Fulminant rhabdomyolysis after prolonged radical prostatectomy managed with continuous veno-venous haemodialysis, regional citrate anticoagulation, and a polysulphone high-flux filter

We present data from a patient with fulminant rhabdomyolysis (RML) after radical prostatectomy, which could be managed successfully with early initiation of continuous veno-venous haemodialysis (CVVHD). A 58-yr-old male was admitted to our hospital with the diagnosis of a carcinoma of the prostate, planned for a radical prostatectomy. The patient had a history of essential hypertension, hyperlipidaemia, and penicillin allergy, and was on atorvastatin, nebivolol, perindopril, indapamide, and spiranolactone. The radical prostatectomy was performed under balanced anaesthesia with fentanyl, desflurane, and atracurium.

After laparotomy, surgery was continued in the extreme Trendelenburg position (at an angle of 45°) and controlled hypotension during exposure of Santorini’s venous plexus. Because of technically difficult surgical conditions, a blood loss of ≈2100 ml occurred. In total, the patient received 600 ml of erythrocyte-concentrate, 1500 ml of hydroxyethyl starch, and 3000 ml of crystalloid fluids. Arterial blood gases, obtained after 4 h of surgery, showed stable conditions. After 7 h, the operation was finished and the extreme Trendelenburg position was ended. Haemodynamic conditions became unstable despite adequate volume load, and norepinephrine was administered up to 0.20 μg kg⁻¹ min⁻¹.

About 1 h later, the patient was transferred to the intensive care unit (ICU) where blood gases showed a severe lactic acidosis (pH 7.26; lactate 5.7 mmol litre⁻¹) and a serum potassium of 5.7 mmol litre⁻¹. Laboratory tests revealed an increase in creatine kinase (>8000 U litre⁻¹) and myoglobin (586 μg litre⁻¹) confirmed a diagnosis of RML, and forced diuresis was started. About 8 h after surgery, oligo-anuria and acute renal failure (ARF) were apparent. Because of the high risk of postoperative bleeding, CVVHD with regional citrate anticoagulation was established (Multifiltrate® with integrated Ci-Ca® system; Fresenius Medical Care, Germany). After 7 days of successful CVVHD, intermittent dialysis was established and after a further 5 days, renal replacement therapy (RRT) could be ceased. The maximum of CK was reached at day 2 (74 072 U litre⁻¹), whereas myoglobin reached the maximum at day 3 (20 600 μg litre⁻¹). CVVHD, continuous veno-venous haemodialysis.

Fig 1 Values of urea, creatinine, creatine kinase, and myoglobin during ICU stay and at day 35 (hospital discharge). The peak value of creatine kinase was reached at day 2 (74 072 U litre⁻¹), whereas myoglobin reached the maximum at day 3 (20 600 μg litre⁻¹). CVVHD, continuous veno-venous haemodialysis.

systemic anticoagulation or to commence CVVHD with regional citrate anticoagulation. It is reported that continuous veno-venous haemofiltration is more effective in myoglobinuric ARF at clearing myoglobin, a major factor in renal toxicity, from the systemic circulation.³ However, in our case, ARF occurred immediately after a surgical procedure with a high blood loss, and a recent study⁴ described the effective elimination of myoglobin by haemodialysis. As we had the opportunity to use the same polysulphone high-flux filter (Ultraflux AV 1000 S®, Fresenius Medical Care) as described in this study, we elected to start CVVHD with regional citrate anticoagulation and this filter to avoid secondary bleeding. After an increase in myoglobin during the first 3 days on ICU due to ongoing RML (maximum at day 3: 20 600 μg litre⁻¹; Fig. 1), this management resulted in a good clearance of myoglobin and return of normal function after a long recovery. The recent development of high-flux filters with an enhanced middle molecule clearance could even improve the efficacy of CVVHD in myoglobinuric ARF. To our knowledge, there are no other reports of fulminant RML immediately after surgical procedures managed with CVVHD and regional citrate anticoagulation yet.

Declaration of interest

None declared.

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Efficacy of rocuronium and sugammadex in a patient with dermatomyositis

Editor—We report a patient with dermatomyositis who showed a slow onset of action of rocuronium-induced neuromuscular block and a slow recovery of neuromuscular function with sugammadex. A 75-yr-old male patient with dermatomyositis was undergoing open reduction of an elbow fracture under general anaesthesia. Preoperative muscle testing showed mild-to-moderate weakness in his extremities. Anaesthesia was induced with fentanyl and propofol and after loss of consciousness, the left ulnar nerve was stimulated at the wrist by a 5 s, 50 Hz tetanic stimulus, followed by supramaximal and square-wave stimuli of 0.2 ms duration, delivered in a train-of-four (TOF) mode at 2 Hz every 15 s. Contraction of the ipsilateral adductor pollicis muscle was measured using an acceleromyograph (TOF-Watch SX; Organon, Dublin, Ireland). Immediately after obtaining baseline TOF responses, the patient received 0.6 mg kg⁻¹ rocuronium. Complete neuromuscular block was obtained in 315 s and the patient’s trachea was intubated thereafter without any difficulty. Anaesthesia was maintained with 1% end-tidal sevoflurane, 0.2 μg kg⁻¹ min⁻¹ remifentanil, and fentanyl as required. Twenty-five minutes after rocuronium administration, the first twitch (T1) of the TOF spontaneously recovered to 10% of the control. At that time, 2 mg kg⁻¹ sugammadex was administered to antagonize rocuronium-induced neuromuscular block. Lag time to the beginning of abrupt increase in T1 was 120 s, while the time to reach a TOF ratio of 0.9 was 345 s after sugammadex administration. Recurarization and respiratory complications were not seen after operation.

Our patient seemed to show normal sensitivity to rocuronium-induced neuromuscular block. It is not likely that there were abnormalities in the properties and number of nicotinic acetylcholine receptors on the motor endplate. However, our patient showed a slower onset of action of rocuronium. Maximal block after 0.6 mg kg⁻¹ rocuronium is normally obtained around 100 s. Considering the normal sensitivity to rocuronium, the slow onset of action of rocuronium was probably due to slow diffusion of rocuronium from the plasma to the neuromuscular junction. In dermatomyositis, there is perivascular inflammation and intramuscular blood vessels show endothelial hyperplasia, fibrin thrombi, and obliteration of capillaries, resulting in a reduction in capillary blood flow to the muscles. Therefore, the slower delivery of rocuronium to the neuromuscular junction may result in slower onset of action of rocuronium in patients with dermatomyositis. This could also explain the longer reversal time. The time to recovery from rocuronium-induced neuromuscular block to a TOF ratio of 0.9 after administration of a sufficient dose of sugammadex is generally 1.1–1.3 min, regardless of the depth of neuromuscular block. Sugammadex may be restricted to the intravascular space due to its low volume of distribution and act mainly in the plasma, resulting in a rapid decrease in plasma concentrations of free rocuronium, which induces rocuronium molecules to extensively diffuse from the neuromuscular junction into plasma, along the concentration gradient. This probably leads to a rapid dissociation of rocuronium from the nicotinic acetylcholine receptors and restoration of normal neuromuscular transmission. Therefore, the delivery rate of sugammadex to the peripheral muscles has profound effects on recovery of the TOF ratio to 0.9. An adequate dose of sugammadex can completely restore neuromuscular function even in dermatomyositis patients. However, it is still recommended that these patients should be observed for much longer than is usual practice in patients with normal neuromuscular function.

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