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Flash Pulmonary Edema Complicating Viral Pneumonia in a Patient with Preserved Cardiac Function

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ABSTRACT

Background: Flash pulmonary edema (FPE) is a syndrome of rapid-onset alveolar flooding leading to acute hypoxemic respiratory failure. It classically arises from acute cardiac triggers such as myocardial infarction, hypertensive emergencies, or abrupt volume overload . Rarely, severe infections (e.g. pneumonia) can precipitate FPE despite normal cardiac function by causing inflammatory injury to the alveolar-capillary barrier . Elevated blood pressure often accompanies acute pulmonary edema, especially in patients with underlying hypertension , potentially exacerbating fluid transudation into the lungs.

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INTRODUCTION

Pulmonary edema is defined as an abnormal accumulation of fluid in the lung interstitium and alveoli, resulting in impaired gas exchange and potentially respiratory failure . It is broadly classified into cardiogenic (hydrostatic) and non-cardiogenic (permeability) types based on the underlying mechanism . Flash pulmonary edema (FPE) refers to an acute, life-threatening form of pulmonary edema that develops rapidly over minutes to hours. While FPE is most often associated with cardiogenic causes (such as acute myocardial infarction, acute valvular regurgitation, or hypertensive crisis leading to a sudden rise in pulmonary capillary pressure) , it is also recognized that severe systemic insults like sepsis or pneumonia can trigger FPE in the absence of intrinsic cardiac failure . Infections – particularly severe viral pneumonias (e.g. influenza or COVID-19) – can cause a form of non-cardiogenic pulmonary edema by acutely increasing pulmonary capillary permeability and inducing diffuse alveolar damage

Distinguishing between cardiogenic and non-cardiogenic pulmonary edema is critical, as their management differs significantly. Clinically, cardiogenic edema (acute heart failure) often presents with elevated cardiac filling pressures, S_3 gallop, jugular venous distention, peripheral edema, and radiographic signs of vascular congestion or cardiomegaly . In contrast, non-cardiogenic edema is characterized by acute onset hypoxemia, bilateral infiltrates on imaging without cardiomegaly, and normal cardiac function indices . Laboratory and point-of-care tools aid this differentiation: B-type natriuretic peptide (BNP or NT-proBNP) tends to be high in cardiogenic pulmonary edema and low in non cardiogenic ; bedside echocardiography can rapidly assess ejection fraction and filling pressures; and ultrasound or CT imaging can help identify patterns (e.g. “ground-glass” infiltrates in non cardiogenic vs. vascular redistribution and interstitial edema lines in cardiogenic edema). In practice, a mixed picture can occur—for example, a patient with flash edema in pneumonia might have a reactive surge in blood pressure and some diastolic dysfunction, blurring the line between hydrostatic and permeability edema. Careful evaluation of cardiac function and volume status is therefore necessary in such scenarios.

Acute pulmonary edema accompanied by severe hypertension is a known presentation of acute heart failure, often seen in patients with long-standing hypertension and preserved ejection fraction . The sudden blood pressure elevation increases left ventricular end-diastolic pressure and pulmonary capillary pressures, precipitating edema even if systolic function is normal . Interestingly, systemic infections can act as a trigger for such hypertensive pulmonary edema. Observational studies have noted that in community-acquired pneumonia, acute cardiac events (including acute heart failure and pulmonary edema) occur in a significant subset of patients, especially those with risk factors like chronic hypertension . Therefore, clinicians should maintain vigilance for flash pulmonary edema in the context of severe pneumonia. We present a case of influenza pneumonia leading to flash pulmonary edema with a preserved left ventricular function, notable for a transient hypertensive crisis at the onset of respiratory failure. This case highlights the pathophysiologic interplay between infection-induced lung injury and acute hypertension, and discusses the management approach for this uncommon clinical scenario.

CASE PRESENTATION

Patient History: A 67-year-old woman with a medical history of essential hypertension and type 2 diabetes mellitus presented to the emergency department in the midst of winter season. She reported a 3-day history of high fever, productive cough, myalgias, and progressive dyspnea. There was no chest pain or orthopnea. She had been compliant with her antihypertensive medications,

and her baseline blood pressure at home was around 130/80 mmHg. On examination in the ED, she appeared acutely ill, tachypneic, and mildly cyanotic. Her oral temperature was 38.7°C, blood pressure 140/90 mmHg, heart rate 112/min (sinus rhythm), respiratory rate 30/min with accessory muscle use, and oxygen saturation 85% on room air (improving to 94% on 4 L nasal cannula). Lung auscultation revealed crackles over both bases. Heart sounds were regular with no gallop; jugular venous pressure was not elevated and there was no pedal edema.

