Section: Anaesthesiology

Case Series

UNEXPECTED DELAYED REVERSAL OF VECURONIUM INDUCED NEUROMUSCULAR BLOCKADE USING SUGAMMADEX- A CASE SERIES

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ABSTRACT

Background: Sugammadex reliably reverses amino steroid neuromuscular blockade, but unexpected delayed clinical emergence after its administration has been reported anecdotally. We present a case series describing delayed recovery of consciousness and motor responsiveness following sugammadex given to reverse vecuronium, and we discuss possible explanations and practical recommendations. **Materials and Methods:** Retrospective case series of four patients in whom sugammadex (2 mg·kg^{-1}) was given to reverse vecuronium at the end of general anaesthesia. Clinical data extracted included anaesthetic and adjunct drug exposures, timing of last vecuronium dose and sugammadex administration, presence of spontaneous ventilation, and time to eye opening and extubation. Quantitative neuromuscular monitoring (TOF ratio) was not available for these patients. **Results:** All four patients demonstrated showed delayed recovery (delayed eye opening and motor responsiveness) inconsistent with rapid clinical emergence expected after adequate reversal. Common co-administered agents included dexmedetomidine, systemic lidocaine, opioids and volatile anaesthetics. No metabolic, temperature, or renal dysfunction explanations were identified in the available records. No objective TOF data were available to confirm residual peripheral neuromuscular blockade. **Conclusions:** These cases are hypothesis-generating and do not prove causality. Delayed clinical emergence after sugammadex may reflect central sedation from concurrently administered sedative/analgesic agents, co-administration or physical incompatibility, or true residual neuromuscular blockade that could not be documented in the absence of quantitative monitoring. We recommend routine use of objective neuromuscular monitoring (TOF ratio/acceleromyography) when reversing blockade, avoidance of injecting sugammadex through lines with active infusions without flushing, and careful documentation of recent sedative/analgesic dosing. Prospective studies with standardized neuromuscular and sedation monitoring are needed to determine whether pharmacologic or physical interactions contribute to delayed emergence.

INTRODUCTION

Sugammadex, a modified γ-cyclodextrin compound, is specifically utilised to reverse the impact of amino steroidal neuromuscular blocking agents (rocuronium and vecuronium).[1] In this process, amino steroidal neuromuscular blocking agents are encapsulated within the central core of sugammadex, rendering it irreversibly fixed and neutralised. Unlike acetylcholinesterase inhibitors such as neostigmine, which are employed to counteract partial neuromuscular blockade by non-depolarising muscle relaxants, sugammadex offers a safer profile by avoiding the induction of cholinergic effects. A recent Cochrane review highlighted that the use of neostigmine is associated with a higher risk of adverse events compared to sugammadex (28% vs. 16%, respectively).[2] The risk of significant adverse

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outcomes, such as residual paralysis, hypersensitivity and allergic reactions, postoperative nausea and vomiting, and bradycardia, was also lower with sugammadex than with neostigmine. However, the incidence of serious adverse events was similar between sugammadex and neostigmine, both hovering around 1%.[2]

Case Series

After approval from Institutional Research Committee and Institutional Ethical Committee retrospective case series of four patients who had a delayed recovery after giving sugammadex was analysed.

Case 1

24 years old female, with weight of 45 kg, ASA class 1 posted for endoscopic tympanoplasty of right ear under general anaesthesia. After attaching monitors, the patient was pre-oxygenated with 100% Oxygen for 3 minutes, pre-medicated with Inj. glycopyrrolate 0.2mg iv, Inj. midazolam 1 mg iv and Inj Fentanyl 2 mcg/kg iv. Patient was induced with Inj. Propofol 2 mg/kg iv and relaxed with Inj Vecuronium 0.08 mg/kg iv. Patient was intubated and anaesthesia was maintained with a mixture of O2, N2O, sevoflurane with intermittent positive pressure ventilation. Vecuronium .02mg/kg iv was repeated every 30 min. Inj. paracetamol 20 mg/kg iv was given after 1 hour of surgery and Inj. tranexamic acid 1g iv infusion was administered towards the end of surgery as per request of the surgeon. Surgery lasted 2.5 hours and the last dose of relaxant was given 21 minutes before skin closure. N2O was stopped before graft placement and sevoflurane was stopped 10 min prior to the administration of the reversal agent Inj. sugammadex 2 mg/kg iv. The drug was given in the same intravenous line of tranexamic acid infusion. There was a delayed incomplete action of sugammadex even after 15 min of injection. So Inj.Neostigmine 0.05mg/kg iv and Inj.glycopyrrolate 0.01mg/kg iv was given. After complete recovery the patient was extubated (24 minutes after Sugammadex injection) and shifted out to the recovery room after 5 minutes.

