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Case Report

Spontaneous seizures after ECT in a patient medicated with bupropion, sertraline and risperidone

Convulsões espontâneas após ECT em doente medicada com bupropiona, sertralina e risperidona

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Abstract

Objective: To report a case of post-electroconvulsive therapy spontaneous seizures in a patient medicated with sertraline, bupropion and risperidone.
Case description: A 53-year-old woman with recurrent major depression was admitted to our psychiatry department for a major depressive episode of 6 weeks' duration, with psychotic symptoms. She was already on 200 mg/day of sertraline and 2 mg/day of risperidone. After 8 weeks on 200 mg/day of sertraline, 4 mg/day of risperidone and slow release bupropion (titrated to 300 mg/day), with no objective improvements, the decision was taken to initiate a course of 8-10 electroconvulsive therapy (ECT) sessions. Two days after the first treatment, three generalized tonic-clonic seizures occurred within 6 hours. Phenytoin and sodium valproate were added to the patient's daily medication and no further spontaneous seizures were observed. After neurologic assessment and discussion of the case, phenytoin and bupropion were withdrawn at once (two days after the spontaneous seizures) and the decision was taken to resume the ECT treatment. No further spontaneous seizures occurred and, at discharge, the patient exhibited significant improvements and was free from major depressive symptoms.
Comments: This report illustrates a case of post-ECT spontaneous seizures that might have been due to a specific pharmacological etiological pathway, namely, bupropion's proconvulsive properties, although both sertraline and risperidone also lower the convulsive threshold.
Keywords: Electroconvulsive therapy, spontaneous seizures, major depressive disorder, bupropion, sertraline, risperidone.

Resumo

Objetivo: Descrever o caso de uma paciente medicada com sertralina, bupropiona e risperidona que apresentou três crises tônico-clônico generalizadas espontâneas após eletroconvulsoterapia (ECT).
Descrição do caso: Uma mulher de 53 anos com antecedentes de perturbação depressiva maior recorrente foi internada em nosso serviço devido a novo episódio depressivo maior com sintomas psicóticos e 6 semanas de evolução. Ela já estava medicada com 200 mg/dia de sertralina e 2 mg/dia de risperidona. Após 8 semanas usando 200 mg/dia de sertralina, 4 mg/dia de risperidona e bupropiona de liberação lenta (titulada até 300 mg/dia), sem melhoras objetivas, decidiu-se iniciar 8-10 sessões de ECT. Dois dias após a primeira sessão, ocorreram três crises tônico-clônico generalizadas num espaço de 6 horas. Foram introduzidos fenitoína e valproato de sódio no esquema terapêutico da paciente, e não ocorreram mais crises. Após avaliação neurológica e discussão do caso, optou-se por suspender a fenitoína e a bupropiona (2 dias após as crises espontâneas) e retomar a ECT. No restante do tratamento não ocorreram mais crises convulsivas espontâneas e, até data da alta, a paciente apresentava melhorias significativas e estava livre de transtorno depressivo maior.
Comentários: Este relato ilustra a ocorrência de crises convulsivas espontâneas após a ECT, as quais parecem ter sido causadas por mecanismos farmacológicos etiológicos específicos, a saber, as propriedades pró-convulsivantes da bupropiona, mesmo com o uso de sertralina e risperidona, que baixam o limiar convulsivante.
Descritores: Eletroconvulsoterapia, crises espontâneas, perturbação depressiva major, bupropiona, sertralina, risperidona.

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Introduction

Electroconvulsive therapy (ECT) is widely used to treat refractory or recurrent disorders (such as severe depression, mania, and schizophrenia). Epidemiologic data indicate that ECT does not precipitate epilepsy. In fact, ECT may be used to reduce the frequency of spontaneous seizures, although late seizures related to this treatment modality have also been reported.[1]

Current ECT protocols suggest that keeping patients with severe depression disorder on their antidepressants during the course of treatment has a beneficial effect.[2] However, an increased incidence of seizures is a well known dose-related side effect of bupropion. There is scant scientific literature addressing the potential interaction between bupropion and induced electroconvulsive seizure activity, whether administered as a monotherapy or in combination with other drugs that may lower the seizure threshold.

