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FROM CRASH TO RELIEF: RAPID REVERSAL OF SYMPATHETIC CRASHING ACUTE PULMONARY EDEMA IN EMERGENCY DEPARTMENT

Emergency Medicine

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ABSTRACT

Introduction: Sympathetic Crashing Acute Pulmonary Edema (SCAPE) is a rapidly progressive and life-threatening form of acute heart failure characterized by sudden respiratory distress and severe hypertension due to intense sympathetic activation. Prompt recognition and early aggressive afterload reduction are critical to prevent respiratory failure.**Case Report:** A 65-year-old male with a history of hypertension and hyperlipidemia, non-compliant with medications, presented to the emergency department with acute onset severe dyspnea, diaphoresis, and agitation. On arrival, he was markedly hypertensive, tachypneic, and hypoxic. Clinical examination, chest radiography, and point-of-care ultrasound confirmed pulmonary edema. The patient was treated with bilevel positive airway pressure ventilation and high-dose intravenous nitroglycerin. Within one hour, there was rapid hemodynamic and respiratory improvement, allowing discontinuation of ventilatory support. **Conclusion:** Early identification of SCAPE and prompt initiation of high-dose nitrates with non-invasive ventilation can rapidly reverse symptoms and reduce the need for endotracheal intubation and prolonged intensive care.

KEYWORDS

INTRODUCTION

Sympathetic Crashing Acute Pulmonary Edema (SCAPE) represents a severe manifestation of Acute Heart Failure Syndrome [1]. SCAPE tends to progress more rapidly due to heightened sympathetic activity, which causes a sudden surge in catecholamine release. This increases both venous and arterial tone [2]. Therefore, treatment strategies should focus on mitigating the effects of elevated catecholamines, which in turn reduces the afterload and resultant pulmonary Edema. Clinical guidelines advise reducing Blood pressures by 25% in the acute setting using nitroglycerin (NTG) within the first hour.It was first described by Matthew et al [3] in a case series of 3 patients, highlighting the use of ultra-high dose NTG and non-invasive positive pressure ventilation (NIPPV).

CASE REPORT :

A 65-year-old male, chronic smoker with past history of hypertension and hyperlipidaemia, non-complaint with medications presented to Emergency Department with sudden onset of breathing difficulty since past 3 hours, which aggravated upon lying down. Breathlessness was accompanied by severe diaphoresis, agitation, and palpitations.

On arrival to ED, his airway was patent, had a respiratory rate of 34/min with oxygen saturation (Spo2) at room air of 80% and BP was recorded as 280/130 mmHg with a heart rate of 128/min. On auscultation, the patient had reduced air entry and coarse crepitations bilaterally. ECG showed sinus tachycardia with RV strain pattern (S1Q3T3). A bedside Chest X-Ray was suggestive of Pulmonary Edema.

POCUS: Lung ultrasound showed extensive B profile in all three zones. IVC was dilated with <50% collapsibility.

The patient was first put on high-flow oxygen via a non-rebreather mask. The patient was further put on bilevel positive airway pressure (BiPAP) support with a 12/6 (inspiratory/expiratory positive airway pressure).1000 mcg of intravenous NTG was given as a bolus dose followed by a 100-mcg/min infusion. The infusion was tapered down every 10 minutes as the BP reduced. At 60 minutes, the BP of the patient had improved to 150/113 mmHg, and his heart rate was 89/min, his respiratory rate was 20/min, and his Spo2 was 95% on room air. He had improved air entry with significant reduction in the crepitations.

BiPAP was removed about 1 hour after initiation. Following which the patient was transferred to ICU for further management.

DISCUSSION:

SCAPE arises from a harmful cycle of increased sympathetic outflow, excessive afterload, and worsening heart failure. The hallmark pathophysiological feature of SCAPE is significantly elevated afterload, driven by systemic vasoconstriction and hypertension. Patients with SCAPE may have the primary issue as fluid shifting into the lungs rather than fluid overload. It is a high-priority emergency that presents in the ED, where prompt and effective treatment can not only save a life but also prevent the need for intubation and ICU care [1]. Treatment strategies for pulmonary edema have shifted from relying on diuretics to using vasodilators, particularly high-dose nitrates, in combination with non-invasive positive pressure ventilation. The main aim is to decrease the afterload at the earliest to cut the vicious cycle caused by sudden sympathetic upsurge [1-3].

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