LETTER

Cardiac arrest following sugammadex administration during inguinal hernia repair

Dear Editor,

We present a case of sustained cardiac arrest after the administration of sugammadex in a patient undergoing inguinal hernia repair. A 62-year-old man was scheduled for elective inguinal hernia repair. The patient was classified American Society of Anesthesiologists physical status (ASA-PS) II, suffered from arterial hypertension, and had a history of transient ischemic attack three years before (without residual impairment). His metabolic equivalents (METs) were evaluated over four METs; he was a previous smoker, made daily use of alcohol, and did not report allergies. Preoperative chest X-ray, electrocardiogram (ECG), and laboratory tests were within the normal range.

The patient was transferred to the surgical room, and initial standard monitoring included noninvasive blood pressure (NIBP), ECG, oxygen saturation (SpO_2), and end-tidal carbon dioxide. His vital signs were initially NIBP 135/85 mmHg, heart rate 85 bpm, and SpO_2 96 %. General anesthesia was induced with propofol at two mg/kg, fentanyl at four mcg/kg, and rocuronium at 0.6 mg/kg. Desflurane was set for anesthesia maintenance. The total duration of the operation was about 45 minutes. Before the end of the operation, intravenous (IV) ondansetron 4 mg and paracetamol 1 g were administered. At the end of the operation, sugammadex 2 mg/kg IV was administered to reverse the remaining neuromuscular blockade (TOF 25 %). The patient smoothly opened his eyes with a good level of consciousness, hemodynamic profile, and oxygenation, so he was extubated. A few seconds after the extubation, the patient developed bradycardia with concomitant ST-segment elevation. Atropine 1 mg was intravenously administered without success. The ST elevation was changed to slow idioventricular, rapidly turning into asystole. Cardiopulmonary resuscitation (CPR), according to 2015 guidelines, was initiated, and epinephrine 1 mg was given. Spontaneous circulation was returned several times (the first after 20 min of CPR). Unfortunately, it could not be maintained for a significant period, and multiple episodes of asystole and ventricular fibrillation occurred. Finally, a return of spontaneous circulation was achieved and maintained after about one hour of CPR. The patient was immediately transferred to the cardiac catheterization lab for coronary angiography, which showed no pathological findings. After the angiography, the patient was transferred to the intensive care unit and was extubated two days later without any neurological deficit. A month later, the patient underwent a cardiac spect tomography scan, which was not positive for induced ischemia.

Most sugammadex's anaphylactic side effects were presented as bradycardia, asystole, and cardiovascular collapse[1]. In a report by Samara et al, a 54-year-old patient developed sugammadex-induced cardiac arrest with shockable rhythm[2]. The sugammadex briefing document states that an episode of ventricular fibrillation was mentioned in the Pooled Phase 1-3 phases. The specific episode occurred eight days after administration and was not attributed to sugammadex. However, post-market experience from 25/07/2008 to 22/04/2015 described six episodes of ventricular fibrillation[3].

The sequence of ECG changes (bradycardia with ST elevation, ventricular fibrillation) in our case led us to suspect intraoperative ischemia. Coronary angiography revealed neither the presence of a thrombus nor critical stenosis.

Finally, we believe that we can rule out the possibility of an anaphylactic reaction. Although a tryptase check was not performed due to the inability of our biochemical lab to perform the test, the absence of any other clinical signs (skin reactions, edema, and bronchospasm) made the diagnosis of anaphylaxis rather unlikely.

Keywords: Suggamadex, cardiac arrest, ventricular fibrillation

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Conflicts of interest
None.

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