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Case Report

Probable Ticagrelor-Associated Acute Asthma Exacerbation Following Percutaneous Coronary Intervention: A Case Report

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Abstract:
A 57-year-old female with well-controlled bronchial asthma underwent percutaneous coronary intervention (PCI). Three hours after administration of a ticagrelor 180 mg loading dose, she developed severe bronchospasm, with oxygen saturation decreasing from 100% to below 90%. The patient received nasal mask oxygen, repeated nebulization with budesonide suspension, salbutamol, and ipratropium bromide, along with concurrent intravenous methylprednisolone. She was discharged from the ICU within 48 hours; clopidogrel 75 mg was substituted, and there was no recurrence. Severe acute asthma attacks, though rare, possibly induced by ticagrelor, require prompt recognition and aggressive management to prevent respiratory failure during PCI.

Keywords: Ticagrelor; Asthma; Antiplatelet Therapy; Percutaneous Coronary Intervention

Introduction

Ticagrelor has rapid, potent, and reversible antiplatelet effects unaffected by genetic variations, greatly reducing the mortality of acute coronary syndrome (ACS) patients [1]. Thus, major global guidelines recommend it as the primary anti - platelet therapy for ACS patients. However, the incidence of ticagrelor - associated dyspnea is 10% - 20%, with most cases being mild and self – resolving [1-4]. Currently, comprehensive epidemiological data on ticagrelor-induced severe acute asthma exacerbation are scarce. Although the incidence is low, clinicians should exercise heightened vigilance due to the potential for fatal risks. This case report presents a 57-year-old female patient with a history of bronchial asthma who developed acute bronchial asthma attack shortly after ticagrelor administration post-PCI, highlighting the need for vigilance in such high-risk populations.

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

Case Presentation

A 57-year-old non-smoking female patient was diagnosed with bronchial asthma four years ago. During attacks, she had shortness of breath. She was treated with inhaled budesonide and formoterol and remained asthma-free for a long time.

Approximately six weeks before hospitalization, the patient started having chest tightness during physical activity, which eased after about two - minute rest and was accompanied by fatigue. After referral to another hospital, pulmonary function tests showed normal ventilation. During admission, physical examination showed stable vital signs, normal transcutaneous oxygen saturation, and no significant cardiac or pulmonary function abnormalities.

Upon admission, comprehensive examinations were done. Biochemical tests showed elevated lipid levels (total cholesterol 5.27 mmol/L, triglycerides 1.92 mmol/L and LDL 3.14 mmol/L) , while routine blood tests, troponin, BNP, D - dimer, and glycated hemoglobin were normal . Electrocardiogram (ECG) showed a heart rate of 103 bpm and abnormal Q waves in leads III and aVF(Figure1A). Chest CT revealed mild emphysema, right middle lobe atelectasis, bronchitis in both lungs and scattered nodules (Figure 2).

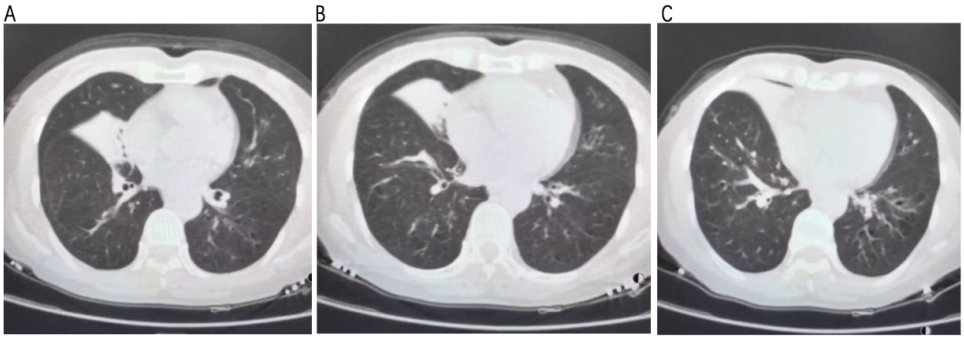

Figure 2. Chest CT upon admission. The chest CT scan reveals atelectasis in the right middle lobe, mild emphysema, bronchitic changes in both lungs, and scattered small nodules.

