CASE REPORT

Ventricular tachycardia and hypertensive crisis induced by routine neuromuscular blockade reversal – a case report and literature review

Ksenija Jovanović1, 2, Ranko Trailović1, 2, Perica Mutavdžić2, 3, Ivan Tomić2, 3, Miloš Sladojević2, 3, Andreja Dimić2, 3, Marko Dragaš2, 3, Igor Končar2, 3, Lazar Davidović2, 3

1 Center for Anesthesiology and Resuscitation, University Clinical Center of Serbia, Belgrade, Serbia; 2 Faculty of Medicine, University of Belgrade, Belgrade, Serbia; 3 Clinic for Vascular and Endovascular Surgery, University Clinical Center of Serbia, Belgrade, Serbia.

Summary

Introduction: Neuromuscular blockade reversal may provoke cardiovascular events. We present the case of a middle-aged, vascular patient in whom ventricular tachycardia and hypertensive crisis occurred immediately after the initiation of neuromuscular blockade reversal.

Patient Review: A 56-year-old man was admitted to our institution for emergency surgical treatment of left extremity ischemia. Besides hypertension, the patient’s medical history was negative for any other significant acute or chronic diseases. Routine preoperative analyses, ECG, and echocardiography showed normal findings. The patient underwent urgent trans popliteal thrombectomy under general endotracheal anesthesia. Immediately following the initiation of neuromuscular blockade reversal, with a diluted mixture of atropine and neostigmine (1 mg/2.5 mg, respectively), ventricular tachycardia and hypertensive crisis developed. Arterial blood gas analyses and electrolyte values were within the reference limits at the moment. Adequate therapeutic measures were undertaken immediately, and sinus rhythm was restored shortly. Later, serial ECG records showed negative T waves in the precordial leads, with an increasing trend of troponin I values (ranging from 0.59 to 5.45 μg/L). Coronary angiography was later performed and revealed a normal coronary angiogram. During the next three days, ischemic ECG alterations showed resolution and the patient was hemodynamically stable. On the fifth postoperative day, the patient was discharged, in good general medical condition.

Conclusion: A careful administration of the atropine/neostigmine mixture is advised. Malignant arrhythmias must be recognized and treated promptly by an anesthesiologist, in order to achieve positive treatment outcomes.

Keywords: neuromuscular blockade reversal; ventricular tachycardia; hypertensive crisis.
INTRODUCTION

Neuromuscular blockade reversal (NMBR), along with anesthesia awakening and tracheal extubation, represents one of the most critical points of general endotracheal anesthesia (GETA). Among various complications that may emerge during that phase of anesthesia management, cardiac arrhythmias deserve special attention. Although cardiac rhythm disorders that develop during this period are often transient and benign, more complex dysrhythmias may develop, as well. Some of those arrhythmias, if not treated promptly, may be life-threatening, even in patients without cardiac comorbidities. Ventricular tachycardia (VT), a malignant cardiac rhythm disorder, is a wide-QRS-complex tachycardia, with a sudden onset, a regular rhythm, and heart rate (HR) higher than 100 beats per minute (bpm). VT most commonly occurs in the presence of structural heart disease. (1) Still, in up to 10% of patients, an obvious structural heart disease has not been identified. (2)

We present a case of simultaneous development of VT and hypertensive crisis (HC) following a routine NMBR in a middle-aged healthy vascular patient.

CASE REPORT

A 56-year-old male was admitted to our institution, due to pain and coldness in his left leg. Pedal pulses were absent and Color Doppler Sonography of left limb blood vessels showed distally occluded left popliteal artery, as well as its branches, by a hypoechoic mass. The patient’s medical history, besides hypertension, was negative for any acute or chronic diseases (including cardiovascular system-related events, such as myocardial infarction, significant arrhythmias, and symptoms of angina pectoris). The patient was a smoker, he denied allergies, previous operations and narcotic consumption. Routine blood, blood gas, and urine analyses showed no disturbances from reference values, except for the deviations related to an acute vascular disease. Preoperative electrocardiogram (ECG) and echocardiography (ECHO) were performed and they showed no disorders either (ECG: sinus rhythm with no changes in the ST segment and T wave; ECHO: structurally normal heart, left ventricle ejection fraction 62%, with no kinetics disorders). (Figure 1)

Due to acute left limb ischemia, the patient underwent trans popliteal thrombectomy under GETA. Anesthesia was induced with 5 mg of midazolam, 80 mg of propofol, and 100 μg of fentanyl. Succinylcholine (80 mg) was used to facilitate tracheal intubation and rocuronium-bromide was subsequently added to maintain muscle relaxation. In order to reach adequate anesthetic depth, sevoflurane, in an oxygen–air mixture was administered. Vital parameters were continuously monitored: oxygen saturation, end-tidal CO\(_2\), blood pressure (BP), HR, and ECG. The patient was hemodynamically stable during the whole procedure and the intraoperative course was uneventful. An average heart rate was 75 bpm and mean arterial pressure was 100 mmHg. (Figure 2)

Following the initiation of NMBR, by diluted atropine–neostigmine mixture(1 mg–2.5 mg, respectively), ECG showed ventricular tachycardia (HR=157 bpm) with cardiac axis deviation, bizarre wide QRS-complexes and capture beats, accompanied by the hypertensive crisis (BP=235/171 mmHg). (Figure 3)