Investigations: Initial laboratory tests showed leukocytosis (14,200/μL with neutrophil predominance), elevated C-reactive protein (18 mg/dL), and normal troponin-I (<0.01 ng/mL). Arterial blood gas on 4 L O_2 revealed PaO_2 68 mmHg, $PaCO_2$ 32 mmHg, and pH 7.46. A chest X-ray demonstrated bilateral patchy infiltrates, more pronounced in the peripheral and lower lung fields, without cardiomegaly or pleural effusions. High-resolution chest CT was obtained and showed extensive bilateral ground-glass opacities with subpleural distribution, consistent with viral pneumonia. An influenza A PCR from a nasopharyngeal swab was positive; tests for other respiratory viruses (including COVID-19) were negative. Bedside transthoracic echocardiogram at admission showed normal left ventricular size and vigorous systolic function (estimated EF ~70%), no regional wall motion abnormalities, normal right ventricle size/function, mild concentric LVH and no significant valvular lesions. The inferior vena cava was small and collapsible, suggesting an euvolemic state. These findings, initially pointed toward a primary pneumonia with hypoxemia rather than cardiogenic pulmonary edema.

Clinical Course: The patient was started on supplemental oxygen, intravenous fluids at maintenance rate, and empirical antibiotics (IV ceftriaxone and azithromycin) along with oseltamivir for confirmed influenza. After 24 hours, her clinical status worsened abruptly. She became extremely dyspneic and hypoxemic despite high-flow oxygen, with pink, frothy sputum noted from her endotracheal suction after she required emergent intubation (the frothy sputum is a classic sign of acute pulmonary edema). Chest auscultation revealed diffuse crackles. Repeat vital signs showed a marked elevation in blood pressure to 190/110 mmHg and a heart rate of 130/min. Repeat arterial blood gas on 100% FiO_2 (post-intubation) showed PaO_2 55 mmHg, $PaCO_2$ 40 mmHg, pH 7.30, indicative of severe hypoxemia. A stat portable chest X-ray now demonstrated diffuse bilateral alveolar infiltrates and perihilar opacities (with a “white-out” appearance in some areas) superimposed on the earlier ground-glass changes. Importantly, there were still no signs of cardiomegaly or vascular redistribution. An emergent focused cardiac ultrasound confirmed continued normal LV contractility with no pericardial effusion; there was no evidence of acute volume overload (no engorgement of the IVC) and no significant change in right ventricular size (arguing against massive pulmonary embolism). Nonetheless, to definitively exclude pulmonary embolism as a cause of her sudden collapse, a bedside Doppler ultrasound of the legs was performed (no deep venous thromboses were found) and D-dimer was only mildly elevated (consistent with inflammation). Serial cardiac biomarkers remained normal (troponin I remained <0.01, BNP 128 pg/mL). Given the combination of severity and acute hypertension without cardiac dysfunction, a diagnosis of flash pulmonary edema precipitated by viral pneumonia was made, likely due to acute lung injury from the infection compounded by a hypertensive surge.

Management: The patient was transferred to the intensive care unit and management intensified. Ventilatory support was provided with a mechanical ventilator (tidal volume 6 mL/kg PBW, high PEEP strategy). In addition, she was placed in the prone position for about 16 hours to improve oxygenation. Recognizing the hypertensive nature of her pulmonary edema, we instituted aggressive blood pressure control: a titrated infusion of intravenous nitroglycerin was started, to reduce afterload and pulmonary capillary pressure . Within the first hour of therapy, her blood pressure was brought down to 150/80 mmHg. We administered low-dose IV furosemide (initial bolus of 20 mg followed by 5 mg/hour infusion) to aid diuresis while carefully avoiding excessive volume depletion. Fluid management was conservative – maintenance IV fluids were stopped and a net negative fluid balance (~1.5 L over 24h) was targeted, showing benefit of conservative fluid strategy . Broad-spectrum antibiotics were continued until secondary bacterial infection was ruled out (blood and sputum cultures ultimately remained negative). Antiviral therapy with oseltamivir was maintained for the recommended course. Notably, high-dose systemic corticosteroids were not given, since this was a viral pneumonia (influenza) without evidence of refractory shock or another indication . Throughout, the patient’s hemodynamics were closely monitored: despite initial severe hypertension, she never developed hypotension or shock once vasodilator therapy was carefully titrated. No inotropes or vasopressors were needed. Repeated echocardiography during ICU stay continued to show normal cardiac function, reinforcing that the pulmonary edema was non-cardiogenic.