Echocardiography detected minimal tricuspid regurgitation. Holter monitoring showed normal rhythm. Two days after admission, coronary angiography revealed 95% stenosis in the anterior descending artery and 70% stenosis in the right coronary artery.

(Figure 3). 300mg of aspirin and 180mg of ticagrelor were given, and a stent was placed in the anterior descending artery.

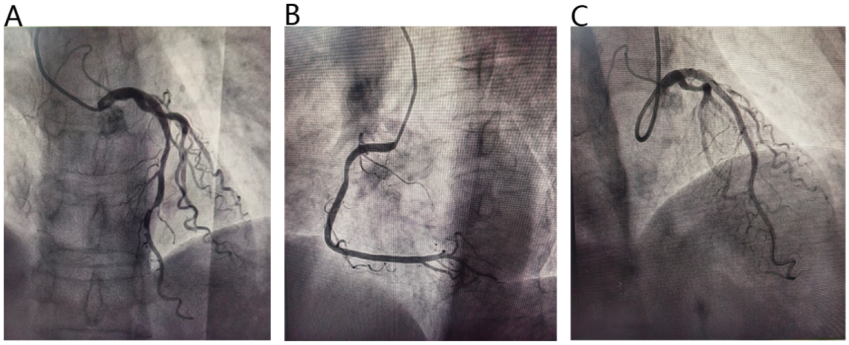

Figure 3. Coronary Angiography.Coronary angiography revealed a 95% stenosis in the middle segment of the left anterior descending artery (A) and a 70% stenosis in the middle segment of the right coronary artery (B). The angiographic image (C) depicts the placement of a Firebird 2 (2.5×33 mm) stent and a Lepu (3.0×24 mm) stent in the left anterior descending artery

Postoperatively, the patient reported mild chest tightness and slight dry cough. Vital signs were stable and peripheral oxygen saturation was 100%. Cardiopulmonary examinations showed no significant abnormalities. Immediate follow - up ECG had no significant changes compared to preoperative results (Figure 1B). About 3 hours later, the patient suddenly developed wheezing and dyspnea, with marked congestion of the bulbar conjunctiva and chest wall skin. The heart rate reached 104 bpm, and the arterial oxygen saturation dropped to 90% and below. Distinct wheezing sounds were heard in both lungs. Subsequently, the classic triad of retractions (sunken intercostal spaces, suprasternal notch, and supraclavicular fossa) gradually developed. Blood pressure rose to 200/110 mmHg, and the heart rate increased to 150 bpm. The patient received oxygen therapy via nasal mask, with repeated nebulization of budesonide suspension, salbutamol, and ipratropium bromide. Concurrent intravenous methylprednisolone was administered. Three hours later, the patient's dyspnea showed slight improvement with blood oxygen saturation reaching 94%. Immediate blood gas analysis indicated an oxygen partial pressure of 79mmHg (under high-flow oxygen therapy).The blood cell count indicated a slight elevation in the total white blood cell count (10.9×10^9/L, reference range 3.5-9.5×10^9/L), neutrophils (7.89×10^9/L, reference range 2.0-7.0×10^9/L), and eosinophils (0.45×10^9/L, reference range 0.1-0.4×10^9/L.Subsequent serial monitoring of BNP, troponin, myoglobin, and creatine kinase isoenzymes (CK-MB) showed no significant abnormalities. Revised electrocardiogram confirmed sinus tachycardia with a heart rate of 140 bpm (Figure1C). The cardiac ultrasound revealed no significant structural or functional bnormalities. The patient and family declined invasive mechanical ventilation, opting instead for repeated nebulization therapy with glucocorticoids and short-acting$\beta_2$agonists. Within 8 hours, the patient's chest tightness and dyspnea symptoms had largely resolved. Subsequent blood gas analyses conducted without supplemental oxygen showed that oxygen partial pressure and other parameters had normalized to preoperative levels . The postoperative follow-up ECG on the second day showed flattened and inverted T waves in leads II, III, aVF, and V1-V6 (Figure1D).

