Case report

Sudden cardiac death after modified electroconvulsive therapy

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Summary: Sudden deaths associated with the use of electroconvulsive therapy are rare. In this case report a 58-year-old male with a 20-year history of bipolar disorder and no history or signs of cardiac illness died from cardiac arrest within one hour of receiving an initial session of modified electroconvulsive therapy (MECT) to treat a recurrent episode of non-psychotic mania. The patient regained consciousness and was medically stable immediately after the MECT session (which did not produce a convulsion) but deteriorated rapidly after transfer to the recovery room. It was not possible to conduct an autopsy, but the authors surmise that the most probable cause was that the use of haloperidol 17 hours prior to MECT exacerbated the cardiac effects of nonconvulsive MECT. The case highlights the need for a thorough cardiac work-up on patients being considered for MECT (possibly including assessment of cardiac enzymes in older individuals) and careful consideration of the concurrent use of antipsychotic medications and MECT.

Keywords: electroconvulsive therapy; sudden death; antipsychotic medication; bipolar disorder; China

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1. Case history

A 58-year-old unmarried man with a 20-year history of intermittent episodes of bipolar disorder was admitted to the hospital at 15:20 on December 19, 2013 because he had become excessively talkative and irritable over the previous 5 days. On admission he had clear consciousness and an untidy appearance. He was talkative, had flight of ideas, fluctuated between an elevated and an irritable mood, and was uncooperative during the examination (spitting at hospital staff). His orientation and intelligence were not checked because of his uncooperativeness. Family members reported that over the last few days he had been quite impulsive and his sleeping and eating were irregular. Based on ICD-10 diagnostic criteria, his admission diagnosis was bipolar disorder with a current episode of non-psychotic mania.

He had no history of coronary heart disease, diabetes, or other medical disorders. His admission electrocardiogram (ECG) showed a regular heartbeat of 100/minute and a QTc of 432 ms. Baseline blood work at admission showed that all electrolytes were within normal limits, a total white blood count of 9.56×10^9 /L, and a platelet count of 293×10^9 /L.

At 16:17 (one hour after admission), he was administered 5 mg of olanzapine. His excitement did not remit so an hour later he was also given 5 mg haloperidol and 0.3 mg scopolamine intramuscularly. However, his manic symptoms persisted so, after discussion among treatment staff, it was decided to start a course of modified electroconvulsive therapy (MECT) because, based on the report of the patient and family member, he had been successfully treated with MECT at a local hospital 6 years previously. The anesthesiologist made a preoperative visit to confirm the safety of anesthesia in the patient, family members signed the informed consent form, and ward staff made the preoperative preparations.

The patient entered the MECT observation room at 9:24 a.m. of the second day of admission. Preoperative preparations included confirming his identification and the status of food and water restriction, drug allergies, and stool and urine evacuation. His body weight was 65 kilograms, temperature was 36.5 °C, pulse rate was 96 beats/min, respiratory rate was 20 times/ min, and blood pressure was 120/86 mmhg. After an intravenous line was established, atropine 0.5 mg was

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administered – a routine preoperative procedure. The treatment procedures started when the patient was wheeled into the MECT treatment room and connected to an ECG monitor and a Spectrum Electroconvulsive Therapy Device. The preoperative exam had the following results: blood pressure was 127/84 mmhg, respiratory rate was 18 times/min, pulse rate was 99 times/min, blood oxygen saturation was 100%, and the resistance was 680 ohms. Oxygen was given via a mask attached to the anesthesia machine. The anesthesia nurse then administered 16 mg etomidate and 70 mg succinylcholine. At 10:00 a.m. the patient was in a state of deep anesthesia and bitemporal MECT was administered. The electric charge was 280 mc, the energy was 37.2 J, the static resistance was 530 ohms, the dynamic resistance was 161 ohms, the current was 800 mA, and the duration was 1.750 sec. The electroencephalogram showed that no epilepsy waveform occurred. At that time, the blood pressure was 158/103 mmhg, the pulse was 132 times/min, and the blood oxygen saturation was 100%.

At 10:09 a.m., the patient woke up and began spontaneous breathing with a respiratory rate of 12 times/min, blood pressure of 148/100 mmgh, pulse rate of 102 beats/min, and a blood oxygen saturation of 100%. He had eyelash reflexes and responded to pressure on the orbital ridge, thus the Steward Recovery Awake Score was rated as 2. The patient continued to receive oxygen via a mask. At 10:15 a.m., the Steward Recovery Awake Score was 3, spontaneous breathing was steady, strength of limb muscle was grade V, muscle tension was normal, both pupils reacted to light, his respiratory rate was 15 times/min, his blood pressure was 146/96 mmhg, his pulse was 96 beats/ min, and his blood oxygen saturation was 100%. Thus he met the standard for transfer to the observation room, so the patient's bed was wheeled into the observation room and he was connected to a palmstyle blood oxygen saturation monitor and an electronic sphygmomanometer. At that time his blood pressure was 140/92 mmhg, his pulse was 90 times/min, and his blood oxygen saturation was 100%.

However, after transfer to the recovery room his condition deteriorated rapidly:

- At 10:22 a.m. his pulse was 70 beats/min, his blood pressure was 130/70 mmhg, and his blood oxygen pressure was 95%.
- At 10:23 a.m. his pulse was 58 beats/min, his blood oxygen saturation was 82%, and he became confused though he was able to respond when called. Pressurized oxygen was given through a simple respirator. There was some resistance, but the ventilation was fair.
- At 10:24 a.m. his pulse was 41 beats/min and his blood oxygen saturation was 86%.
- At 10:25 a.m. his pulse suddenly dropped to 0 beats/min, his blood pressure dropped to 60/20 mmhg, and his blood oxygen saturation

was 72%. His face became pale, his lips became cyanotic, he did not respond to being called, the light response in both pupils disappeared, no carotid pulse was detectable, and his breathing was weak.

