Letters to Editor

Cardiac arrest after administration of sugammadex as neuromuscular blockade reversal agent and full recovery from anesthesia

Madam,

We report a case of cardiac arrest after the administration of sugammadex as neuromuscular blockade reversal agent and full recovery from anesthesia.

A 54-year-old male who was 175 cm tall and weighed 75 kg was scheduled for elective hernia-plasty repair under general anesthesia. The patient smoked 20 cigarettes/day for the past 28 years but had no other specific past medical history. He did not report any allergies, medication taken at home, or previous surgeries. A preoperative electrocardiogram (ECG) was done, and the patient was evaluated by a cardiologist, with no abnormal findings. The results of the chest X-ray examination and other preoperative laboratory tests were normal. The patient entered the operating room without premedication. ECG, noninvasive blood pressure (NIBP), end-tidal carbon dioxide, and oxygen saturation (SpO₂) were monitored. The patient’s initial vital signs were: NIBP 143/77 mmHg; SpO₂ 98%; and heart rate 67 beats·min⁻¹. General anesthesia was induced with midazolam 1 mg, fentanyl 100 mcg, propofol 150 mg, and rocuronium 50 mg. Mask ventilation was applied with 100% oxygen, and tracheal intubation was done without incidents 3 min after rocuronium administration. Another 150 mcg fentanyl was administered and the anesthesia was maintained with propofol and remifentanil continuous infusion and O₂/NO₂ mixture. The operation was completed uneventfully and lasted 1 h. The total doses of propofol and remifentanil were 450 mg and 30 mcg, respectively. The patient also received ranitidine 50 mcg, ondansetron 8 mg, and dexamethasone 8 mg. An additional 20 mg of rocuronium was administered during the operation to maintain muscle relaxation, so the total dose of rocuronium was 70 mg. The total fluid input was 800 ml crystalloid with no significant bleeding. At the end of the surgery, the administration of propofol and remifentanil was stopped, the infusion set was discarded, and the intravenous cannula was flushed with saline. O₂ 100% was delivered. Since no neuromuscular monitoring was used, the patient was waited to make a random movement before reversing the neuromuscular agent. Soon after, the patient not only had automatic breathing but also responded to commands, so sugammadex 200mg was given IV and trachea extubated. Right after the extubation, the patient’s vitals were: NIBP 145/82, HR 65 beats·min⁻¹, and SpO₂ 99%. He was asked his name and responded appropriately. The patient was taken off monitor, ready to be transferred to postanesthetic care unit. At that point, the nurse noticed that the IV flow was obstructed due to wrong position of the patient’s hand and it was then moved to restore the flow. During the next minute, and while still in the operating room, the patient got unresponsive, apneic, and pulseless. Immediate monitoring revealed asystole as cardiac rhythm and a SpO₂ 45%. CPR was started immediately. Resuscitation lasted 40 min, during which epinephrine 8 mg and amiodarone 450 mg were administered and the patient was defibrillated 6 times, and reintubation done, according to the advanced life support algorithm of 2015. After 5 min of CPR EtCO₂ was 15 mmHg and after 40 min the patient obtained a sinus rhythm but remained unresponsive. A new blood sample was sent to the biochemistry laboratory, with no significant results, other than troponin t level which was 1227 ng·l⁻¹, which was attributed to the six defibrillations he sustained. There were no echocardiographic findings and no ST-segment abnormalities in the ECG after return of spontaneous circulation (ROSC). Magnesium was also measured 2.2 mg·dl⁻¹. A head CT revealed no significant findings and the patient was transferred to the ICU. Six hours after the sugammadex administration, the patient got hemodynamically stable. Tracheal extubation was performed 2 days later with the patient neurologically intact. He was transferred to general ward the next day and was discharged after 2 more days.

In our case, an otherwise healthy patient had a cardiac arrest after sugammadex administration. ROSC was obtained only after 40 min of CPR and hemodynamic stability after 6 h. The cause of cardiac arrest is unclear. First, we assumed wrong drug administration and specifically rocuronium instead of sugammadex. However, there were no signs of suffocation that should occur before rhythm abnormalities and asystole occurred too soon to be contributed to hypoxemia. Recurarization was another possible scenario. In the absence of neuromuscular blockade monitoring, blind reversal of rocuronium with sugammadex can lead to a train-of-four (TOF) ratio less than desirable, as shown by Kotake et al. Again, limited time and absence of warning signs of recurarization (the patient did a successful head lift and was fully responsive) and hypoxemia before the cardiac arrest made us look for another cause. Second, we thought of a myocardial infarction or coronary spasm, as the patient had history of smoking, but echocardiography did not reveal such findings, and the ejection fraction was 60%. Moreover, a CT pulmonary angiogram excluded pulmonary embolism.

We suspected that sugammadex administration was the cause, based on the timing of the incidents. Moreover, the sugammadex datasheet clearly states “Cases of marked
bradycardia, some of which have resulted in cardiac arrest have been observed within minutes after the administration of [sugammadex].”[2] An intradermal testing for sugammadex and rocuronium hypersensitivity was not performed later, but there was no evidence of an allergic reaction such as rash and urticaria.

In Ko et al.’s case report, a 76-year-old patient developed ventricular premature contraction bigeminy, which led to cardiac arrest. Cardiopulmonary resuscitation was done three times before the patient was stable. The patient was finally diagnosed with variant angina, previously unknown.[3] Sanoja and Toth reported drug-resistant bradycardia, which led to cardiac arrest in a 60-year-old man with no known previous cardiac condition, attributed to sugammadex administration.[4] Bhavani reported two more cases of sugammadex-induced bradycardia and cardiac arrest in individuals with no previous heart history with sugammadex doses similar to ours, in which the patients were also resuscitated successfully.[5] Hoshino et al. also reported a patient who had repeated cardiac arrests due to coronary vasospasm related to sugammadex administration.[6] In at least three of the reported cases the patients had severe comorbidities or unknown previous cardiac disease.[3,5] Moreover, two of the cases had mild respiratory acidosis, as CO$_2$ was used during the endoscopic procedures.[5] These facts may have acted in favor of the cardiac arrest.

To our knowledge, this is the first such incident to be reported in European territory, and also the first sugammadex‑related cardiac arrest which has developed a shockable rhythm. Sugammadex was approved in Europe in 2008 for reversal of neuromuscular blockade induced by rocuronium and vecuronium and has been widely used since. However, data from recent case reports indicate serious side effects associated with the use of sugammadex.[3,6] Considering that sugammadex has become an everyday ally in clinical practice, we can only expect more and more such incidents. Therefore, we need to be extremely cautious with its use and never limit our monitoring of the patient during sugammadex administration.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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