

Rapid respiratory failure due to influenza A with superimposed methicillin-resistant *Staphylococcus aureus* pneumonia: a case report and review of diagnostic and therapeutic challenges

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Abstract

Influenza is a viral respiratory infection affecting millions annually and can cause complications like superimposed Methicillin-Resistant *Staphylococcus aureus* (MRSA) pneumonia, which has high morbidity and mortality. This case describes a 50-year-old female with severe persistent asthma who had acute shortness of breath. Workup revealed influenza A with superimposed pneumonia on chest X-ray. She rapidly decompensated, requiring intubation and Intensive Care Unit (ICU) admission. Testing revealed MRSA-positive pneumonia. Despite early treatment with broad-spectrum antibiotics, including MRSA coverage, her course remained complicated by ICU admission and respiratory support. The Infectious Diseases Society of America guidelines emphasize treating influenza and recognizing bacterial coinfections but focus more on testing rather than prophylactic antibiotics. This case emphasizes the complexities of managing superimposed MRSA and the need to better understand its pathophysiology. Early antibiotic treatment, especially in high-risk individuals, may mitigate disease severity, reduce ICU admissions, and improve outcomes.

Introduction

Influenza is a viral respiratory infection that infects millions of individuals, is associated with 100,000-710,000 hospitalizations, and causes 4,900-51,000 deaths in the United States each year.¹ Influenza A is one of the most common subtypes of influenza infections, more often infecting individuals with chronic medical conditions, such as asthma or chronic obstructive pulmonary disease, and is more virulent than influenza B.²

Given the high morbidity and mortality rates of influenza, early diagnosis and treatment are crucial.³ Predictors of severe outcomes, such as acute respiratory failure and Intensive Care Unit (ICU) admission, include age extremes, comorbid chronic illness, and delayed presentation.⁴ Chronic illnesses such as chronic obstructive pulmonary disease, cardiovascular disease, malignancy, diabetes, obesity, and chronic kidney disease are also associated with higher rates of prolonged hospitalizations.⁴ A recent retrospective study noted that with each day of delayed antiviral treatment, the probability of severe clinical outcomes, including ICU admissions and death, increased, supporting immediate antiviral initiation upon diagnosis.⁵ Moreover, influenza A is more likely to result in a greater number of hospital admissions and longer hospitalizations, although the rates of complications between influenza A and B are similar.² Identifying high-risk individuals is critical to preventing the progression of influenza to respiratory failure

and other severe complications. Bacterial coinfection occurs in approximately 11.2% of influenza cases, most often caused by *Streptococcus pneumoniae* (*S. pneumoniae*) and *Staphylococcus aureus* (*S. aureus*).^{3,6} More rarely is the development of superimposed Methicillin-Resistant *Staphylococcus aureus* (MRSA) pneumonia.⁶ In a recent retrospective study, bacterial pneumonia and severe influenza causing acute respiratory failure were identified as leading to prolonged mechanical ventilation and worsened health outcomes.⁷ However, the use of prophylactic antibiotics within hospitalized influenza patients remains controversial, with limited supporting data. Some case reports of influenza with MRSA pneumonia illustrate diagnostic and treatment challenges due to the overlap of symptomatology; in these reports, authors advise for the need for improved understanding, and in some cases, recommend the use of empiric antibiotics in situations of high clinical suspicion.⁸ A 2023 systematic review highlights the intricacies of recognizing bacterial coinfection and preventing the overuse of antibiotics.⁶ Due to limited data and clinical complexity, uncertainty in diagnosis and treatment persists. Lack of clear guidelines and varied clinical management may lead to delays in treatment and increased complications.

This paper describes a case of influenza with superimposed MRSA pneumonia and the associated morbidity despite early recognition. It also explores the recommendations of prior case reports and reinforces the need for early diagnosis and treatment.

