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Case report

THE USE OF ELECTROCONVULSIVE THERAPY TO TREAT SCHIZOAFFECTIVE DISORDER IN A PATIENT WITH PACEMAKER: A CASE REPORT

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INTRODUCTION

Athough being one of the most controversial treatments in psychiatric history, contemporary literature and treatment recommendations support the use of electroconvulsive therapy (ECT) as a valid method in the treatment of psychiatric disorders. While in Croatia there are no guidelines on the use of the ECT, the usual procedure followed here is in line with the existing recommendations released by the established professional associations (APA 2001, Rush et al. 2007). In short, the ECT is recommended for the treatment of certain types of schizophrenia and affective disorders, especially in the presence of factors such as resistance to pharmacotherapy, side effects to pharmacotherapy positive response to ECT treatment in the earlier acute episodes of the illness (APA 2001, Rush et al. 2007).

Here we present a case of a female patient suffering from schizoaffective disorder with implanted pacemaker to her congenital total AV block who was successfully treated with the ECT. While considering the treatment options for this patient, we became aware of the lack of studies specifically designed to explore the risks of the use of ECT in patients with a pacemaker. Based on the reported data summarized in a recent review, the ECT appears safe in patients with implanted pacemakers treated from depression (Korkas et al. 2011).

CASE REPORT

Female patient, aged 47 was admitted to our ward accompanied by her husband presenting with hetero aggressive behavior.

Anamnestic and heteroanamnestic data obtained from her husband, and the medical chart review revealed she had her first psychotic outbreak at the age of twenty one, when she was diagnosed with schizoaffective disorder, manic episode. She was treated with clozapine and twenty applications of the ECT achieving remission. In the following years she was hospitalized twice, both times after stopping her medication and treated with clozapine and lithium to achieve remission. After that, her illness entered in a phase of stabile remission which lasted for the next twenty years during which she married, started a family and sustained her job. During that period she was treated only with clozapine in minimal daily doses (25mg/day). Two weeks ago a relapse occurred for no apparent reason.

At the time of the admission, her psychiatric status indicated logorrhea, affective instability, with sudden mood swings from elevated to lowered mood on daily bases, dissociative speech, paranoid and grandiose delusions, ideas of reference, delusions of guilt, auditory hallucinations, ambivalence, suggestibility and disorganized behavior.

Further medical evaluation revealed that she developed cardiac symptoms (bradicardia, fainting) five years ago due to her congenital total AV block and was implanted a pacemaker (St. Jude, DDD). Apart that she was overweight (body mass index of 28 kg/m2), she had no other cardiovascular risks and no other somatic illnesses.

Upon admittance her physical examination revealed blood pressure of 120/80 mmHg, hearth rate of 70 BPM, and electrocardiogram (ECG) showed normal pacemaker function.

She was diagnosed with schizoaffective disorder, mixed episode (F25.2), according to the International Classification of Disorders 10th revision (ICD-10). The initial treatment plan included a combination of pharmacotherapy, supportive individual and family psychotherapy. She was initially treated with a combination of clozapine (50mg bid) and lithium (900mg tid) for three weeks, but at the 3rd week assessment only psychomotor agitation was reduced. Dose elevation of clozapine (100mg quid) induced only sedation. For the next two weeks she was treated with olanzapine (10mg once a day), lithium (900mg tid), valproate (900mg tid) and haloperidol (10mg bid). However, after several days she developed polyuria and polydypsia and was diagnosed with litium-induced diabetes insipidus, which led to the discontinuation of lithium. During this period of time one could discern more pronounced delusions of guilt and therefore sertraline (100 mg bid) was added, but with no positive treatment response.

After the evaluation of an internal medicine specialist and an anesthesiologist, we consulted a cardiologist for the evaluation of the patient cardiologic status and the state of the pacemaker. After the cardiologist's final approval and after the patient and her husband have given a written consent for the ECT treatment, we started the treatment. The ECT team (composed of a psychiatrist, an anesthesiologist, a cardiologist familiar with the pacemaker programming, a psychiatric nurse and an anesthesiology nurse) applied the standard protocol. To avoid a possible unfavorable parasympathetic reflex after the ECT, atropine (0.01 mg/kg) was given as premedication five minutes before the treatment. Following preoxigenation (100% O_2) general anesthesia was induced with propofol (1mg/kg). After the loss of consciousness and eyelash reflex, intravenous succinylcholine (0.5 mg/kg) was administered for muscle relaxation and ventilation was assisted with a face mask and 100% oxygen. ECT was administered using the Thymatron Modell DG, Somatics Inc, 1995 device. Electrodes were placed bilaterally, frontotemporally, followed by the application of 800 mA, 90 Hz, short pulse wave of 1 ms, with the duration of the stimulus for 3.36 s - 3.92 s. The energy delivered to the patient was 35% - 40%, or (151.2 mC/ 34.79 J – 174.4 mC/ 39.76 J). The ictal response was monitored by observing the leg reflex response and by the electroencephalogram (EEG). The treatment produced a 25-28 seconds modified generalized seizure.