Case 2

43 years old female, weighing 60 kg, belonging to ASA PS class 2 with past history of total abdominal hysterectomy and bilateral salpingo-oophorectomy 4 years back under general anaesthesia, was posted for laminectomy and discectomy (L4,L5,S1). After attaching monitors, pre-oxygenated with 100% Oxygen for 3 minutes. Pre-medicated with Inj. glycopyrrolate 0.2mg iv, Inj. midazolam 1 mg iv and Inj fentanyl 2mcg/kg iv. Patient was induced with Inj. propofol 2 mg/kg iv and relaxed with Inj succinylcholine 1.5 mg/kg iv. After intubation anaesthesia was maintained with Intermittent positive pressure ventilation with O2, N2O and isoflurane. Inj. vecuronium 0.08mg/kg iv was given as bolus, followed by intermittent doses of .02 mg/kg iv every 30 minutes. Patient was kept in a prone position for surgery. Inj dexamethasone 8 mg iv, Inj ondansetron 4mg iv and Inj. paracetamol 1g infusion was given during the intraoperative period. Inj dexmedetomidine 0.5 mcg/kg/hour infusion was given during surgery. Inhalational agents stopped at time of skin suturing. After the procedure the patient was turned supine and Inj sugammadex 2 mg/kg iv was given. Last dose of vecuronium was given around 30minutes prior. A delayed eye opening and reversal of vecuronium (12 minutes after sugammadex injection) was noticed. Patient was extubated after attaining adequate response and shifted to the recovery room.

Case 3

A 22 years old male, weighing 68 kg, belonging to ASA PS class 2 with controlled hypertension was posted for arthroscopic repair of Bankart lesion under general anaesthesia. After attaching monitors, preoxygenation and premedication was done according to standard hospital protocol. Patient was induced with Inj. propofol 2 mg/kg iv, relaxed with Inj vecuronium 0.08mg/kg iv. After intubation anaesthesia was maintained with intermittent positive pressure ventilation with oxygen, nitrous oxide and sevoflurane, followed by intermittent doses of .02 mg/kg iv every 30 minutes. Inj dexmedetomidine was given as 1 mcg/kg iv slow bolus followed by 0.5 mcg/kg/Hour infusion. Inj dexamethasone 8 mg iv, Inj ondansetron 4 mg iv, Inj paracetamol 20 mg/kg infusion, and Inj tramadol 50 mg iv were given during the intraoperative period. Procedure lasted around 3.5 hours. Inj dexmedetomidine infusion stopped towards the end of surgery (30 minutes prior) and inhalational agents stopped 10 minutes prior. Intraoperative vitals were stable and hypothermia was prevented with warmer. After completion of procedure Inj. sugammadex was given 2 mg/kg iv. Last dose of vecuronium was given 20 minutes prior to the injection of sugammadex. Adequate spontaneous tidal volume was generated within 2 minutes. But there was no eye opening and motor activity for verbal commands. So, it was decided to extubate and keep an oral airway to prevent tongue fall. After extubation it was noticed that respiration was adequate and there was no motor activity of limbs. Vitals were stable. Patient was kept under close monitoring. It took 30 minutes for eye opening and motor activity of extremities to return, after which the airway was removed and shifted to the recovery room.

Case 4

24 years old female, weighing 40 kg, belonging to ASA PS class 1 posted for septoplasty under general anaesthesia. After attaching monitors, pre-oxygenated with 100% Oxygen for 3 minutes. Pre-medicated with Inj. glycopyrrolate 0.2mg iv, Inj. midazolam 1 mg iv and Inj fentanyl 2 mcg/kg iv. Patient was induced with Inj. propofol 2 mg/kg iv, pre-curarization done with Inj vecuronium 1mg iv and relaxed with Inj succinylcholine 1.5mg/kg iv. After intubation anaesthesia was maintained with intermittent positive pressure ventilation with oxygen, nitrous oxide and sevoflurane. Inj. vecuronium 0.08mg/kg iv was given as bolus,

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followed by intermittent doses of .02 mg/kg iv every 30 minutes. Inj dexamethasone 8 mg iv, Inj ondansetron 4 mg iv and Inj. paracetamol 20 mg/kg infusion was given during the intraoperative period. Intraoperative period was uneventful. Total anaesthesia time was 1.5 hours. Last dose of vecuronium was given 20 minutes prior. Inhalational agents stopped 10 minutes prior. Preservative free Inj lignocaine 1.5mg/kg iv was given just prior to reversal. Inj sugammadex 2 mg/kg was given at the end of surgery. It was noticed that the eye opening was 13 minutes after injection of Sugammadex. After complete recovery the patient was extubated and shifted to the recovery room.

