

Case Report
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Sympathetic Crashing Acute Pulmonary Edema (SCAPE): A Race Against Time in the Emergency Department

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ABSTRACT

Sympathetic Crashing Acute Pulmonary Edema (SCAPE) represents the extreme end of the heart failure spectrum, characterised by hemodynamic instability and severe respiratory distress. Prompt recognition and intervention are crucial to reducing morbidity and mortality in patients with SCAPE. High-Dose Nitroglycerin (HDN) and Non-Invasive Ventilation (NIV) are pivotal in managing these cases. We present a case of a 56-year-old man who presented to our Emergency Department (ED) with SCAPE. He was successfully managed with HDN at a dosage of 100 µg/min and positive pressure ventilation. This case underscores the importance of early recognition and aggressive intervention in SCAPE, highlighting the effectiveness of HDN and NIV in stabilising hemodynamics and improving outcomes.

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Introduction

Sympathetic Crashing Acute Pulmonary Edema (SCAPE) represents a severe manifestation of acute heart failure characterised by sudden respiratory distress and hemodynamic instability [1]. Sudden deterioration in cardiac function leads to profound pulmonary congestion and hypoxia. The term “sympathetic crashing” underscores the sympathetic nervous system’s hyperactivation, contributing to the acute decompensation of heart failure. This emergent condition demands immediate attention from emergency physicians due to its life-threatening nature [2]. Early recognition is crucial for initiating aggressive management strategies that aim to mitigate congestion, optimise oxygenation, and stabilise hemodynamics. We describe a case of SCAPE, highlighting the importance of prompt recognition and aggressive treatment with positive pressure ventilation and nitroglycerin infusion.

Case Report

A 56-year-old male with a history of hypertension and anxiety presented to the Emergency Department (ED) with a chief complaint of worsening Shortness of Breath (SOB) over the past 2 weeks. He had been treated by his general practitioner with antibiotics for a presumed respiratory infection. However, over the last few hours, his SOB worsened acutely, and the patient said he was struggling to breathe. On arrival, the patient was in severe respiratory distress, hypoxic, tachypneic, having pursed lip breathing and sitting in a tripod position. His oxygen saturation was 90% on a non-rebreathing face mask at 15L/min, respiratory rate was 32, and there were global crepitations on auscultation.

His blood pressure was markedly elevated at 229/135 mmHg, heart rate was 132/min, and he appeared agitated and hungry for air (Table 1).

Bedside ultrasound demonstrated severely impaired left ventricular function and a bilateral B profile. Blood gas showed type 1 respiratory failure (pO₂-8.47 on 0.85 FiO₂, pH-7.07, pCO₂-8.6) (Table 2). Electrocardiogram revealed sinus tachycardia, and chest X-ray showed pulmonary edema with cardiomegaly.

Based on these findings, a diagnosis of SCAPE was made. The patient was extremely agitated and was in severe respiratory distress. To bridge him to get on NIV, we used a water circuit and a 2 mg dose of Morphine. The patient was then started on NIV with an EPAP of 6cm H₂O, an IPAP of 12cm H₂O, and an FiO₂ of 28%. We planned to increase EPAP by 2cm H₂O if no improvement. He was started on nitroglycerin infusion at 100 microgram/min.

Within 2 hours of initiating treatment with high-dose nitroglycerin (HDN) and NIV, the patient’s symptoms improved significantly. His respiratory distress improved, tachypnea reduced, and the patient appeared much calmer. At 1.5 hours of treatment, his blood pressure reduced to 150/100 mm Hg, heart rate dropped to 100 beats/min, and respiratory rate decreased to 24 breaths/min. Repeat blood gas analysis showed improvement in acid-base balance. The patient was off NIV after 6 hours of treatment, and his oxygen requirement was only 40%. The patient was subsequently discharged the following day.

Discussion

SCAPE lies in the extreme severe end of the spectrum of acute heart failure. It is characterised by a rapid onset of hemodynamic

instability, which can lead to high morbidity and mortality if not treated promptly and aggressively. The pathophysiology involves a complex interplay of neurohormonal activation, increasing afterload, and myocardial dysfunction, leading to acute elevation in left ventricular filling pressures, pulmonary edema, and subsequent hypoxia [3]. The hypoxia exacerbates the sympathetic activation, which further worsens the cardiac function.

The diagnosis is mainly clinical with the patient presenting with an acute onset of dyspnea, and sympathetic overactivity - tachypnea, hypertension, hypoxia, bilateral crepitations and agitation [2]. A bedside ultrasound can identify severely impaired ventricular function, bilateral pulmonary edema as B profile and a collapsing inferior vena cava [4]. The condition should be immediately recognised by the emergency physician and treatment started at the earliest.

The vicious cycle of sympathetic overactivity, excessive afterload, cardiac dysfunction and hypoxia can be halted by rapidly reducing

the afterload with HDN. Nitroglycerin (NTG) can be given as boluses or an infusion. The dose of bolus NTG is not well established, with some advocating a bolus of 1-2mg every 3-5 minutes [5,6]. A case report mentions the use of ultra-high dose 9mg in a patient presenting with a BP of 292/154mm Hg [4]. However, a blood pressure-based NTG bolus followed by infusion has been described, which seems a more practical approach [2]. NTG infusion has also been described as infusion at 100ug/min without boluses [7-9]. We started our patient on NTG infusion at 100ug/min, which led to a significant improvement in the patient's status.

Positive pressure from NIV helps in redistributing the fluid from the lungs into systemic circulation, reducing the work of breathing and improving ventilation. Aggressive use of NIV with high PEEP initially can also prevent endotracheal intubation and ventilation [10-11]. The main pathophysiology in SCAPE is excessive afterload rather than volume overload, and therefore, diuretics are not recommended [2,9].

Table 1: Vitals at Various Time Periods of Patient Presentation

Time	Baseline*	20 mins	40 mins	80 mins	2h	3h	4h
Blood pressure (mm Hg)	229/135	196/105	160/90	150/100	136/77	144/80	126/74
Heart rate (beats/min)	132	126	105	100	88	87	82
Respiratory rate (breaths/min)	32	30	26	24	22	20	20
Oxygen requirement (FiO2)	0.85	0.85	0.85	0.60	0.60	0.40	0.40
Saturation (%)	90	96	97	96	98	98	98

* Baseline is the observations just before starting NTG infusion and NIV. All timelines are minutes from starting the treatment.

Table 2: Acid Base Values at Various Time Periods of Patient Presentation

Time	On arrival	1 hour after treatment	5 hours after treatment
PH	7.07	7.3	7.4
PCO2	8.6	5.2	4.5
PO2	8.47	10.7	12.1
Fio2	0.85	0.4	0.4
HCO3	15	20	22.6
Base Excess	-12	-5	-2.2
Lactate	4.0	2.0	0.8

Conclusion

In conclusion, SCAPE represents a life-threatening condition that requires immediate recognition and aggressive intervention. This case underscores the significance of raising awareness among ED physicians regarding this condition and its management. Further research is needed to investigate optimal dosing and duration of NTG boluses and infusions

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