Outcome: After 12–18 hours of intensive therapy, the patient’s oxygenation improved markedly. Her FiO_2 requirements were weaned to 40% by the next day, and arterial oxygenation correspondingly improved by ICU day 2). The pulmonary infiltrates on chest X-ray began regressing within 24–48 hours. Her blood pressure gradually normalized as well, settling around 130/80 mmHg by the second hospital day with the IV nitroglycerin tapered off. By ICU day 3, she was awake and stable enough to be extubated to high-flow nasal cannula, and subsequently to low-flow oxygen. She had no recurrence of pulmonary edema symptoms. By hospital day 5, she was breathing comfortably in room air. Echocardiography prior to discharge showed an EF of 70% and normal ventricular contractility and mild concentric LVH but no heart failure. The final diagnoses were severe influenza A pneumonia complicated by flash pulmonary edema, in the context of preserved systolic cardiac function and an acute hypertensive response. She was discharged on hospital day 7 on her prior antihypertensive regimen (with good blood pressure control) and supplemental home oxygen for short-term use during exertion. At follow-up 2 weeks later, she had returned to her baseline functional status and oxygenation, and her blood pressure remained at her normal baseline. Preventive measures were reinforced, including annual influenza vaccination and optimization of her chronic disease management, to mitigate future risk.

DISCUSSION

This case illustrates an unusual convergence of non-cardiogenic and cardiogenic factors in flash pulmonary edema. The patient’s clinical course strongly suggests that her viral pneumonia (influenza) was the primary trigger for acute lung injury, while a surge in blood pressure at the critical moment added a hydrostatic component to her pulmonary edema. We discuss below the important aspects of differentiation, pathophysiology, and management in such a scenario, with reference to current literature.

Differentiation between Cardiogenic and Non-Cardiogenic Edema: Distinguishing flash pulmonary edema due to acute heart failure from that due to viral etiology is challenging but essential. In classic cardiogenic FPE, patients often present with markedly elevated blood pressure, high filling pressures, and signs of acute left heart failure (e.g. orthopnea, engorged neck veins). Brain natriuretic peptide is usually elevated, and chest imaging may show cardiomegaly, vascular congestion, and interstitial edema (e.g. Kerley B lines) . Echocardiography typically reveals left ventricular dysfunction or valvular abnormalities. In contrast, non-cardiogenic pulmonary edema is caused by inflammatory capillary leak; these patients may not have hypertension and often have normal cardiac size and function. Imaging classically shows diffuse bilateral infiltrates without cardiomegaly, corresponding to protein-rich edema in the alveoli rather than transudative fluid . Our patient had features favoring a non-cardiogenic cause: low BNP and normal echocardiogram indicated that her flash edema was not due to acute systolic heart failure. The presence of preceding viral pneumonia and the "ground-glass" appearance on CT along with acute severe hypertension at the time of decompensation added complexity – raising consideration of a hypertensive cardiac flash edema. The absence of diastolic or systolic dysfunction on echo argued against pure cardiogenic edema. It is likely that her longstanding hypertension had caused some degree of diastolic stiffness, which, combined with the catecholamine surge (and possibly pain/anxiety), led to a transient increase in left atrial pressure when her blood pressure spiked . In effect, her case may represent a mixed-mechanism flash edema, where infection-mediated lung injury was exacerbated by acute hypertension. This underscores the importance of a broad evaluation: relying on a single parameter (for instance, a high blood pressure) could mislead toward a cardiogenic diagnosis, whereas comprehensive assessment (including imaging and echo) confirmed the predominant non-cardiogenic nature in this case.

Pathophysiology: Severe viral pneumonia can provoke flash pulmonary edema through several interconnected mechanisms. The patient's sympathetic nervous system was likely intensely activated during her respiratory decompensation. Hypoxemia, hypercapnia, and distress can drive a surge in catecholamines, leading to systemic vasoconstriction and hypertension . This acute hypertension can have deleterious effects even in a heart with normal pump function. A sudden rise in afterload and blood pressure increases left ventricular end-diastolic pressure (due to impaired diastolic filling and possible mitral regurgitation from acute pressure overload), which is transmitted back to the pulmonary circulation, causing hydrostatic pulmonary edema . In essence, the patient experienced what has been termed "sympathetic crashing acute pulmonary edema (SCAPE)", where a vicious cycle of hypertension and pulmonary edema develops rapidly . Patients with chronic hypertension are especially prone to this, as they often have left ventricular hypertrophy and diastolic dysfunction; when systemic vascular resistance surges, the stiff LV cannot accommodate the pressure, leading to acute pulmonary congestion despite preserved systolic function . Secondly, mechanical factors may have contributed: the patient's struggle to breathe against an increasingly fluid-filled lung could generate strong negative intrathoracic pressures (especially prior to intubation). Such negative-pressure pulmonary edema can further draw fluid into alveoli. Although typically described in airway obstruction cases, a similar effect can occur in acute heart failure when patients make forceful inspiratory efforts against a closed glottis or severe obstruction. Thus, the pathophysiology in this case likely involved a combination of inflammatory capillary leak and acute hydrostatic forces (from the hypertensive crisis and diastolic dysfunction), together precipitating fulminant pulmonary edema.