During the procedure vital functions were monitored using the non invasive blood pressure, continuous ECG, peripheral oxygen saturation (SpO2) and end-expiratory CO2 partial pressure (end-tidal CO2) at the nostrils. No adverse effects occurred throughout the procedures. Hemodynamic parameters were stabile during the ECT and the patient was discharged from the hospital recovery room after 45 minutes.

Before each ECT application, the cardiologist switched the pacemaker to asynchronous mode (DOO), by placing a magnet on a skin above the pacemaker following the recommendation for the ECT application. After each treatment the pacemaker was set to synchronous mode again. The pacemaker was interrogated prior to each treatment, and monitored on the ECG during the treatment. Final interrogation of the pacemaker showed no changes in relevant pacemaker parameters (amplitude, threshold and impedance).

The ECT was applied three times per week and in total she received 15 applications. While her symptoms significantly reduced, around the 15th application she started having anterograde memory loss, and we had to discontinue further treatment with the ECT. After 90 in-hospital days she was demitted to outpatient treatment, and after 6 months she achieved her prior level of functioning within the family and returned to work. Cognitive symptoms recovered shortly after the ECT discontinuation. Supportive family psychotherapy helped the stabilization of her family relations.

DISCUSSION

To the best of our knowledge, this is the first documented case of the ECT application in a patient with a pacemaker in Croatia. In general, the use of ECT may induce few cardiac complications which are usually mild and transient, such as bradiarrhythmias, tachyarrhythmias atrial flutter, atrial fibrillation (Tess et al. 2009, Nuttall et al. 2004). However, more severe side effects such as ventricular tachycardia, asystolic cardiac arrest, ischemic complications and congestive heart failure have also been reported (Yu et al. 2009). Elderly patients and patients with preexisting heart condition seem to be the most vulnerable population group (Tess et al. 2009). In patients with implanted pacemakers, potential cardiac adverse effects result from the actions of the ECT on preexisting hearth conditions (Tess et al. 2009, Nuttall et al. 2004) and from the interference of the ECT on the work of the pacemaker, as it was reported that rate responsive pacemakers can wrongly sense electrical activity as the hearth activity (MacPherson et al. 2006). Therefore, according to manufacturers and the currently existing recommenddations (APA 2001), it is advised to adjust the pacemaker in the asynchronous mode (in which the pacemaker acts as rate non responsive) during the ECT. However, it should be noted that in the available case reports of patients with pacemakers treated with ECT, the majority of patients had their pacemakers in synchronous rate during the ECT (Korkas et al. 2011). In addition, while it was found that the ECT interferes even with the newer types of pacemakers, this interference is generaly regarded as clinically insignificant (Giltay et al. 2005). In our opinion both options are acceptable (synchronous vs. asynchronous). If a magnet or preprogramming to VOO or DOO mode (asynchronous) is used, the patient is guaranteed a minimum heart rate. On the other hand, synchronous mode is also viable option, especially with shorter bursts of the ECT energy, as it is unlikely that the ECT energy and muscle fasciculation can inhibit or reprogram the pacemaker functions due to the low response voltages measured in the electrode system (Giltay et al. 2005). The advantage is that we do not have to use a magnet or pacemaker preprogramming.

The next important cause of ECT-induced cardiac adverse effects in patients with pacemakers is related to the concomitant use of medication with known cardiac effects: 1) anticholinergic medication which is commonly used during the ECT to reduce the possibility for ECT-induced bradicardia and asystole; 2) medications which are taken by the patient for her or his psychiatric or somatic condition. While the use of anticholinergic medication was recommended as part of the standard ECT protocol, newer reports showed that anticholinergic medications may increase the risk of cardiac adverse effects such as the cardiac output workload (Mayur et al. 1998) and hypertension (Korkas

et al. 2011). Therefore the use of anticholinergics has been recommended only in patients with previous history ECT-induced asystole, and in minimal effective dose (Tess et al. 2009). A number of psychiatric drugs, especially antipsychotics can affect the hearth conductance for example by the prolongation of the QTc interval, by inducing bradicardia, hypotension and compensatory tachycardia (Glassman et al. 2001). The concomitant use of these drugs during ECT might therefore additionally increase the risk of ECT-induced adverse effects in treated patients (i.e. Nahshoni et al. 2004).

Based on the review all available reported case reports and case series in the literature, Korkas et al. (2011) have proposed recommendations for the safe use of the ECT in depressed patients with pacemakers. Compared to patients with depression, patients with schizoaffective disorders are usually treated with antipsychotics which can affect cardiac conductance (Glassman et al. 2001) and with combinations of drugs which might interact with each other on their pharmacokinetic and pharmacodinamic levels. Both could result in more often or severe side effects, including the ones related to the cardiac conductance. Also, the long term use of antipsychotics is associated with obesity, diabetes, cardiovascular disorders (De Hert et al. 2011) and unhealthy life style (Pesek et al. 2011) which make. These patients especially vulnerable for ECT-induced adverse effects (Tess et al. 2009).