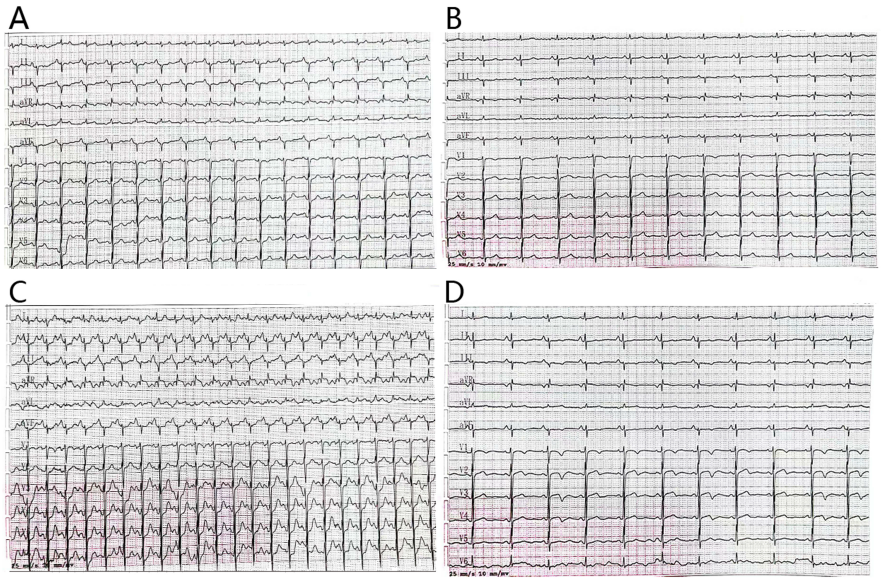

Figure 1. ECG upon admission, immediately post-PCI, during the asthma attack, and the following day after the asthma had subsided.The admission ECG revealed a slightly elevated heart rate of 103 bpm with abnormal Q waves in leads III and aVF (A). The ECG immediately post-PCI showed a slower heart rate (of 70 bpm compared to the admission reading (B). During an asthma attack, the ECG indicated a marked increase in the ventricular rate to 140 bpm (C). The following day, after the asthma had subsided, the heart rate returned to 67 bpm, and significant T-wave inversions were observed in leads V1-V6.

Based on these findings, the diagnosis was confirmed as a bronchial asthma attack rather than cardiogenic asthma. Ticagrelor was identified as the likely trigger, leading to the adjustment of the antiplatelet regimen to aspirin combined with clopidogrel. Subsequent

follow-ups before discharge and during the months following discharge showed no recurrence of dyspnea episodes, further confirming that the asthma attack was most likely triggered by ticagrelor. The event timeline content is as follows (Table 1).

Table 1. The event timeline content

Time Point	Event
4 years before admission	The patient was diagnosed with bronchial asthma and remained asymptomatic following regular inhaled therapy
~6 weeks before admission	Stress-induced chest tightness occurred during physical activity and was relieved with rest, accompanied by fatigue. Pulmonary function tests conducted at external hospitals were normal.
2 days after admission	Coronary angiography confirmed coronary artery stenosis. A dose of 300mg aspirin and 180mg ticagrelor were administered, and stent implantation was performed in the left anterior descending artery.
Immediately after PCI	The patient reported mild shortness of breath, with no abnormalities noted in vital signs or physical examination.
~3 hours after PCI	Sudden wheezing and dyspnea occurred, with arterial oxygen saturation dropping to less than 90%. Bilateral wheezing was detected, and aggressive anti-asthma treatment was administered immediately.
3 hours after treatment	Dyspnea improved slightly, oxygen saturation increased to 94%
8 hours after treatment	Chest tightness and dyspnea were predominantly relieved, and blood gas parameters had returned to preoperative levels.
2nd day after surgery	Suspected diagnosis: Ticagrelor-induced acute asthma exacerbation. Ticagrelor was discontinued, and the antiplatelet regimen was adjusted to aspirin plus clopidogrel.
3 months after discharge	Follow-up assessment demonstrated normal pulmonary function with no recurrence of asthma symptoms.

The Naranjo Adverse Drug Reaction Probability Scale was applied, yielding a score of 7, indicating a probable relationship between ticagrelor and the asthma attack. The patient was followed up for 3 months after discharge, with pulmonary function tests showing normal ventilation capacity and no recurrence of asthma symptoms (Table 2).

