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Original Article

Acute Myocardial Infarction in Severe Acute Exacerbations of Chronic Obstructive Pulmonary Disease: Surviving Cases

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Abstract

Background: COPD is a disease signed by chronic respiratory symptoms with progressive-irreversible structural abnormalities and impaired lung function. Previously COPD was known as a condition that only affected the airways and lungs, but recent studies have revealed the incidence of cardiovascular disease in this population as prevalence increased.

Case: A 68-year-old male came to the emergency room fully conscious, complaining of shortness of breath since the afternoon. A physical examination detected tachypnea, desaturation of oxygen, and additional breath sounds in both lungs. Based on the blood gas analysis with the result of respiratory acidosis, supporting the diagnosis with AECOPD (acute exacerbations of chronic obstructive pulmonary disease), impending-type II respiratory failure, cor-pulmonale. During the treatment in the intensive unit, the physicians recognized deteriorating patients' conditions as unconsciousness, unstable vital signs, and changing the ST-T segment on ECG with an elevated cardiac marker. Other medications (antiplatelet, LMWH, statin) had given immediately. The patient's condition improved. On the ninth day of the treatments, the patients can discharge home.

Discussion: Acute exacerbations of COPD have a higher risk of developing ischemic heart disease with varying underlying mechanisms (atherosclerosis process and oxygen supply-demand imbalance). Understanding the numerous pathways that contribute to AMI (acute myocardial infarction) in COPD will help physicians to determine the therapy.

Conclusion: Based on this case, the ECG and cardiac enzymes warrant immediate evaluation, as must symptoms, vital signs, clinical findings, and other changes. Delays in case finding and treatment can worsen the prognosis.

Keywords: AECOPD, AMI, respiration, atherosclerosis, hypoxia

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INTRODUCTION

COPD is a disease signed by chronic respiratory symptoms with progressive and irreversible structural abnormalities and impaired lung function. Previously COPD was known as a condition that only affected the airways and lungs, but recent studies have revealed the incidence of cardiovascular disease in this population as prevalence increased.(1) Various processes might contribute to the association between COPD and ischemic heart disease. There are similarities in risk factors profile in the same degree of smoking habits and elderly age, as well as increased secretion of pro-inflammatory cytokines. Exacerbation inflammation is not restricted to the airways but composes a systemic inflammatory process. Platelet activation and enhanced thrombotic processes are all present in AECOPD. Those affect the formation of atherosclerosis.(1,2) Besides that process, ischemic heart disease in people with COPD is more common due to hypoxia. The incidence of acute myocardial infarction not

only exists in acute exacerbation of COPD patients but in moderate-grade stable COPD conditions and cardiovascular comorbidities.[3]

There are 30% of COPD deaths incidence due to cardiovascular problems. Because of unspecified signs, acute myocardial infarction (AMI) is rarely detected in acute exacerbations of COPD (AECOPD) patients, resulting in diagnosis delays and treatment.[1,3] The existence of a close relationship between COPD and the risk of acute myocardial infarction and death, therefore, requires appropriate identification, evaluation, and treatment.[1] Complementary 12-lead electrocardiogram, transthoracic echocardiography, pulmonary function test, laboratory parameters for inflammation and myocardial injury, and also coronary computed tomography angiography (CCTA) have been used to evaluate impaired cardiac function in COPD patients in the absence of specific cardiac disease.[1,4] Therefore, this case report aimed to give an example of rapid diagnosis and comprehensive treatment of AMI in AECOPD, showing a specific outcome to patients' condition, including saving the patients' life.

CASE

A 68-year-old male came to the emergency room fully conscious, complaining the shortness of breath since the afternoon. Patients frequently experience breathing difficulties, especially on long walks or strenuous activities. Breathing difficulties decrease with rest. The patient denied the other symptoms, which included cough, fever, chest pain, and partial weakness on one side of the body. There is nothing wrong with appetite and also complaining about urination or defecation. The patient previously got several medications such as salmeterol xinafoate 50 mcq combined with fluticasone propionate 250 mcq, and orally furosemide 20 mg, carvedilol 6.25 mg, and candesartan 8 mg as his medication history. However, the medicines are only sometimes obtained and consumed. On physical assessment in the emergency room, the consciousness level was E4V5M6, blood pressure 102/71 mmHg, heart rate 70 times per minute regularly, respiratory rate 28 times per minute, oxygen solubility in blood 89%, and temperature 36 degrees Celsius. A physical examination of the thorax detected additional breath sounds (rhonchi and wheezing) in both lungs. The other organ evaluation results were within normal.

