

CASO CLÍNICO

Ketamine and Electroconvulsive Therapy: A Case Report

Cetamina e Eletroconvulsivoterapia: Um Caso Clínico

Daniela Pereira^{1*} , Catarina Silva¹ , Ana Paulino¹ , João Rema¹ 

Afiliação

¹ Department of Anesthesiology, Centro Hospitalar Universitário Lisboa Norte, EPE, Lisbon, Portugal.

Keywords

Anesthesia; Depressive Disorder/complications; Electroconvulsive Therapy; Ketamine; Psychotic Disorders/complications

Palavras-chave

Anestesia; Cetamina; Perturbação Depressiva/complicações; Perturbações Psicóticas/complicações; Terapia Eletroconvulsiva

ABSTRACT

Electroconvulsive therapy (ECT) is the gold standard therapy for treatment-resistant depression and is usually performed under general anesthesia. A variety of induction agents may be used. Ketamine is an induction agent with increasing evidence suggesting an antidepressant effect. However, ketamine is known to produce psychotic symptoms and therefore its use has been avoided in psychosis. The authors explore the case of a 30-year-old man, who was diagnosed with affective psychosis with predominance of the negative symptoms. He had two major depressive episodes resistant to pharmacological therapy requiring long term permanence in the psychiatry ward and underwent twelve ECT sessions. Ketamine was the most frequently used induction agent. Its use as induction agent in ECT sessions allowed the use of a decremental electrical charge, was associated to a sustained remission of depressive symptoms and was not accompanied by psychotic or dissociative phenomena during the treatment and follow-up period.

RESUMO

A eletroconvulsivoterapia (ECT) é o tratamento de primeira linha na depressão resistente, sendo habitualmente realizado sob anestesia geral. Muitos agentes indutores podem ser utilizados. A cetamina é um indutor anestésico que apresenta evidência crescente de efeito antidepressivo. Contudo, a cetamina é conhecida por poder produzir sintomas psicóticos, e por este motivo tem sido evitada em doentes com patologia psicótica. Os autores apresentam o caso clínico de um homem de 30 anos, com o diagnóstico de psicose afetiva com predomínio de sintomas negativos. O doente apresentou dois episódios depressivos graves, com necessidade de internamento prolongado, tendo sido submetido a doze sessões de ECT. A cetamina foi o agente indutor mais frequentemente utilizado, tendo-se associado à utilização de uma carga elétrica total progressivamente menor, com uma remissão dos sintomas depressivos sustentável, sem episódios psicóticos ou dissociativos durante o tratamento ou seguimento posterior.

INTRODUCTION

Electroconvulsive therapy (ECT) is a nonpharmacologic treatment which has been proven to be highly effective in many psychiatric conditions. The safety and tolerability of this type of treatment have been enhanced by the use of modified stimulation techniques and anesthesiology support. A variety of induction agents may be used depending on clinical characteristics of the patient. Anesthetic agents frequently used and cited are propofol, thiopental, methohexital and ketamine. Propofol is being widely used among the anesthesiologists because of its popularity, yet it could shorten the duration of the seizure.¹ Ketamine has become an agent of interest in the treatment of depression and as a induction agent for ECT, with a potential neuroprotective effect. However, ketamine administration in patients with psychotic symptoms has been avoided because of its potential psychotomimetic effects.²

CASE REPORT

The authors present a 30-year-old man, with no history of psychiatric symptoms until the age of 27 when he first reported an episode of body spasms followed by a feeling of “an acid running through his body”. The following year was characterized by progressive social isolation, anhedonia, unusual behavior along with emergence of psychotic symptoms.

The patient was admitted to the psychiatric ward after a first psychotic episode followed by two major depressive episodes resistant to pharmacological therapy. After the second depressive episode he agreed with twelve ECT sessions. The treatment was performed in collaboration with the Anesthesiology Department in the Post-anesthesia Care Unit (PACU) once/twice a week. It is important to highlight that different anesthesiologists were involved in this process and the induction agent was an individual choice of each one. Before every session, written consent and fasting times

Autor Correspondente/Corresponding Author:

Daniela Trindade Pereira

Morada: Centro Hospitalar Universitário Lisboa Norte, EPE, Avenida Professor Egas Moniz, Department of Anesthesiology, 1649-028 Lisbon, Portugal.