Table 2. The Naranjo Adverse Drug Reaction Probability Scale

Adverse Drug Reaction Probability Scale				
Score Interpretation: ***Definite (≥9), Probable (5-8), Possible (1-4), Doubtful (≤0)***				
Question	Yes	No	Unknown	Score
1. Are there previous conclusive reports on this reaction?	**1**	**0**	0	1
2. Did the adverse event appear after the suspected drug was administered?	**2**	**-1**	0	2
3. Did the adverse reaction improve when the drug was discontinued or a specific antagonist was administered?	**1**	**0**	0	1
4. Did the adverse reaction reappear when the drug was readministered?	**2**	**-1**	0	0
5. Are there alternative causes (other than the drug) that could on their own have caused the reaction?	**-2**	**1**	0	1
6. Did the reaction reappear when a placebo was given?	**-1**	**1**	0	1

Table 2. *Cont.*

7. Was the drug detected in the blood (or other fluids) in concentrations known to be toxic?	**1**	**0**	0	0
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	**1**	**0**	0	0
9. Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	**1**	**0**	0	0
10. Was the adverse event confirmed by any objective evidence?	**1**	**0**	0	1
Total Score	7			

Discussion

The patient in this case had a history of bronchial asthma. After long - term medication, the asthma was controlled for an extended period, and preoperative examinations showed no abnormalities in lung function or blood oxygen saturation. The patient was admitted due to chest tightness during physical activity and was initially diagnosed with angina.

However, within hours after PCI, the patient developed severe dyspnea, decreased blood oxygen saturation, tachycardia, and abnormal hypertension.

Etiology of Acute Postoperative Dyspnea

In the post - percutaneous coronary intervention setting, the causes of acute dyspnea can be categorized into cardiac, pulmonary, medication - related, and miscellaneous etiologies.

Acute thrombosis within the stent and other relevant causes also need to be differentiated (Table 3). In this case, acute intrastent thrombosis was definitely ruled out. ECGs were reviewed immediately after the operation and during episodes of dyspnea, and no significant ST - T dynamic changes were observed. Although partial T - wave inversion was noted in some leads on the second postoperative ECG, the patient did not experience chest pain and had normal myocardial markers. This was considered a T - wave memory phenomenon, which reflects transient T - wave inversion or biphasic patterns resulting from delayed electrophysiological recovery after revascularization.

Table 3. Key points for the differential diagnosis of postoperative acute dyspnea-related diseases.

Differential Diagnosis	**Key supporting points**	**Points for exclusion**	**Consistency with this case**
Contrast agent allergy	Conjunctival congestion; skin flushing; auscultation reveals expiratory wheezing	It typically occurs within 5 - 30 minutes after injection, without urticaria or rash, and the skin or mucosal reactions disappear after blood pressure decreases.	moderate
Anxiety-related hyperventilation	Stress related to PCI and tachypnea	There is no perioral or extremity numbness, and blood gas analysis reveals no respiratory alkalosis. Wheezing sounds are present in the lungs.	low

Differential Diagnosis	Key supporting points	Points for exclusion	Consistency with this case
Acute pulmonary embolism	Sudden onset of dyspnea accompanied by hypoxemia and tachycardia	D-dimer levels are normal, with improvement in chest tightness after bronchodilator administration	low
Acute left heart failure related to hypertensive crisis	Stress during PCI procedures may lead to hypertension, manifested as dyspnea and orthopnea.	Intraoperative and postoperative vital signs remained stable within 3 hours, with no pink frothy sputum. Significant chest tightness persisted despite normalized blood pressure, and no moist rales were detected in the lungs. BNP and echocardiography results were normal.	low
Acute bronchial asthma	Dyspnea with three-depression sign, auscultation reveals wheezing sounds, and gradual drug response after repeated use of corticosteroids and bronchodilators	Ticagrelor-induced severe acute asthma exacerbations are relatively rare.	high
		Aspirin exacerbation of respiratory diseases is relatively more common, but subsequent continued aspirin use did not present with symptoms such as chest tightness.	