The initial therapy was administered immediately, with ten drops per minute of normal saline and oxygen supplementation through a non-rebreathing mask of 10 liters per minute. Additionally, blood samples were collected for complete blood count examinations, blood chemistry evaluations, and blood gas analysis. Electrocardiography and chest X-rays were also done (Figure 1, Table 1). The complete blood test revealed an increase in absolute neutrophil count followed by an elevation of NLR, and decreased kidney function which was marked by a slight increase in blood urea nitrogen-serum creatinine levels. The blood gas study revealed a decline in pH, an elevation in pCO2, with an increase in HCO3.

Based on history, physical examination, and laboratory findings, the diagnosis came with an acute exacerbation of COPD, respiratory acidosis, impending type II respiratory failure, and cor-pulmonary. Oxygenation therapy via NRM eight liters per minute was continued, with normal-saline eight drops per minute as fluid resuscitation. Patients received hydrocortisone 100 mg intravenously every 12 hours, cetirizine

10 mg every 12 hours orally, salbutamol-ipratropium bromide-budesonide nebulization every 12 hours, and pursed lip breathing exercises every hour for 10 minutes from the lung department. Meanwhile, the patient received diuretic therapy with furosemide 20 mg intravenously every 8 hours and spironolactone 25 mg every 24 hours from the cardiology department. The patient was treated in the high-care unit (HCU) for close observation and further treatment.

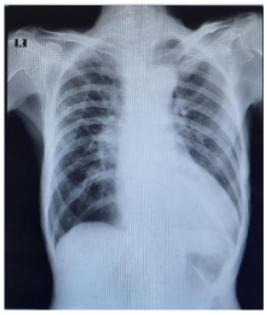


Figure 1. Bilateral infiltrates, with a CTR >50%. Costophrenicus sinuses and diaphragm within normal limits. Impression: bronchopneumonia, cardiomegaly.

After three days treated at the HCU, the patient experienced a wet cough and fever. Sputum is thick with yellowish white. Physical examination indicates the patient's condition is unstable, where blood pressure is 83/46 mmHg, heart rate is 137 beats per minute regular, respiratory rate is 16 times per minute, the solubility of oxygen in the blood is 98% with an NRM of 8 liters per minute and a temperature of 39.1 degrees Celsius. The patient went into shock with blood pressure below 90 MAP and did not respond to fluid resuscitation. Further investigations were made to find the cause of the decline in the patient's condition. There were ST segment changes on the EKG and increased cardiac enzymes. The elevated WBC from 8.840 to 11.680 indicated a bacterial infection from the respiratory system. The AGD results show an improvement.

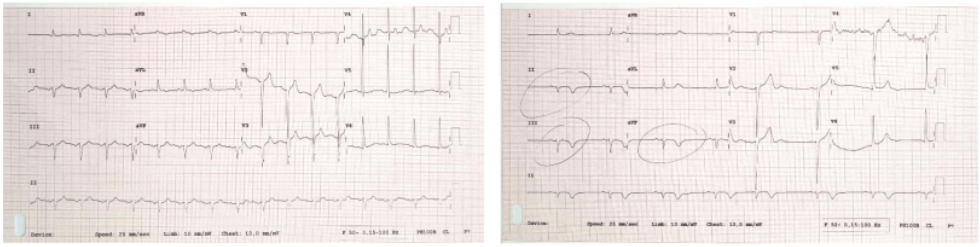
Vasopressor was given to a patient, starting from the lowest dose of 0.05 mcq/kgbb/minute until titrated to a target MAP of 65 mmHg. Fluids are maintained with an infusion pump of 50 cc/hour. The patient received antibiotic therapy with suspected pneumonia (HAP), septic or cardiogenic shock. Additionally, Moxifloxacin 400 mg every 24 hours and Cefoperazone 1 gram every 12 hours were administered intravenously, along with Paracetamol 1 gram every 8 hours as antipyretics. As well as the oral drug N-Acetyl Cysteine (NAC) 200 mg every 8 hours and salbutamol 2 mg every 12 hours. The shock condition continued to degrade the patient's condition on the fifth day of treatment. Decreased state with E2V4M5 uncooperative on examination. Evaluation of the EKG assessment found inferior ischemia and increased cardiac enzymes (Table 2.). An additional diagnosis of NSTEMI was established, with adjuvant therapy in the form of Clopidogrel 75 mg, Acetosal 80 mg, Atorvastatin 40 mg, and LMWH 0.4 cc every 24 hours.