DISCUSSION

Sugammadex is a recently available drug for reversing neuromuscular blockade induced by rocuronium and vecuronium. It is a credible alternative to anticline esterases in anaesthesia.[3] A Cochrane review done by Abrishami et al in 2009 have found sugammadex to be 17 times faster in reversing routine to moderate neuromuscular block in comparison to neostigmine.[4] In this case series there was a delay in recovery following sugammadex administration in patients following tranexamic acid, dexmedetomidine and lignocaine. None of the four patients had hypothermia, hypothyroidism, neuromuscular diseases, renal disease or electrolyte abnormalities which can delay the return of motor function.

Lobaz S et al have advised against using sugammadex with verapamil, ranitidine and ondansetron. They have further explained that the drug interactions are mainly due to displacement and capture reactions, and the potential drugs causing displacement of sugammadex are toremifene, fusidic acid, flucloxacillin and diclofenac.[5] In the first case we found that co administration of sugammadex in the same intravenous line of tranexamic acid has resulted in delayed recovery of adequate motor function. After 15 minutes of waiting it was decided to use conventional neostigmine and glycopyrrolate, following which patient had sufficient motor power and respiration and extubated. There was no literature showing an interaction of sugammadex with tranexamic acid.

Second case underwent laminectomy under general anaesthesia with routine drugs used in our institution. Since the procedure demanded a hypotensive anaesthesia inj dexmedetomidine infusion was started during the intra operative period and mean blood pressure was kept above 65mmHg through out to allow adequate organ perfusion. After giving sugammadex, patient had delayed recovery and was extubated 13 minutes later.

Case 3 also had delayed motor power recovery after administration of dexmedetomidine. Sugammadex was administered after 20 minutes of last vecuronium dose. Even though spontaneous respiration was present there were no adequate motor power. Patient needed close monitoring with nasal airway and oxygen supplementation. Full motor power was regained after half an hour. There are many case reports of bradycardia associated with the use of sugammadex.[6-8] It is more profound in higher doses.[7] Though dexmedetomidine also causes bradycardia and hypotension these patients did not develop any significant bradycardia or hypotension. No cases of delayed reversal of sugammadex with dexmedetomidine have been reported so far.

Case 4 patient was given preservative free lignocaine 1.5mg/kg i.v just before extubation in addition to the regular drugs used for general anaesthesia in our institution. Rocuronium is found to have enhanced action with anti arrhythmics and local anaesthetics.[9] The increased adult muscle-type nicotinic acetylcholine receptor inhibition produced when local anaesthetics are combined with non depolarising muscle relaxants may contribute to the clinical enhancement of neuromuscular blockade by local anaesthetics.[10]

Neuromuscular monitoring is advised for reversing a patient from general anaesthesia. Since this facility is not available in our institution we used our vast clinical experience in using neostigmine to compare the effect of sugammadex.

These four cases describe delayed clinical emergence after administration of sugammadex following vecuronium even though it is claimed that sugammadex causes early recovery even in the absence of neuromuscular monitoring.[11] In the absence of quantitative neuromuscular monitoring, causation cannot be established; the observations instead raise the possibility that co-administration/line-compatibility issues (notably administration through an active tranexamic-acid infusion in one case), concurrent sedative/analgesic drugs (dexmedetomidine, opioids, volatile agents, lidocaine), or transient reduction in available free sugammadex may contribute to delayed clinical recovery. Clinicians should use objective TOF monitoring, avoid administering sugammadex through lines with other active infusions (flush lines), and interpret delayed emergence cautiously. above four cases had significant delay in recovery from vecuronium.

CONCLUSION

The present case series raise the possibility of interactions and/or co-administration/compatibility issues that warrant further study of sugammadex with drugs commonly used in the intra operative period like tranexamic acid, dexmedetomidine and lignocaine. Being a relatively new drug one should be cautious about these possibilities while using sugammadex. Prospective studies with standardized neuromuscular and sedation monitoring are needed to determine whether true pharmacologic interactions exist.

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