

ASSESSMENT OF ETOMIDATE-ASSOCIATED MYOCLONUS FOR PROCEDURAL SEDATION IN A TERTIARY CARE EMERGENCY DEPARTMENT

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Abstract

Background: Etomidate is a frequently employed drug for procedural sedation within the emergency department (ED), often associated with a reported myoclonus incidence rate of 33%. This study seeks to challenge the prevailing notion that etomidate-induced myoclonus holds lesser significance than the reported frequency. **Methods:** Conducted prospectively for six months at a tertiary care hospital, this investigation focused on procedural sedation in the ED, overseen by physicians. Adult patients undergoing procedural sedation with etomidate were recruited for participation. **Results:** Among the 212 individuals (148 males and 64 females) enrolled in the study for procedural sedation with etomidate (administered at a dose of 0.3 mg/kg), myoclonus was observed in only 6 cases (2.83%). The myoclonus scale was employed to note its presence and duration. The mean age was determined for both male and female participants. Contrary to reported values, the incidence of etomidate-induced myoclonus during procedural sedation in the ED was found to be less significant. **Conclusion:** This study concludes that the occurrence of myoclonus following etomidate administration is lower when compared to findings from other ED studies.

INTRODUCTION

In emergency settings, a range of medications have been employed to achieve procedural sedation in patients, among which etomidate stands out as the preferred choice for its efficacy as a sedative-hypnotic agent. Etomidate, classified as a carboxylated imidazole, exerts its depressant effects on the central nervous system by modulating gamma-aminobutyric acid activity [1-4]. Characterized by rapid onset, minimal cardiovascular risk, negligible respiratory depression, and consistent sedative properties, etomidate emerges as the prime candidate for procedural sedation within the emergency department (ED). Additionally, etomidate demonstrates neuroprotective effects in cerebral and myocardial ischemia scenarios, exhibits a straightforward dosing regimen, induces minimal respiratory depression, and attenuates histamine release, rendering it suitable as an induction agent for hemodynamically unstable patients [5-7]. Furthermore, in individuals with traumatic brain injury, etomidate effectively lowers intracranial pressure while preserving normal arterial pressure. Etomidate exhibits high plasma protein binding and undergoes hepatic and plasma esterase metabolism [8,9].

Common adverse reactions associated with etomidate administration include myoclonus and adrenal suppression, with additional occurrences of nausea, vomiting, and injection site discomfort [10]. Myoclonus induced by etomidate during both anesthesia and emergency department sedation has been documented in the literature, with a reported incidence of approximately 33% at ED sedation dosages [11]. Our investigation aims to ascertain whether the incidence of etomidate-induced myoclonus in patients is lower than the reported percentage.

MATERIALS AND METHODS

This prospective study spanned six months at a tertiary care hospital. Patients undergoing sedation in the ED with etomidate were enrolled in the study. The procedural sedation in our ED was overseen by the attending emergency physician. During the procedure, patients' cardiac function, pulse oximetry, blood pressure, and end-tidal carbon dioxide levels were continuously monitored in accordance with ED guidelines. Data collection and organization were carried out using Microsoft Excel. Patient

demographics, etomidate dosage, and the presence of myoclonus were documented and tabulated.

All patients undergoing procedural sedation with etomidate were included in the study, with exclusion criteria applied to pregnant individuals, those with neuromuscular disorders, individuals with a history of adverse reactions to etomidate, and those unable to provide informed consent.

Patients who developed myoclonus following etomidate administration during procedural sedation in the ED were assessed for the severity of myoclonus using a predefined scale. Vital signs including cardiac function, blood pressure, and pulse oximetry were monitored per ED guidelines.

The percentage of patients was computed with respect to various variables such as age, gender distribution, total mean dose of etomidate, severity of

myoclonus, and time to onset of myoclonus after etomidate administration. Statistical significance was determined using P-values.

RESULTS

In our investigation involving 212 patients (comprising 148 males and 64 females), 6 patients (2 males and 4 females) exhibited myoclonus following the administration of etomidate during procedural sedation in the ED (refer to Table 1 and Table 3). The calculated P-value for this observation was 0.039, indicating statistical significance at a threshold of <0.05. Table 2 delineates the Myoclonus scale employed for assessing the severity of myoclonus.

Table 1: Demographic parameters of study patients

Parameters	n	%
Cases	212	100.00
Procedural sedation	212	100.00
Age in years (mean ± SD)	43.26 ± 7.56	
Males	148	69.81
Females	64	30.19
Weight in kg (mean ± SD)	68.47 ± 8.54	
Height in inches (mean ± SD)	57.32 ± 5.67	

Table 2: Myoclonus scale utilized for assessment of the degree of myoclonus

Scale	Degree of Myoclonus	Description
0	None	No myoclonus
1	Mild	Minor tremors/One extremity affected
2	Moderate	Myoclonus in 2-3 extremities
3	Severe	All extremities affected or myoclonus requiring extremity stabilization or premature stoppage of procedure

Table 3: Details of etomidate-associated myoclonus

Variables	Value
Mean Initial dose of etomidate; mg/kg	0.14
Mean total dose of etomidate; mg/kg	0.16
Total number experiencing myoclonus	6 (2.83%)
Mild	3
Moderate	2
Severe	1
Time till onset of myoclonus following etomidate; seconds; mean ± SD	50.12 ± 2.45
Duration of myoclonus; seconds; mean ± SD	94.59 ± 5.14

DISCUSSION

Yates et al. [1] reported that approximately 75% of patients developed myoclonus. Conversely, in our investigation, a mere 2.83% (6 out of 212 patients) experienced myoclonus following etomidate administration during procedural sedation in the ED, with manifestations predominantly of a minor nature. The intensity of myoclonus was evaluated utilizing a scale outlined in Table 2. Instances of mild-to-moderate myoclonus observed in the ED during sedation were notably fewer in comparison to findings from prior studies.

Pretreatment with lidocaine, midazolam, dexmedetomidine, among others, has been demonstrated to decrease both the incidence and severity of etomidate-induced myoclonus [12-15]. Numerous studies by Doenicke et al. [16] and

Stockham et al. [17] have indicated that myoclonus can be mitigated through the administration of premedications such as benzodiazepines or Stockham et al. [17]. Contrarily, in our investigation, we refrained from administering any premedications prior to etomidate administration [18]. Moreover, our study specifically focused on detecting myoclonus, thereby heightening our sensitivity to its occurrence. The reduced incidence of myoclonus, coupled with the procedural sedation's efficacy, suggests that etomidate can indeed serve as a suitable sedative agent for procedural sedation.

Given the lower incidence of etomidate-induced myoclonus observed in our study compared to previous research, it can be inferred that etomidate holds promise as a sedative agent for procedural sedations in the ED. Our investigation was conducted to demonstrate the diminished occurrence of

etomidate-induced myoclonus in comparison to previous studies.

CONCLUSION

Based on our findings, we conclude that the incidence of myoclonus occurring with the administration of etomidate is lower compared to that reported in other studies conducted in the emergency department.

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