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Anesthesia for Electroconvulsive Therapy: A Case Report

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ABSTRACT

Background: Electroconvulsive therapy (ECT) is one of the therapeutic options in the field of psychiatry. ECT causes several conditions such as stimulation of the general autonomic nervous system, increased intracranial pressure and conditions such as headaches, myalgias, swelling, weakness, nausea and even musculoskeletal complications. Anesthetic management is required to prevent this complication in addition to its primary goal of providing total unconsciousness. The choice of anesthetic agent is chosen based on its rapid onset and duration and not interfering with the patient's seizure activity.
Case Report: We present the case of a 24-year-old female who is diagnosed with schizophrenia hebephrenic. She scheduled for ECT because her symptoms were worsening over time and did not respond to oral medications. Anesthesia is given during ECT. As premedication used atropine sulfate 0.5 mg, then induced with propofol 100 mg and pethidine 50 mg. We did not use neuromuscular blocking agents and only managed ventilation patients with Jackson-Rees circuit. After multiple ECT treatment, we observed no significant clinical effect on seizure duration or hemodynamic profile. The patient's clinical and functional status also significantly improved with ECT treatment.
Conclusions: Anesthesia during ECT creates a safe and comfortable environment for generalized epileptic seizures to occur. Multiple anesthetic agents are acceptable for use during ECT. Although the anesthesia given in this case has several limitations, it still produces good clinical outcomes and minimal side effects.

Keywords: Electroconvulsive Therapy, Anesthesia, Propofol

BACKGROUND

Electroconvulsive therapy (ECT) is one of the therapeutic options in the field of psychiatry for patients with schizophrenia, catatonia, mania, psychotic depression and, especially, drug-resistant depression. This technique uses an electric current to induce epileptic seizures for therapeutic purposes [1]. 70 to 120 volts of pulsed electricity are applied unilaterally or bilaterally to elicit a seizure [2].

ECT causes generalized autonomic nervous system stimulation, initially producing bradycardia induced by parasympathetic nerve stimulation, followed immediately by more prominent sympathetic stimulation that results in transient tachycardia and hypertension. On cerebral the effects include cerebral oxygen consumption, blood flow, and intracranial pressure all increase [3]. This condition causes symptoms of headache, myalgia, drowsiness, weakness, and nausea. Increased salivation can occur, as can dental damage and oral cavity lacerations [4].

During ECT, various anesthetics, including methohexital, sodium thiopental, propofol, etomidate, ketamine, and benzodiazepines, have been used either to induce anesthesia or as premedication [5]. Some of the anesthetic agent requirements for ECT include control of these and related hemodynamic changes and complications,

along with major requirements amnesia and muscle relaxation. Despite this, the goal of treatment cannot be such a deep level of anesthesia as to overly suppress seizure activity [4,5].

CASE REPORT

The patient was a 24-year-old female who was diagnosed with schizophrenia hebephrenic. Before presenting to the hospital, the patient displayed symptoms of hallucinations, delusions, severely disturbed thinking and behavior that impairs daily functioning. The patient had her first episode of hallucinations when she was 19 years old. She was then given trifluoperazine/stelazine 5 mg 2 times a day. Her symptoms were worsening over time and did not respond to oral medications. The results of testing for organic neuropsychiatric conditions and drug-related psychosis were negative. She also had no history of significant systemic disease. Then, she was scheduled to have an ECT session after 2 months of admission. MECTA spectrum 5000 was used to perform unilateral ECT sessions. The anesthetic protocol dictated that sessions of electroconvulsive therapy were carried out twice per week.

Prior to the procedure, the patient is pre-anesthetic evaluated, including anamnesis and physical examination. Laboratory tests carried out included a complete blood count, kidney function tests, liver function tests and blood sugar. In addition, a 12-lead ECG examination was also performed on this patient.

The patient was treated with ECT for a total of nine sessions. An intravenous catheter (IV) is placed before the induction of anesthesia. Then, the patient is given 100% oxygen for pre-oxygenation. As premedication used atropine sulfate 0.5 mg. General anesthesia was induced with weight-based propofol and pethidine. We intravenously administered 100 mg of propofol and 50 mg of pethidine for the procedure. The patient's ventilation was managed with Jackson-Rees circuit by the anesthesia provider. In order to prevent muscle relaxants from reaching the affected area, a tourniquet is applied to one of the lower extremities. By allowing personnel to observe the tonic and clonic activity of the lower extremity, this facilitates monitoring of the motor portion of the seizure. The patient is then given a soft bite block to stop the seizure from causing an oral injury. A processed Electroencephalogram (EEG) is used to monitor the electrical stimulus that is given to start the seizure. The psychiatrist then examined the available visual EEG output for evidence of good seizure intensity and generalization.

