

# Metoclopramide and Bronchospasm in Patients on the Mechanical Ventilation: A Rare Case Report

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Received January 04, 2021; Revised January 25, 2021; Accepted February 02, 2021

**Abstract** This case report aims to draw attention to the possible occurrence of metoclopramide-induced bronchospasm. We report a case of bronchospasm reaction to metoclopramide in a mechanically ventilated patient treated for cardiogenic shock. Metoclopramide can cause severe adverse events, such as bronchospasm, and should be used with caution in patients on mechanical ventilation.

**Keywords:** metoclopramide, bronchospasm

**Cite This Article:** Eman EL-Sawalhy, Muna Shaaeli, and Essa Kadiri, "Metoclopramide and Bronchospasm in Patients on the Mechanical Ventilation: A Rare Case Report." *American Journal of Medical Case Reports*, vol. 9, no. 4 (2021): 236-237. doi: 10.12691/ajmcr-9-4-8.

## 1. Introduction

Initially approved in 1979, metoclopramide has been used to treat a variety of gastrointestinal disorders. It is a dopamine-2 receptor antagonist that acts on the chemoreceptor trigger zone in the central nervous system, which in turn prevents nausea and vomiting triggered by most stimuli [1,2]. Bronchospasms have been demonstrated as a side effect of several medications. Multiple pathways have been postulated, most importantly, cholinergic pathways influencing airway reactivity [3,4]. The respiratory tree is supplied by parasympathetic postganglionic nerves and the terminals are largely cholinergic. Metoclopramide can release acetylcholine from postganglionic cholinergic nerve terminals, potentially precipitating bronchospasms. A literature search demonstrated a case of metoclopramide associated with bronchospasm in an asthmatic patient [5], however to our knowledge no cases have been reported on bronchospasm in patients who have no history of reactive airway disease.

## 2. Case Presentation

A 74-year-old African American woman with type 2 diabetes, hypertension, paroxysmal atrial fibrillation, and morbid obesity presented to the hospital with a 2-day history of progressively worsening fatigue and confusion after being found on the floor by her daughter. In the emergency department, she was noted to be altered, hypoxic with a SpO<sub>2</sub> of 84%, bradycardia with a heart rate in the 40s, and melena/bright red blood noted on her bed linen after a bowel movement. An EKG demonstrated atrial fibrillation with a slow ventricular rate. Urinalysis was suspicious for a urinary tract infection which later

cultures confirmed to be E. Coli. Leukocytosis of 15.2 thous/uL was noted to be predominantly neutrophilic and acute kidney injury with a Cr 1.70 (up from a normal baseline). COVID-19 testing was negative. Chest x-ray demonstrated right basilar opacity and bilateral congestion with small bilateral effusions. CT of the head was unremarkable with no evidence of hemorrhage or midline shift. She was initiated on oxygen therapy via nasal cannula and AVAPs, antibiotics with Vancomycin/ Zosyn, a dose of atropine was given with improvement of heart rate and discontinuation of her Lopressor and Xarelto. Cardiology, electrophysiology, infectious disease, and general surgery were consulted.

Over the next few days, her mentation and respiratory status improved. She underwent an EGD which showed gastritis and refused a colonoscopy. Cardiology was planning to place a pacemaker, however, on day six, she developed respiratory distress and altered mentation which led to intubation. She was subsequently transferred to the ICU for closer monitoring. On day 2 of intubation, she was started on Metoclopramide 5 mg every 6 hours for gastroparesis. On day 3 of metoclopramide and day 5 of intubation, lung auscultation revealed severe wheezing associated with ventilator asynchrony which did not improve with inhaled bronchodilator therapy or ventilator setting adjustments. Paralysis with Cisatracurium led to the resolution of wheezing and synchronization with mechanical ventilation. A CT scan of the chest demonstrated a moderate right-sided pleural effusion. A thoracentesis was performed and 400 cc of transudative fluid was removed. Multiple attempts to wean Cisatracurium led to recurrent severe wheezing and ventilator asynchrony. Hospital medications were postulated as a possible etiology and after a thorough review, it was noted that Metoclopramide's insert label documented side effects of bronchospasm, laryngospasm, laryngeal edema, and pulmonary edema which can occur within 24-48 hours after initiation of

therapy. Metoclopramide was discontinued and after 24 hours, paralytic weaning was successful without recurrence of wheezing or ventilator asynchrony.

Unfortunately, despite successful weaning, extubation was not achieved due to a lack of improvement in mental status while off of sedation. Imaging, as well as neurology consultation, were unrevealing and after approximately 3 weeks, per family wishes and goals of care, comfort measures were pursued.

### 3. Discussion

Metoclopramide belongs to a class of drugs called prokinetic agents and has been approved as first-line therapy for gastroparesis. It acts both centrally as an antiemetic and peripherally by accelerating gastric emptying. The exact mechanism has not been fully established, however, it appears to interact with central dopaminergic receptors and sensitize peripheral tissues to the actions of acetylcholine, resulting in stimulation of upper GI motility.

Adverse reactions can occur within 24-48 hours and have been reported more in women than in males and more in nondiabetic patients than diabetic patients [1,2,3,4,5,6]. In 2009 a black box warning was added to its label due to an adverse reaction of tardive dyskinesia. Rare adverse reactions including dystonia due to laryngospasms resulting in stridor and dyspnea, cardiovascular side effects like acute congestive heart failure, possible AV block, bradycardia, hypotension, hypertension, fluid retention by a transient increase in plasma aldosterone have been reported [1,2,3,4,5,6,7]. In our case, our patient developed a bronchospastic reaction 3 days after initiation of metoclopramide therapy, resulting in severe wheezing on auscultation and ventilatory asynchrony which was unresponsive to bronchodilator therapy and only improved with paralytic therapy. Unfortunately, recurring upon weaning attempts of paralytic therapy and only resolving after discontinuation of metoclopramide therapy. The bronchospastic reaction of metoclopramide can be explained by its ability to release acetylcholine from the

postganglionic cholinergic nerve terminals [5]. This case illustrates the necessity to use caution when prescribing metoclopramide, especially in patients with reactive airway disease. Further clinical observations and studies are needed to confirm the bronchospastic effect of the metoclopramide.

### 4. Conclusion

Metoclopramide is not a benign medication and can cause rare, but potentially life-threatening adverse reactions, including bronchospasm, laryngospasm, and laryngeal edema. Caution and close attention should be observed when prescribing to patients on mechanical ventilation and/or with reactive airway disease.

### Conflict of Interest

None of the authors have any conflicts of interest to declare.

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