The patient's chest CT scan revealed right middle lobe atelectasis, mild emphysema, bilateral bronchial inflammatory changes, and scattered small nodules. These findings are consistent with the pulmonary imaging manifestations observed in some chronic asthma patients. In such patients, chronic inflammation, airway remodeling, and mucus plug obstruction lead to middle - lobe - syndrome - like presentations, characterized by middle lobe atelectasis and chronic inflammatory changes in adjacent bronchi.

Pulmonary embolism, pneumothorax, and contrast - agent - induced hypersensitivity are potential factors contributing to sudden dyspnea in patients after PCI. However, pre - hospital pulmonary function tests at another hospital showed no abnormalities, and transcutaneous oxygen saturation remained at 100% from preoperative to 3 hours post - operatively. D - Dimer levels showed no elevation before or after the onset of dyspnea, effectively ruling out pulmonary embolism.

Although CT scans revealed mild emphysema, physical examination and subsequent clinical progression did not align with the clinical features of tension pneumothorax.

Contrast - agent - induced allergic asthma typically manifests as an immediate reaction, with peak symptoms occurring 5 - 30 minutes after injection. Respiratory symptoms include laryngeal edema and bronchospasm - related manifestations, accompanied by systemic allergic reactions such as cutaneous urticaria, hypotension, tachycardia, nausea, and dizziness. While this patient exhibited bronchospasm, systemic allergic signs were absent, and the presentation consisted of abnormal blood pressure elevation rather than hypotension indicative of anaphylactic shock.

Through clinical evaluation and follow - up tests, including electrocardiogram, blood gas analysis, BNP, cardiac enzyme tests, and echocardiography, the preliminary diagnosis indicates that ticagrelor may have triggered a severe acute asthma attack. Since the patient

was experiencing severe dyspnea during the acute episode, spirometry was not performed. After ruling out cardiac causes, the diagnosis of an acute asthma attack was mainly based on clinical auscultation findings, hypoxemia, and the fact that the patient's symptoms improved rapidly after treatment with nebulized bronchodilators and systemic corticosteroids.

Mechanisms underlying ticagrelor - induced asthma

Pre - existing asthma history is the most definitive risk factor[5]. Patients with underlying asthma have airways in a hyperreactive state, where ticagrelor may act as a trigger to induce severe bronchospasm. The precise mechanism by which ticagrelor induces bronchospasm and persistent asthma has not been fully elucidated. Current hypotheses include the Adenosine Hypothesis, the P2Y12 receptor - mediated mechanism, and the Non - specific stimulation response[6-10].

Mechanism of ticagrelor inhibition of adenosine uptake by cells

The mechanism by which ticagrelor inhibits adenosine uptake by cells is its suppression of the equilibrative nucleotide transporter 1 (ENT1). This is the core mechanism responsible for elevating extracellular adenosine levels induced by ticagrelor.

Under physiological conditions, extracellular adenosine is primarily transported into various cells (such as red blood cells, endothelial cells, and platelets) via ENT1. Subsequently, it is metabolized intracellularly (e.g., degraded into inosine by adenosine deaminase), thereby maintaining low and stable adenosine concentrations in the extracellular fluid.

When ENT1 is inhibited, the uptake of extracellular adenosine by cells is impaired, leading to delayed adenosine clearance. Consequently, the half - life of adenosine is prolonged, and its concentrations in extracellular fluids (including plasma and interstitial fluid) are significantly elevated. This effect is particularly pronounced in areas where adenosine production is inherently high, such as during local ischemia or inflammation.

Therefore, in bronchial asthma patients with well - controlled asthma, chronic local inflammatory responses in the airways may lead to significant adenosine accumulation after administration of ticagrelor.

Potential role of adenosine in inducing bronchial smooth muscle contraction

The mechanism by which adenosine induces bronchial contraction does not involve direct action on smooth muscle but primarily occurs through indirect pathways, particularly by activating inflammatory cells within the airways. Adenosine binds to A2B receptors on the surface of mast cells in the airway mucosa, leading to their activation and degranulation. Simultaneously activated mast cells release a series of preformed and newly synthesized spasmogenic mediators, including: Histamine: one of the primary mediators responsible for rapid bronchial contraction. Cysteine leukotrienes: such as LTC_4, LTD_4, and LTE_4, which are potent bronchoconstrictors. Prostaglandin D_2 (PGD_2): which also induces contraction of airway smooth muscle. These mediators collectively act on corresponding receptors on airway smooth muscle, ultimately resulting in bronchial smooth muscle contraction and lumen narrowing, clinically manifesting as bronchospasm.

