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CLINICAL VIGNETTE

Clinical decisions and consequence in critical care: To intubate or not to intubate ... and use NIV instead

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Case Presentation

A 53-year-old man presented in the middle of the night to the Emergency Department (ED) with 5 days of progressive dyspnea. He reported initial symptoms of malaise, fever, and chills, followed by cough, progressive dyspnea, mild hemoptysis, emesis, diarrhea, and myalgias. The patient's daughter experienced symptoms of fever and cough one week prior but recovered within three days.

The patient was previously prescribed azithromycin and over-the-counter cold medications without improvement. Of note, he had not received Influenza vaccine.

On the morning of presentation, he developed small volume hemoptysis, estimated at 15 to 20 mL of blood mixed with sputum expectorated over 24 hours. On arrival to the ED, he was tachypnic with increased work of breathing and was started on non-invasive, bi-level positive airway pressure (henceforth referred to as non-invasive ventilation – NIV, commonly referred to as BiPAP) at 14/5 cm H₂O with immediate relief of dyspnea. He was admitted to the ICU.

Additional history included possible exposure to bat feces while cleaning an attic. The patient smoked cigarettes “socially” in his youth and denies alcohol use. No history of IV drug use or HIV risk factors. He had no significant occupational exposures to pulmonary toxins.

Past medical history included elevated lipids, lumbar spondylosis, and BPH, as well as prior hernia repair. Family history was non-contributory.

Physical exam in the ICU revealed a calm, cooperative male, mildly tachypnic to the high teens on NIV but otherwise without distress. Lung exam was notable for diminished breath sounds at bilateral bases, worse on the right than left. The exam was otherwise unremarkable.

Labs were remarkable for leukopenia (1.96×10^3), thrombocytopenia (87×10^3 K), and an acutely elevated creatinine of 1.6 mg/dL. Arterial blood gas with the patient on NIV 14/5 cm H₂O showed pH 7.41, pCO₂ 38 mm Hg, and pO₂ 110 mm Hg on FiO₂ 1 (100%).

Chest X-ray showed right lower lobe consolidation with associated volume loss (suggesting an element of mucous impaction and distal atelectasis).

The patient was admitted to respiratory isolation in the ICU and treated with oseltamivir and broad spectrum antibiotics pending results of viral PCR, bacterial and fungal cultures, and fungal serologies.

He was observed overnight on NIV with plans for intubation if there was worsening hemoptysis or further clinical deterioration.

The morning after admission to the ICU, the patient's Influenza PCR returned POSITIVE. The ICU team discussed the likelihood of worsening pneumonia and recommended semi-elective intubation to the patient and family. *The patient who was alert and capable of decision making declined intubation in part because his primary symptom of dyspnea improved with NIV treatment.* Over the next 24 hours, the patient remained persistently febrile with progressive hypoxemia despite increasing levels of NIV support (18/10 cm H₂O with 100% oxygen). The patient's respiratory rate on NIV was recorded as “20s to 30s.”

On the evening of the second hospital day, at approximately 18:30, just prior to shift change, the patient complained of worsening shortness of breath and fatigue despite NIV and requested intubation.

The patient was difficult to sedate, requiring high doses and multiple rounds of sedatives and paralytics. The intubation was difficult due to the presence of bloody secretions and was complicated by severe oxygen desaturations that required prolonged manual bagging and hypotension. The patient was successfully intubated on the 4th attempt. Arterial and central venous catheters were emergently placed for hemodynamic monitoring and administration of pressors.

The patient's subsequent hospital course was characterized by severe acute respiratory distress syndrome (ARDS), requiring salvage oxygenation techniques such as inverse ratio pressure controlled and prone ventilation, as well as septic shock with multi-organ system failure that required multiple pressors and continuous renal replacement therapy. Complications included ICU delirium, critical illness myopathy, and hospital-acquired

ventilator associated pneumonia with *Acinetobacter* spp. The patient eventually underwent tracheostomy tube placement for prolonged respiratory failure and gastrostomy tube placement for nutrition.

The patient was eventually stabilized and was discharged home on hospital day 75. His mental status improved though he had persistent anxiety and memory deficits. The tracheostomy tube was de-cannulated, and the G-tube was removed prior to discharge home.

Discussion

Modern critical care medicine is equipped with an impressive arsenal of medical technology, which can be applied to save lives of profoundly ill patients. Our case also demonstrates the limitations in our understanding of how and when to best apply our tools. Many of the advances in critical care medicine are related to limiting harm in our efforts to heal – decreasing medical device related infections and ventilator induced lung injury, judicious use of sedation with daily interruptions, and promoting sleep and early mobilization in even the most critically ill patients. Treatment bundles, checklists, and protocol-driven care have helped increase adoption of proven best practices; however, there remain gaps in how best to implement and sustain quality improvement.¹⁻⁵

The data supporting use of NIV is primarily in acute exacerbations of chronic heart failure and acute exacerbations of COPD with limited data in acute pneumonia in immunocompromised patients.⁶⁻⁸

In our case, the decision to start NIV acutely was related to its rapid availability, the relative ease of application, prompt symptomatic improvement, and the possibility of a bias that “non invasive” is equivalent to “benign.” Recognizing NIV failure or detecting the presence of contraindications early and transitioning to invasive mechanical ventilation is a key issue underlying NIV management. While successful application of NIV can be associated with improved survival, in the presence of predictors of NIV failure, delayed intubation results in increased mortality, particularly in patients with hypoxemic and “de novo” acute respiratory failure.⁹ The extended use of continuous NIV in this case resulted in a delayed and very difficult intubation. The patient that fails NIV should be regarded as an inherently difficult intubation. The clinician should be prepared for acutely worsening hypoxemia and hypercapnia due to rapid alveolar de-recruitment and hemodynamic instability related to dynamic changes in cardiac output, volume status, and the effects of sedation.

As clinicians, it is easy to assume the “sleeping patient” on NIV with normal oxygenation and vital signs is well compensated. One must recognize some of the limitations of NIV in comparison to invasive mechanical ventilation including, but not limited to, decreased ability to perform effective airway clearance while applying NIV, inability to reliably monitor end tidal CO₂, less reliable ventilation due to mask leaks and quantification of effective ventilation, and the need to account for upper airway resistance when applying a

“pressure controlled” ventilation mode. These factors are dynamic, especially in the critically ill individual where airway clearance, atelectasis, neuromuscular strength, and sleep may all affect the ability to reliably ventilate the patient with a non-invasive strategy. Simply said, most patients on BiPAP are usually sicker than they look.

Widely accepted protocols for the application, titration, and discontinuation of NIV are lacking despite its increasing clinical use. Until best practices are clearly established and employed, clinicians treating acutely ill patients with advanced ventilator support, including NIV, must not only know the indications, contraindications, and limitations of NIV but also seek expert consultation and be prepared to promptly transition to an invasive strategy in the event of NIV failure.

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