

Correspondence

Rocuronium-induced anaphylaxis not improved by low dose sugammadex: a case report

Recently, sugammadex has been used to treat rocuronium-induced anaphylaxis. We report a case of rocuronium-induced anaphylactic shock, which failed to improve with sugammadex. The patient was a healthy 19-year-old man, scheduled for endoscopic sinus surgery. Written informed consent was obtained from the patient and his parents for publication of these details.

After arrival in the theatre, his baseline vital signs were normal. General anaesthesia was induced with propofol and remifentanyl. Rocuronium 50 mg was injected after the patient lost consciousness. Immediately after rocuronium injection, his heart rate (HR) increased, and airway resistance gradually increased. Three minutes after rocuronium injection, his trachea was intubated and mechanical ventilation was started. The peak inspiratory pressure increased to 27 cmH₂O and his systolic blood pressure (BP) fell to below 80 mmHg. Fluid resuscitation with crystalloid was commenced and intermittent doses of IV phenylephrine were given. After phenylephrine 1 mg had been administered, his systolic BP remained below 50 mmHg and his HR was 130 /minute. Anaphylaxis was clinically diagnosed and adrenaline 0.3 mg, chlorpheniramine 10 mg, and hydrocortisone 100 mg were administered. Immediately following IV adrenaline, his BP increased to 112/69 mmHg and the peak inspiratory pressure decreased. Because rocuronium was the most suspicious drug, sugammadex 200 mg (2.7 mg/kg) was administered, 24 minutes after rocuronium. However, hypotension and tachycardia persisted. After repeated adrenaline bolus (0.3 mg) injections, a continuous infusion was started. Blood analysis confirmed hypoxaemia, metabolic acidosis, and haemoconcentration; however, no hyperlactataemia was observed. The surgery was cancelled, and the patient was transferred to our intensive care unit.

Six hours after rocuronium injection, the patient's BP was maintained within the normal range with continuous infusion of adrenaline, and he was extubated. A total of 2.5 mg adrenaline had been given over the 12 hours since rocuronium administration. His tryptase level was 13,400 ng/ml and histamine level was 45.2 nmol/ml. Sixty days after the event, a skin prick test was performed. Rocuronium and vecuronium showed a positive reaction.

Recently, some case reports have suggested that sugammadex is effective for treating rocuronium-induced anaphylaxis¹⁻⁵. In the current case, we used 2.7 mg/kg of sugammadex because this was the smallest dose in the current case reports, but this seemed to have no effect. Some reports suggest that sugammadex does not behave in

a dose-dependent manner for the treatment of rocuronium-induced anaphylaxis¹⁻⁴. Early administration of sugammadex seemed to improve the outcome in some reports. Conte et al reported that sugammadex was administered six minutes after rocuronium injection, and improved the patient's haemodynamic state⁵. But other cases have been reported where sugammadex was injected 18 to 20 minutes after rocuronium injection, and adrenaline was required until 60 minutes later^{2,3}. Leysen et al suggest from their in vitro experiments that sugammadex can prevent, but not stop, basophil activation by rocuronium⁶. Furthermore, Platt et al have reported in their case control study that sugammadex did not improve the clinical course of rocuronium-induced anaphylaxis⁷. All cases received fluid resuscitation, adrenaline, and steroid treatment in addition to sugammadex.

Our case reinforces previous findings that suggest sugammadex does not have an immunological role in the management of anaphylaxis.

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