Planned magnetic resonance imaging for a patient with a permanent pacemaker in situ with suspected spontaneous intracranial hypotension

Editor—A female patient, aged 77yr, with a past medical history of hypothyroidism, was admitted after gradual neurological deterioration over 6 weeks, including increasing confusion and unsteady gait. A permanent pacemaker (PPM) had been sited after collapse secondary to sinus arrest 2 weeks previously.

On examination, her Glasgow coma scale (GCS) was 13 with no localizing neurology. Computed tomography (CT) of the head demonstrated bilateral subdural collections with subfalcine and tonsillar herniation. Bilateral burr holes were drilled, revealing cerebrospinal fluid (CSF) collection not under pressure, rather than the expected subdural haematoma. This raised the possibility of spinal CSF leak, resulting in hygroma formation and brain compression.

The GCS deteriorated to seven on day 3. Repeat CTs were unchanged and further investigation for spinal CSF leak by CT myelography or magnetic resonance imaging (MRI) was deemed necessary.

The radiologist’s opinion was that CT myelography would cause unacceptable risk of potentially fatal brain herniation, as central and tonsillar herniation were present. The opinion of a cardiologist was sought regarding PPM and MRI. The PPM was interrogated and revealed 1:1 atrioventricular conduction with no evidence of distal conduction disease, suggesting the bradycardia leading to collapse was due to intracranial pathology rather than a primary cardiac cause. The cardiologists felt the risks of removal of the PPM outweighed the risks of MRI with PPM in situ.

MRI was performed with neurointensivist and electrophysiologist presence after temporary reprogramming of the PPM. It revealed a dural tear at the level of the C2 cervical vertebra. The PPM was interrogated post-MRI and showed no parameters that required electrophysiologist alteration.

Diagnosis enabled treatment with CT targeted C3 epidural blood patch (EBP) under general anaesthesia. There was rapid improvement over 48h to GCS 14. The patient has since regained her pre-morbid state.
Spontaneous intracranial hypotension (SIH) is a poorly understood entity resulting from CSF leakage or from venous hypotension causing increased CSF absorption.\(^1\) 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 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 retrosinal CSF collections, and superior detection rates in spinal leaks.\(^2\)

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.\(^3\)

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.\(^4–7\)

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

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**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.\(^1\) 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 predni solone 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 \(\mu\)g 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 accelerometerographically (TOF-Watch SX, Organon Teknik, Ireland). Stabilization, calibration, and baseline responses were recorded at anaesthesia induction before rocuronium administration, and neuromuscular monitoring was continued until the TOF ratio returned to \(\geq 1.0\). NMB was achieved with a rocuronium 0.9 mg kg\(^{-1}\) bolus before tracheal intubation. This reduced the TOF ratio from 1.07 to 0.0 in 80 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-suppurative inflammatory infiltrates leading to muscle fibre degeneration.\(^1–4\) Clinically, it causes symmetrical, proximal muscle weakness. There is no evidence that the disease affects

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