Case description

A 53-year-old woman with a history of recurrent major depression was admitted to our psychiatry department for a major depressive episode with psychotic symptoms (mood congruent delusions) that had lasted 6 weeks. She was already on 200 mg/day of sertraline and 2 mg/day of risperidone. Organic etiology assessment included laboratory tests (complete blood cell counts, electrolytes, liver and renal functions, thyroid-stimulating hormone [TSH], blood fat level), electrocardiogram (ECG), computed tomography (CT) of the head and electroencephalogram (EEG). All results were within normal parameters. There was no history of head trauma or seizure disorders. The patient's past medical history was otherwise unremarkable and she was on no other medication besides psychotropics. She had received an uncomplicated and effective course of ECT treatment for an earlier depressive episode almost 20 years previously.

She was prescribed 200 mg/day of sertraline, 4 mg/day of risperidone and slow release bupropion (titrated at 300 mg/day), but no objective improvements were perceptible after eight weeks of hospitalization. The decision was taken to initiate a course of 8-10 ECT sessions (twice a week; with bilateral electrode placement). A MECTA Spectrum 5000Q was available at our department.

The ECT treatment was initiated on June 18, 2014. The patient's medication was not administered on the morning of the first treatment session. Prior to the first ECT session the patient was medicated with a total of 275.00 mg of thiopental, 55.00 mg of succinylcholine, and 0.50 mg of atropine. In accordance with our department's ECT protocol, the seizure threshold was determined via a method-of-limits procedure. A 35-second seizure occurred after ECT stimulus using the following parameters: charge 384 mC, energy 49.9 joules, dynamic impedance 162 Ohms, frequency 40 Hertz, duration 6 seconds, pulse width 1.0 msec, and current 800 mA. No immediate intercurrent conditions or complications were observed.

Two days later, three generalized tonic-clonic seizures occurred within 6 hours. A CT scan of the head and CBC detected no abnormalities and phenytoin and diazepam were administered at our emergency department. Phenytoin and sodium valproate were added to the patient's daily medication and no more spontaneous seizures were observed. After neurologic assessment and discussion of the case, phenytoin and bupropion were withdrawn from the patient's daily medication at once (two days after the spontaneous seizures) and the decision was taken to resume ECT treatment.

For the last 9 ECT sessions, the patient's medication was not administered on the mornings of treatment days and sodium valproate was not administered on the night before treatment.

These 9 ECT treatments took place, twice a week, with the following parameters: charge 576 mC, energy 88.1 joules, dynamic impedance 184 Ohms, frequency 60 Hertz, duration 6 seconds, pulse width 1.0 msec, and current 800 mA. The anesthetic drug dosages used were: 175.00 mg of thiopental, 25.00 mg of succinylcholine and 0.50 mg of atropine. The seizures induced by each ECT treatment all lasted from 30 to 35 seconds and no other episode of spontaneous seizures occurred.

At discharge the patient exhibited significant improvements and no major depressive symptoms.

In October 2014 sodium valproate was discontinued with no consequences. In November 2014, an EEG assessment found no signs of epileptic activity and the patient remained free from depressive symptoms, medicated with 200 mg/day of sertraline, and 2 mg/day of risperidone.

Discussion

The American Psychiatric Association (APA) defines post-ECT spontaneous seizures as those that occur days, weeks, or longer after the treatment.[3] Recent studies[4] reaffirm the APA's recommendations[5] on the importance of the patient's underlying organic condition in cases in which seizures occur, rather than the etiological role of ECT in epilepsy.

This report illustrates a case of post-ECT spontaneous seizures that might have been due to a specific pharmacological etiological pathway, namely, bupropion's proconvulsive properties, although sertraline and risperidone also lower the convulsive threshold.

The seizure risk related to bupropion has been shown to be dose-dependent, even when administered at therapeutic doses.[6] The pharmacology of the convulsive liability of bupropion has been studied recently, to determine whether it can be accounted for by the parent drug or one of its active metabolites.[7]

Bupropion's pharmacological characteristics and the clinical evolution of the case reported here suggest that this drug had a major influence on the spontaneous seizures. However, the patient was also medicated with another two drugs with the potential to lower the convulsive threshold. The current state of theory regarding this complex drug interaction demonstrates the importance of conducting further studies to attempt to identify whether the effect is cumulative or independent before definitive conclusions can be drawn.

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