Notably, once the flash edema was treated and resolved, the patient's blood pressure normalized – reflecting the transient nature of the hypertensive trigger. In hypertensive acute heart failure, blood pressure often drops rapidly with appropriate therapy and as pulmonary edema abates . Our patient's BP returning to baseline without the need for escalation of chronic therapy suggest s that the extreme hypertension was reactive (stress-induced and pain-induced) rather than a permanently uncontrolled blood pressure. This pattern is consistent with reports that SCAPE episodes often resolve quickly with aggressive blood pressure management, and overshooting with long-term antihypertensives should be avoided until the acute phase is over .

Management Considerations: The management of flash pulmonary edema in the setting of viral pneumonia and hypertension requires addressing both the respiratory failure and the hemodynamic derangements. Key principles include:

- Respiratory Support: Immediate support of oxygenation and ventilation is critical. High-flow nasal cannula or NIPPV (CPAP/BiPAP) can be used initially to improve oxygenation and reduce the work of breathing. In our case, the severity of hypoxemia necessitated rapid intubation and invasive mechanical ventilation. A lung-protective ventilation strategy was employed (low tidal volume around 6 mL/kg and adequate PEEP) to prevent ventilator-induced lung injury .
- Afterload and Blood Pressure Control: In flash pulmonary edema with elevated blood pressure, rapid reduction of afterload is a cornerstone of therapy. Intravenous vasodilators such as nitroglycerin (often first-line) or nitroprusside are recommended to swiftly lower blood pressure and pulmonary capillary pressures . In fact, current heart failure guidelines endorse prompt vasodilator use in hypertensive acute heart failure presentations . We started IV nitroglycerin early, which likely helped abort the vicious cycle of hypertension and pulmonary edema. Titrating these medications requires careful arterial pressure monitoring to avoid overshooting into hypotension. In our patient, blood pressure was brought under control within an hour, which coincided with stabilization of her respiratory status.
- Diuresis and Fluid Management: Careful diuretic therapy and fluid management are important. Even though our patient was not grossly volume-overloaded, we administered low-dose furosemide to encourage diuresis, knowing that any reduction in pulmonary venous volume can help respiratory function. In non-cardiogenic edema, aggressive diuresis is not as fundamental as in cardiogenic edema, but a conservative fluid strategy is still beneficial . Landmark studies in ARDS have shown that limiting fluids and achieving a negative fluid balance can improve lung function and outcomes . We therefore avoided unnecessary IV fluids and achieved net negative fluid balance. Close monitoring of renal function and perfusion is needed during diuresis, especially when using vasodilators concomitantly.
- Treating the Underlying Cause: It is imperative to treat the triggering insult. In this case, appropriate antiviral therapy for influenza (oseltamivir) was given promptly. We also provided empiric antibiotics until bacterial superinfection was excluded, following sepsis management protocols . Adherence to Surviving Sepsis Campaign guidelines – including early pathogen-

directed therapy and organ support – is associated with better outcomes in infection-related acute lung injury . Notably, we avoided corticosteroids, given that this was influenza pneumonia; evidence does not support routine steroid use in viral pneumonia unless there is refractory shock or another indication (steroids may even worsen outcomes in influenza by prolonging viral replication).

- Supportive ICU Care: Standard ICU measures were applied, including deep vein thrombosis prophylaxis, stress ulcer prophylaxis, tight glucose control for her diabetes, and vigilant prevention of ventilator-associated pneumonia. Frequent assessment of neurological status and sedation needs were done to ensure comfort without delaying weaning from the ventilator.

With these interventions, our patient's condition improved rapidly. This rapid recovery is in line with the concept that flash edema (whether cardiogenic or not) can often reverse quickly if the precipitating factor is addressed and supportive care is optimal . In cases of SCAPE, for instance, aggressive treatment can result in dramatic resolution within hours .

