

CORRESPONDENCE

Slow recovery after sugammadex bolus after rocuronium-induced anaphylaxis

Editor—Some case reports have suggested the use and efficacy of sugammadex during rocuronium-induced anaphylactic shock.^{1–3} This was associated with rapid restoration (under 2 min) of the clinical signs of shock. We report a case who required a longer resuscitation after similar treatment. The patient gave approval to publish the case.

A female patient of 65 yr age, 70 kg, 1.60 m was to undergo a colectomy for adenocarcinoma. Her medical history included previous hysterectomy, and ventricular extrasystoles treated with flecainide (stopped the morning of surgery). She gave no history of any allergies. The day before the surgery, she received an oral premedication with hydroxyzine 50 mg. Routine monitoring was used and the variables were recorded every 5 min.

On arrival in the operating theatre, the baseline observations were: arterial pressure (AP) 145/96 mm Hg, heart rate (HR) 78 beats min⁻¹, and oxygen saturation (Sp_O₂) 99%. General anaesthesia was induced with midazolam (2 mg), sufentanil 25 µg (0.35 µg kg⁻¹), propofol 150 mg (2.2 mg kg⁻¹), and ketamine 20 mg (0.3 mg kg⁻¹). Rocuronium 50 mg (0.7 mg kg⁻¹) was then administered to facilitate tracheal intubation. No antibiotic was administered. Two minutes after receiving rocuronium, the patient developed sinus tachycardia (130 beats min⁻¹), hypotension (65/45 mm Hg), hypocapnia, bronchospasm, and oxygen desaturation (92%). Airway pressures increased. These clinical signs suggested a rocuronium-induced, grade II anaphylactic reaction. The patient was treated with oxygen 100%, i.v. epinephrine 100 µg and 10 µg (repeated 10 times), and a rapid infusion of 1000 ml Ringer's lactate solution. Thirteen minutes after appearance of the first clinical signs of anaphylaxis, and despite these treatment, AP was not measurable. With evolution to a grade III anaphylactic reaction, we increased the intermittent bolus dose of epinephrine to 100 µg and administered an i.v. bolus of sugammadex 1000 mg (14 mg kg⁻¹). The train-of-four count increased from 0/4 to 4/4 over 1 min. In a 7 min period after sugammadex administration, the patient was still being treated with i.v. epinephrine 100 µg × 3 and rapid infusion of 500 ml 0.9% NaCl and 500 ml hydroxyethylamidon. Ten minutes after sugammadex, the haemodynamic variables were: HR 118 beats min⁻¹, AP 47/28 mm Hg, Sp_O₂ no trace, F_{E'}CO₂ 2 kPa. The inflation pressures remained high. The clinical improvement from the signs of anaphylaxis was seen after two boluses of epinephrine 1 mg. Fifteen minutes after sugammadex injection, haemodynamics parameters were: HR 115 beats min⁻¹, AP 85/35 mm Hg, Sp_O₂ 92%, F_{E'}CO₂ 4 kPa. The inflation pressures decreased. Continuous epinephrine was then given at 2 mg h⁻¹ until tracheal extubation 2 h later. Surgery was deferred. The patient made an uncomplicated recovery and was informed that a likely anaphylactic reaction had occurred during anaesthesia.

The anaphylactic reaction was confirmed by the mast cell tryptase from blood sample drawn during the event; the level reached 200 µg litre⁻¹ (normal <13.5 µg litre⁻¹). Serum-specific IgE against neuromuscular blocking drugs were 7.3% of fixation for quaternary ammonium (confirmation of allergy >2%) and 49% of inhibition for rocuronium (interpretation: allergy >20%). Six weeks after the event, skin prick tests were positive to rocuronium (1:10 dilution), vecuronium (4 mg ml⁻¹), and succinylcholine (10 mg ml⁻¹). Skin prick tests were negative to non-steroidal neuromuscular blocking drugs, midazolam, sufentanil, and latex. Intradermal skin testing was performed using 1:100 dilution of mivacurium and atracurium and 1:10 of cisatracurium. They remained negative.

To our knowledge, this is the first published case of allergy to rocuronium confirmed by positive skin testing, where sugammadex administration did not induce recovery from the clinical signs of anaphylaxis as in the earlier reports.^{1–3} Dosage of sugammadex could be an important issue.³ Fifteen minutes after an injection of rocuronium 0.6 mg kg⁻¹, sugammadex 6 and 8 mg kg⁻¹ are needed to obtain a T4/T1 count 0.9, respectively, in 2.0 and 1.5 min.⁴ However, in our case, there was no underdosing (14 mg kg⁻¹ sugammadex). It is not clear whether sugammadex would be of benefit in these situations. One plausible mechanism is that formation of the rocuronium–sugammadex complex leads to the elimination of the quaternary ammonium epitopes on rocuronium molecules from circulation.⁵ However, it is difficult to understand in which patients sugammadex could alleviate the anaphylactic process so quickly. Further studies and publications of clinical cases are needed to understand and confirm if sugammadex can be efficient during rocuronium-induced anaphylactic reaction or if recovery was due to conventional treatment by epinephrine and fluid loading.

Declaration of interest

None declared.

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Interesting artifact: distortion of invasive arterial line trace from somatosensory evoked potential signal

Editor—An otherwise healthy 14-yr-old child underwent T3–L2 posterior spine fusion for progressively worsening idiopathic scoliosis. Invasive arterial pressure and intraoperative neurophysiological monitoring (IONM) were planned in addition to standard ASA monitoring for the procedure. After uneventful induction of general anaesthesia, a radial arterial line and electrodes for somatosensory evoked potential (SSEP—bilateral median and posterior tibial nerves) and trans-cranial motor evoked potential (MEP) were placed. General anaesthesia was maintained with 0.6 MAC of sevoflurane supplemented with remifentanyl infusion. Neuromuscular blockers were avoided in order to optimize MEP signals.

An hour into the procedure, without any abrupt changes in the depth of anaesthesia or surgical stimulus, we noted the sudden appearance of a persistent double systolic peak in the arterial line tracing with concurrent changes in the plethysmographic trace (Fig. 1). ECG tracing, arterial pressure, and SSEP signals remained stable during this episode. The arterial line tracing looked similar to pulsus bisferiens which has been described in patients with hypertrophic obstructive cardiomyopathy (HOCM) and aortic regurgitation.¹ The tracing suddenly reverted back to normal only to reappear intermittently. We noted that the distortion of arterial and plethysmographic waveform was temporally associated with repeated flexion of the wrist secondary to median nerve stimulation for SSEP monitoring and correlated with the stimulation frequency (~3 Hz) which was being used for SSEP.

Invasive arterial monitoring is a commonly used haemodynamic monitoring tool in the operating theatre and intensive care units and its signals are subject to artifacts arising from catheter clotting, transducer flushing, over- and under-damping, and various movements.²

Eipe and Bertram³ have reported similar interference to arterial line tracing from SSEP signals. In this particular scenario, the temporal association of the aberrant arterial and plethysmographic trace with the SSEP stimulation, approximation of the plethysmographic heart rate tracing with SSEP stimulation frequency, unaltered ECG trace, and stable haemodynamics all pointed towards a mechanical artifact rather than the unmasking of a potentially ominous clinical scenario (HOCM



Fig 1 Photograph of the screen of the monitor to show the artifacts.