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A CASE REPORT: SEVERE CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) EXACERBATION DUE TO COMMUNITY-ACQUIRED PNEUMONIA (CAP) INFECTION IN SMOKERS

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Abstract: A Case Report: Severe COPD Exacerbation Due to Community Acquired Pneumonia (CAP) Infection in Smokers. *COPD, also know as Chronic Obstructive Pulmonary Disease, refers to a lung condition marked by persistent and progressive airflow blockage. This is usually the result of a chronic inflammatory reaction in the respiratory tract brought on by exposure to harmful gases or particles. Respiratory tract infections like Community-Acquired Pneumonia (CAP) frequently cause COPD exacerbations, while smoking is the main risk factor that contributes to the onset and worsening of COPD. The goal of this case report is to provide an understanding of the underlying mechanism of severe COPD cause by CAP infection in smokers and to diagnose and manage the condition effectively. A 72-year-old man complained of shortness of breath that he had been having for the last year, and that had worsened over the previous three days when he arrived at the emergency unit of Abdoel Moeloek Hospital in Lampung. In addition, he also had a fever and severe cough with yellow sputum. Following a comprehensive review of medical history, physical examination, and supporting examination, it was concluded that a CAP infection was linked to severe COPD exacerbation. The patient received antibiotic medication and hospitalization treatment for symptomatic therapy.*
Keywords *CAP, COPD Exacerbation, Smokers*

Abstrak: Laporan Kasus: Eksaserbasi PPOK Parah Akibat Infeksi Pneumonia yang Didapat dari Masyarakat (CAP) pada Perokok. PPOK atau Penyakit Paru Obstruktif Kronis, mengacu pada kondisi paru-paru yang ditunjukkan dengan penyumbatan aliran napas yang terus-menerus dan berkembang secara progresif. Hal ini umumnya terjadi akibat reaksi peradangan kronis pada saluran pernapasan yang disebabkan oleh paparan gas atau partikel berbahaya. Infeksi saluran pernapasan seperti *Community-Acquired Pneumonia* (CAP) sering menyebabkan eksaserbasi PPOK, sementara merokok adalah faktor risiko utama yang berkontribusi terhadap timbulnya dan memburuknya PPOK. Tujuan dari laporan kasus ini untuk memberikan wawasan tentang mekanisme mendasari PPOK berat akibat infeksi CAP pada perokok dan mendiagnosis serta mengelola kondisi tersebut secara efektif. Seorang pria berusia 72 tahun mengeluhkan sesak napas yang telah dialaminya selama satu tahun terakhir dan semakin memburuk dalam tiga hari terakhir ketika ia tiba di unit gawat darurat Rumah Sakit Abdoel Moeloek di Lampung. Selain itu, ia juga mengalami demam dan batuk parah dengan dahak berwarna kuning. Setelah dilakukan tinjauan menyeluruh terhadap riwayat medis, pemeriksaan fisik, dan pemeriksaan penunjang, diketahui mengalami eksaserbasi

PPOK berat yang berhubungan dengan infeksi CAP. Pasien diberi perawatan rawat inap untuk terapi simtomatik dan pengobatan dengan antibiotik.
Kata kunci : CAP, COPD Eksaserbasi, Perokok

INTRODUCTION

The lung disorder known as chronic obstructive pulmonary disease (COPD) is marked by a persistent and progressive blockage of airflow, usually brought on by an inflammatory reaction in the airways. COPD exacerbations are acute events where respiratory symptoms worsen significantly, often requiring additional medical intervention such as hospitalization. Respiratory infections, such Community-Acquired Pneumonia, are recognized as significant contributors to exacerbations of COPD (Wang and Sun, 2024). The occurrence of CAP tends to increase with age, smoking habits, and any history of additional medical disorders (Braeken et al., 2017).

Respiratory infections like CAP often lead to exacerbations in COPD patients due to impaired pulmonary defence mechanisms. The presence of new types of bacteria, such as *Streptococcus pneumoniae* and *Haemophilus influenza*, increases the risk of significant exacerbations in COPD patients. Severe acute and chronic respiratory failure can result from acute exacerbations of COPD, with persistent hypercapnia representing a significant risk for morbidity and mortality (Mustofa *et al*., 2023). Smoking greatly increases the risk of developing and worsening COP. Cigarette smoke causes lung tissue damage and triggers a chronic inflammatory response, resulting in airflow obstruction. COPD patients who start smoking at a young age have a worse prognosis, with lower lung function and higher exacerbation frequency compared to those who start smoking later in life. Patients who persist in smoking undergo a more accelerated decrease in pulmonary function and increased risk of mortality linked to COPD (Ko *et al*., 2015).

