Critical Respiratory Events in the Post Anaesthesia Care Unit: A Case Report and Overview of the Literature

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Introduction

Complications in the post anaesthesia care unit (PACU) occur frequently [1] yet there appears to be a lack of recent data documenting the overall occurrence. The Association of Anaesthetists of Great Britain & Ireland (AAGBI) has published guidelines on the standards of management for patients in PACUs. We present a case of respiratory compromise in the PACU and suggest that sugammadex should be considered where rocuronium has been administered during anaesthesia in the management of patients in PACU experiencing airway problems.

Case and Method

A 69 year old male, ASA 2 was anaesthetised for elective inguinal hernia repair via the TEP (Total extra-peritoneal) laparoscopic technique. This procedure requires neuromuscular block to enable good operating conditions and facilitate optimum laparoscopic visualisation of the surgical field.

The patient’s past medical history was unremarkable aside from well controlled hypertension. There were no concerns regarding the patient’s airway. His weight was 90 kg. He had an uneventful induction of anaesthesia using propofol with neuromuscular blockade achieved with a single dose of rocuronium of 70 mg. The patient received intermittent boluses of fentanyl to a total dose of 200 mcg and anaesthesia was maintained with desflurane. The procedure was completed uneventfully, lasting approximately 120 minutes in total.

At the end of the procedure, the train-of-four (TOF) ratio was estimated at >0.7 by subjective assessment using a conventional nerve stimulator. The patient was given a full reversal dose of 5 mg neostigmine and glycopyrrolate 1 mg. After reversal the patient had a normal respiratory rate and a tidal volume of in excess of 450 ml. The patient was extubated uneventfully and transferred to the PACU.

After 5 minutes in the PACU the patient developed stridor and became anxious. He continued to have good tidal volumes greater than 450 ml and was able to vocalise. He maintained his oxygen saturations above 95%.

Immediate management of administering PEEP with Water’s Circuit and high flow oxygen was commenced. This failed to resolve the stridor and senior help was summoned. Whilst awaiting assistance the patient was given chlorphenamine 10 mg IV, hydrocortisone 100 mg IV and an adrenaline 1 mg nebuliser were also commenced in case of developing upper airway oedema from a hypersensitivity reaction.

A senior anaesthetist was called to the PACU who reviewed the history and clinical situation. The decision was made to give sugammadex and a 200 mg dose of sugammadex was administered IV. This resulted in complete resolution of the patients’ stridor symptoms within 30 seconds.

Discussion

Patients in the PACU can present a wide range of clinical scenarios ranging from post-operative nausea through to cardiovascular compromise. In their 1992 survey Hines et al. quote a total complication rate in the PACU of 23.7%. The rate of respiratory compromise in studies ranges from 1.3-6.9% [1-3], a more recent study showed critical respiratory events occurring in 7.2% of patients who received intermediate acting muscles relaxants [4]. Interestingly in this study there was also evidence that the use of neostigmine for reversal without the routine use of neuromuscular monitoring increased the risk of post-operative hypoxemia and re-intubation.

A separate study by Hayes et al. showed presence of neuromuscular block despite the use of reversal agents [5]. Others have shown that in patients who have spontaneously recovered from neuromuscular block, reversal with neostigmine may potentially cause a neuromuscular block [6]. The potential for neostigmine causing a neuromuscular block after spontaneous recovery of initial neuromuscular block was discussed at our institution’s local review of untoward clinical anaesthesia events. Our patient received 5 mg of neostigmine. This equates to a dose of 55 µg/kg in line with the recommended dosing regimen.

Debaene at al measured the degree of residual paralysis in patients who received a single dose of intermediate acting neuromuscular blocking agents. Patients were shown to have a TOF of less than 0.7 and 0.9 in 10% and 37% of cases respectively 2 hours after administration of neuromuscular blocking agents, but no reversal [7].

Murphy et al. in 2008 showed that critical respiratory events in the PACU are strongly associated with post-operative residual neuromuscular blockade [8]. They recorded an incidence of 0.8% of CRE in patients despite the use of intraoperative TOF monitoring.

Current UK practice of assessing the presence of residual block is made subjectively by assessing either clinically (presence of head lift, adequate tidal volumes, hand grip) or measuring with a nerve stimulator (TOF ratios greater then 0.7, lack of fade on Double Burst Stimulation [DBS]). Collectively the evidence suggests that these subjective methods used to assess the adequate return of neuromuscular function by the time that patients reach the PACU are suboptimal.

The only effective way to ensure recovery is at a TOF>0.9 is by objective monitoring of neuromuscular block for example using intraoperative acceleromyographic monitoring; further work by Murphy et al has shown that this reduces the incidence of CREs in the PACU [9].

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The muscles of the upper airway are more sensitive to neuromuscular block. One of the authors of this case report suggests that where patients have received rocuronium who present with airway compromise after extubation, the possibility of post operative residual neuromuscular block should always be considered and the administration of sugammadex in these circumstances be mandatory [10].

We suggest that anaesthetists whose routine clinical practice is to reverse neuromuscular blockade with neostigmine should have a low threshold for administering sugammadex to patients who have been given rocuronium during anaesthesia and are experiencing airway difficulties in the PACU which may be as a direct consequence of unrecognised post operative residual neuromuscular block.

References

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