Spontaneous intracranial hypotension (SIH) is a poorly understood entity resulting from CSF leakage or from venous hypotension causing increased CSF absorption. It causes a range of symptoms from orthostatic headaches to coma.

MRI and CT myelography are the investigations of choice in demonstrating CSF leaks as presumed sites of dural defects. CT myelography has traditionally been the first diagnostic tool, but this necessitates lumbar puncture for contrast injection and this is both invasive and of significant risk with infra-tentorial herniation. MR myelography has been shown to have comparable detection rates in nerve root leaks and high cervical retrospinal CSF collections, and superior detection rates in spinal leaks.

A diagnostic quandary arose in this patient who had serious contraindications to MRI and CT myelography. The presence of a PPM is widely recognized as a contraindication to MRI due to the risk of harmful arrhythmias and heating or displacement of the device or its leads. In a survey asking radiologists and cardiologists whether they would submit a patient to MRI with a PPM in situ, 97% radiologists responded that they would not, whereas 34% of cardiologists responded that they would under appropriate circumstances.

There is now a substantial evidence base providing safety data on a large number of MRI studies with PPMs. The devices for which there is most evidence include those manufactured after 1998, in place for over 6 weeks and without epicardial or abandoned leads.

The number of PPMs will continue to increase due to an ageing population and increasing indications. However, the number of patients with an expanding range of co-morbidities that will benefit from the diagnostic advantages offered by MRI will also multiply. MRI should only be considered when there is no lower-risk alternative to answer clinical questions essential to patient management.

This case highlights the commonly overlooked pathology of SIH and raises awareness of the increasing evidence that PPMs are becoming relative rather than absolute contraindications to MRI.

**Declaration of interest**

None declared.

C. C. MacFie*

P. G. Laws

Newcastle upon Tyne, UK

E-mail: carolinemacfie@doctors.org.uk

1. Rahman M, Bidari SS, Quisling RG, Friedman WA. Spontaneous intracranial hypotension: dilemmas in diagnosis. Neurosurgery 2011; 69: 4–14


doi:10.1093/bja/aet390

**Sugammadex in a patient with Sjogren’s syndrome and polymyositis**

Editor—Polymyositis was considered responsible for a delayed onset and reversal of rocuronium-induced neuromuscular block (NMB) by sugammadex. We recently cared for a female patient, aged 67 yr (weight, 60 kg; height, 155 cm), with polymyositis associated with Sjogren’s syndrome, undergoing laparoscopic sigmoid resection for diverticulitis. Her medications included prednisone 12.5 mg daily and methotrexate 15 mg weekly, and on preoperative evaluation, she was noted to have severe weakness of the extremities. Anaesthesia was induced with fentanyl 3 μg·kg⁻¹ and propofol 2 mg·kg⁻¹ and maintained with desflurane and remifentanil, titrated to a state entropy value of 35 (5). After loss of consciousness, the left ulnar nerve was supramaximally stimulated near the wrist with square-wave 0.2 ms pulses, delivered as 2 Hz train-of-four (TOF) pulses at 15 s intervals. The resulting adductor pollicis muscle contractions were quantified acceleromyographically (TOF-Watch SX, Organon Teknik, Ireland). Stabilization, calibration, and baseline responses were recorded at anaesthesia induction before rocuronium-induced NMB, from 1 PTC to a TOF ratio of 1.11, was achieved within 90 s. NMB was maintained with subsequent boluses of rocuronium, titrated to achieve moderate NMB for the first 30 min, and then, in the following period, deep NMB [1–5 post-tetanic counts (PTCs)] to ensure adequate surgical conditions (rocuronium 220 mg total dose) (Fig.1). At the end of the uneventful, 210 min surgical procedure, remifentanil was stopped and sugammadex 4 mg·kg⁻¹ was administered to reverse the deep NMB. Complete reversal of NMB, from 1 PTC to a TOF ratio of 1.11, was achieved within 90 s. Desflurane was then discontinued, the patient awakened, and the tracheal tube removed. The patient had no evidence of residual or recurrent NMB in the postoperative period.

Polymyositis is histopathologically characterized by perivascular non-suppurrative inflammatory infiltrates leading to muscle fibre degeneration. Clinically, it causes symmetrical, proximal muscle weakness. There is no evidence that the disease affects...
the neuromuscular junction itself.2–4 Thus, the onset of action of rocuronium and the reversal of rocuronium-induced NMB by sugammadex should be within the normal range, as observed in our case, and not affected by the inflammatory processes of the muscle.1 The patient in the Suzuki and colleagues1 report was 75 yr old, which may have been an important factor contributing to his delayed onset and recovery from rocuronium-induced NMB by sugammadex.5–7 The onset of rocuronium is longer in older than in younger adults.5 This is primarily attributed to diminished cardiac output, which increases the time to equilibrium between the plasma and the neuromuscular junction.56 The time for reversal of rocuronium-induced NMB by sugammadex may also be prolonged in the elderly.67 Although sugammadex facilitates rapid reversal from rocuronium-induced NMB in adults of all ages, reversal of rocuronium-induced NMB with sugammadex is somewhat slower in the elderly.2 When sugammadex was administered at the time of spontaneous reappearance of the second twitch of TOF, the geometric mean time from sugammadex administration to recovery of the TOF ratio to 0.9 was longer in patients ≥65 yr than those aged 18–64 yr (2.9 vs 2.3 min, P=0.022).7 With increasing age, the geometric mean time increased from 2.3 min (adults) to 3.6 min (patients ≥75 yr).7 This difference has been attributed to the lower cardiac output in older patients, which leads to a slower distribution of sugammadex.67 The elderly may also experience altered perfusion within the muscles, thus changing the distribution and redistribution rates of rocuronium, sugammadex, and the rocuronium–sugammadex complex.6 7

In conclusion, in patients with Sjogren’s syndrome, polymyositis alone seems to not affect the onset of action of rocuronium and the reversal of rocuronium-induced deep NMB by sugammadex. The observations of Suzuki and colleagues1 may have been at least partly attributed to the older age of their patient.

**Declaration of interest**

M.C. has received payments for lectures from MSD. C.O. has received payments and travel funding for lectures and as a member of the MSD Advisory Board.

M. Carron*

G. Ieppariello

C. Ori

Padova, Italy

*E-mail: michele.carron@unipd.it


doi:10.1093/bja/aet391