



Case Report

High-Dose Nitroglycerin Bolus and Non-Invasive Ventilation Prevent Intubation or ICU Admission on Hypertensive Emergency and Acute Pulmonary Edema: Sympathetic Crashing Acute Pulmonary Edema (SCAPE) Treatment on STEMI Patient: A Case Report

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ABSTRACT

Background: A marked elevation in blood pressure that causes acute heart failure and fluid accumulation in the lungs is known as sympathetic crashing acute pulmonary edema (SCAPE). Here, we present a SCAPE patient with severe respiratory failure who was successfully treated with high-dose nitroglycerin (NTG) bolus and non-invasive ventilation (NIV). **Case summary:** A 48-year-old male was referred to our center with typical chest pain and dyspnea. Physical and additional examination found hypertensive emergency (196/66 mmHg), acute pulmonary edema, and acute heart failure, accompanied by anterior STEMI and severe respiratory failure (P/F ratio of 72) in need of intubation. He was given 2 mg intravenous NTG bolus twice supported with NIV, diuretics followed by the primary percutaneous coronary intervention (PCI) stenting in mid left anterior descending (LAD). The patient recovered from acute pulmonary edema, and the P/F ratio improved from 72 to 321 without intubation. **Discussion:** SCAPE could lead to respiratory failure requiring intubation and intensive care unit (ICU) admission when not treated promptly. The administration of large doses of NTG and NIV was safe and resulted in a speedy improvement in symptoms. **Conclusion:** SCAPE can be resolved without invasive procedures, using high-dose NTG and NIV support to overcome hypertensive emergency and pulmonary edema.

Highlights:

1. SCAPE could lead to respiratory failure when not treated promptly.
2. High-dose NTG and NIV support can be used as an alternative treatment to overcome hypertensive emergency and pulmonary edema, thus preventing the need for endotracheal intubation and ICU admission.

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Introduction

A marked elevation in blood pressure that causes acute heart failure and fluid accumulation in the lungs is known as “SCAPE” (sympathetic crashing acute pulmonary edema) [1]. SCAPE's pathophysiology differs from those of other acute heart failure (AHF) syndromes in that it develops rapidly as a result of a sympathetic surge. Even in euvolemic patients, the excessive release of catecholamines induces an abrupt increase in arterial pressure and acute pulmonary edema [2]. Without prompt recognition and treatment, the condition often progresses rapidly to respiratory failure requiring intubation and intensive care unit (ICU) admission.

It has been proposed that high-dose nitroglycerin in combination with non-invasive positive pressure ventilation is the mainstay of therapy for patients with SCAPE. The American College of Emergency Physicians Clinical Policy on AHF syndromes provides level B recommendations for the use of CPAP and intravenous nitrates in patients with AHF and dyspnea [3]. Only a handful of study exists that specifically reports the use of high-dose nitroglycerin in SCAPE [4,5]. Multiple studies revealed variable dosing of nitroglycerin given to SCAPE patients, in which some of them are

significant to reduce symptoms and had better resolution while others were not significant [5-7]. We present a SCAPE patient with severe respiratory failure who was successfully treated with high-dose nitroglycerin (NTG) bolus and non-invasive ventilation (NIV). The patient rapidly improved after treatment and did not require endotracheal intubation or admission to the ICU.

Case Presentation

A 48-year-old male was referred to our center with typical chest pain five days prior and dyspnea two days prior. In addition to that, he had a cold sweat, but he did not experience any chest tightness, palpitations, nausea, or vomiting. He had been a heavy smoker for over 15 years. The patient's family history did not include any incidences of diabetes, hypertension, or cardiovascular disease. On the physical examination, it was discovered that the patient had hypertensive emergency of 196/66 mmHg, an increased jugular venous pressure (JVP), and bilateral lung rales. Further testing indicated that the patient had an anterior STEMI (figure 1), a high-sensitivity Troponin I level of 17.31 ng/L, and severe respiratory failure with a P/F ratio of 72. These findings indicate that the patient needed to be intubated.

