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Severe angina pectoris in asthma attack: a case report

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Abstract

Asthma is a chronic inflammatory disorder of the airways related to the obstruction of reversible airflow. Asthma presents as recurrent attacks of cough and dyspnea. Poor control causes recurrent admissions to the ICU, and mortality is related to poor drug compliance and follow-up. Angina pectoris is a syndrome of recurrent chest discomfort related to myocardial ischemia. The presence of these two disorders rarely has been reported. We reported a 12-year-old boy who was referred with exacerbation of asthma and developed angina pectoris during hospitalization. He had labored breathing and diffuse wheezing. During treatment of the asthma, the patient developed severe chest pain due to shunt formation and coronary hypoxia, caused by the sole administration of ventolin, since oxygen had been disconnected. After receiving appropriate therapy, both his asthma and angina recovered, and, to date, he has not experienced angina pectoris again.

Keywords: severe asthma, angina pectoris, pediatrics

1. Introduction

Asthma is a chronic inflammatory disorder of the airways related to the obstruction of reversible airflow, hyper responsiveness, and respiratory symptoms. Bronchial asthma is a chronic disease that affects individuals of all ages. There are approximately 300 million asthma patients worldwide, and experts believe that its prevalence will increase in the next decade. In France and Germany, America, and the United Kingdom, its current prevalence is about 6-7, 11, and 15-18%, respectively (1). The prevalence of asthma in Iranian children is between 1.26-11.6%, depending on gender, race, and socioeconomic status (2). Insufficient asthma management could cause repeated emergency department visits by patients who are in asthma crisis (3). Exacerbation of acute bronchial asthma is a common medical emergency (4). All patients with asthma are in danger of having exacerbations presented by severe cough, shortness of breath, wheezing, or chest tightness (5). Angina pectoris is a syndrome of recurrent chest discomfort related to myocardial ischemia but without myocardial necrosis (6). The patients typically describe this chest discomfort as a burning sensation or pressure and tightness in the chest. Patients presenting with symptoms in favor of angina pectoris almost always are referred for the evaluation of cardiac problems, despite the fact that there is a variety of differential diagnoses for angina pectoris in the literature (7). Angina pectoris is caused by coronary artery hypoxemia and necrosis. Hypoxemia is the insufficiency of oxygen in the circulating blood, which can be caused by changes in the respiratory rate, ventilation, or perfusion. Cardiovascular causes also can contribute to hypoxemia, e.g., shunts. The most common causes of hypoxemia are shunts, hypoventilation, and mismatches in the

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ventilation/perfusion rates. Profound hypoxemia is a reversible cause of death due to asthma (8). Therefore, the Asthma Guideline of the British Thoracic Society recommends oxygen for all patients with severe, acute asthma. During asthma attacks, nebulized beta-2 agonists may worsen hypoxemia, so the combination of beta-2 agonists with oxygen is recommended. Three to five percent of deaths in asthma patients are due to hypoxemia. Ventilation/perfusion mismatch-induced hypoxemia will be expected if the forced expiratory volume in one second (FEV1) is less than 25% of the predicted value, causing alveolar hypoventilation. Administering only ventolin to predisposed patients increases the ventilation/perfusion mismatch, leading to critical acute desquamation. Therefore, oxygen is the first line of treatment and must be administered prior to and simultaneously with nebulized bronchodilators (9).

2. Case presentation

2.1. Clinical Presentation

A 12-year-old boy presented to the emergency department with an acute exacerbation of asthma. On arrival, he had labored breathing. Initial findings included a respiratory rate of 35 breaths/min, heart rate of 96 beats/min, pulse oxymetry reading of 90% in ambient air, temperature of 37.3 °C, nasal flaring, and diffuse wheezing in all lung fields.

2.2. History

The patient had had an upper respiratory tract infection for two days and a 10-year history of asthma. The patient had poor drug compliance and had no outpatient follow-up during the past four years. Despite suffering from cough and shortness of breath during the day and at night, he was using only a salbutamol inhaler to relieve his symptoms. He also had a past history of allergic rhinitis and was on a corticosteroid nasal spray. The patient's family history also was positive (his mother and sister) for allergic rhinitis.

2.3. Laboratory and Imaging Findings

The arterial blood gas (ABG) showed pH: 7.40, carbon monoxide pressure (pCO_2): 30 mmHg, oxygen pressure (pO_2): 60 mmHg, sodium bicarbonate (HCO_3-): 20.4 mEq/l. His chest X-ray revealed hyperinflation with no evidence of either a lung infiltrate or pneumothorax. The electrocardiogram (ECG) showed a sinus tachycardia. The pulse oxymetry probe was still reading below 90%, and the patient had air hunger and angina pectoris after broncodilator therapy. Since the patient had experienced angina pectoris, we checked CKMB (creatine kinase-MB) and Troponin I, and both were normal.