Smoking impairs pulmonary defence mechanisms, which increases a patient's vulnerability to infections like CAP. Consequently, such infections worsen the symptoms of COPD and increase the severity and frequency of exacerbations (Yang *et al.,*2022). It highlights how crucial it is for COPD patients to smoke cessation and aggressive infection management in COPD patients to lower the possibility of severe exacerbations and enhance patient outcomes (Woodhead *et al*., 2015; Restrepo *et al*., 2016).

CASE

On March 16, 2024, a male patient aged 72 years with a background as a construction worker for 30 years was brought to the emergency unit of Abdoel Moeloek Hospital with complaints of shortness of breath that had been intermittent over the past year, worsening three days before the hospital admission (DBA). The shortness of breath was accompanied by wheezing and worsened with activity, especially when walking more than 100 meters. The patient also experienced an intermittent productive cough for the past year, which had worsened over the past three days with yellow sputum that was difficult to expectorate. There was no hemoptysis or chest pain, but the patient had a fever for three days DBA. The patient had a history of smoking 12 cigarettes per day for 50 years (600 pack-years, heavy).

On physical examination, the patient appeared moderately ill with alert consciousness. The vital signs showed a 37.2°C temperature, 140/93 mmHg blood pressure, 96x/ minute pulse rate, 26x/ minute respiratory rate, and 50% oxygen saturation (SpO_2) in room air, which increased to 92% with 10 lpm oxygen via a non-rebreathing mask (NRM). The patient weighed 50 kg and measured 150 cm in height, with a Body Mass Index (BMI) of 20.83 kg/m². A neck examination revealed a jugular venous pressure (JVP) of 5+3 cm H_2O without lymphadenopathy. Chest examination showed no venous distension. Cardiac examination

revealed regular heart sounds I-II without murmurs or gallops. Pulmonary examination revealed symmetrical inspection, increased tactile fremitus bilaterally, resonant percussion notes on both hemi thoraces and vesicular breath sounds with prolonged expiration, accompanied by bilateral crackles and wheezing. Abdominal and extremity examinations were within normal limits (WNL) without edema, with muscle strength 5/5/5/5. The laboratory and radiological examinations of this patient revealed a decrease in Hb 9.8 g/dl and Ht 31%, an increase in leukocytes of 15.460 sel/µl, an increase in platelets of 471.000, and an increase in neutrophils of 88% (Table 1). It is indicate acute infection.

Table. 1. Laboratory Assessments

	The Findings of Clinical Labs	**Standard Range**
Complete Blood Counts:		
Hemoglobin (Hb)	**9,8***	13,2 -17,3
Hematocrit (Ht)	**31***	40-52
White Blood Cells (WBC)	**15.460***	3.800-10.600
Trombosit	**471.000***	150.000-440.000
Differential count:		
Lymphocytes	8	25-40
Monocytes	4	2-8
Neutrophils segment	**88***	50-70
Eosinophils	0	2-4
Basophils	0	3-5
K/Na/Cl	4,2/130/8,6	3,5-5,0/135-147/8,95-105
Ureum / Creatinine	51/1,06	18-55/0,6-1,2
Albumin	3,4	3.5-5.2
Current blood sugar	116	70-200
Bilirubin T/D/I	-	<0.1/<0.2/<1
SGOT/SGPT	27/14	0-35/0-35
HbsAg/HAV/HCV	-	Non-reactive
HIV	-	
***Rapid molecular test* using Xpert MTB/RIF**	Negative	

The radiographs were administered. The initial report showed an air space consolidation appearance, which is typically a lobar pattern in pneumococcal disease, is seen on the chest X-ray (Figure 1). The clinical diagnosis for the patient was severe COPD exacerbation with CAP infection, as determined by the medical history, physical examination, and supporting examination. The patient was hospitalized for symptomatic therapy and medical treatment. The patient was given 10 lpm oxygen via a nasal mask (NRM) and an infusion of 0.9% NaCl 500cc every 12 hours. Administration of nebulization with a combination of ipratropium bromide 0.52 mg and salbutamol 3.01 mg every 6 hours, as well as nebulization with budesonide 0.5 mg every 6 hours was also performed. The oral drugs prescribed were N-acetyl cysteine (NAC) at a dosage of 200 mg three times daily, azithromycin at a dosage of 500 mg every 24 hours, and paracetamol at a dosage of 500 mg every 8 hours. The patient was administered intravenous methylprednisolone at a dosage of 40 mg three times a day, along with ceftriaxone at a dosage of 2 g every 24 hours.