Case Report

A 50-year-old female with a history of severe persistent asthma, hypertension, iron deficiency anemia, and a recent left lower extremity below knee amputation due to a brown recluse spider bite arrived at the Emergency Department (ED) with three days of shortness of breath. She also reported cough, congestion, fever, and intermittent midsternal chest pain. Believing it was an asthma exacerbation, she utilized her home albuterol without relief before seeking medical care.

On arrival, she was afebrile (37.8°C), tachycardic (heart rate 126), normotensive (blood pressure 123/61), tachypneic (respiratory rate 28), and saturating 97% oxygen on room air. On the initial exam, she had tachypnea and bilateral wheezing without acute distress. She received ipratropium, albuterol, and prednisone for asthma exacerbation. Workup included: complete blood count with White Blood Cell (WBC) $10.6 \times 10^3/\mu\text{L}$ ($3.9\text{--}11.2 \times 10^3/\mu\text{L}$), hemoglobin 9.9 g/dL ($11.3\text{--}15.1$ g/dL), platelets $253 \times 10^3/\mu\text{L}$ ($165\text{--}366 \times 10^3/\mu\text{L}$), and electrolytes and creatinine within normal limits. Coronavirus/Influenza/Respiratory Syncytial Virus (COVID/Flu/RSV) swab was positive for Influenza A and blood cultures were obtained. Despite respiratory treatments, she worsened, requiring Bilevel Positive Airway Pressure (BiPAP) and magnesium. Chest X-ray (Figure 1) showed right-sided opacification concerning for pneumonia, for which she was started on vancomycin and ceftriaxone. Venous blood gases showed a pH of 7.37 (7.32–7.42), carbon dioxide of 45 (mmHg) (42–55 mmHg), oxygen of 60 mmHg (25–40 mmHg), and bicarbonate of 25 mmol/L (22–29 mmol/L), consistent with respiratory acidosis with hypercapnia. With worsening distress, altered mentation, and fatigue, she was intubated and admitted to the ICU for acute hypoxic hypercapnic respiratory failure.

In the ICU, MRSA Nucleic Acid Amplification Test (NAAT) was positive and a pneumonia panel revealed 3+ *S. aureus*, influenza A, and the methicillin resistance (*MecA*) gene. Respiratory culture showed 4+ polymorphonuclear neutrophils and

3+ gram-positive cocci. She was started on cefepime, linezolid, and oseltamivir, along with albuterol, ipratropium bromide/albuterol treatments, and intravenous (IV) steroids. She was briefly placed on heliox while intubated. On day two of admission, she tolerated a respiratory support wean, was extubated to 4-liters nasal cannula, and transferred to the medicine service. From day three to eight of admission, she continued a slow wean in oxygen to 1 liter but had persistent wheezing and diminished air movement bilaterally. She completed a course of linezolid and prophylactic cefepime, a five-day course of steroids, and a five-day course of oseltamivir. She continued respiratory treatments as guided by respiratory therapist assessments.

On day nine of admission, due to persistent oxygen requirements, Pulmonary Medicine was consulted and recommended a pulmonary embolism workup, Immunoglobulin E (IgE) level testing, fungal cultures, as well as extended inhaler treatments, and a prolonged steroid taper. Computed Tomography (CT) (Figure 2) was obtained and was negative for pulmonary embolism. It did however show areas of focal bronchiectasis and cystic changes, which together with her IgE elevated to 901.6 and repeat IgE 1166 (0–99.9 IU/mL), produced concern for Allergic Bronchopulmonary Aspergillosis (ABPA). However, sputum fungal cultures were overall negative, and she was not treated for ABPA. With continued inhalers and steroids, she was weaned to room air and discharged on day eleven without home oxygen. At discharge, she was continued on *Pneumocystis jirovecii* pneumonia prophylaxis with trimethoprim-sulfamethoxazole and a prolonged steroid taper. At outpatient pulmonary follow-up, further workup was negative for additional lung pathologies.