Clinical Implications: This case emphasizes a few important clinical lessons. First, severe pneumonia can masquerade as acute heart failure – and vice versa – or present as a combination of both. Clinicians should suspect flash pulmonary edema in any patient with sepsis or pneumonia who has a sudden unexplained respiratory collapse, even if there is no prior history of heart failure. Basic investigations (BNP levels, troponin, chest imaging, bedside echo) are invaluable to discern the dominant mechanism of edema. In our patient, these tools helped confirm that we were dealing largely with a flash episode of pulmonary edema rather than primary cardiogenic shock, which guided us to continue infection-focused therapy and avoid unnecessary heart failure medications like inotropes. Second, the presence of hypertension in acute respiratory failure should prompt aggressive afterload management, but also careful search for underlying triggers. The fact that our patient's pulmonary edema resolved as her infection was treated and blood pressure controlled underscores the importance of addressing both components. Third, prevention is key: patients with chronic conditions (hypertension, diabetes) and the elderly are at higher risk of complications from influenza and other respiratory viruses . Vaccination against influenza (and COVID-19, when applicable) and early treatment of infections may prevent the cascade that leads to such life-threatening scenarios . Finally, this case adds to the growing recognition of the complex cardiac-pulmonary interactions during systemic infections. Even in the absence of traditional heart failure, an infection can acutely strain the cardiovascular system (through cytokines, fever, tachycardia, etc.), and if compounded by factors like hypertension, can result in acute pulmonary edema. As such, a multidisciplinary approach – involving intensivists, cardiologists, and pulmonologists – is often needed to manage these patients optimally.

KEYWORDS

Flash pulmonary edema, Influenza A pneumonia, Preserved cardiac function, Hypertensive crisis, Non-cardiogenic pulmonary edema, Cardiogenic pulmonary edema, Inflammatory lung injury, Early diagnosis, Supportive care

CONCLUSION

Viral pneumonia can precipitate flash pulmonary edema even in patients with normal cardiac pump function, especially when compounded by acute factors such as a hypertensive surge. This case demonstrated that an influenza infection led to flash pulmonary edema , despite a preserved ejection fraction. Early differentiation between cardiogenic and non-cardiogenic pulmonary edema was pivotal in guiding therapy: the use of cardiac biomarkers and point-of-care ultrasound allowed appropriate identification of the edema's etiology. The patient's successful recovery was achieved and measures tailored to hypertensive acute heart failure, including rapid afterload reduction, ventilatory support, conservative fluids, and timely antiviral treatment. Clinicians should be aware of this atypical presentation and remain vigilant for flash pulmonary edema in severe respiratory infections – even when cardiac function is intact. Prompt, aggressive supportive care and treatment of the underlying cause can lead to excellent outcomes. This case also underlines the importance of preventive strategies such as vaccination and optimal control of chronic conditions like hypertension, which may help avert the cascading events that result in flash pulmonary edema.

REFERENCES

1. King KC, Goldstein S. Congestive Heart Failure and Pulmonary Edema. StatPearls Publishing; 2022 .
2. Malek R, Soufi S. Pulmonary Edema. StatPearls Publishing; 2023 .
3. Xu Y, Xin J, Sun Y, et al. Mechanisms of sepsis-induced acute lung injury. Pharmaceuticals (Basel). 2024;17(4):472 .
4. Zanza C, Saglietti F, Tesauro M, et al. Cardiogenic pulmonary edema in emergency medicine. Adv Respir Med. 2023;91(5):445–463.
5. Prescott HC, Ostermann M. Surviving Sepsis Campaign 2021 updates. Med Klin Intensivmed Notfmed. 2023.
6. Wiedemann HP, Wheeler AP, Bernard GR, et al. Comparison of two fluid-management strategies in acute lung injury. N Engl J Med. 2006;354(24):2564–2575.
7. Arabi YM, Fowler RA, Hayden FG. Management of severe viral pneumonia (including COVID-19). Intensive Care Med. 2020;46(2):315–328.
8. Kumar R, Gandhi SK, Little WC. Acute heart failure with preserved systolic function. Crit Care Med. 2008;36(1 Suppl):S52–S56 .
9. EMCrit – Farkas JD. Sympathetic Crashing Acute Pulmonary Edema (SCAPE) (Internet Monograph) . Accessed 2025.
10. Chen L, Han X, Li Y, et al. Complications of cardiovascular events in patients hospitalized with influenza pneumonia. Infect Drug Resist. 2021;14:1899–1910