CONCLUSIONS

In general, the use of ECT in patients with a pacemaker should follow existing guidelines (APA 2001, Rush et al. 2007) and recommendations (Korkas et al. 2011, Tess et al. 2009). In line with those but acknowledging the illness and treatment-related specificities we suggest several additional steps: 1) The decision on the preferred treatment (pharmacotherapy vs. ECT) should be personalized accounting for the patient's cardiac status and the presence of cardio-vascular risks (such as obesity, diabetes); 2) The use of concomitant medication should be carefully analyzed. It is recommended to avoid psychiatric medications with known cardiac effects or use the minimal possible dose. It is recommended to avoid the concomitant use of psychiatric drugs which can interact on the pharmacokinetic level altering drug concentrations in blood or to modify the dose of the drugs accordingly, preferably after a consultation with a clinical pharmacologist. During the ECT it is recommended to use nonpolarizing muscle relaxants if muscle fasciculations are present, and to avoid anticholinergics if possible, or to use the lowest effective dose; 3) Before starting with the ECT it is necessary to examine the status of the pacemaker and to ensure a doctor familiar with pacemaker programming who will interrogate pacemaker and be present during ECT; 4) Whereas both synchronous vs. asynchronous mode of the pacemaker are acceptable, it is recommended to readjust the pacing mode of the pacemaker, if advised by the manufacturer; 5) The work of the pacemaker should be monitored before and during the ECT with the ECG; 6) After the ECT it is recommended to interrogate pacemaker and ensure its proper function during recovery and readjust the pacing mode if modified before ECT.

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REFERENCES

1. *American Psychiatric Association: The Practice of ECT: Recommendations for Treatment, Training and Privileging. Revised edition. Washington, DC: American Psychiatric Press; 2001.*
2. *De Hert M, Cohen D, Bobes J, Cetkovich-Bakmas M, Leucht S, Ndetei DM et al.: Physical illness in patients with severe mental disorders. II. Barriers to care, monitoring and treatment guidelines, plus recommendations at the system and individual level. World Psychiatry 2011; 10: 138-51.*
3. *Giltay EJ, Kho KH, Keijzer LT, Leijenaar M, van Schaick HW, Blansjaar BA: Electroconvulsive therapy (ECT) in a patient with a dual-chamber sensing, VDDR pacemaker. J ECT 2005; 21: 35-8.*
4. *Glassman AH, Bigger JT: Antipsychotic drugs: prolonged QTc interval, torsade de pointes, and sudden death. Am J Psychiatry 2001; 158: 1774-82.*
5. *Kokras N, Politis AM, Zervas IM, Pappa D, Markatou M, Katirtzoglou E et al.: Cardiac rhythm management devices and electroconvulsive therapy: a critical review apropos of a depressed patient with a pacemaker. J ECT 2011; 27: 214-220.*
6. *MacPherson RD, Loo CK, Barrett N: Electroconvulsive therapy in patients with cardiac pacemakers. Anaesth Intensive Care 2006; 34: 470-474.*
7. *Mayur PM, Shree RS, Gangadhar BN, Subbakrishna DK, Janakiramaiah N, Rao GS: Atropine premedication and the cardiovascular response to electroconvulsive therapy. Br J Anaesth 1998; 81: 466-7.*
8. *Nahshoni E, Manor N, Bar F, Stryjer R, Zalsman G, Weizman A: Alterations in QT dispersion in medicated schizophrenia patients following electroconvulsive therapy. Eur Neuropsychopharmacol 2004; 14: 121-5.*
9. *Nuttall GA, Bowersox MR, Douglass SB, McDonald J, Rasmussen LJ, Decker PA, Oliver WC Jr, Rasmussen KG: Morbidity and mortality in the use of electroconvulsive therapy. J ECT 2004; 20: 237–241.*
10. *Pesek MB, Mihoci J, Medved K, Solinc NP: Long term groups of patients with psychosis: physical activity and medical treatment. Psychiatr Danub 2011; 23: S149-54.*
11. *Rush G, Kimmich O, Lucy JV: Electroconvulsive therapy: International guidelines, clinical governance and patient selection. Ir J Psych Med 2007; 24: 103-107.*

12. *Tess AV, Smetana GW: Medical evaluation of patients undergoing electroconvulsive therapy. N Engl J Med 2009; 360: 1437-44.*
13. *Yu SW, Vallurupalli S, Arnoldi J, Holloway R: Atrioventricular dissociation after electroconvulsive therapy. Cardiol Res Pract 2011; 2011: 746373.*

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