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Etomidate use in septic patients requiring rapid sequence intubation

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Disclosure Statement

The listed individuals have the following to disclose regarding financial or personal relationships with commercial entities (or their competitors) that may be referenced in this presentation:

- Elizabeth Osmon, Pharm.D. – Nothing to disclose
- Nishika Patel, Pharm.D., BCPS, BCCCP – Nothing to disclose

Boca Raton Regional Hospital

- Not-for-profit 400 bed advanced academic tertiary medical center
- **Recognized leader in:**
  - Cardiovascular Care
  - Oncology
  - Women's Health
  - Orthopedics
  - Emergency Medicine
  - Neurosciences
- Predominantly elderly patient population
- Highest ranked hospital in Palm Beach County
  - Listed by U.S. News & World Report 2019-2020
- Lynn Cancer Institute is one of the largest cancer programs in the state of Florida and accredited by the American College of Surgeons

Presentation Objective

Identify the effect etomidate has on cortisol production

Background

During sepsis, pro-inflammatory markers stimulate the upregulation of cortisol release

An increase in cortisol production results in metabolic, cardiovascular, and anti-inflammatory benefits in order to maintain homeostasis during stress

A disruption in this mechanism causes primary adrenal insufficiency and a lack of adequate stress response

Prigent H, Maxime V, and Annane D. Science review: Mechanism of impaired adrenal function in sepsis and molecular action of glucocorticoids. *Crit Care* 2004;8:243-252.

Background

- Etomidate is a short-acting, sedative hypnotic that is often used as an induction agent for rapid sequence intubation (RSI)[1]
- Etomidate inhibits the enzyme 11-β hydroxylase, which is responsible for the conversion of 11-deoxycortisol to cortisol[2]
- Reduced plasma cortisol levels have been reported with a typical induction dose (0.3 mg/kg) of etomidate[1]
- Although the role of etomidate in adrenal suppression has been established, the clinical consequences of this mechanism are controversial[2]

1. Amidate (etomidate) [package insert]. Lake Forest, IL: Hospira, Inc.;2017.
2. Thompson Baastin ML, Baker SN, and Weant KA. Effects of etomidate on adrenal suppression: A review of intubated septic patients. *Hosp Pharm* 2014;49(2):177-183.

Literature Evaluation

Study	Design	Arms	Results
Ray, et al.[1] 2007 n = 159	Single-center, retrospective	Etomidate vs. other induction agents	Induction agent did not affect duration (P = 0.54) or dose (P = 0.53) of vasopressor therapy
Elliot, et al.[2] 2012 n = 50	Single-center, retrospective	Etomidate vs. other induction agents	No difference in the mean dose of vasopressor in norepinephrine equivalents (P = 0.61)
Alday, et al.[3] 2014 n = 411	Multicenter, retrospective	Etomidate vs. other induction agents	No difference in need for vasopressor support with etomidate vs. non-etomidate (P = 0.88)

1. Ray DC, et al. Effect of etomidate agent on vasopressor and steroid use, and outcome in patients with septic shock. *Crit Care*. 2007;11:R56.
2. Elliot M, et al. Does etomidate increase vasopressor requirements in patients needing mechanical ventilation? *Can J Hosp Pharm*. 2012;65(4):272-276.
3. Alday NJ, et al. Effects of etomidate on vasopressor use in patients with sepsis or severe sepsis: a propensity-matched analysis. *J Crit Care*. 2014;29(4):517-22.

Purpose

To analyze if etomidate exhibits a dose dependent effect on the duration of intravenous (IV) vasopressor support and other clinical outcomes in septic patients

Study Outcomes

Primary outcome

- Duration of IV vasopressor support between low dose (≤ 0.3 mg/kg) and high dose (> 0.3 mg/kg) etomidate

Secondary outcomes

- Number of patients requiring initiation of stress dose steroids
- Intensive care length of stay
- Duration of mechanical ventilation
- Inpatient mortality

