Anesthetic and psychiatric implications of accidental awareness under general anesthesia during electroconvulsive therapy

ABSTRACT
Accidental awareness under general anesthesia (AAGA) is a well-known phenomenon. However, little literature exists in its relation to the psychiatric field, particularly within the electroconvulsive therapy (ECT) setting. This report explores the case of a 52-year-old woman that describes her own experience of AAGA during ECT. Relevant anesthetic details are also provided along with its possible implications in AAGA. The aim of this case report is to increase awareness among clinicians in regard to AAGA and its occurrence during ECT.

Key words: Anesthetic awareness; anxiety disorders; electroconvulsive therapy; maintenance of anesthesia; posttraumatic stress disorder; psychiatry

Introduction
Electroconvulsive therapy (ECT) is an effective medical therapy that has been utilized in the field of psychiatry since 1938. The most current guidance published in 2003, by the National Institute of Clinical excellence, recommends that ECT should be used “for the treatment of severe depressive illness, a prolonged or severe episode of mania, or catatonia.”[1] In particular, “ECT should be used to gain fast and short-term improvement of severe symptoms after all other treatment options have failed or when the situation is thought to be life threatening.” It is usually reserved for cases in which patients have had a poor response to both psychological and pharmacological interventions.[2] ECT is currently administered only under general anesthesia, known as modified ECT.[3] Modification in ECT is performed by either the use of intravenous or inhalational anesthetics. Both propofol and etomidate are common intravenous anesthetic agents used for this purpose with their associated advantages and side effects.[4] Alternatively, inhalational anesthetics such as sevoflurane and isoflurane can also be used. These are particularly useful for gas induction of anesthesia, for instance, when intravenous access can be difficult to obtain. In either case, a muscle relaxant such as suxamethonium can be administered to reduce the activity of a tonic–clonic seizure, therefore minimizing the musculoskeletal injuries.[5]

Accidental awareness under general anesthesia (AAGA) refers to the unintended intraoperative experience of awareness accompanied by subsequent recall and psychological trauma, which can lead to anxiety, depression, and post-traumatic stress disorder.[6] Understanding the implications of AAGA during ECT is crucial for both the anesthesiologist and the psychiatrist, as it can affect the patient’s post-operative recovery and overall psychological well-being.

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consequences. The incidence of AAGA remains controversial. Most papers that are dated within the last 10 years have reported the incidence as approximately one in every six thousand to twenty thousand anaesthetics.

In addition, AAGA has shown to be a potent precipitant of posttraumatic stress disorder (PTSD). Up to fifty percent of patients go on to develop overt psychological consequences. Although many of the adverse effects are benign in nature, AAGA has the propensity to lead to disengagement with ECT treatment, further mental health services, and possible future anesthetic procedures.

This report describes the experience of AAGA following ECT in a 52-year-old patient. Here, the patient went on to complete the course of twelve bilateral ECT sessions, following the addition of a volatile anesthetic agent and progressing to full recovery.

**Case Report**

A 52-year-old Caucasian female attended ECT clinic for the treatment-resistant depressive disorder. She was due to commence a third set of bilateral ECT treatments. The patient was prescribed lamotrigine 100 mg twice a day, agomelatine 25 mg twice a day, and buspirone 5 mg three times a day.

In the first ECT treatment, the anesthesia was induced with intravenous etomidate, and intravenous suxamethonium was given to provide muscle relaxation; oxygen was administered with intermittent positive pressure ventilation. A dose range of 0.15–0.3 mg/kg body weight of intravenous etomidate for induction of anesthesia and 0.5–1 mg/kg body weight intravenous suxamethonium for muscle relaxation are normally used in our hospital for ECT treatment under anesthetics. The first ECT treatment was uneventful. The patient had a motor seizure of 63 s duration, and an electroencephalogram (EEG) recording showed seizure activity lasting 121 s. The initial dose given was 75 mC.

In the second ECT treatment, when AAGA was reported to have taken place, the anesthetist was not a regular ECT anesthetist. Anesthetic agents used were again intravenous etomidate induction and suxamethonium. The ECT treatment was uneventful. This time there was no evidence of a motor seizure; however, the EEG recording illustrated spike and wave activity lasting 76 s. The stimulus dose was set at 75 mC.

After the second ECT was completed, the patient was taken into the recovery room where she was reported being aware and unable to move within the ECT suite. She was also able to recall the movement of being wheeled into the recovery room. The patient said that “... all I remember is being aware of voices. I couldn't make out what the voices were saying but I can remember trying to open my eyes, trying to speak, trying to move my feet but I could not move any part of my body, and it felt like forever. I didn't feel like I could breathe properly. I was desperate, thinking ‘I've got to let them know.’ As soon as I could, I let out a noise and cried out. I heard a voice saying ‘it's ok P’ and I recognized her voice, she was my nurse. I did not think anyone knew until I yelled and I vividly remember being wheeled through to recovery.” Further clinical consultation confirmed that the patient had in fact experienced true AAGA following delivery of the treatment.