Discussion

This case exemplifies the high morbidity of influenza with MRSA pneumonia, even with early recognition and treatment. Our patient's clinical course demonstrates four important aspects of the disease process that require a deeper understanding and guidelines on approach to management. These aspects are: i) guidelines on

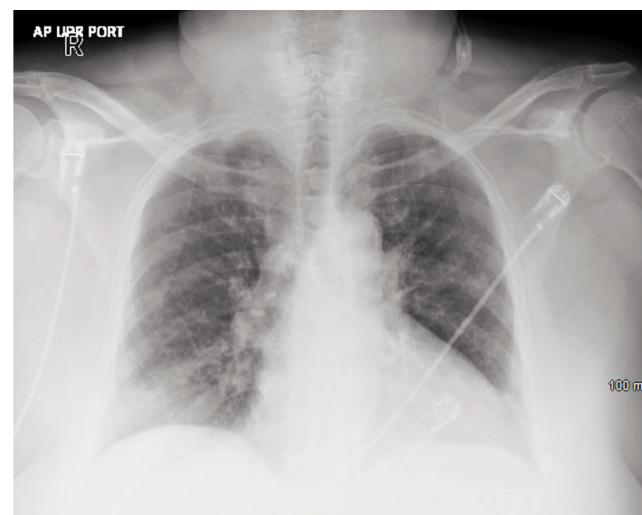


Figure 1. Chest X-ray obtained in the ED on presentation was notable for hazy bibasilar patchy opacities bilaterally, right greater than left. Mild increased interstitial markings as well.

indications to initiate prophylactic antibiotics with MRSA coverage in influenza patients, ii) understanding existing diagnostic challenges for patients with superimposed MRSA pneumonia, iii) understanding of the impact of comorbid asthma as a risk factor for severe influenza-related complications, iv) understanding the pathophysiology associated with influenza and MRSA with comorbidities to develop more targeted therapeutics.

Current treatment guidelines for prophylactic antibiotic use

Current treatment guidelines for influenza alone are well-established and widely understood. According to the Infectious Diseases Society of America (IDSA) Clinical Practice Guidelines, antiviral treatment is recommended for individuals with influenza with the following criteria: hospitalization for influenza, severe out-patient influenza infection or at high risk for complications, pregnancy, individuals less than two weeks postpartum, children under 2 years old, and adults over 65 years old.⁹ Oseltamivir is most commonly used for the prevention and treatment of influenza and is effective in hospitalized patients 4-5 days after the initiation of symptoms.¹⁰ In adults with influenza A or B, it is given as a 75 mg dose twice daily for five days. For individuals with renal dysfunction, the dosages are adjusted appropriately; 30 mg twice daily is given to individuals with a creatinine clearance of 30-60 mL/min, 30 mg daily is given to individuals with a creatinine clearance of less than 30 mL/min, and 30 mg are given after dialysis for those who require hemodialysis.¹⁰

The IDSA recommends increased clinical suspicion for bacterial coinfection with influenza in individuals with a severe disease process (*i.e.*, respiratory failure, hypotension, and fever), individuals who do not respond to treatment within the first three to five days, and individuals whose health further declines after initiation of treatment. In these cases, the American Thoracic Society (ATS)-IDSA recommends providers to work up for possible bacterial coinfection when the aforementioned criteria are met and MRSA risk factors are present, suggesting empiric treatment with vancomycin or linezolid.^{9,11} However, the IDSA does not recommend standardized prophylactic antibiotic use with MRSA coverage in influenza patients due to the accessibility and reliability of MRSA nasal screening for diagnosis.¹¹ They also recommend discontinuing MRSA treatment when testing is negative.¹¹ Additional prior

studies suggest the need for earlier empiric antibiotic treatment for the prevention of MRSA pneumonia complications.⁸

