available.

Case Report

Patient Information and Presenting Symptoms

A 55-year-old man presented with progressive shortness of breath, cough and increased sputum production for one week. He had undergone left pneumonectomy 43 years earlier. The original operative records and definitive surgical indication were unavailable; the patient recalled an episode of haemoptysis around the time of surgery. Information on baseline spirometry, smoking exposure, previous exacerbation frequency and home oxygen use was not included in the source record.

Clinical Findings

At presentation, temperature was 98 °F, pulse 60 beats/min, blood pressure 130/70 mmHg, respiratory rate 22 breaths/min and oxygen saturation 84% on room air. A 36-cm healed left posterolateral thoracotomy scar was present. Left hemithoracic movement and expansion were markedly reduced, and the trachea was slightly deviated to the left. Percussion was dull over the left hemithorax and resonant over the right. Breath sounds were absent over most of the left hemithorax, with transmitted bronchial sounds superiorly; diffuse wheeze was audible over the right lung.

Diagnostic Assessment

Investigation	Result
Haemoglobin	11.4 g/dL
Total leukocyte count	4,300 cells/μL
CRP	60 mg/L
Procalcitonin	0.5 ng/mL
ABG	pH 7.32; $PaCO_2$ 50 mmHg; PaO_2 89 mmHg; HCO_3^- 26 mmol/L
2D echocardiography	LVEF approximately 52%; mild TR; trace MR; normal IVC

The arterial blood gas indicated acidemia with hypercapnia; the inspired oxygen concentration at the time of sampling was not documented. Chest radiography showed complete opacification and volume loss of the left hemithorax with ipsilateral mediastinal shift, compatible with previous pneumonectomy, and no new infiltrate in the right lung. Computed tomography confirmed non-visualization of the left lung, marked left hemithoracic volume loss, ipsilateral shift and mild compensatory hyperinflation of the right lung.

Figure 1: Anonymized Chest Radiograph Showing Complete Opacification of the Left Hemithorax with Ipsilateral Mediastinal Shift and Compensatory Expansion of the Right Lung.

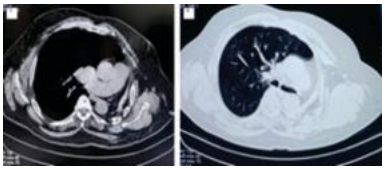

Figure 2: Axial Computed Tomography in Mediastinal (A) and Lung (B) Windows Demonstrating Post-left-

pneumonectomy Anatomy, Marked Left Hemithoracic Volume Loss and Compensatory Right-lung Hyper-inflation.

Therapeutic Intervention and Outcome

Controlled oxygen was titrated to a target saturation of 88-92%, with repeat clinical and blood-gas monitoring. This target is recommended for patients at risk of hypercapnic respiratory failure.[5] Nebulized short-acting bronchodilators, intravenous corticosteroids followed by oral corticosteroids, empirical antibiotics and chest physiotherapy were administered. Intravenous fluids were given cautiously. Because of respiratory acidosis and distress, bilevel non-invasive ventilation (BiPAP) was initiated; evidence-based guidelines and randomized trial data support NIV for acute hypercapnic respiratory failure during AECOPD.[6,7]

Respiratory distress and oxygenation improved progressively over three days. The patient was subsequently discharged once clinically stable on a long-acting β_2-agonist/long-acting muscarinic antagonist/inhaled corticosteroid combination, with advice regarding vaccination, smoking cessation, adherence, follow-up and pulmonary rehabilitation. Long-term inhaled therapy should be individualized to symptoms, exacerbation history and treatment response.[3,8] Pulmonary rehabilitation after hospitalization for a COPD exacerbation is strongly recommended when feasible.[9]

DISCUSSION

This case illustrates how a moderate physiological disturbance can become clinically important after pneumonectomy. Long-term adaptation includes hyper-inflation of the residual lung and changes in cardiopulmonary mechanics; however, loss of parenchyma and vascular bed leaves limited reserve when airflow obstruction, infection or respiratory muscle fatigue develops.[1,2]

Controlled oxygen delivery is essential. Unrestricted oxygen can aggravate hypercapnia in susceptible patients, whereas undertreatment of hypoxaemia is also dangerous. Titration to 88-92% with early blood-gas reassessment is a pragmatic approach until the individual risk of carbon dioxide retention is clarified.[3,5] In this patient, the combination of acidemia, hypercapnia and respiratory distress justified early BiPAP, with close observation for treatment failure.[6,7]

Fluid management warrants particular caution because all pulmonary blood flow is directed through the remaining lung. Historical post-pneumonectomy series associated a more positive fluid balance with pulmonary oedema.[10] Although those data concern the perioperative period, the physiological principle supports avoiding unnecessary fluid loading during acute respiratory illness in a single-lung patient.

If invasive ventilation becomes necessary, clinicians should minimize volutrauma and barotrauma, use predicted-body-weight tidal volumes, monitor plateau and driving pressures, and individualize positive end-expiratory pressure. Thoracic-surgery guidance supports lung-protective ventilation principles during one-lung ventilation, although direct evidence in remote post-pneumonectomy AECOPD is limited.[11]

Important limitations are the absence of the original pneumonectomy records, baseline and post-recovery spirometry, smoking history, sputum microbiology, eosinophil count, exact oxygen conditions during arterial blood-gas sampling, detailed BiPAP settings and exact length of hospital stay. These gaps limit confirmation of COPD phenotype and assessment of the indication for long-term triple inhaled therapy. The report was organized with reference to CARE reporting principles where the available source information allowed.[12]

CONCLUSION

AECOPD after pneumonectomy is a high-risk presentation because deterioration in the only functioning lung can rapidly lead to respiratory failure. Prompt recognition, controlled oxygen therapy, standard exacerbation treatment, early NIV when indicated, cautious fluid administration and lung-protective escalation strategies can support a favorable outcome. Comprehensive documentation of baseline lung function, ventilatory settings and follow-up is essential for future reports.

Learning Points

- A patient with previous pneumonectomy has limited reserve despite long-term compensatory adaptation.
- Oxygen should be titrated to a defined target, with early repeat arterial blood-gas assessment when hypercapnic failure is possible.
- Early non-invasive ventilation is appropriate for AECOPD with respiratory acidosis when no contraindication is present.
- Avoid unnecessary intravenous fluid loading and protect the remaining lung from ventilator-induced injury.
- Document baseline spirometry, smoking exposure, oxygen conditions, NIV settings and follow-up outcomes to strengthen diagnostic certainty and case-report quality.

Patient Perspective

The patient reported marked improvement in breathlessness after treatment and stated that he was able to resume routine daily activities after discharge while adhering to inhaled medication and follow-up advice.

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