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CLINICAL REPORT

A case of nonsustained ventricular tachycardia immediately following modified electroconvulsive therapy in a depressive patient

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Abstract Modified electroconvulsive therapy (mECT) with the use of hypnotics and muscle relaxants is an optional and prevailing treatment for depression in patients who have failed on antidepressant regimens. We describe a patient who developed ventricular tachycardia (VT) immediately after mECT. A 64-year-old man with no remarkable past history underwent a course of mECT for drug-resistant depression. Anesthesia was induced with intravenous thiopental (150 mg) followed by rocuronium (50 mg). Three minutes after the administration of rocuronium, the brain was electrically stimulated using a pulse wave. The first mECT session was performed uneventfully. However, the second session 2 days later elicited acute hypertension (182/134 mmHg) and tachycardia (130 bpm), resulting in the appearance of single and couplets of premature ventricular contractions on the electrocardiogram followed by VT lasting about 10 s. The chest was immediately compressed several times, then normal sinus rhythm was spontaneously restored without administering antiarrhythmic agents. The patient recovered from anesthesia without complications. Postoperatively, close examination was unable to definitively determine the cause of VT, resulting in the cancellation of subsequent mECT sessions. It is important to bear in mind that mECT can induce life-threatening arrhythmias such as VT.

Keywords Modified electroconvulsive therapy · Hypertension · Tachycardia · Arrhythmia · Ventricular tachycardia

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Introduction

Modified electroconvulsive therapy (mECT) under general anesthesia with hypnotics and muscle relaxants has been extensively applied for the treatment of drug-resistant depression, schizophrenia with depression, and other conditions [1, 2]. It is well known that mECT is accompanied by significant hemodynamic responses such as hypertension and tachycardia. In general, however, these are transient and resolve without detrimental sequelae [3–5]. Here we report, in contrast, a case of ventricular tachycardia (VT) immediately following mECT for depression.

Case description

A 64-year-old man (178 cm in height and 66 kg in weight) with no contributory family or past history, and who was otherwise healthy, had been diagnosed with depression approximately 6 months earlier. Thereafter, the following antidepressants had been administered (mg/day): mirtazapine (15), sertraline (25), flunitrazepam (3), brotizolam (0.25), aripiprazole (3), and midodrine (2). As the patient began to manifest suicidal thoughts, regardless of medication, mECT was scheduled on alternative days for 2 weeks in order to mitigate the depressive disorder. The preoperative laboratory data, including an electrocardiogram (ECG), were observed to be within normal limits.

The mECT was performed in the same way for both the first and the second scheduled mECT as follows. Infusing an acetated Ringer's solution into a peripheral vein at a rate of 300 mL/h, premedication with intramuscular atropine (0.5 mg) and intravenous famotidine (20 mg) was administered 30 min before transfer to the operating theater. The