MECTA settings for the first four ECT sessions were the following: pulse width 1.0 meter per s, frequency 60 hertz, duration 2.0 s, and amplitude 0.8 meters. The fifth session onwards the duration was increased to 3.0 s, while the others remained the same. Patient hemodynamics (blood pressure and pulse) before and after ECT and duration of seizures are shown in Table 1.

Table 1. Summary of the 9 ECT treatments over 29 days with clinical and anesthetic details.

ECT No.	Day No.	Anesthetic	Pre-procedure Blood Pressure	Pre-procedure Pulse	Time of Tonic Seizure	Time of Clonic Seizure	Post-procedure Blood Pressure	Post-procedure Pulse	Post-ECT Condition
1	1	Propofol 100 mg Pethidine 50 mg	110/83 mmHg	72 bpm	3	36	110/54 mmHg	94 bpm	No agitated, sleep, no apnea
2	3	Propofol 100 mg Pethidine 50 mg	108/76 mmHg	93 bpm	5	8	110/52 mmHg	94 bpm	No agitated, sleep, no apnea
3	7	Propofol 100 mg Pethidine 50 mg	103/61 mmHg	68 bpm	2	11	110/60 mmHg	120 bpm	No agitated, sleep, no apnea

Table 1 To Be Continued…									
4	10	Propofol 100 mg Pethidine 50 mg	100/80 mmHg	71 bpm	2	26	110/60 mmHg	125 bpm	No agitated, sleep, no apnea
5	15	Propofol 100 mg Pethidine 50 mg	100/80 mmHg	84 bpm	3	23	110/60 mmHg	100 bpm	No agitated, sleep, no apnea
6	17	Propofol 100 mg Pethidine 50 mg	99/76 mmHg	80 bpm	2	14	150/60 mmHg	110 bpm	No agitated, sleep, no apnea
7	22	Propofol 100 mg Pethidine50 mg	99/73 mmHg	83 bpm	7	16	90/50 mmHg	70 bpm	No agitated, sleep, no apnea
8	25	Propofol 100 mg Pethidine 50 mg	102/74 mmHg	102 bpm	3	19	110/80 mmHg	100 bpm	No agitated, sleep, no apnea
9	29	Propofol 100 mg Pethidine 50 mg	100/80 mmHg	70 bpm	4	18	140/80 mmHg	88 bpm	No agitated, sleep, no apnea

After the seizure ends, the patient is given breathing assistance until she can breathe spontaneously. The patient is then transferred to the recovery room for observation until she regains consciousness.

During ECT, the patient did not experience significant complaints. She felt no pain and was always asleep and relatively calm after the procedure. When compared to pharmacologic therapy alone, the patient's clinical and functional status significantly improved with subsequent ECT treatments. Prior to the initial treatment, she was combative, uncooperative, and unable to communicate. After treatments 4–6, however, she was able to speak in short sentences and express herself. During treatments 8 and 9, she was still able to answer questions, and she was able to participate in social activities.

DISCUSSION

At first, ECT was performed on awake patients. As a result, musculoskeletal complications affect as many as 40% of patients. Providing general anesthesia for ECT is standard and has been the case since the 1950s to improve patient safety and admission [1,5,6]. The goals of anesthetic management during ECT are to: (i) provide fast-track anaesthesia with a minimum of convulsive motor activity to prevent fractures; (ii) provide a complete unconsciousness with minimal anticonvulsant side effects; (iii) prevent hypoxia; and (iv) manage the adverse effects of ECT itself [7].

Physical examination, blood tests and ECG should be performed to make a preoperative assessment [4]. Atropine (0.01 mg/kg) as anticholinergic agent was given to antagonize the initial parasympathetic discharge. It is administered intravenously just before injecting the induction agent. In 1998, a report by Mayur et al. demonstrated that patients who did not receive atropine had significantly lower rate pressure product (RPP) values after all stimulus recordings. Additionally, there were no clinically significant bradyarrhythmias in those not receiving atropine [8]. Although their routine use is still debated, they may be particularly useful in patients with unknown seizure thresholds, as in this case. [5].

The electrical stimulus for ECT usually lasts 2–8 seconds and is followed by seizure activity of 30–60 seconds. As a consequence, anaesthesia needs no longer than 3–5 minutes. In this case, a short acting anaesthetic, propofol, was used, so intubation was not necessary and could have been avoided [7]. The patient should be pre-oxygenated. After induction, ventilation can be gently assisted with a Jackson-Rees

circuit. The limitations of ventilation machines in our hospital and the relatively inexpensive, made us consider using this system.