2.4. Treatment and Follow-up

Oxygen was administered promptly with a reservoir mask. He was given multiple nebulizer treatments with ventolin every 20 min for one hour; he also was given an anticholinergic agent and oral corticosteroids. As the patient`s condition showed no improvement, he also received magnesium sulfate with a slow intravenous (IV) infusion. During the patient's hospital stay, he felt slightly better; his respiratory rate became 25 breaths/min, and his heart rate was 90 beats/min. Diffused wheezing could still be detected, so we decided to continue ventolin nebulization every hour. He also was receiving oxygen, and, after two hours, the patient developed compressive retrosternal chest discomfort with no change in intensity while breathing or applying local pressure. This occurred about 10 min after receiving the last dose of nebulized ventolin. The patient became agitated and was tachypneic and screaming with pain. His heart rate was 110 beats/min, and his BP was 130/90; despite not having significant central cyanosis, pulse oxymetry was showing an oxygen saturation of 89% while the patient was receiving supplementary oxygen with a reservoir mask, and expiratory wheezes were audible all over the chest. Transient ischemia was responsible, but it did not cause any permanent sequela. About five minutes later, the patient's condition had improved, and, 20 minutes after that, he no longer had any sign of chest discomfort. The patient was admitted to the ward where he was observed closely. His asthma attack was managed well, and the patient was discharged two days later in good condition. He has never again experienced the angina-like chest pain.

3. Discussion

Asthma is an inflammatory disease that affects the small airways related to reversible airflow obstruction, hyper reactivity in airways, and variable pulmonary symptoms, from dyspnea to respiratory failure (10). Status asthmaticus also is one of the most common medical emergencies in children and adults, and it is related to vital morbidity and mortality (11). In asthma exacerbation, narrowing of the airway occurs due to mucosal edema, bronchospasm, and increase in secretions (12). Short-acting beta-2-agonists have the effect of relaxing the bronchial smooth muscles. Several randomized controlled trials have been designed for testing the efficacy and safety of inhaled beta-2-agonist

therapy in both adult and pediatric asthmatic patients (9, 11). In the management of a severe asthma attack, nebulized or inhaled short acting β2-agonists will support ventilation and oxygenation until the anti-inflammatory effects of all types of corticosteroids take effect (11). Almost all patients with asthma exacerbation have some hypoxemia due to ventilation perfusion (V/Q) mismatching. Short-acting Beta-2 agonists (nebulized or metered dose inhaler) might aggravate it by pulmonary vasodilation in areas of the lung that are not ventilated properly (11, 13, 14). We have described a teenage boy, a known case of asthma, admitted and treated as an asthma exacerbation, during which he developed chest discomfort and hypoxemia while continuous salbutamol nebulization and supplementary oxygen was disconnected accidentally. Pneumothorax and rib fracture was excluded by chest X-ray, and the patient's ECG (Electrocardiogram) had no sign of ischemic changes. The patient had no prior history of chest wall trauma or sickle cell anemia. Blood tests and electrolytes also were normal. We cannot exclude myocardial necrosis as a result of hypoxia and severe asthma, since under conditions of hypoxemia, such as a severe asthma attack, activation of beta agonists may cause significant impairment in the myocardial oxygen supply/demand relationship, and this can result in myocardial damage (15). So, we checked CKMB and troponin I, which were in the normal range, and the ECG showed no ischemic changes. The patient was not receiving oxygen while ventolin was being administered, and, shortly after receiving supplementary oxygen, the patient`s chest discomfort improved. Electrolyte imbalance and myocardial necrosis as side effects of continuous salbutamol nebulization were excluded (16-19).

The patient improved following asthma therapy, with a decrease in heart rate and respiratory rate, but he developed chest discomfort and decreased oxygen saturation several hours later. This was in favor of ventilation perfusion mismatch. The cause of the mismatch was nebulization of salbutamol without supplementary oxygen, which resulted in pulmonary vasodilatation, increasing perfusion to lung areas that were not properly ventilated, and ventilation perfusion mismatch, thus aggravating hypoxemia (12), which clinically presented with angina-like pain in this case. In the literature, it has been recommended that ventolin be administered along with oxygen, but the incidence of chest pain hasn't been mentioned before.

4. Conclusions

A 12-year-old boy presented with asthma exacerbation and developed an episode of angina pectoris during ventolin nebulization without oxygen therapy. Although pediatricians may be familiar with the chest pain described by some asthmatic patients during severe attacks, angina-like pain is not a common symptom in asthma exacerbation. While treating status asthmaticus patients, supplementary oxygen should be provided in order to keep an oxygen saturation of ≥ 92%. All nebulized medications also should be delivered with oxygen in order to avoid causing or worsening the ventilation/perfusion mismatch.

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Conflict of Interest:

There is no conflict of interest to be declared.

Authors' contributions:

All authors contributed to this project and article equally. All authors read and approved the final manuscript.

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