- At 10:26 a.m. he was returned to the MECT treatment room for cardiopulmonary resuscitation. He was given oxygen by the manual oxygen mask attached to the anesthesia machine. The ECG monitor showed that his pulse was 0 beats/min, his ECG was a straight line, his blood pressure could not be detected, and his blood oxygen saturation was 52%. The cardiopulmonary resuscitation continued. A laryngeal mask airway was established and oxygen was given using the anesthesia machine.
- At 10:29 a.m. the ECG monitor showed a straight line. The cardiopulmonary resuscitation continued and 1 mg of adrenaline was intravenously injected every three to five minutes.
- At 11:08 a.m. the ECG monitor still showed a straight line, spontaneous breathing had not recovered, there was no carotid pulse, and his pupils had become dilated and fixed, so he was declared dead.

2. Discussion

We were unable to obtain familial consent for an autopsy, so the definitive cause of death in this case remains unknown.

Reports from other countries^[1,2] indicate that sudden death associated with use of MECT is quite rare, occurring in approximately two cases for every 100,000 patients who receive MECT. In this case pretreatment food and water restriction, pre-operative use of atropine, and the lack of excessive oral secretions during resuscitation rules out the possibility of asphyxia induced by inhalation. That leaves the possibility that a fatal arrhythmia or cardiac arrest was induced by the MECT treatment, by the anesthetic, or by the antipsychotic medications and scopolamine the patient received approximately 18 hours prior to the MECT treatment.

Transient arrhythmia or cardiac arrest lasting up to 30 seconds can occur during or immediately after MECT,^[1,3] but there are no reports of this resulting in death. In this case the patient experienced no cardiac abnormalities during MECT and regained consciousness and was medically stable immediately after MECT, so the subsequent cardiac arrest was probably not a direct result of the anesthesia or MECT treatment. Moreover, the patient had received a course of MECT six years previously.

Subconvulsion stimulation from MECT can enhance stimulation to the parasympathetic nerve by inducing a sympathetic response and, thus, is potentially dangerous to patients with pre-existing heart disease. This patient had no history of heart disease and no symptoms or signs suggesting cardiac disease. However, he was 58 years old, so it is possible that he had an underlying heart condition not identified in the usual cardiac work-up for MECT.

The patient had been given 5 mg of olanzapine 18 hours before the MECT. The cardiac effects of olanzapine are minimal;^[4,5] dose-related sinus arrhythmias and ST-T changes can occur, but these changes are reversible and there is no evidence of more serious EEG changes, such as prolongation of the QT interval. We do not believe the use of olanzapine was related to the subsequent sudden cardiac death, though its use may have exacerbated the cardiac effects of the haloperidol that was given one hour later.

Haloperidol is widely used in clinical psychiatry. It is a relatively safe drug, and, like other antipsychotic medications, is considered safe when used in patients receiving MECT.^[6] But some earlier case reports from China show an association between haloperidol use and cardiac arrhythmias and sudden cardiac death.^[7-10] An intramuscular injection of 5 mg can cause elevated cardiac enzymes and ECG changes, which typically return to normal over 72 hours.^[11] Another study found that haloperidol can cause a significant decrease of effective circulating blood volume, increased myocardial oxygen consumption, and injury to myocardial cells.^[12]

In this case one 5 mg dose of haloperidol was administered with 0.3 mg of scopolamine about 17 hours prior to MECT. We surmise that this resulted in subtle cardiac changes that predisposed the patient to

改良电抽搐治疗后心源性猝死

王智慧,王继禹

概述: 与电抽搐治疗相关的猝死极其罕见。本文报告 中一例 58 岁男性患者在本疗程首次改良电抽搐治疗 (modified electroconvulsive therapy, MECT) 后 1 小 时 内死于心脏骤停。该患者有双相障碍病史 20 年,既 往无心脏疾病病史或指征,本次实施 MECT 为治疗不 伴精神病性症状的复发性躁狂发作。本次 MECT 并未 产生惊厥。治疗结束后病人立即恢复意识、生命体征 稳定,但被推入观察室后迅速恶化。我们未能进行尸 检,但推测最可能的原因是 MECT 前 17 小时使用了氟 哌啶醇,加剧了无抽搐 MECT 对心脏的影响。该案例

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asystole when exposed to the cardiac stress induced by nonconvulsive MECT. A pre-existing but undetected cardiac problem may have been an additional factor.

This case highlights several basic principles about the use of MECT. MECT is an effective therapy that is usually quite safe. Sudden death during or immediately after the use of MECT is rare, but it can occur in individuals with pre-existing heart conditions or in other, as yet unspecified, circumstances. Clinicians need to be diligent in conducting pre-treatment cardiac workups and, possibly, consider assessing cardiac enzymes in older patients who have no overt signs or symptoms of cardiac disease. The pre-treatment use of antipsychotic medications is not contraindicated, but antipsychotic medications that commonly induce arrhythmias should be used with caution, particularly in older patients.

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Conflict of interest statement

The author reports no conflict of interest related to this manuscript

Informed consent

The patient's brother provided written informed consent to publish this case history.

强调对准备用 MECT 的患者进行全面的心脏检查是必须的(对老年人也许还要检测心肌酶谱),并且需慎 重考虑同时使用抗精神病药物和 MECT。

关键词: 电抽搐治疗; 猝死; 抗精神病药物; 双相障碍; 中国

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