The patient’s condition improved. Vital signs were stable, so the patient was transferred to the regular treatment room, and on the ninth day of treatment, the patient was given permission to return home.

Table 1. Blood chemistry and blood gas analysis

Indicator	Result			Reference interval
	18/02/2023	22/02/2023	25/02/2023	
Blood chemistry test				
Random blood sugar	201	(-)	(-)	70-140 mg/dL
SGOT	23	(-)	(-)	11-33 U/L
SGPT	12	(-)	(-)	11-50 U/L
Ureum	62^	(-)	76^	15-45 mg/dL
Creatinin	1.9^	(-)	1.4^	0.70-1.20 mg/dL
eGFR	35	(-)	51	mL/min/1.73 m 2
Na	141	(-)	(-)	136-145 mmol/L
K	4.5	(-)	(-)	3.5-5.1 mmol/L
Cl	108	(-)	(-)	94-110 mmol/L
Blood gas analysis				
Lactic acid	1.12	0.97	(-)	0.36-1.25 mmol/L
pH	7.262*	7.413	(-)	7.35-7.45
pCO2	79.7^	64.1 ^	(-)	35-45 mmHg
pO2	321^	176 ^	(-)	80-100 mmHg
BE ecf	9 ^	16 ^	(-)	(-2) – (+2) mmol/L
HCO3	35.9 ^	40.9 ^	(-)	23-26 mmol/L
CO2 total	38.0 ^	43 ^	(-)	24-30 mmol/L
SO2	100	100	(-)	95-99%

*Notes: ^ above normal; * below normal*



(a) (b)

Figure 2. Patients ECG, (a). Left ventricle hypertrophy with left axis deviation and old myocardial infarction on II, III, avF lead; (b). ST-T changes on inferior lead

Table 2. Lipid profile, cardiac marker, uric acid examination

Indicator	Result		Reference interval
	23/02/2023	24/02/2023	
Troponin - I	95.5^	90.2^	<19 ng/L
CKMB	16.6	28.1^	≤25 U/L
Total cholesterol	125	(-)	<200 mg/dL
HDL	22*	(-)	35-60 mg/dL
Trigliserida	103	(-)	<150 mg/dL
LDL	82	(-)	<130 mg/dL
Uric acid	4.2	(-)	2.0-7.0 mg/dL