Study Design

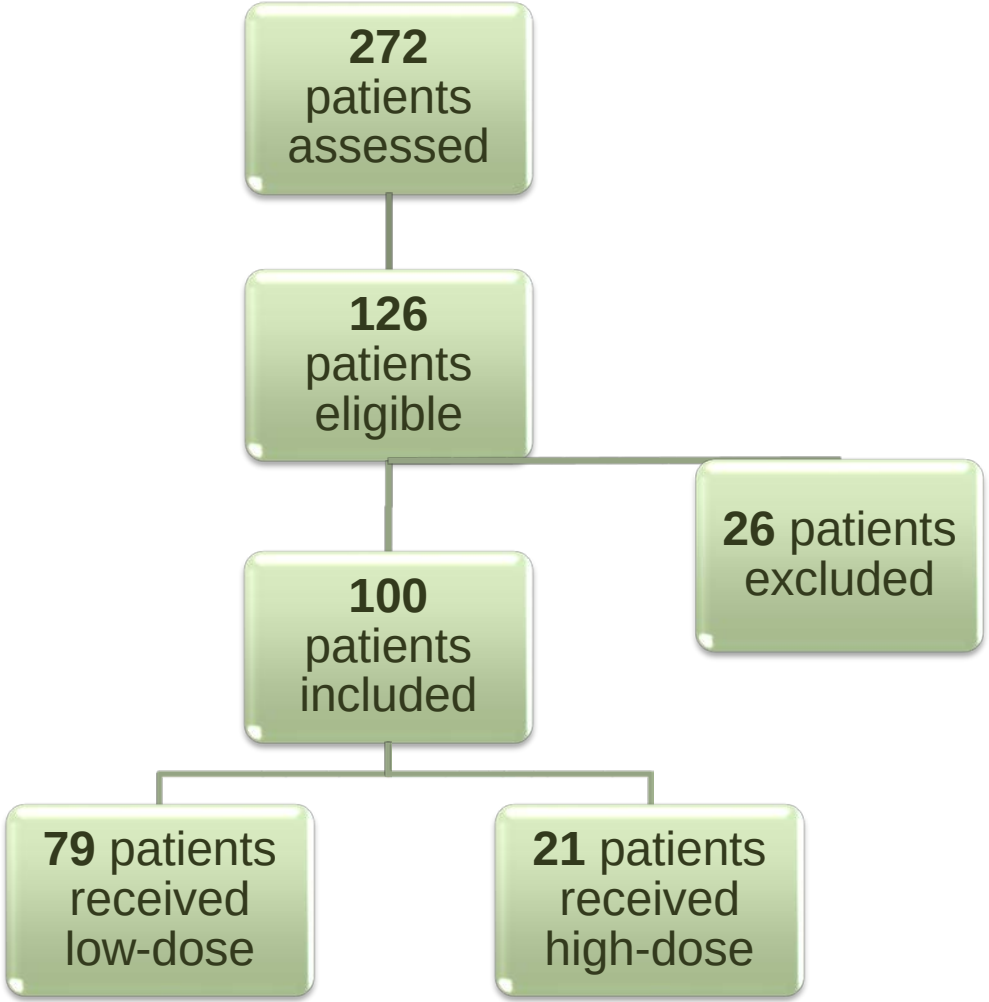
Methods: Retrospective chart review using an electronic medical record (EMR)-generated report from October 21, 2017 to December 31, 2019

Inclusion Criteria	Exclusion Criteria
• Age ≥ 18 years • Differential diagnosis of sepsis or septic shock based on provider documentation • Received etomidate as an induction agent for RSI	• History of an adrenal disorder • Taking medications that directly impact adrenal function prior to admission

Statistical Analysis

Primary Outcome	• Mann-Whitney U test
Secondary Outcomes	• Descriptive statistics

