We did not consider a rectal agent particularly in view of her incontinence. We opted for ketamine i.m., as its use in Caesarean sections is well described. It has good haemodynamic properties and does not cause neonatal depression. Its use by the i.m. route is less common, and is reserved mainly for the management of difficult children. It is associated with nightmares in the recovery period, but in this case none were reported. We did not give any opioids or benzodiazepines, which are often used to attenuate these, but did ensure a quiet environment for recovery. Further, ketamine has analgesic properties, which undoubtedly contributed to the pain-free state of the patient postoperatively. However, the complete lack of analgesic requirements may not be attributable solely to the ketamine, and may be because of an altered sensation on the part of the patient.

We used a 'normal' induction dose of thiopentone (5 mg kg⁻¹) before intubation. We acknowledge that this was probably a larger dose than was needed in a lady who had already been given ketamine but wished to ensure unconsciousness in a patient in whom ketamine had yet to reach full effect.

This case illustrates the need for close cooperation between the obstetric and anaesthetic departments. This was a patient in whom obstetric intervention was highly likely, and who was clearly going to be difficult to manage. In addition, she was an inpatient for steroid therapy, because of the risk of early labour. However, she was not brought to our attention until late at night when the decision to perform an emergency Caesarean section had been made. Early referral to an anaesthetist may not have altered our subsequent management but would have given us time to plan. Furthermore, interview of the patient under less fraught circumstances, and frank discussion of all the options and risks may have resulted in a more co-operative and less anxious patient and would have expedited the delivery of the distressed baby.

References


Intracranial subdural haematoma is an exceptionally rare complication of spinal anaesthesia. A 20-yr-old male underwent appendicectomy under partial spinal and subsequent general anaesthesia. A week later, he presented with severe headache and vomiting not responding to bed rest and analgesia. Magnetic resonance imaging showed a small acute subdural haematoma in the right temporo-occipital region. The patient improved without surgical decompression.
The pathogenesis of headache and subdural haematoma formation after dural puncture is discussed and the literature briefly reviewed. Severe and prolonged post-dural puncture headache should be regarded as a warning sign of an intracranial complication.

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The most common complication of spinal anaesthesia is headache. Post-dural puncture headache (PDPH) is classically postural and responds within 48 h to increased fluid intake and bed rest. Prolonged PDPH may be caused by subdural haematoma1–4 or intracerebral haemorrhage.5 Few anecdotal cases of this neurological complication are described in the literature. We report a case of cranial subdural haematoma in a patient who underwent spinal anaesthesia for appendicectomy.

Case report

A 20-yr-old male was admitted with abdominal pain and vomiting. Acute appendicitis was diagnosed and appendicectomy was performed under spinal anaesthesia. A 23G Quincke point spinal needle was introduced through the L4–5 interspace. After dural puncture at the first attempt, 1.8 ml of 0.5% heavy bupivacaine was injected. This was only partially effective, so general anaesthesia was induced with thiopentone 200 mg, succinylcholine 80 mg and atropine 0.6 mg. Anaesthesia was maintained with 0.5% halothane, pentazocine 30 mg and midazolam 1 mg. Muscle relaxation was maintained by a further dose of succinylcholine 80 mg given 20 min later. The patient remained haemodynamically stable. The excised appendix was grossly inflamed; histopathology confirmed the diagnosis of acute appendicitis.

The patient recovered fully and was discharged on the third day after the operation. He had no headache at the time of discharge. However, a week later, he developed a severe diffuse headache which did not subside with analgesia and bed rest. When he started to vomit, an intracranial lesion was suspected and a neurosurgical consultation was sought. There was no history of fever, trauma or bleeding diathesis. On examination, the patient was conscious and oriented: Glasgow coma score was 15, fundi were normal and there was no focal neurological deficit. He had no neck rigidity or Kernig’s sign. Haemoglobin, total and differential leucocyte counts, platelet count, bleeding time, clotting time, prothrombin time and activated partial thromboplastin time (APTT) were normal. Biochemical values, including liver and renal function tests, were also normal.

Magnetic resonance imaging of the head showed a small acute subdural haematoma in the right temporo-occipital region with no mass effect. The patient was managed conservatively with bed rest, analgesia and intravenous fluids. He recovered completely and was discharged after a week.

Discussion

Headache is the most frequent complication after lumbar puncture, occurring in ≤40% of cases.6 Such headaches are thought to be caused by excessive (≤250 ml day−1)7 leakage of cerebrospinal fluid (CSF) through the dural puncture, causing caudal displacement of intracranial structures.8 This displacement then stretches the intracranial pain-sensitive dura, sinuses and blood vessels, causing pain. Typical PDPH may occur soon after spinal anaesthesia and usually subsides in a few days with bed rest and analgesia. Recently, Suess and colleagues found that headache lasting >5 days was the chief complaint in 17 reported cases of intracranial haemorrhage after myelography.9 Macon and colleagues reported that headache of subdural haematoma was more severe than PDPH and persistent, even in the recumbent position.10 PDPH was the most frequently encountered complication with traditional Quincke point spinal needles of large size. Its incidence has been minimized with the use of 29G needles11 and pencil-point needles.12 Epidural blood patching has been described for stopping CSF leakage, thereby relieving the headache.13 Subdural haematomas have occurred after lumbar puncture in association with cerebral aneurysm, brain tumour, recent cerebrovascular accident and meningovascular syphilis. It is postulated that the haemorrhage is caused by a sudden decrease in intracranial pressure consequent to the loss of CSF at the lumbar puncture site. Sudden caudal shift of the brain may cause traction on the arachnoid mater and/or venous structures and may lead to bleeding from ruptured vessels. Thorsen has described multiple petechial haemorrhages on the surface of the brain after spinal anaesthesia.14 Pavlin and colleagues reported two cases of large subdural haematoma, which required surgical evacuation.1 Mantia reported a case of intracerebral haemorrhage after lumbar puncture with a 26G spinal needle; his patient improved with conservative treatment.5 The true incidence of subdural haematoma after dural puncture is not known. Most patients with headache are...
probably treated without investigation. Subdural haematomas are known to resolve spontaneously\textsuperscript{15} but they may be catastrophic as evident from the deaths recorded as a complication of lumbar puncture.\textsuperscript{9,16} In the case described here, the patient developed a subdural haematoma despite the use of a narrow gauge (23G) spinal needle. Altered CSF dynamics probably caused the rupture of a cerebral vein, resulting in the development of a subdural haematoma. Fortunately, the haematoma was only small and did not require surgical decompression.

Severe and prolonged PDPH should be regarded as a warning sign of an intracranial haematoma. In these patients, early neurosurgical consultation is recommended.

References