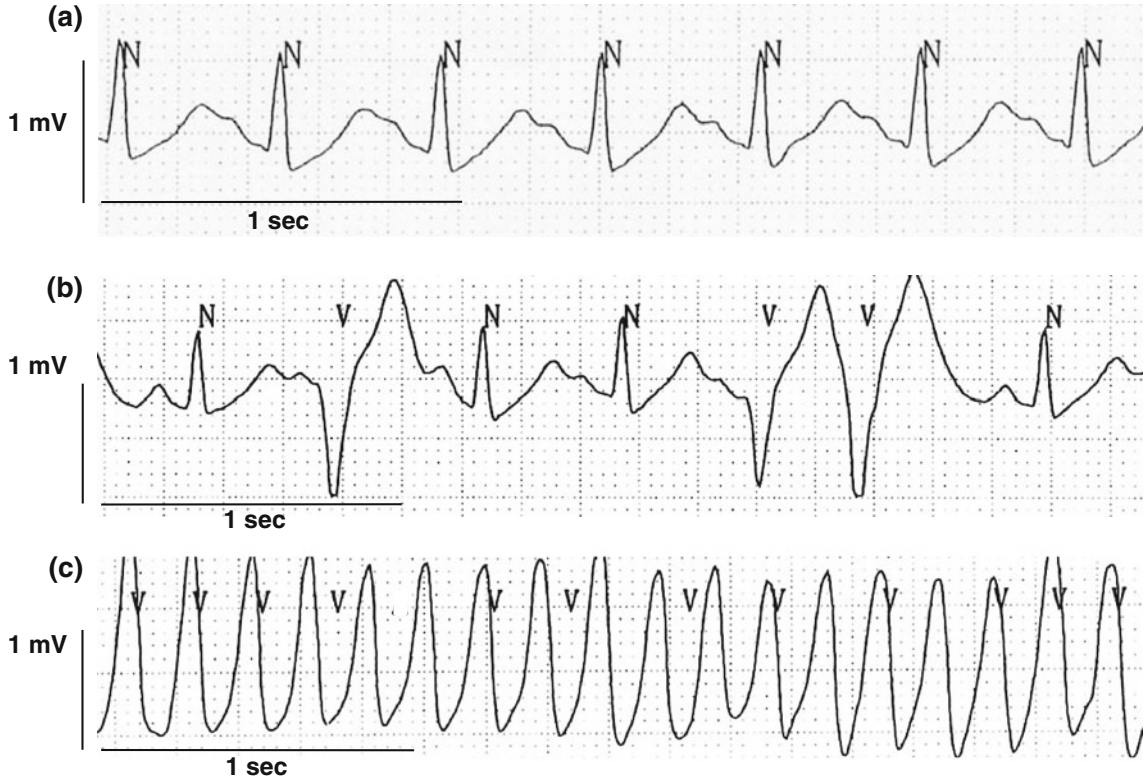

Fig. 1 Findings of the electrocardiogram on the heart monitor (lead II), showing sinus tachycardia (130 bpm) with a QTc interval of 0.46 s (**a**), single and couplets of premature ventricular contractions (**b**), and ventricular tachycardia (**c**)

monitor in the operating room included an ECG, noninvasive blood pressure (BP), SpO_2, capnogram, and axillary body temperature. Following the inhalation of oxygen (6 L/min) via a face mask for a few minutes, anesthesia was induced with intravenous thiopental (150 mg). About 10 or so seconds later, after checking for loss of consciousness, intravenous rocuronium (50 mg) was administered and bag-mask ventilation was performed with 100% oxygen. Three minutes after the administration of rocuronium, the brain was electrically stimulated using a pulse wave generator (Thymatron®, Somatics, Palo Alto, CA, USA). The mode of stimulation was as follows: pulse width, 0.5 ms; frequency, 20–30 Hz; current, 900 mA; duration, 7.0–7.5 s, and quantity of electricity, 120–200 mC, with successful mECT indicated by persistent tonic and clonic convulsions lasting longer than 20–30 s on the electroencephalogram (EEG). At the end of the seizures, the effect of rocuronium was reversed by intravenous sugammadex (200 mg), resulting in the prompt restoration of spontaneous respiration.

The first mECT was completed uneventfully without significant increases in BP and heart rate (HR). On his second scheduled mECT 2 days later, BP and HR with normal sinus rhythm were 100/60 mmHg and 69 bpm, respectively, on arrival in the operating room. The first stimulus, which induced considerable hypertension (BP 182/134 mmHg), sinus tachycardia (HR 130 bpm), and a slight prolongation (0.46 s) of QTc intervals (Fig. 1a), could not achieve the expected efficacy of seizure. Therefore, about 1 min after the first stimulus, a second one was applied consecutively without additional administration of thiopental, and effective convulsions were obtained. However, this second stimulus triggered monofocal isolated and couplets of premature ventricular contractions lasting for approximately 20 s (Fig. 1b) on ECG, followed by about 10 s of VT causing a loss of pulse and the disappearance of the finger plethysmogram on a pulse oximeter (Fig. 1c). Immediately, chest compression was performed several times, resulting in the successful restoration of spontaneous and normal sinus rhythm using no antiarrhythmic agent. Intravenous 0.5 mg of nicardipine for persistent hypertension after the restoration of sinus rhythm restored a normotensive state. SpO_2 was maintained at between 97 and 100% throughout the course of events. He recovered smoothly from anesthesia without any adverse consequences or postoperative complications. Because of this incidental VT, all subsequent attempts to perform mECT were canceled.