Following a bolus injection of lidocaine (1.5 mg/kg), the continuous infusion was initiated, at the rate of 1 mg/kg/hr. Within two minutes, VT converted to sinus rhythm. Glycerol nitrate was fractionally administered intravenously and as continuous infusion, with metoprolol boluses, so blood pressure lowered. At the time, arterial blood gas analysis and blood electrolytes revealed normal values. When the patient was hemodynamically stabilized, no other active intervention was done, and he was transferred to the Intensive care unit. Three hours later, a serial 12-lead ECG revealed negative T waves in
precordial leads. Troponin I value showed an increasing trend (from 0.59 up to 5.48 μg/L). Later, coronary angiography was performed and it showed a normal coronary angiogram. (Figure 4) During the next three days, negative T waves showed resolution; the patient was hemodynamically stable. On the fifth postoperative day, the patient was discharged, in good general medical condition.

DISCUSSION

Anesthesia emergence and tracheal extubation often provoke hypertension and tachycardia. These cardiovascular alterations may precipitate the occurrence of additional cardiac rhythm abnormalities and may even lead to the development of myocardial ischemia in patients with coronary arterial disease. (3) On the other hand, in patients without structural heart disease, hemodynamic disturbances which develop spontaneously and with no obvious reason during anesthesia awakening may be associated with NMBR.

In an anesthetized patient, without structural heart disease, VT may develop due to many reasons, such as metabolic/electrolyte abnormalities, hypoxia, hypercarbia, anesthetic overdose, myocardial ischemia, and IV use of adrenaline and other catecholamines. Furthermore, life-threatening ventricular arrhythmias may develop in patients without coronary artery disease due to Takotsubo cardiomyopathy. (4) This stress-induced cardiomyopathy which occurs due to the massive release of catecholamines may lead to cardotoxicity, multivessel coronary vasospasm, and abnormalities in coronary microvascular function. (5) Still, all the above-mentioned factors were ruled out in our patient. Since the cause of this malign arrhythmia may remain unknown, (6) we can only assume that the atropine-neostigmine mixture, along with anesthesia awakening, over-stimulated our patient’s cardiovascular system, predisposed the coronary arteries to spasm and thus the autonomic control of the heart was altered. Our assumption is supported by the fact that, although generally considered safe, this anticholinesterase-anticholinergic combination may have a potential proarrhythmic effect. (7)

In order to further investigate the possible cause of this substantial but, fortunately, timely managed cardiovascular event in our patient, we have performed a literature review. Several relevant electronic databases were systematically searched using a combination of the following keywords: “neuromuscular blockade reversal”, “atropine and neostigmine”, “ventricular tachycardia” and “hypertensive crisis”. The results of our review suggest that, although routine NMBR may precipitate significant hemodynamic disturbances, only a few similar cases were described. Back in 1995, Rodríguez J. et al. presented a case of bradycardia and asystole following atropine-neostigmine administration in a patient who underwent a Caesarean section. The authors partially attributed the event to the effects of methyldopa, which was used for the management of pregnancy-induced hypertension, but they believed that NMBR agents might have contributed. (8) In 2004, Liaquat et al. presented the case of successfully managed cardiac arrest induced by routine NMBR. (6) Similar events were also described in children - Tüfek A. et al. published a case of cardiac arrest in a healthy18-month-old child. Besides the immaturity of the parasympathetic nervous system, the authors associated this event to the direct effects of the atropine/neostigmine mixture. (9) The rest of the cases in the available literature which describe similar cases refer to the patients with structural heart abnormalities (10, 11) or to another combination of medicaments used for NMBR. (12) So, to the best of our knowledge, no other report presented the exact case as ours.
CONCLUSION

The report emphasizes the need for careful atropine-neostigmine mixture administration, even in healthy patients, without structural heart diseases. It also suggests that malignant cardiac rhythm disorders must be recognized and treated promptly, in order to achieve positive outcomes, which mostly depend on the anesthesiologist’s timely intervention.

Author Contributions

1) the conception or design of the work: K.J., R.T., P.M., M.S.; 2) the acquisition: R.T., I.T., P.M., A.D.; 3) analysis, or interpretation of data: AD, MS, IK; 4) preparing the draft of the manuscript: K.J., I.T., M.D., L.D.; 4) interpretation of revised version of manuscript: K.J., I.K., M.D., L.D.

References


VENTRIKULARNA TAHIKARDIJA I HIPERTENZIVNA KRIZA IZAZVANE RUTINSKOM REVERZIJOM NEUROMUSKULARNE BLOKADE: PRIKAZ SLUČAJA I PREGLED LITERATURE

Ksenija Jovanović1,2, Ranko Trailović1,2, Perica Mutavdžić2,3, Ivan Tomić2,3, Miloš Sladojević2,3, Andreja Dimić2,3, Marko Dragaš2,3, Igor Končar2,3, Lazar Davidović2,5

Sazetak

Uvod: Reverzija dejstva miorelaksanasa može dovesti do nastanka kardiovaskularnih poremećaja tokom buđenja iz anestezije. Prikazujemo vaskularnog bolesnika kod koga je došlo do nastanka ventrikularne tahikardije i hipertenzivne krize, neposredno nakon započetka reverzije neuromuskularne blokade.


Ključne reči: reverzija neuromuskularnog bloka; ventrikularna tahikardija; hipertenzivna kriza.

Primljen: 06.05.2022. I Revizija: 16.08.2022. I Objavljen: 20.08.2022