Case Report

Negative Pressure Pulmonary Edema Following Septoplasty Surgery Triggering Acute Subendocardial Myocardial Infarction

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ABSTRACT

Negative pressure pulmonary edema (NPPE) is defined as fluid transudation into the pulmonary interstitium which occurs as a result of elevated negative intrathoracic pressure caused by the upper respiratory tract obstruction and strong inspiratory effort. NPPE is usually seen during emergence from general anesthesia in the early post-operative period especially after upper respiratory tract surgery. We present a case of a 37-year-old male patient who underwent septoplasty operation and developed NPPE which could not diagnosed and progressed to acute subendocardial myocardial infarction.

Key words: Acute subendocardial myocardial infarction, negative pressure pulmonary edema, septoplasty, upper respiratory tract obstruction

INTRODUCTION

NPPE is an uncommon complication of extubation in the early post-operative period and most commonly caused by laryngospasm. Acute laryngeal spasm results in airway obstruction and can cause life threatening pulmonary edema due to negative intrathoracic pressure. The incidence of NPPE has been reported to be 0.05 to 0.1% of all anesthesiology practices however it may be underreported due to failure of recognizing or misdiagnosing. The morbidity and mortality associated with under recognised NPPE is as high as 40%. We present a case of NPPE which progressed to acute subendocardial myocardial infarction.

CASE REPORT

A 37-year-old male patient was referred to the emergency department due to developing of severe chest pain, hypotension, and dyspnea following septoplasty operation. On admission, the patient had on-going chest pain and was markedly agitated. Physical examination revealed blood pressure of 70/40 mmHg, heart rate of 75/min, bilateral diffuse crackles at the lung and wheezy breathing. Oxygen saturation was 60-70% while breathing high-flow oxygen through a face mask.

Twelve-lead resting electrocardiography (ECG) revealed deep and down-sloping ST-segment depressions of 3 mm in the precordial leads [Figure 1]. Due to elevated cardiac biomarkers on admission (troponin I level: 2.1 ng/dl and Creatine Kinase Myocardial Band (CK-MB) level: 7.4 mg/dl) he immediately underwent cardiac catheterization with the diagnosis of non-ST elevation myocardial infarction, acute pulmonary edema and cardiogenic shock. However coronary angiography showed normal coronary arteries. Bedside transthoracic echocardiography showed normal ejection fraction. A 3D transthoracic echocardiography revealed normal left ventricular function with mild mitral regurgitation. The thoracic CT scan did not show aortic dissection and there was no evidence of thrombus in the main pulmonary artery.

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Based on these laboratory parameters, imaging results and clinical course, the patient was diagnosed as hypoxia-induced acute subendocardial myocardial infarction, which is an unexpected result of negative pressure pulmonary edema. NPPE is one of the causes of non-cardiogenic pulmonary edema which occurs with increased inspiration effort and upper respiratory tract obstruction following upper respiratory, oropharyngeal,

pulmonary embolism. There was only increase in diffuse density of the lungs supporting congestion: [Figure 2].

After coronary angiography, the patient was followed in the intensive care unit. During the follow-up, his oxygen saturation and blood pressure levels were increased, chest pain, and dyspnea were improved with high-flow oxygen, inotropic support, and diuretics.

Figure 1: Twelve-lead resting electrocardiography showing deep and down-sloping ST segment depressions in the precordial leads

Figure 2: Thoracic computerized tomography angiography demonstrating increase in diffuse density of the lungs supporting acute diagnosis acute pulmonary edema
and nasal surgeries. The patient was discharged with full wellness on the fourth day of admission.

DISCUSSION

NPPE, usually occurs during emergence from general anesthesia in the early post-operative period especially after upper respiratory tract surgery and may less frequently occur in various clinical situations (electroconvulsive therapy, etc.). Upper respiratory tract obstruction caused by laryngospasm which occurs especially after extubation has been suggested as the most common cause of the NPPE. Increased inspiration effort caused by upper respiratory tract obstruction elevates the negative intrathoracic pressure and this condition progresses to the NPPE.

There are some investigations which reports healthy, young, and well-built adults are more prone to NPPE because of their ability to further increase the negative intrathoracic pressure. An entirely different population carries high NPPE risk which consist of the patients who are obese and patients who have obstructive sleep apnea syndrome, mediastinal mass, and upper respiratory tract obstruction. Depending on the clinical circumstance, if diagnosed early, many patients do not need aggressive therapies but some patients will need invasive or non-invasive mechanical ventilation. Patients usually respond to well fluid restriction, diuretics and corticosteroids.

To the best of our knowledge, we present the first NPPE triggered acute subendocardial myocardial infarction case in the literature. An interesting point need to be mentioned in our case. The diagnosis of NPPE was missed in the early post-operative period, and therefore, the clinical status of the patient deteriorated with time. The complaints of chest pain and dyspnea were misdiagnosed as acute coronary syndrome, acute pulmonary edema and hypotensive shock in the emergency service. In conclusion, other than anesthesiologists, the cardiologists, pulmonologists, and emergency physicians must be aware of the NPPE, a diagnosis of exclusion, in the differential diagnosis of acute pulmonary edema.

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