Diagnostic challenges

The IDSA supports the MRSA nasal screening as a tool to rule out suspicion of MRSA superimposed pneumonia, and in the cases of positive screenings, they suggest blood and sputum samples to also confirm the presence of MRSA.¹¹ However, the reliability of the MRSA nasal screening to rule out MRSA may not capture all the individuals affected by MRSA-superimposed pneumonia. In a recent retrospective study that included both culture-based and Polymerase Chain Reaction (PCR) testing, it was noted that four out of every ten individuals with MRSA pneumonia and influenza had a negative MRSA nasal screen, demonstrating that the reliability of these screening tests is not adequate to diagnose the number of individuals infected.³ Another recent retrospective study found the MRSA PCR nasal swab had a sensitivity of 48% and a Negative Predictive Value (NPV) of 97%.¹² The authors highlighted the importance of understanding the disease prevalence at the time of testing to accurately interpret MRSA PCR nasal swab results.¹² Moreover, PCR testing primarily identifies MRSA through the detection of the *MecA* gene; however, false-positive results can arise from the presence of Methicillin-Sensitive *S. aureus* (MSSA) or the SCC cassette that houses the *MecA* gene if it is present.¹³

Culture-based testing in the past was primarily completed in two steps: culturing *S. aureus* for identification and then testing for oxacillin resistance.¹³ However, chromogenic medium has become more prevalent in culture-based samples, allowing a one-step culture to identify *S. aureus* and resistance to oxacillin.¹³ While the earliest detection can result within 24 hours, there is an increased sensitivity when incubating until 48 hours, meaning that there is still a need for a high-sensitivity, rapid test for patients experiencing severe respiratory distress, such as the patient in this case.¹³ Furthermore, the ambiguity of diagnosis given the classification opacification seen on this patient's X-ray demonstrates the importance of having a reliable and rapid test for diagnosis (Figure 1).

Recently developed molecular assays have demonstrated much higher sensitivity and specificity values, although the risk of false-positive results still exists.¹³ For instance, the Xpert MRSA NxG assay has a sensitivity of 92.9% and specificity of 97.6%, com-

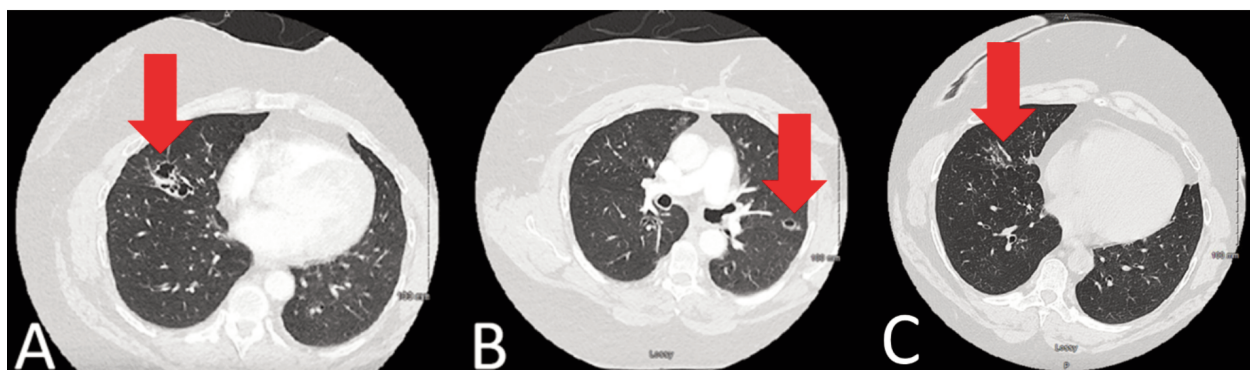


Figure 2. Computed Tomography (CT) pulmonary embolism with angiography and contrast obtained on day nine of admission due to persistent oxygen requirements and prolonged hospital course to assess for pulmonary embolism. Imaging negative for acute pulmonary embolism. Noted **A**) new diagnosis of right middle lobe predominant bronchiectasis; **B**) left upper lobe with small cystic areas near the fissure and with prominent apical ground glass opacities. Repeat CT chest without contrast six weeks following discharge with noted **C**) interval resolution of cystic lung changes with residual scarring and no focal consolidative lung changes or ground glass opacities.