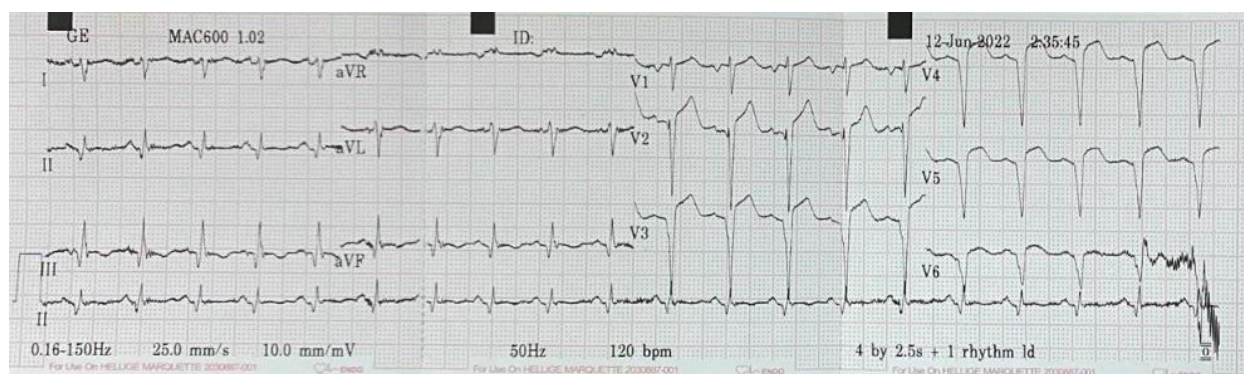


Figure 1. Electrocardiography showed sinus tachycardia 120 bpm with anterior STEMI

An enlarged cardiothoracic ratio (CTR) of 55% was observed on the chest X-ray, and the presence of bilateral cephalization was suggestive of acute pulmonary edema (figure 2). The ejection fraction (EF) as measured by TEICH was 27%, which confirmed that the patient had heart failure-reduced ejection fraction (HFrEF) (figure 3). Throughout the course of the coronary angiography investigation, we found significant stenosis on the distal left circumflex (LCX) artery, critical stenosis on the mid-left anterior descending (LAD) artery, and chronic total occlusion (CTO) on the ostial right coronary artery (RCA) (figure 3).

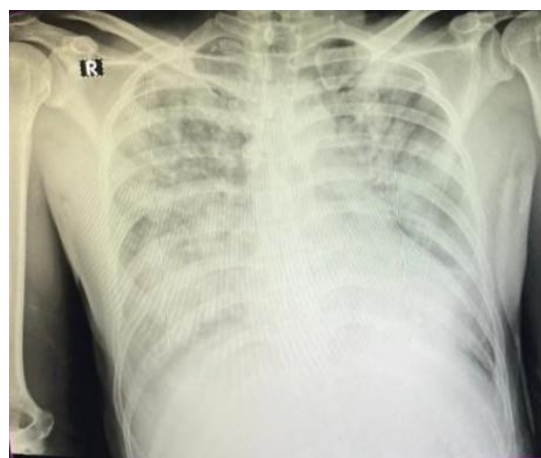


Figure 2. Chest radiograph demonstrated bilateral edema suggestive of acute pulmonary edema at presentation

Afterward, a bolus dose of 2 milligrams of intravenous NTG was administered twice, and also given intravenous furosemide for the SCAPE condition. In addition, ticagrelor was administered to him, and this was followed by primary percutaneous coronary intervention (PCI) stenting in the mid-LAD. The patient made a full recovery from acute pulmonary edema, and the P/F ratio improved to the point at which it no longer required endotracheal intubation. The ratio went from 72 to 321 throughout the patient's recovery. The patient has agreed and

consented to be discussed in this paper. Written informed consent was obtained from the patients.

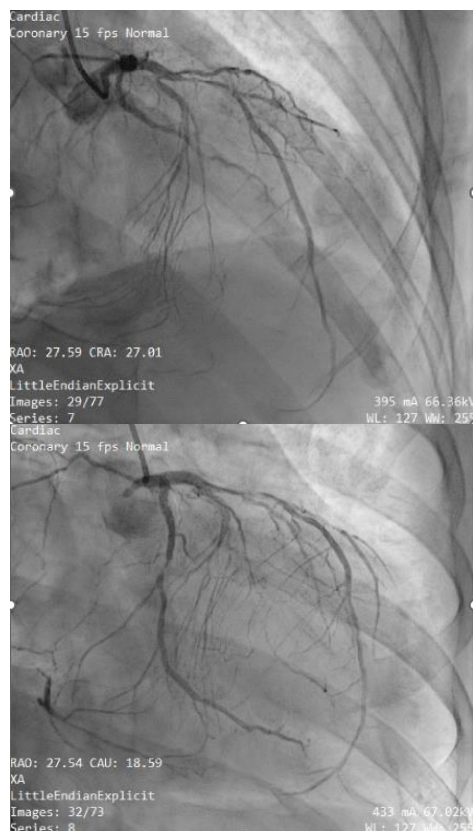


Figure 3. Coronary angiography examination of the patient. Critical stenosis in mid left anterior descending artery and significant stenosis in distal left circumflex artery