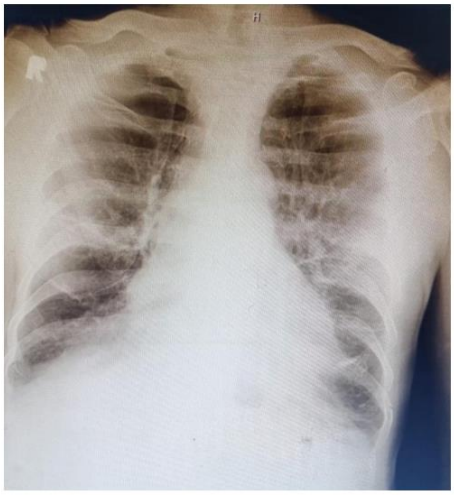

Figure 1. A radiological assessment on March 16, 2024, an air space consolidation appearance, which is typically a lobar pattern in pneumococcal disease, is seen on a chest radiograph.

DISCUSSION

Severe exacerbations in COPD are often triggered by respiratory infections, including community-acquired pneumonia (CAP). These infections make COPD patients' respiratory symptoms worse and raise their risk of morbidity and death (Restrepo et al., 2018). Studies show that bacteria such as Streptococcus pneumonia, *Haemophilus influenza*, and *Moraxella catarrhalis* are frequently involved in these exacerbations, while viral infections also contribute to about 30% of COPD exacerbation cases (Ouaalaya et al., 2020).

The patient showed typical symptoms of a COPD exacerbation, including increased breathlessness, wheezing, and a productive cough with difficulty in excreting sputum. The presence of fever indicates an active infection exacerbating the COPD condition. This combination of symptoms highlights the importance of early identification and appropriate management to prevent further complications (Han et al., 2019; Badr et al., 2017).

Community-acquired pneumonia (CAP) diagnosis is determined by the patient's medical history, physical examination, and other diagnostic tests (Ko et al., 2016). The diagnosis is certain community-acquired pneumonia is established if a chest x-ray shows new infiltrates or progressive infiltration plus two or further symptoms like changes in the characteristics of phlegm/purulence, the coughing increase, body temperature > 38^{o}C (axillary)/ history of fever, in the physical examination found leukocyte counts greater than 10,000, bronchial breath sounds, crackles and consolidation symptoms were discovered. Blood tests may provide data on the inflammatory condition, such as leukocyte and CRP cell counts and features, as well as indications of accompanying organ damage, such as acute renal failure. These tests can help determine the severity of illnesses in individuals whom physicians suspect of having community-acquired pneumonia. (Wunderink and Watever, 2014).

One of the primary risk factors for the development and progression of COPD is a prolonged time of smoking (Chung et al., 2023). Smoking causes lung tissue damage and triggers a chronic inflammatory response, resulting in airflow obstruction. In this patient, a history of smoking 12 cigarettes per day for 50 years has likely contributed significantly to the severe development of COPD and worsening symptoms. Physical examination reveals signs of COPD exacerbation with increased tactile

fremitus, crackles, and wheezing, along with abnormal auscultation findings. The significant drop in oxygen saturation indicates severe ventilation-perfusion impairment (Woodhead et al., 2015).

In terms of management, combining oxygen therapy, bronchodilators, and systemic corticosteroids is crucial to reduce symptoms and control inflammation. Antibiotics such as azithromycin and ceftriaxone are necessary to treat bacterial infection that may exacerbate the patient's condition. In managing CAP, the main principle is administering antibiotics as quickly as possible and evaluate the clinical condition within the first 72 hours (Mustofa et al., 2023). Using N-acetylcysteine (NAC) helps to reduce sputum viscosity, making it easier to expectorate and improving respiratory function (Ouaalaya et al., 2020). The mechanism of action of NAC occurs through interaction with oxygen radicals (ROS), increasing the body's antioxidant capacity. Clinically proven utilization of N-acetylcysteine (NAC) is useful for treating pulmonary diseases with a high dose of 1200 mg/day (Poty et al., 2024).

A comprehensive and timely strategy for treating COPD exacerbations, which includes addressing the underlying infection, is crucial for improving clinical outcomes and reducing further problems. Close observation and treatment modification in following the patient's reaction are essential to the efficiently controlling COPD exacerbations (Badr et al., 2017). Proficient management of exacerbations associated with COPD is critical in reducing potential complications, given that such exacerbations correlate with increased healthcare expenditure and decreased quality of life (Khan et al., 2023).

CONCLUSION

Community-acquired pneumonia (CAP) and other respiratory infections are common causes of severe exacerbations of COPD. The patient's long history of smoking significantly contributes to the severe development of COPD. Physical examination and diagnostic tests confirm the presence of serious ventilation-perfusion impairment. Comprehensive management with oxygen therapy, bronchodilators, corticosteroids, antibiotics, and N-acetyl cysteine (NAC) effectively reduces symptoms and improves respiratory function. Timely intervention is crucial to prevent further complications and improve clinical outcomes.

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