Why patients with a history of asthma may be particularly sensitive

Patients with a history of asthma exhibit greater sensitivity to the adenosine - related effects of ticagrelor, primarily because their airways are already in a state of "allergy" and "hyperresponsiveness."

Elevated baseline adenosine levels in airways: Studies have shown that asthma patients exhibit higher adenosine levels in their airway secretions compared to healthy individuals. This indicates that their airway tissues are already exposed to elevated levels of adenosine.

Mast cells are in a "pre - activated" or hyperresponsive state: Asthma is an airway inflammatory disease characterized by the activation and increased number of mast cells. The expression of adenosine receptors on the surface of these cells may be upregulated, rendering them more sensitive to adenosine stimulation.

A complete effector system exists: In the airways of asthma patients, not only are activated mast cells present, but there is also a reserve of smooth muscle and neural pathways that can be activated by mediators released by mast cells.

Synergistic effect: When ticagrelor further increases extracellular adenosine concentration by inhibiting ENT1, it is equivalent to adding fuel to the fire in an already sensitized and hyperinflamed environment. The increased adenosine interacts with pre - activated or proliferated mast cells in the airways, triggering the release of bronchial constriction mediators at levels far exceeding those in healthy individuals, thereby inducing or exacerbating bronchospasm.

In summary, although adenosine accumulation is a well-established pharmacological effect of ticagrelor and may theoretically induce bronchospasm in asthma patients, clinically, ticagrelor-related dyspnea, which is more commonly observed, manifests as central or sensory respiratory abnormalities without airway obstruction.

For patients with a history of asthma, the potential risk of ticagrelor-induced bronchospasm warrants particular vigilance.

Here, we summarized the comparison between ticagrelor-induced asthma and non-allergic bronchial asthma (Table 4).

Table 4. Comparison between Ticagrelor - Induced asthma and non - allergic bronchial asthma.

Feature	Ticagrelor-Induced Asthma	Non-Allergic Bronchial Asthma (Intrinsic Asthma)
Pathogenesis	Ticagrelor inhibits the equilibrium nucleotide transporters (ENT 1 and 2), thereby blocking adenosine uptake and increasing extracellular adenosine concentration. Adenosine stimulates C fibers of the vagus nerve in the bronchial wall through A1 and A2A receptors, which results in bronchial smooth muscle contraction.	The underlying mechanisms remain incompletely understood and may involve abnormal neural regulation, airway hyperresponsiveness, immune cell activation induced by physical stimuli (air particles, temperature changes), or nonspecific T-cell activation caused by "superantigens".
Allergic history	No history of specific allergies	No history of confirmed allergy
Trigger factor	Ticagrelor Administration	Respiratory tract infections (the most common cause, with approximately 60% of acute exacerbations associated with infection), cold air, exercise, emotional fluctuations, air pollution, and occupational exposure
Inflammation pattern	Adenosine-mediated bronchospasm and inflammatory response	Inflammatory response predominantly characterized by neutrophil infiltration (non-eosinophil-mediated)
Therapeutic Strategy	Discontinue the medication or switch to another antiplatelet agent; theophylline (an adenosine receptor antagonist) may be considered to alleviate symptoms.	Control infection, avoid irritants, and administer high-dose corticosteroids combined with long-acting bronchodilators
prognosis	Symptoms typically alleviate after discontinuation of medication.	The condition is typically more severe than allergic asthma, more prone to chronicity, and has a relatively poorer prognosis.

Conclusion

This case highlights the importance of recognizing ticagrelor as a potential trigger for severe asthma exacerbations, even in patients with well-controlled respiratory conditions. In the context of acute dyspnea post-PCI, clinicians should include drug-induced bronchospasm in the differential diagnosis. Prompt recognition and substitution of the antiplatelet agent are critical for a favorable outcome.

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Informed Consent Written, informed consent was obtained from the patient.

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