Possible mitigation of rocuronium-induced anaphylaxis after administration of sugammadex

Sir,

We describe a case of rocuronium-induced anaphylaxis, which was possibly mitigated after administration of sugammadex. A 61-year-old woman (45 kg, 155 cm) was scheduled to have bone cementoplasty for pain reduction in relation to bone metastasis of a thyroid cancer. She previously had multiple thyroid surgeries without any allergy report and a history of hypertension. Her medication included propranolol, L-thyroxine, and opioids for chronic pain.

In our hospital, this procedure is generally performed under remifentanil sedation; however, because of severe pain she refused to have this procedure under sedation only, therefore general anesthesia was decided. Premedication was propranolol, 40 mg, and hydroxyzine, 50 mg, one hour before surgery. Standard monitoring was done with electrocardiogram, pulse oximetry, noninvasive blood pressure, and neuromuscular monitor. Her initial blood pressure was 120/65 mmHg with a pulse rate of 80/min. The following drugs were administered before tracheal intubation: propofol 2.5 mg/kg, remifentanil, target-controlled infusion 2 ng/ml, and rocuronium 0.6 mg/kg. After tracheal intubation, an initial end-tidal carbon-dioxide ($EtCO_2$) of 33 mmHg was recorded.

The blood pressure after intubation decreased to 100 mmHg, inhalational anesthesia was started with sevoflurane (end tidal - 1.5%). Five minutes after intubation, the blood pressure dropped to 65 mmHg, with a pulse rate of 110/min. 6 mg of ephedrine intravenous (IV) was injected and sevoflurane decreased to 0.5%. However, 2 min later, her pulse rate significantly increased to 150/min in addition to a decrease in $EtCO_2$ (15 mmHg) concomitant to an upper body rash and high respiratory airway pressure (40 mmHg), and auscultatory wheezing.

Ten minutes after intubation, blood pressure further decreased to 45 mmHg (systolic). At this point, we suspected an anaphylactic reaction and an IV bolus of epinephrine 0.2 mg was administered. At the same time, blood samples (total IgE and tryptase) were taken for allergic assessment. Twelve minutes after intubation, blood pressure increased to 75 mmHg but tachycardia, rash, wheezing, and high airway pressure persisted.

Decision was made to cancel the case. The neuromuscular monitor showed five responses under post-tetanic count stimulation, we decided to administer sugammadex 4 mg/kg only to reverse paralysis (14 min after intubation). A train of four ratio of 100 was achieved, 3 min after administration of sugammadex, surprisingly blood pressure increased to 100 mmHg, pulse rate dropped to 100/min, airway pressure decreased to 25 mmHg and $EtCO_2$ increased to 33 mmHg; however, her upper body rash remained (16 min after intubation). The patient was therefore awakened and trachea extubated and transferred to ICU for surveillance. No further hemodynamic instability was noticed and the rash disappeared a few hours later. She was transferred the day after to regular ward. Laboratory results, received a few weeks later, indicated to an anaphylactic reaction to rocuronium [tryptase > 36.5 μg/l (normal < 11.4), histamine > 100 nmol/l (normal < 10), specific antirocuronium IgE 74% inhibition (normal < 1%), and specific IgE for propofol (within normal range)]. The patient could not go for further cutaneous test because of a decline in her general status due to the cancer which needed rehospitalization.

We agree that specific antirocuronium IgE alone might not be sufficient to confirm rocuronium anaphylaxis, nevertheless a high predictive positivity of the specific antirocuronium IgE[1] and clinical events in the case of our patient made us almost certain of the diagnosis.[2] Mitigation of rocuronium-induced anaphylactic reaction by sugammadex is now under debate.[3-5] Since randomized studies are impossible, reporting of similar events and cases are mandatory. In our case, we only wanted to reverse paralysis; thus a dose of 4mg/kg according to manufacturer was used, but the subsequent hemodynamic stability and improvement of patient's condition surprised us. In contrast to Mc Donnell's case,[4] we had an initial good response to a single dose of epinephrine. Sugammadex is unlikely to have antianaphylactic properties, but could have improved the situation either by recovery of muscle tone when the patient awakened or by encapsulating rocuronium molecules and thus removing the antigen molecules from circulation. However, we cannot rule out spontaneous recovery. Caution is still necessary and before drawing any conclusions, more case reports should be analyzed.

Cyrus Motamed, Pascal Baguenard, Jean louis Bourgain

Service d'anesthésie Institut Gustave Roussy. Villejuif France. Rue Edouard Vaillant villejuif France

Address for correspondence: Dr. Cyrus Motamed,
Service d'anesthésie, Institut Gustave Roussy. Villejuif France.
114 rue Edouard Vaillant Villejuif France94805
E-mail: motamed@igr.fr

References

1. Leysen J, Bridts CH, De Clerck LS, Vercauteren M, Lambert J, Weyler JJ, *et al.* Allergy to rocuronium: From clinical suspicion to correct diagnosis. Allergy 2011;66:1014-9.
2. Ebo DG, Venemalm L, Bridts CH, Degerbeck F, Hagberg H, De Clerck LS, *et al.* Immunoglobulin E antibodies to rocuronium: A new diagnostic tool. Anesthesiology 2007;107:253-9.
3. Leysen J, Bridts CH, De Clerck LS, Ebo DG. Rocuronium-induced anaphylaxis is probably not mitigated by sugammadex: Evidence from an in vitro experiment. Anaesthesia 2011;66:526-7.
4. McDonnell NJ, Pavy TJ, Green LK, Platt PR. Sugammadex in the management of rocuronium-induced anaphylaxis. Br J Anaesth 2011;106:199-201.
5. Baldo BA, McDonnell NJ, Pham NH. Drug-specific cyclodextrins with emphasis on sugammadex, the neuromuscular blocker rocuronium and perioperative anaphylaxis: Implications for drug allergy. Clin Exp Allergy 2011;41:1663-78.

Access this article online	
Quick Response Code:	Website: www.joacp.org
	DOI: 10.4103/0970-9185.92464