INTRODUCTION
Profound hypotension is a recognised clinical feature of both anaphylaxis and an acute hypoadrenal state. Central hypoadrenalinism is seen in intra-cranial disease processes affecting the hypothalamic–pituitary–adrenal axis. We present an unusual case of apparently typical peri-operative anaphylaxis in a man with intra-cranial lymphoma, the lymphoma was diagnosed and treated as such but the anaphylactic testing led to a subsequent diagnosis of hypocortisolaemia being made. The patient and his relatives gave permission for his case to be discussed in medical literature.

Case description
A 64 year old man presented to a neuropsychiatric centre for urgent biopsy of a posterior fossa lesion (see Image 1). He had been transferred from a DGH where he had received initial treatment for a presumed ischaemic cerebellar stroke after presenting with vertigo and vomiting. 4 months prior to this, he was diagnosed with diabetes insipidus and hypogonadotropic hypogonadism. Initial assessment of his adrenocortical function appeared normal. An MRI of the pituitary was also normal.

The biopsy procedure was deferred for 1 week due to previous anticoagulant therapy. Desmazhame was withheld to avoid tumour shrinkage prior to the biopsy. (1) The patient underwent neurosurgical biopsy which was also given to aid intubation. Immediately following induction, he became profoundly hypotensive, this was unresponsive to first measures (ephedrine) but ultimately responded to intravenous fluid and adrenaline (boluses followed by an infusion). Intravenous hydrocortisone and chlorphenamine were given, as per AAGBI guidelines. (2) He was admitted to the Neurosciences Intensive Care Unit (NICU) with a working diagnosis of anaphylaxis. The suspected trigger agents were remifentanil, propofol and rocuronium; with the latter deemed the most likely.

Desmazhame was commenced on NICU. Unfortunately, the patient developed a Takotsubo cardiomypathy secondary to resuscitative adrenaline. In terms of allergy testing, blood which had been taken prior to the reaction was tested for specific IgG antibodies for phosphocline and suxamethonium (which could have been suggestive of possible rocuronium allergy), and for chlorhexidine. These were all negative. Serum tryptase levels were taken at 1, 3 and 24 hours after the event. No deflection from a normal baseline was demonstrated. None of these results were considered to have made the diagnosis of anaphylaxis less likely.

The neuropsychiatric plan was to wean desmazhame and plan for a repeat procedure at a later date, pending a full assessment in the anaeasthetic allergy clinic. Desmazhame was weaned on NICU. Prior to stopping it completely, the NICU medical team ensured that this patient did not previously take any steroids regularly. Following an endocrine review, he was discharged home on his previously prescribed desmopressin, levothyroxine and testosterone.

19 days after being discharged, he required a short course of desmazhame for neuropsychiatric symptom control. A concurrent repeat CT brain revealed stable intracranial appearances with no change in ventricular volume. 22 days later, he presented to anaeasthetic allergy clinic for formal allergy testing at which time he was no longer taking desmazhame.

Unfortunately, he was unwell with sepsis due to a likely aspiration pneumonia. This resulted in a second NICU admission. Desmazhame was re-started.

The scheduled allergy testing was carried out during this inpatient admission, approximately 8 weeks following the index event. A Basophil activation assay (Histamine release assay, Reflab Copenhagen) did not show positive results for any neuromuscular blocking drugs or chlorhexidine. Skin prick and intradermal tests were negative for all drugs tested (propofol, remifentanil, chlorhexidine, rocuronium, fentanyl and cisatracurium). This panel of negative allergy tests, in combination with the observed clinical deteriorations when off steroids, alongside other endocrine abnormalities, led to a suspicion of hypocortisolaemia being the underlying reason for the index episode of cardiovascular collapse. Regular oral hydrocortisone supplementation was started. A general anaesthetic with additional steroid cover was deemed safe.

On day 12 of this admission, he underwent the planned neuropsychiatric procedure (posterior fossa biopsy) via a burrhole incision. The anaeasthetic was uneventful, and the drugs used were propofol, remifentanil and fentanyl. Additional perioperative steroids were given as planned. After initially waking uneventfully postoperatively, he rapidly developed complications related to a posterior fossa bleed. This necessitated emergency posterior fossa craniectomy and haemotoma evacuation (see Image 2). Sadly, he failed to recover neurologically from this complication and died from a hospital acquired pneumonia 2 months later. Histology did indeed confirm a non GC type diffuse lymphoma of the posterior fossa (see Image 1). He had received propofol alongside other endocrine abnormalities, led to a suspicion of anaphylaxis. The suspected trigger agents were remifentanil, propofol and Rocuronium; with the latter deemed the most likely.

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References