During ECT, a variety of anesthetics, including methohexital, thiopental sodium, propofol, etomidate, ketamine, and benzodiazepines, have been used to induce anesthesia. An effective induction agent ought to have a short half-life, a rapid onset and recovery, maintain hemodynamic stability, and not affect seizure duration or threshold [3,5]. Therefore, at first, drugs such as propofol, lorazepam, and midazolam (oral and intranasal) which reduce seizures were not given much attention for ECT use [10]. Due to its availability at the time, sodium pentothal (2–4 mg/kg) was the first induction agent used. Methohexital, a newer barbiturate (0.5–1.0 mg/kg), gained popularity after its development. Methohexital and thiopental, two barbiturates with comparatively weak anticonvulsant effects, were regarded as the gold standard for ECT anesthetic induction agents. But due to recent worldwide shortages of these agents, now, other drugs such as propofol, etomidate and ketamine have become more widely used [3,5,11].

Despite disagreements on the relationship between the effectiveness of ECT treatment and seizure duration, the suggested time for clinical effectiveness is 25–30 seconds [3,12]. Compared with other anesthetic agents, the duration of seizures due to propofol has been shown to be shorter and requires a higher stimulus loading with more failed seizures compared to other anesthetic agents. However, more recent tissue meta-analyses cast doubt on the significance of the difference between propofol and the other agents. ECT given with propofol was found to have a shorter duration of seizures than methohexital but not the other agents. In addition, the use of propofol at minimal hypnotic doses (0.75 mg/kg) was associated with seizure duration comparable to standard hypnotic doses of methohexital. Even the largest doses of propofol (1.5 mg/kg) may result in a duration of EEG seizure activity that is considered clinically acceptable [3,13].

Despite the disadvantages are a much greater anticonvulsant effect and a higher cost. The advantages of propofol over methohexital include a quicker and smoother emergence from anesthesia and a somewhat more favorable hemodynamic profile [11]. The induction with propofol has been associated with decrease in blood pressure, but it's better haemodynamic control with lower postictal blood pressure and heart rate control compared to etomidate, ketamine, sevoflurane, thiopental and methohexital [3,13,14]. In two other studies, propofol was compared with thiopental sodium with respect to a variety of anaesthesia and ECT relevant parameters. Propofol was superior to thiopental sodium in attenuating the physiological response to ECT, with milder haemodynamic changes and a less vigorous tonus and possibly clonus [7].

Propofol also has better recovery times compared to most agents with less risk of nausea and vomiting compared to methohexital and is superior to thiopental in terms of cognitive adverse effects [13]. Propofol was also associated with an earlier ability to walk. Thus, it may favour use in high-risk elderly patients requiring ECT [7].

Induction with propofol has some untoward effects. These include pain on injection, myoclonus, apnea and rarely thrombophlebitis. Because the ECT procedure itself causes discomfort and the administration of propofol causes pain, analgesics such as opioids are considered in these patients. Pethidine, an opioid with anticholinergic activity, causes tachycardia, which overcomes the bradycardia caused by propofol induction. When pethidine is combined with propofol, it can maintain hemodynamics variables within 20% of the baseline [14]. Opioids can act as a “seizure enhancers' ' by reducing the required hypnotic dose. Hence, short-acting opioids are effective in patients with an insufficient duration of seizures following ECT. Actually, pethidine is not recommended for

use with ECT because it can interact with other antidepressants (e.g. monoamine oxidase inhibitors or potentially serotonin reuptake inhibitors), potentially causing a hypertensive crisis and/or serotonin syndrome [15]. Since the patient was not diagnosed with depression and had no history of taking these medications, this could be ruled out.

Neuromuscular blocking agents reduce muscular convulsions and decrease the risk of serious injury. Visible muscle activity and, more accurately, EEG monitoring are used to monitor seizure activity. Succinylcholine (0.5 mg kg21) is most commonly used. If contraindicated, a non-depolarizing agent, mivacurium, can be used [4]. We did not use neuromuscular blocking agents because it is not available in our hospital.

CONCLUSION

ECT is a safe and effective treatment for various psychiatric disorders. Anesthesia during ECT creates a safe and comfortable environment for generalized epileptic seizures to occur. Multiple anesthetic agents are acceptable for use during ECT, and the choice of this drug should be considered for any underlying comorbidities that the patient has. Since propofol is a short-acting anesthetic with a rapid onset of action and hemodynamic changes under propofol anesthesia are more stable than those under barbiturate anesthesia, it is used in many reports. Induction using propofol and pethidine, has been proven to produce good clinical outcomes and minimal side effects, even though it is not accompanied by the use of neuromuscular blocking agents and ventilation machines

Declaration by Authors

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