Postoperatively, close examination—including an ECG at rest, Holter ECG, echocardiogram, chest roentgenogram, and biochemical laboratory data—failed to clarify any possible cause of VT, such as long QT syndrome, Brugada

syndrome, early repolarization syndrome, torsades de pointes, ischemic heart diseases, disordered electrolytes of calcium, magnesium, potassium or other conditions.

Discussion

In recent years, mECT under general anesthesia has been widely employed to treat various kinds of drug-resistant depressive states. It is well known that mECT induces stimulation of the autonomic nervous system with initial parasympathetic outflow immediately followed by sympathotonic responses. Most of these responses, consisting of initial bradycardia, arrhythmias, and subsequent hypertension and tachycardia, are in general transient and recover spontaneously [3–9]. However, there have been several reports of life-threatening arrhythmias in relation to mECT, indicating that predisposing factors to these arrhythmias might involve pre-existing ischemic heart disease, suxamethonium-induced hyperkalemia, and tachycardia due to the use of atropine as premedication [10–16]. Concerning atropine, the authors administered it 30 min before transferring the patient to the operating room as routine premedication for mECT in order to decrease the salivary secretion, which is occasionally facilitated by antipsychotics. However, the patient did not demonstrate tachycardia, possibly induced by atropine, on arrival in the operating room. In our patient, therefore, these factors could be ruled out.

With respect to the QTc interval, it has been pointed out that mECT can provoke prolongation of QTc intervals [17, 18]. In our patient, the QTc interval spread to 0.46 s following the convulsions. However, the degree of prolongation was so small that it appears to suggest little clinical significance. Drug-induced QTc prolongation has been indicated for antipsychotics such as chlorpromazine and haloperidol, tricyclic antidepressants such as imipramine, amitriptyline and doxepin, and selective serotonin receptor inhibitors like fluoxetine [19], while none of the drugs potent enough to induce prolongation of the QTc interval were prescribed for our patient.

Macro- and microshock can be responsible for cardiac arrhythmias through electrical implements. The leakage currents derived from the pulse wave generator used for him, measured using a tester (3155 Leak Current HiTester®, Hioki E.E., Ueda, Nagano, Japan), were 11–17 μA: well below the values permitted by Japanese Industrial Standards for medical equipment. Accordingly, macroshock was ruled out as the reason for his arrhythmias.

Thus, in our case, the reason that the first electric stimulus failed to produce an effective convulsion and VT is still unclear. However, it is clear that, under the condition of uncontrolled hemodynamics, considerable sympathotonic increases in BP and HR following the electric stimuli augmented the cardiac oxygen demand, myocardial contractility, and serum catecholamine level, resulting in relative myocardial ischemia [20]. Therefore, the use of calcium channel and/or beta blockers before applying electric stimuli should be considered in order to attenuate hemodynamic hyperactivity [8, 15, 17]. We used nicardipine, a calcium channel blocker, against persisting hypertension even after the restoration of normal sinus rhythm. Had the nicardipine been administered prophylactically, these adverse events could have been prevented (although, to our regret, the BP and HR were not measured just before and after the second electric stimulus).

The authors did not administer thiopental additionally before the second stimulus. However, it might be necessary to check the level of awareness with the EEG in order to prevent awareness and, if necessary, additional thiopental should be given if the first stimulus fails.

The precise incidence of mECT-induced arrhythmias is unknown, but caution is advised, especially in patients at high risk for cardiac arrhythmias [21]. It is highly important to be aware that mECT has the potential to induce unpredictable and life-threatening arrhythmias like VT.

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