pared to Cobas MRSA/SA assay with a sensitivity of 93.9% and specificity of 99.6%.¹³ Additionally, blood culture assays have proved to be a rapid and comprehensive test, searching for multiple pathogens and various resistance genes. For instance, the BCID Panel tests 24 pathogens and 3 antibiotic genes, with a manufacturer reported *S. aureus* sensitivity of 96% and specificity of 97.6% along with a *MecA* detection sensitivity of 97.6% and specificity of 100%.¹³

The patient in this case received PCR testing via NAAT, a pneumonia panel, and a respiratory culture, all of which reaffirmed a MRSA diagnosis. The rapid availability of the NAAT allowed for a quick diagnosis to be made in a patient who was having respiratory failure, understanding the risk for a false-negative or false-positive. The pneumonia panel and respiratory culture confirmed the presence of MRSA, through understanding of the high sensitivities of these tests. By detecting MRSA early in an individual's hospital course, treatment can be initiated and lower the risk for severe complications or ICU admission.^{5,8} Due to the vast availability of MRSA diagnostic testing, understanding of the unique characteristics of each test, including but not limited to, time, sensitivity, and specificity, allows a care team to minimize resources while adequately interpreting results and considering the clinical context of the patient.

Understanding the role of comorbidities

This patient's prior diagnosis and impacting variable of severe persistent uncontrolled asthma further complicate the case and clinical course. Studies demonstrate that asthma exacerbations in individuals with severe asthma increase the risk for progression to respiratory failure, respiratory acidosis, and intubation, necessitating intensive care.¹⁴ According to the 2024 Global Initiative for Asthma (GINA) guidelines, individuals at high risk for ICU admission include those who have severe exacerbations characterized by drowsiness, confusion, or have an absence of wheezing and breath sounds, also known as silent chest.¹⁵ Moreover, those who fail to improve with therapy, require ventilation or have worsening respiratory distress characterized by hypoxemia or hypercapnia should be considered for ICU admission.¹⁵

Additionally, this patient underwent a diagnostic workup for ABPA. ABPA presents in patients with asthma as poorly controlled asthma and persistent eosinophilia with mucoid impaction, bronchiectasis, or chronic pulmonary aspergillosis on CT imaging.¹⁶ According to the IDSA, last updated in 2016, diagnostic workup for ABPA in a patient with asthma includes CT chest imaging to assess for the criteria as mentioned above along with serum IgE, Aspergillus-specific IgG, and eosinophilia.¹⁶ Management includes oral corticosteroids and antifungal therapy, often itraconazole.¹⁶ While the patient in this case had elevated IgE levels, and some bronchiectasis on imaging as seen on her CT (Figure 2), her sputum fungal cultures were negative. Due to this, she was not treated for ABPA.

It remains uncertain the degree to which this patient's asthma impacted her requiring intubation and ICU admission versus the influenza and MRSA pneumonia as a driving factor. However, this patient had multiple prior exacerbations that did not require intubation or ICU admission, leading to suspicion that influenza with superimposed MRSA pneumonia played a large role.

It is known that asthma is associated with increased susceptibility to respiratory infections and pneumonia, and should be recognized as a risk factor for potential severe illness, however, the pathophysiology by which this happens is not well understood.¹⁷ In studies examining the rapid progression of respiratory infections in mouse models, early elevations of Interleukin-6 (IL-6) and

Tumor Necrosis Factor-Alpha (TNF- α) are thought to contribute to the rapid progression of infection.¹⁸ Additionally, a decreased rise in Interleukin-10 (IL-10) and Interferon-Gamma (IFN- γ) is thought to contribute to inadequate clearance of viruses.¹⁸ This patient's initial symptom presentation, mistaken for an asthma exacerbation, ultimately delayed her diagnosis and treatment, contributing to the severity of her complications.