Screening

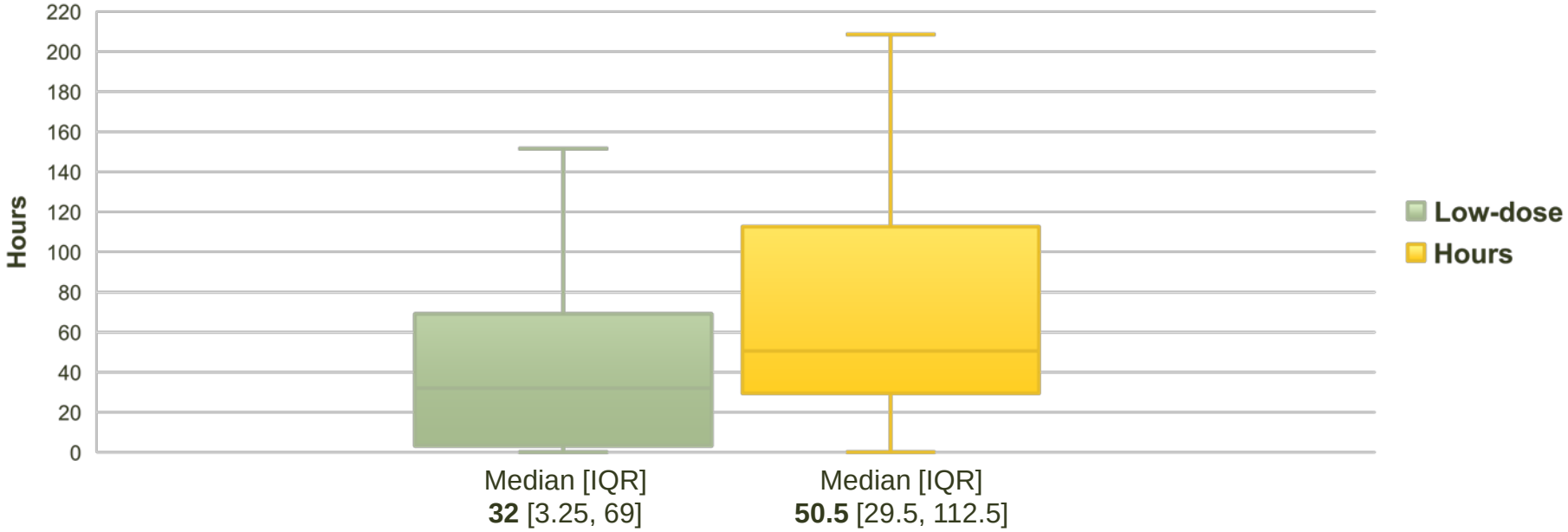

272 patients assessed
126 patients eligible
26 patients excluded
100 patients included
79 patients received low-dose
21 patients received high-dose

Baseline Characteristics

Variable	Low-Dose (n = 79)	High-Dose (n = 21)
Age – years		
Median	77	81
Interquartile range	63 – 86.5	75 – 87
Gender – no. (%)		
Female	31 (39.2)	14 (66.7)
Weight – kg		
Median	77	49.7
Interquartile range	67.9 – 90	45.4 – 57

Results

Primary outcome: median duration of IV vasopressor support was 32 hours vs. 50.5 hours, **P = 0.0455**



Results

Secondary Outcomes	Low-Dose (n = 79)	High-Dose (n = 21)
Initiated on stress dose steroids – no. (%)	24 (30.4)	6 (28.6)
Intensive care length of stay – days		
Median	5	8
Interquartile range	3 – 12	5 – 10
Duration of mechanical ventilation – days		
Median	4	5
Interquartile range	2 – 9.5	4 – 8
Inpatient mortality – no. (%)	27 (34.2)	5 (23.8)

Conclusion

- There was a **statistically significant difference** in duration of IV vasopressor support between the low-dose and high-dose groups
- Secondary outcome results were similar between the low-dose and high-dose groups
- Despite the small sample size, the significant results of this study warrant the need for a randomized controlled trial to be conducted

Limitations

- Inconsistent use of sepsis-3 criteria among providers
- Limited provider documentation
- Oral vasopressor support (i.e. midodrine) was not evaluated
- Small sample size
- Unequal treatment group sizes

Acknowledgment

Nishika Patel, Pharm.D., BCPS, BCCCP

Self-Assessment Question

Which of the following correctly describes the effect etomidate has on cortisol production?

A. Etomidate directly binds to cortisol, which makes it inactive.

B. Etomidate inhibits the enzyme 11-β hydroxylase, which is responsible for conversion of 11-deoxycortisol to cortisol.

C. Etomidate regulates cortisol production through a negative feedback mechanism.

D. Etomidate does not affect cortisol production.

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