Reply from the authors

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symptoms: onset 4 days after lumbar puncture and resolution within hours after treatment in our patient *vs* 4 weeks until onset and several days until remission with treatment in theirs. The imaging findings were unremarkable in our patient, whereas several findings, including downward displacement of the brainstem, were identified in their patient. Based on the findings in these cases, we conclude that (i) psychiatric symptoms can be associated with CSF leakage and (ii) the clinical and radiological aspects of the cases can vary.

Declaration of interest

None declared.

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1 Loures V, Savoldelli GL, Alberque C, Haller G. Post-dural puncture cerebrospinal fluid leak presenting as an acute psychiatric illness. *Br J Anaesth* 2012; **108**: 529–30

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Reply from the authors

Editor—We read with great interest the report by Dr Y. Kakisaka and colleagues of a paediatric case similar to ours.[1] Their report highlights the fact that post-dural puncture headache is not always characterized by orthostatic headache associated with neck stiffness, tinnitus, hypoacusia, photophobia, or nausea.[2] Clinical presentation can sometimes be atypical and patients can present with neuropsychiatric symptoms. The diagnosis may be particularly difficult to make. Computed tomography scan is of limited value for the diagnosis of the perforation as illustrated in their report. Sometimes, images of a subdural fluid collection, obliteration of subdural cistern, and ventricular collapse can be seen and suggest an intracranial hypotension, but this is not always the case.[3] Cranial magnetic resonance imaging (MRI) with gadolinium injection is the radiological test of choice. Images will show a diffuse dural pachymeningeal enhancement, reflecting the presence of small thin-walled dilated blood vessels in the subdural space.[4] We would therefore recommend prompt neurological evaluation (cranial MRI with contrast injection) for patients who present with neuropsychiatric symptoms after dural perforation.

Declaration of interest

None declared.

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Failure of esmolol to control tachycardia associated with thyroid storm after subtotal thyroidectomy

Editor—Thyroid storm is a potentially life-threatening disorder marked by fever, sweating, and tachycardia. It usually requires treatment with β-blocker. Esmolol, a cardioselective β-blocker, has been shown effectively to control tachycardia in patients with thyroid storm.[1 2] Here, we describe a case in which esmolol therapy did not control sinus tachycardia in a postoperative patient with thyroid storm.

A 33-yr-old, 56 kg Chinese female had progressive swelling in the neck for 7 yr. She had been diagnosed with hyperthyroidism 4 yr previously. Because of medical treatment failure, subtotal thyroidectomy was performed under general anaesthesia using propofol and sufentanil infusions. Anaesthesia and surgery were uneventful, and the patient was transferred to the recovery room. After 30 min, she regained consciousness and adequate respiratory strength. However, she was febrile (38.6°C), restless, and had profuse sweating despite adequate analgesia. Arterial pressure was 156/107 mm Hg and an ECG showed a sinus tachycardia (152 beats min^{-1}). As thyroid storm was suspected, esmolol 30 mg was given i.v. over 1 min, followed by midazolam 5 mg. Ice packs and alcohol sponging were used to lower body temperature, methimazole 20 mg was given by the nasogastric tube, and hydrocortisone 100 mg and nicardipine 0.25 mg were administered i.v. After 20 min, the temperature had decreased to 37.4°C, but tachycardia and hypertension persisted. A further dose of esmolol 30 mg was given, followed by an infusion at an initial dose of 50 $\mu g\ kg^{-1}\ min^{-1}$, titrated up to 300 $\mu g\ kg^{-1}\ min^{-1}$ (the maximum recommended dosage) over a 50 min period. However, the patient remained tachycardic and hypertensive. At that time, no signs and symptoms of congestive