Subsequently, in third ECT treatment, the sleep was again induced with the intravenous etomidate, and intravenous suxamethonium was administered for muscle relaxation. The patient had a motor seizure lasting 38 s, and the EEG showed seizure activity for 63 s. The stimulus dose was set at 75 mC. The patient was observed attempting to open her eyes immediately after the cessation of motor seizures. As the muscle relaxant was still in effect and the patient was unable to breathe hence was on assisted manual ventilation with oxygen by face mask. A decision was made to add a volatile anesthetics sevoflurane (minimum alveolar concentration >1) through the facemask until the return of spontaneous breathing. The patient did not recall any of the events during the ECT treatment this time.

Consequently, it was decided that the addition of a volatile anesthetic agent such as sevoflurane to maintain the anesthesia after intravenous etomidate anesthetic induction should be used in this particular patient, and in others, if and when AAGA is suspected, during future ECT treatments.

The patient required ongoing reassurance and dedicated consultation time with the anesthetic team to manage her heightened anxiety. There was no evidence of PTSD. However, the patient continued to be able to recall vivid distressing memories of the event.

The patient completed the course of 12 bilateral ECT treatments without any further anesthetic complications. Subsequently, the patient commenced maintenance ECT treatment and achieved good progress. The patient has since become an “Expert Patient” in advocating ECT to patient groups.

**Discussion**

Limited data exists on the incidence and etiology of AAGA during ECT. A paper published in 2002 studied 81 cases of...
AAGA of which three had occurred within an ECT setting. The study found that two of these occurrences had no identifiable cause. The third had resulted from a drug error leading to the inadvertent paralysis of an awake patient. More recently, an article published in 2015 found 4 out of 68 cases of AAGA occurring in an ECT setting. This paper focused on the patient perceptive of intraoperative awareness. Before this, two case reports were published on AAGA in ECT in the last 10 years. Both patients went on to decline further ECT treatment.

Two theories have been proposed to explain AAGA. One theory suggests that there is a failure to deliver sufficient anesthetic agent to the patient. The second theory proposes that the individual patient has a resistance to an otherwise sufficient dose of anesthetic agent. The most common form of AAGA reported is known as emergence AAGA. It is defined as, “any time after the end of surgery, when the patient reported they were awake when they felt they should still have been unconscious.” Patients are invariably aware and paralyzed. This can be a likely result of inadvertent mismatch between the time course of return of consciousness versus the return of motor capacity. In addition, AAGA specifically in the ECT setting can be explained by the following reasons. First, patients can potentially have an altered seizure threshold having had psychotropic medication. Second, the induction dosage of IV anesthetic agents tends to be as low as possible to a) induce sleep for only a few minutes and b) enable an adequate ECT response. Therefore, the maintenance of anesthesia is a not routinely practiced in this setting.

In the third ECT session, clinical signs of motor activity (flickering of the eyelids) suggested possible awareness and prompted the anesthetist to use volatile anesthetic to maintain the anesthesia. In practice, it is recommended to increase the depth of the induction dose. However, in this setting, a volatile agent sevoflurane was readily available. There are several advantages of utilizing sevoflurane within the ECT setting. First, it can be easily administered through facemask with minimal tracheopharyngeal irritation. Second, it allows a smooth titration leading to rapid induction. Finally, sevoflurane has little or no impact on seizure threshold, permitting a rapid offset and an efficient recovery.

The administration of an IV benzodiazepine has also been recommended in the case of AAGA to reduce the risk of postoperative recall. This was avoided due to evidence on benzodiazepines both altering seizure threshold and delaying recovery.

The maintenance of anesthesia with inhaled anesthetic agents in patients undergoing ECT is a potential area for further exploration. In psychiatry, AAGA is seldom discussed in ECT. Prescribers and other clinicians involved should be aware of the concept and the psychological impact that AAGA can have on patient compliance with further treatment. This report also highlights that there can be a possibility of AAGA despite having undergone previous uneventful ECT treatments under general anesthesia.

Conclusion

The clinicians should be aware of AAGA within the ECT setting and the psychological impact on patient compliance regarding further treatment. A volatile anesthetic agent such as sevoflurane should be readily available for anesthesia maintenance, especially in the eventuality of unexpected awareness indicators or a delayed procedure, where otherwise only a short-term and rapid anesthesia induction is needed. The case report also highlights the possibility of AAGA in patients who have undergone previously uneventful general anesthesia. The AAGA in ECT is a rare but important phenomenon that can lead to distressing consequences, such as PTSD.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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