Discussion

Acute pulmonary edema, which can be life-threatening, may not be caused by fluid buildup but rather by fluid redistribution as a result of heart failure and increased systemic vascular resistance produced by sympathetic neurohormonal activation. The activation of the renin-angiotensin-aldosterone system, also known as the RAAS, causes the peripheral vascular resistance to become even higher, in addition to increasing the amount of salt and water that are reabsorbed. Both of these things

are detrimental to cardiac function and reduce pulmonary vein return. As a result, fluid from the intravascular compartment shifts into the pulmonary interstitium and alveoli, which leads to hypoxia, dyspnea, and increased sympathetic response. [1]

Even if the patient is hypovolemic or euvolemic, SCAPE still has the potential to cause a sudden redistribution of fluid into the lungs [1]. In addition, the maximum effect of loop diuretics such as intravenous furosemide is reached within 30 minutes to two hours, and it is quite improbable that these medications can offer symptomatic relief in the immediate way that is necessary for SCAPE [2]. As a result, the focus on the treatment of pulmonary edema has shifted away from diuretics and toward vasodilators, particularly high-dose nitrates, in conjunction with noninvasive positive pressure ventilation (NIPPV) [7]. Because nitroglycerin is given in low dosages (less than 100 micrograms per minute) and has very little effect on arterial tone, substantially greater doses are necessary for it to be an effective treatment. [2]

Patients diagnosed with AHF with dyspnea are encouraged to utilize continuous positive airway pressure (CPAP) and intravenous nitrates, as recommended by level B of the American College of Emergency Physicians Clinical Policy on AHF syndromes [3]. Patients diagnosed with SCAPE will make a rapid recovery if they receive prompt treatment. Most importantly, the high-dose NTG

bolus strategy might save a significant number of unnecessary intubations and hospitalizations to the intensive care unit.

According to our protocol, the majority of therapy is high-dose NTG and NIV, both of which should be initiated within minutes of the patient's admission at the emergency department (ED). Our NTG dosage protocol calls for the administration of a 0.6 mg sublingual tablet up to three times, followed by 2 mg intravenous boluses every three to five minutes as needed, with a maximum dose of 20 mg. The use of NTG should be avoided in patients who have hypertrophic obstructive cardiomyopathy, recent use of phosphodiesterase type 5 inhibitors (such as sildenafil or tadalafil), significant aortic stenosis, or hypotension at any time.

Hsieh et al.^[8] detailed three cases of SCAPE that were effectively treated with high-dose nitroglycerin bolus (1 mg intravenously every 2 min). Wilson et al.⁷ conducted research that examined the effectiveness of intermittent bolus NTG (two milligrams intravenously every three to five minutes), continuous NTG infusion therapy (beginning at twenty micrograms per minute), and the combined therapy of bolus and infusion. Patients who were given high-dose boluses had considerably shorter durations of stay and decreased rates of admission to the intensive care unit compared to patients who did not receive these types of treatments. Low incidences of hypotension

were observed across the board regardless of dose protocol, including all groups. Despite the findings, a number of the limitations of the study should be mentioned. When compared to the amounts used for IV boluses, the doses that were administered by infusion were much lower. Since of its very short half-life, nitroglycerin remains to be a favorable agent because it enables modest dosage adjustments to improve cerebral autoregulation.

The administration of large doses of NTG and NIV was shown to be safe and resulted in a speedy improvement in symptoms ^[9]. There is a need for more studies to define the appropriate usage and route of administration of high-dose NTG for individuals diagnosed with SCAPE.

Conclusion

In conclusion, our case demonstrated that SCAPE can be resolved without invasive procedures. The administration of high-dose NTG and the use of NIV to overcome hypertensive emergency and pulmonary edema, minimizes the need for endotracheal intubation and ICU admission.

Acknowledgement

There is no conflict of interest.

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