

LETTERS



SEVERE MYOCLONUS PRIOR TO ELECTROCONVULSIVE THERAPY FOLLOWING INTRAVENOUS ETOMIDATE

To the editor:

Electroconvulsive therapy (ECT) is a short and simple procedure, which has been used in psychiatry since 1938, and it remains the “gold standard” for the treatment of severe and/or medication-resistant depression.¹ The acute phase of ECT is typically administered 3 times a week (for a total of 6-12 treatments) followed by the chronic (maintenance) phase performed at progressively increasing intervals (from once a week to once a month) designed to prevent relapses of depression. The efficacy of ECT in the treatment of depression is highly dependent on the duration of an electrical current-induced generalized (motor) seizures with the optimal anti-depressant response achieved with electroencephalographic seizure activity lasting 25 to 50 seconds.²

Although for nearly 30 years ECT was performed without anesthesia,³ the use of rapid onset and short duration intravenous induction agents (eg, etomidate or propofol) followed by the depolarizing muscle relaxant (succinylcholine) facilitates the ECT procedure.² When compared with propofol and thiopental, induction of anesthesia with

intravenous etomidate (0.15-0.3 mg/kg) is generally associated with longer seizure duration, and consequently etomidate remains a popular induction agent for ECT in the United States. To minimize pain on injection, common with intravenous administration of both etomidate and propofol, lidocaine (eg, 1-3 mL) is typically injected into the intravenous catheter immediately prior to either induction agent.² Other adverse effects of etomidate include nausea, vomiting, and transient skeletal muscle movements (myoclonus).

In their practice of anesthesia, the authors of this report encountered a 69-year-old, otherwise healthy, male patient with severe medication-resistant depression scheduled for chronic phase ECT. The patient had received intravenous etomidate for ECT on multiple occasions (over several years) without any problems/adverse reactions (including myoclonus). However, following what appeared to be “routine” administration of 10 mg of intravenous etomidate (etomidate injection, 2 mg/mL, Bedford Laboratories, Bedford, Ohio), the patient developed severe myoclonus lasting approximately 10 minutes (vital signs remained stable and oxygenation via the face mask was easily maintained) and necessitating interruption of induction of anesthesia and delay in the scheduled procedure. Uneventful anesthesia for ECT was induced approximately 45 minutes later with a standard dose of intravenous propofol.

To the best of our knowledge, such a severe prolonged myoclonus complicating intravenous readministration of etomidate for induction of anesthesia for ECT has not been previously reported. We conclude that in the practice of anesthesia, great vigilance and careful observation is required even when routine “gold standard” procedures/medications with good record of safety and efficacy are performed/administered.

REFERENCES

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Krzysztof M. Kuczkowski, MD

Assistant Clinical Professor of Anesthesiology and Reproductive Medicine
Director of Obstetric Anesthesia
Departments of Anesthesiology and Reproductive Medicine
University of California San Diego
San Diego, California

Brian H. Hastings, CRNA, MSN

Staff Certified Registered Nurse Anesthetist
Department of Anesthesiology
University of California San Diego
San Diego, California