Notes: ^ above normal; * below normal

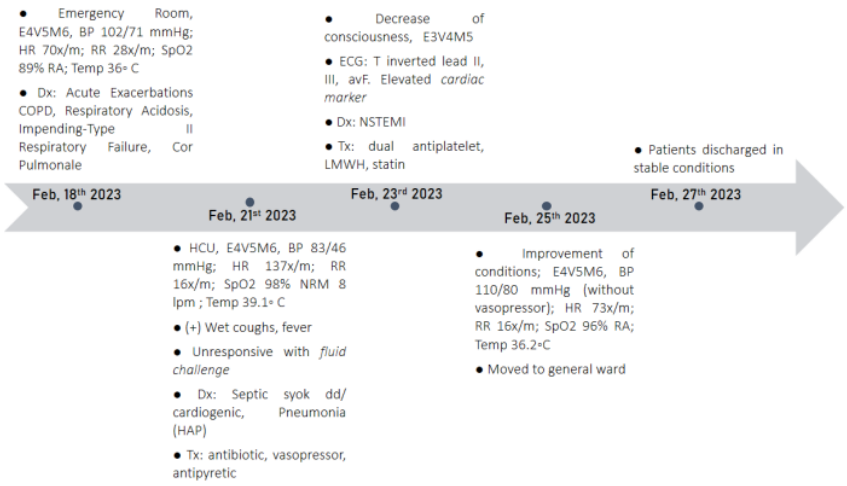


Figure 3. Patients' timeline

DISCUSSION

Cardiovascular is the most comorbidity found in COPD patients. In most patients, the cause of death in COPD patients is from underlying cardiovascular disease rather than respiratory problems.[2] Acute myocardial infarction in COPD patients occurs by various mechanisms, but the precise mechanism is not entirely defined. It is probably derived from two pathogenic causes, acute coronary thrombosis, due to systemic inflammatory reaction and a mismatch between myocardial demand and supply oxygen.[5] The existence of common risk factors like smoking, sedentary behavior, and elderly age is the essential thing that must be discovered.[1,2]

In COPD, a systemic inflammatory process characterized by an increase in pro-inflammatory proteins and cytokines in the acute phase, such as interleukin-6, and interleukin-18. Platelet reactivation is also triggered and increases the risk of thrombosis.[6] The finding of blood vessels that are more rigid in exacerbation conditions by examining the carotid-femoral aortic pulse wave velocity (aPWV) indicates endothelial dysfunction. These factors all enhance the possibility of AMI.[1,2,4]

Besides the atherosclerosis process, Mc Allister's research, cited in Goedemans L et al, stated that the most incidence of myocardial infarction in COPD patients was due to the mechanism of oxygen supply-demand imbalance.[3] Unbalanced between these two mechanisms is part of the pathophysiology of type 2 myocardial infarction (T2M1). T2M1 happened depending on numerous determinants such as tension on a systolic wall, contractility, heart rate, and myocardial oxygen supply that relies on the coronary blood flow and oxygen-carrying capacity.[7]

Acute hypoxemic condition in COPD exacerbations is an example of this situation. It can stimulate sympathetic nerve activity through arterial chemoreceptor stimulation. This sympathetic stimulation will lead to tachycardia and the risk of ischemic heart disease.[3,7] It could cause myocardial cell death, symptoms, abnormalities in the

electrocardiogram, and the release of cTn (cardiac troponin).[7] COPD-related hypoxia is also associated with activation of the renin-angiotensin system, which causes decreased renal blood flow and peripheral vasoconstriction, and increased oxidative stress, ultimately increases the risk of acute myocardial infarction.[1–3]

Acute exacerbation episodes in COPD patients are enforced based on the fulfilled indicators of the cardinal symptoms, including increased dyspnea, improved sputum quantity, and sputum purulence. During the treatment period, a spirometry examination could not be performed, because the patient was still in a hemodynamically unstable condition with findings of myocardial infarction, which is a contraindication to this examination.[8] As a result, the severity of exacerbations is determined based on clinical findings and patient support where there is a risk of life-threatening respiratory failure.[9]

Myocardial infarction is rarely detected in hospitalized patients for AECOPD. Based on clinical manifestations, patients with AMI in COPD had a slight possibility of complaining the typical chest pain. Patients mostly report unusual chest pain along with palpitations and shortness of breath. Similar to this case, the patients didn't feel anything despite of decrease in consciousness. In addition to atypical clinical symptoms, overlapping symptoms make finding cardiac problems in AECOPD more challenging than in regular patients.[3] Patients are more likely to have NSTEMI than STEMI and lower cardiac enzymes (troponin and creatinine kinase) values in further studies.[2,6]

The pathological picture on the ECG might describe a transient secondary ischemic condition triggered by an increase in oxygen demand, while the oxygen supply reduced and insufficient to fulfill those demands. If ischemic heart disease is suspected, echocardiography may adjust to assess ventricular function and identify the location of vascular occlusion. In conditions of exacerbation, this examination is challenging to perform due to the limited acoustic window. Cardiac MRI can be chosen as an alternative in these conditions. Cardiac markers such as Troponin-T, Troponin-I, and brain natriuretic peptide (BNP) are additional tests that physicians can select. However, troponin measurement is not specific for ischemic cases because this cardiac enzyme can increase in conditions of heart failure, renal dysfunction, pulmonary embolism, pulmonary hypertension, tachyarrhythmia, and sepsis.[3]

