CASE REPORT

Sugammadex rescue following prolonged rocuronium neuromuscular blockade with 'recurarisation' in a patient with severe renal failure

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SUMMARY

We describe our experience of a 71-year-old patient with severe renal failure, who exhibited an unusually prolonged rocuronium-induced neuromuscular blockade (>4 h) and apparent recurarisation, following emergency rapid sequence induction (RSI). At the end of operation, 45 min post induction, train-of-four (TOF) testing had been 4/4 prior to wake up. No respiratory effort was seen 150 min postinduction, despite further neostigmine/glycopyrrolate and repeat TOF 4/4. The patient was reseeded and transferred to the intensive care unit (ICU). At 180 min postinduction, fade was evident on TOF, suggestive of rocuronium reblockade. At 285 min, the patient was extubated safely following sugammadex administration and discharged uneventfully from the ICU. An important lesson to recognise is the potential for extremely prolonged neuromuscular blockade following rocuronium in patients with severe renal failure, particularly when using the higher doses (1.2 mg/kg) required for RSI, and that TOF in such cases may not be reliable in detecting residual blockade.

BACKGROUND

We present a case of extremely prolonged rocuronium neuromuscular blockade (>4 h) in a patient with severe renal failure following rapid sequence induction (RSI). Within the literature, there have been a small number of reports of prolonged blockade with rocuronium (some >10 h) in patients with renal failure. Although these reports exist, within the anaesthetic community, most anaesthetists are aware of a potential prolongation of neuromuscular blockade with renal failure, but not to this extent. Also, sugammadex use in severe renal failure is relatively new and appears to be safe, providing a prompt resolution of the situation in this case, as blockade may have persisted for many more hours. We hope that this case report will help increase awareness and promote discussion of the potential for extremely prolonged neuromuscular blockade with rocuronium when severe renal failure exists.

CASE PRESENTATION

A 71-year-old patient with metastatic prostate cancer and acute-on-chronic renal failure secondary to a blocked ureteric stent was to undergo emergency stent change. His urea was 36.9 mmol/l and creatine 1080 µmol/l. Total venous carbon dioxide was noted to be low at 11 kPa. Rocuronium had previously been used uneventfully in this patient for RSI, and seemed a reasonable choice on this occasion.

Thiopentone (1.6 mg/kg, 135 mg), rocuronium (1.2 mg/kg, 100 mg) and fentanyl (50 µg) were administered with the patient being intubated uneventfully. Isoflurane and oxygen/air were used for maintenance of anaesthesia throughout. No further doses of rocuronium were given. Amoxicillin was the only antibiotic used intraoperatively.

The ureteric stent procedure lasted 45 min. Peripheral nerve stimulation at adductor pollicis indicated a train-of-four (TOF) of 4/4. Neostigmine and glycopyrrolate (2.5 mg/0.5 mg) were given to ensure complete reversal. Isoflurane was stopped and high-flow oxygen administered with pressure control ventilation. However, even after 90 min following induction, the patient did not make any respiratory effort. End-tidal CO₂ had risen to 8.0 kPa. A further dose of neostigmine and glycopyrrolate was given.

At this time, the reason for this patient failing to breathe was unclear, as multiple factors could have contributed to this state. TOF remained at 4/4, making inadequate reversal unlikely, and consideration was given to the possibility that metabolic or acid-base disturbances, as well as opiate accumulation, could account for the clinical picture. High flow oxygen and pressure control ventilation were continued. Occasional triggering of the ventilator was seen, but inadequate tidal volumes were reached. Doxapram (40 mg) was tried in an attempt to improve the respiratory effort but was ineffective. Arterial blood gas analysis showed the following: pH 6.8, pCO₂ 13.2 kPa, pO₂ 42.1 kPa and BE-2.0. The ventilation rate was increased, and 500 ml of 8.4% sodium bicarbonate was also administered to correct the acidosis. TOF remained at 4/4. The patient had been given fentanyl for analgesia during surgery. In view of this and possible opiate accumulation in a background of severe renal failure, naloxone (400 µg) was given.

Despite these measures, the patient still failed to make any adequate respiratory effort. At 120 min following RSI, TOF remained 4/4 with improving blood gases (pH 7.18, pCO₂ 9.2 kPa, pO₂ 37.6 kPa, BE 3.2).

At 150 min post induction, the patient was able to open his eyes and move his fingers and toes at will. Pressure control ventilation was continued. It was possible to communicate reasonably well with the patient who appeared calm and reassured by the explanations given. Power appeared to improve slowly, but at 180 min post induction, the patient was still unable to generate an adequate tidal

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volume. Blood gases were normal (pH 7.40, pCO₂ 5.2 kPa, pO₂ 12.8 kPa, Base Excess (BE) 0.5). However, neuromuscular testing showed fade creeping back into TOF with 3/4 twitches. A propofol infusion was started and the patient was transferred to a critical care bed for continued ventilator support, as sugammadex was not immediately available, owing to the drug having to be brought in from an off-site pharmacy.