Management of asthma in the setting of co-infection with influenza and MRSA pneumonia includes not only managing the asthma exacerbation with rapid-acting bronchodilators and corticosteroids but adequately treating the influenza with antivirals and MRSA with linezolid and vancomycin.¹⁹⁻²¹ A study looking at influenza co-infection with MRSA pneumonia in children found that vancomycin monotherapy is insufficient for treatment.²⁰ Rather, vancomycin in combination with linezolid or clindamycin significantly decreased mortality.²⁰ Studies in adults have shown no significant difference in mortality from MRSA infection with combination anti-bacterial therapy compared to monotherapy, but this has not been studied in adults with influenza co-infection.²² In critically ill adults, studies have demonstrated a survival benefit in patients treated with neuraminidase inhibitors, such as oseltamivir.²¹

Emerging pathophysiology

Further complicating our management of influenza with superimposed pneumonia is the lack of a clear understanding of the mechanism of the disease process. One study that investigated the pathophysiology focused on the role of the Nucleotide-Binding Domain-Like Receptor Protein Three (NLRP3) inflammasome on secondary MRSA infections. The NLRP3 inflammasome aids alveolar macrophages in the production of an immune response in both influenza A virus and MRSA.²³ The researchers found that primary infection with influenza A for seven days activated the NLRP3 inflammasome, and the increased expression of the NLRP3 inflammasome increased MRSA infections secondary to influenza but also led to decreased Interleukin-1 β (IL-1 β).²³ The authors suspect that the decreased IL-1 β could potentially impair host immunity, increasing susceptibility to superimposed infections, like MRSA pneumonia, and be a potential therapeutic target.²³ Another study demonstrated that superimposed MRSA infection on influenza could be caused by injury to the lung microenvironment, subsequently causing changes in bacterial toxins and inducing changes in metabolic pathway molecules, such as Leukocidin AB (LukAB), a cytotoxin which subsequently produces further lung injury and inflammation.^{15,17} Studies have demonstrated that the increased inflammation from co-infection of influenza and MRSA is primarily mediated through the lung microenvironment.¹⁷ The injured alveolar lung microenvironment in the presence of influenza infection, regulated by LukAB, produces an environment for increased MRSA virulence.¹⁷

Moreover, this disease process is further exacerbated in individuals with a history of asthma. NLRP3 inflammasome activation has also been found in airway allergen-driven inflammatory responses, such as asthma.²⁴ Current therapeutic uses of the NLRP3 inflammasome nonspecifically target increased IL-1 β levels through an antagonistic mechanism, treating human autoimmune diseases. One study demonstrated the combination of intranasal curcumin and dexamethasone decreased inflammatory cell recruitment in the lungs, particularly by influencing the NLRP3 inflammasome pathway.¹⁹ The development of site-specific NLRP3 therapeutics, particularly for the lung, could contribute to decreased asthma exacerbations and potentially reduce susceptibility to infection.^{19,24}

Conclusions

Enhancing our understanding of the pathophysiology behind superimposed MRSA infection and influenza in the setting of asthma would allow for the potential development of more targeted therapies to prevent severe complications, even with early empirical treatment. Even with influenza treatment initiated upon hospitalization as seen in this patient, MRSA pneumonia remains a challenging complication. Although the MRSA nares test may not reliably detect all cases of MRSA, understanding its limitations and incorporating additional diagnostic tools, such as sputum cultures or pneumonia panels, is essential for accurate diagnosis in cases of high clinical suspicion for superimposed pneumonia. Clinicians should consider initiating empiric MRSA antibiotic treatment early in patients who meet IDSA criteria for antibiotic treatment, including respiratory failure, failure to respond to treatment, or worsening of symptoms with treatment. Empiric MRSA antibiotic coverage should also be considered prophylactically in high-risk individuals with comorbidities, such as asthma, or hospitalizations where the risk of superimposed bacterial infections is increased. Carefully monitoring respiratory symptoms, identifying worsening pulmonary function early, and initiating treatment promptly could help reduce instances of rapid progression to respiratory failure, intubation, and ICU admission, and improve overall patient outcomes. Future studies that focus on risk stratification for patients with suspected MRSA pneumonia, selecting appropriate diagnostic modalities within the clinical context, or the development of targeted therapies through the NLRP3 inflammasome pathway can contribute to more personalized approaches for patients with complex infections.

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