Coronary angiography would evaluate problems inside the coronary arteries with a transfemoral or transradial approach. This test evaluates the atherosclerotic processes, arterial stiffness, and coronary stenosis ≥ 50% which may be a reliable indicator of cardiovascular issues. According to Pizarro et al. study, from 88 patients with AECOPD and increased cardiac troponin who underwent the prospective cohort research, 38.6% of them had angiographically confirmed ischemic heart disease that required revascularization PCI (percutaneous coronary intervention). The operator's expertise and standard procedures decided the interventional method for each patient.[4]

Compared to the non-COPD group, invasive therapy (percutaneous coronary intervention) for AMI patients in COPD is rarely given. The decision-making process's factors may not be completely understood. However, the age aspect, where COPD patients typically manifest in the elderly with a high vulnerability,

is one that may be taken into consideration.[1,4] Several medications are suggested to prevent cardiovascular problems after MI in COPD, including β-blockers, dual anti-platelets (aspirin and P2Y12 receptor antagonist, given for one year), statins, angiotensin-converting enzyme inhibitors, and angiotensin II receptor blockers.[1,2] COPD is a contraindication to using of β-blockers due to the potential risk of bronchospasm. However, many studies have shown that cardio-selective β-blockers that are primarily active at cardiac β1 receptors, not bronchial β2 receptors, are not associated with changes in FEV1 or increased COPD exacerbations.[2]

The danger of acute myocardial infarction in COPD is not only feared to occur in critical conditions but can occur in the advanced phase. It was reported in several studies cited in the study of Goedemans L et al., that the outcome of AMI conditions in COPD patients requires special attention because it can cause heart failure, arrhythmias, and death.[1] No proof exist that COPD patients with AMI are more likely than non-COPD patients to experience significant bleeding, stroke, or recurrent AMI.[2] Based on IRR (incidence rate ratio) and 95% CI (95% confidence interval), it has been concluded that severe airflow limitations on AECOPD (GOLD 3-4) have stronger association with myocardial infarction compared to mild-to-moderate (GOLD 1-2).[6] he more severe AECOPD conditions requiring hospitalization are associated with a higher incidence of myocardial infarction compared to those with mild conditions treated at home (RR 8.00).[5]

The outcome of COPD patients with AMI varies widely, depending on the patient's clinical symptoms and the delay in identifying the condition. Atypical clinical manifestations related to the size and location of infarction due to the delay in therapy might confuse the diagnosis and treatment of AMI. Delays in giving treatment, such as reperfusion in STEMI patients, can cause heart failure and possibly death.[1,2]

LIMITATION

In this case, the causes of myocardial infarction are unclear. The abnormalities in the heart and blood vessels cannot be identified since further investigations have not yet been conducted. Sepsis also cannot be ignored, where the cardiovascular system is affected. In sepsis condition, cardiovascular dysfunction occurs through a complex mechanism. The ejection fraction of both ventricles dropped due to inadequate preload, suppression of myocardial contractility, and reduced ventricular compliance. Depression in cardiac function can cause cardio-toxic inflammatory mediators (IL-1, TNF-α, nitric oxide). So, an increase in heart rate will be found to compensate for and maintain the cardiac output.[10] Circulating chemicals (sepsis-induced), vasopressors, and catecholamine toxicity may also potentially mediate cTn increases in addition to T1MI or T2MI.[7]

CONCLUSION

The ECG and cardiac enzymes must be closely monitored, as must symptoms, vital signs, clinical findings, and other changes. So that cases of acute myocardial infarction in AECOPD patients have not been neglected. Delays in case finding can affect the prognosis. Understanding the numerous pathways that contribute to AMI in COPD will help physicians to determine the therapy.

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Conflict of Interest

None

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None

Disclosure

The scientific poster of this case report was presented at the 9th Pulmonary Update 2023.

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