In the intensive care unit (ICU), TOF continued to deteriorate reaching 2/4 at 4 h 45 min post RSI. At this point, sugammadex (6 mg/kg, 500 mg) was given intravenously. Within 1 min, TOF improved to 4/4. Sedation was stopped and the patient was able to raise both arms. The patient was extubated safely 10 min following sugammadex and discharged uneventfully from the ICU on the next day.

DISCUSSION

Since 1994, rocuronium has become widely available in the UK for neuromuscular blockade (NMB).¹ A monoquaternary aminosteroid, its introduction provided an alternative to suxamethonium for RSI, due to its comparable onset and favourable intubating conditions, at higher doses (1.2 mg/kg).¹² Rocuronium metabolism is mainly through hepatobiliary elimination, with 10–30% being renally excreted.¹³ There are many clinical situations and drugs that have been reported in the literature, which can potentiate or antagonise rocuronium NMB. For example, aminoglycosides are widely known to prolong NMB.^{2–5} Multiorgan failure is associated with prolonged rocuronium NMB, but the clinical significance of renal failure of varying severity and rocuronium metabolism was unclear initially, with conflicting reports of no discernible effect to prolongation of NMB and clearance.^{6–8} More recently, there have been a growing number of cases that have reported prolonged NMB following rocuronium in renal failure (some over 10 h), with most accepting that the contribution that renal clearance plays in the excretion of rocuronium, albeit small, appears to be significant, particularly in renal failure.^{9–13} The introduction of sugammadex has changed the way we use rocuronium, increasing its versatility and safety. Sugammadex is cleared renally. Although clearance of sugammadex is delayed in end-stage renal failure, it has been found to work rapidly and effectively in rocuronium reversal in renal failure without recurrence of NMB.^{14,15}

Our case illustrates prolonged NMB following rocuronium (1.2 mg/kg) in severe renal failure with evidence of recurarisation at 3 h post operation. Similar cases have been seen in the literature, where even smaller doses of rocuronium (0.6 mg/kg) resulted in NMB of over 10 h.^{9,10,12}

The observation that TOF at 3 h in our case began to fade is highly suggestive of rocuronium reblockade/recurarisation. There are also case reports of postoperative rocuronium reparalysis and residual neuromuscular blockade, where TOF was demonstrated to be 4/4 with full neuromuscular recovery post-operatively with unexpected rocuronium reparalysis occurring in the recovery room.^{16–19}

The aetiology behind rocuronium reblockade is likely to be multifactorial. However, in our case, the severity of renal failure and possible saturation of rocuronium excretion, the quick changes in metabolic/respiratory parameters and the sudden reversal of acidosis may be an explanation for persistent neuromuscular blockade and recurarisation.

It is quite likely that NMB would have been extremely prolonged if it were not for the intervention with sugammadex (6 mg/kg). Sugammadex, in this case, rapidly reversed persistent rocuronium NMB in a patient with severe renal failure with no adverse sequelae. The excretion of rocuronium-sugammadex

complex was found by Staals *et al*²⁰ to be prolonged in renal failure; however, clinically, this delayed excretion appears to be insignificant. Recently, elderly patients have been found to require a slightly longer recovery time from profound rocuronium NMB with sugammadex than younger patients, but again this factor did not affect our patient's recovery duration.²¹ It is worth noting that under-dosing of sugammadex has been shown to produce residual rocuronium blockade and reparalysis, but doses above 4 mg/kg are usually sufficient to prevent this occurrence.²²

It is important to recognise the potential for extremely prolonged NMB following rocuronium in patients with a background of severe renal failure, particularly when using the higher doses (1.2 mg/kg) required for RSI. In such cases, TOF may not be reliable in detecting residual neuromuscular blockade and there exists a potential for inadequate reversal and/or recurarisation. Sugammadex appears safe and effective in the reversal of prolonged rocuronium NMB in severe renal failure.

Learning points

- ▶ Important to recognise the potential for extremely prolonged neuromuscular blockade following rocuronium in patients with severe renal failure.
- ▶ Reminder that train-of-four testing may not be reliable in detecting residual neuromuscular blockade and potential for inadequate reversal/recurarisation even when full twitches are present.
- ▶ Sugammadex appears to be a safe and effective reversal agent for prolonged rocuronium-induced neuromuscular blockade in severe renal failure.

Competing interests None.

Patient consent Obtained.

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