E-mail: danielatrindadepereira@gmail.com

were checked and no premedication was administered. The patient was monitored according to American Society of Anesthesia standards and the electrodes were placed in the temporal region bilaterally. The patient was pre-oxygenated and a total intravenous anesthesia was performed with a variable induction agent followed by suxamethonium 1 mg/kg. After loss of conscience, the bite block was inserted, and the patient was ventilated with a bag-valve-mask. The psychiatrist chose the parameters and the electrical discharge administered after turning off temporarily the source of oxygen. Then the electroencephalographic (EEG) and motor responses were analyzed and registered. Hemodynamic stability was maintained in all sessions. Bag-valve-mask ventilation was maintained until returning of spontaneous ventilation. Intravenous acetaminophen 1 g was given to prevent post-procedure myalgias. After the first session, he reported nausea. Since then, monophylaxis of nausea and vomit was taken with intravenous dexamethasone 4mg. No other complications were reported, especially arrhythmias, prolonged seizure or psychotic symptoms. All parameters evaluated are described in Table 1. In most cases, ketamine was the agent of choice. Propofol and Etomidate were only used in the first and sixth sessions, respectively. The patient was discharged and during the ongoing consultations he was prescribed clomipramine 75 mg 3 times a day, clozapine 300 mg/day and topiramate 200 mg/day. The patient got a new job, started feeling better with no detected suicidal ideation or psychotic symptoms during his on-going consultations. Negative symptoms such as asocialism are still present, as to be expected. No major depressive events were reported since then and the current diagnosis proposed by the attending psychiatrist is of schizoaffective disorder. The patient signed the informed consent for this case description.

DISCUSSION

This case report describes a major depressive episode in a young man with a psychotic disorder, who underwent twelve ECT sessions after failure of pharmacological therapy. Despite the hypnotic agent chosen for the first ECT session was propofol, ketamine was chosen in the subsequent

sessions due to reasons not explained in the clinical records. With the use of ketamine EEG convulsive response ranged from 30 to 53 seconds, and motor convulsive response ranged from 25 to 50 seconds, with progressive use of lower total electrical charge. This lower total electrical charge can be associated with less cognitive adverse effects.⁴

Propofol was used in the first ECT session with an EEG convulsive response of 33 seconds and a motor convulsive response of 25 seconds.

Etomidate was used only in the 6th ECT session without EEG or motor convulsive response, in opposition to previous reports,¹ and for reasons that the authors are unable to explain. This treatment regimen led to a sustained remission of depressive symptoms and was not accompanied by psychotic or dissociative phenomena during the treatment and follow-up period. There are some concerns about whether ketamine should be used in people with psychotic disorders. In fact, there are few reports demonstrating that ketamine can be used safely in this group of patients.

Successful treatment of two cases of psychotic depression with off-label intravenous ketamine was reported, with a dramatic mood improvement and dissolution of psychotic symptoms.² Another case report describes intramuscular use of ketamine to facilitate intravenous line and electrode placement, in a patient with schizoaffective disorder, who presented with psychosis and severe refractory agitation.³

A double-blind and placebo controlled study that combined ECT and ketamine showed no difference between ketamine and placebo yet with a trend level difference in rates of early remission with ketamine and possibly with minimum cognitive side effects.⁴ A recent meta-analysis found that ketamine alone does not appear to improve the efficacy of ECT, but the combination of ketamine with other anesthetic agents may be beneficial in improving depressive symptoms at the early phase of ECT.⁵ In conclusion, despite the use of ketamine is controversial and avoided in psychosis, in this case the ECT protocol with use of ketamine as main induction agent, improved depressive symptoms with no exacerbation of psychotic features. Further research in this field is necessary to access ketamine use as an induction agent in ECT and its effects in psychotic disorders.

Table 1. Summary of ECT Sessions

ECT Session	Electrical charge (mC)	Induction Agent	EEG Convulsive Response (sec)	Motor Convulsive Response (sec)
1 st	189	Propofol 250 mg (2.5 mg.kg ⁻¹)	33	25
2 nd	189	Ketamine 100 mg (1 mg.kg ⁻¹)	50	50
3 rd	162	Ketamine 100 mg	50	49
4 th	144	Ketamine 100 mg	50	49
5 th	120	Ketamine 100 mg	52	45
6 th	96	Etomidate 26 mg (0.2 mg.kg ⁻¹)	0	0
7 th	96	Ketamine 100 mg	53	50
8 th -12 th	84	Ketamine 100 mg	>30	>25

ECT: electroconvulsive therapy, mC: millicoulombs, sec: seconds.

Ethical Disclosures

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ORCID

Daniela Pereira  <https://orcid.org/0000-0002-0514-1115>

Catarina Silva  <https://orcid.org/0000-0003-4941-3810>

Ana Paulino  <https://orcid.org/0000-0003-2340-9485>

João Rema  <https://orcid.org/0000-0002-2552-5175>

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