No neuromuscular block after maximal dose of rocuronium: a case report

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Background

Rocuronium is a non-depolarizing neuromuscular blocking agent (NDNMBA), employed in the clinic as an adjunct to general anesthesia to facilitate tracheal intubation rapid sequence, and to provide skeletal muscle relaxation during surgery [1,2]. Many cases of resistance to neuromuscular blocking agents (NMBAs) have been anecdotally reported. There are specific pathologic states, such as upper motor neuron lesions, severe thermal injuries, liver disease, renal failure, disuse atrophy, all of which show an increased resistance to the effects of nondepolarizing muscle relaxants [3]. Also concurrent drug therapy can alter the efficacy of NMBAs such as some classes of antibiotics, furosemide, β receptor agonists, phosphodiesterase inhibitors, calcium antagonists, respiratory stimulants but also ketamine, propofol and barbiturates at high concentrations [4]. In this scenario we describe an unusual case of 20-years-old man who showed a complete resistance to rocuronium maybe due to a glucocorticoids concomitant therapy.

Case report

A 20-years-old man, 177 cm, 78.4 kg, BMI 24.9, was scheduled for renal biopsy as part of diagnostic protocol for undefined glomerulonephritis with nephrosic syndrome. He was hypertensive, thalassaemic, hepato-spleno-maegalic, allergic to latex. The patient took an oral prednisone therapy (30 mg/day) which lasted 140 days, and received the last dose (5 mg) two months before the scheduled surgery. After receiving one-day desensitizing therapy (betamethasone 4 mg twice a day) and ciprofloxacin (200 mg i.v.), the patient was admitted in the Operating Room and was premedicated i.v. with midazolam (1 mg), fentanyl (0.05 mg) and atropine (0.7 mg). Then anaesthesia was induced with propofol (200 mg) and continous remifentanil (0.11mcg/kg/min). Just before the administration of rocuronium 0.6mg/kg (50mg). Three minutes later any depression of twitch height was observed and the patient seemed to be partially awake without clinical signs of muscle relaxation. Additional doses of propofol (200mg) and rocuronium (50mg) from different batches were injected over ten minutes, but any depression in the twitch response was noted over the next seven minutes. Therefore it was decided to attempt neuromuscular relaxation with a benzilisoquinolinic nondepolarizer compound, and cis-atracurium 0.2mg/kg (16mg) was injected. Train-of-four responses disappeared quickly (120sec.) and completely and orotracheal intubation was easily performed.

Conclusion

The case described above represents an example of a drug therapeutic failure (DTF). The DTF is to date included within the wider definition of adverse drug event given by the World Health Organization (WHO) and it is proposed as a peculiar type of adverse drug reaction designated as 'failure'. For this reason pharmacovigilance post-marketing studies are key elements to monitor the safety and effectiveness of approved drugs [5-9].