Meningitis after combined spinal–extradural anaesthesia in obstetrics

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Summary
We report two cases of meningitis which developed after combined spinal–extradural procedures for obstetric analgesia. The first case was thought to be caused by aseptic or chemical meningitis and the second was a case of bacterial meningitis in a patient who also received an extradural blood patch. It is important that meningitis is considered as a differential diagnosis in patients who present with headache after spinal anaesthesia and that antibiotic therapy is selected to cover unusual organisms. (Br. J. Anaesth. 1994; 73: 545-547)

Key words
Anaesthesia, obstetric. Complications, meningitis.

Case report
CASE 1
A previously healthy 34-yr-old woman was admitted at term plus 9 days in early labour. Her first child had been delivered by emergency Caesarean section 3 yr previously. She requested extradural analgesia and a combined spinal–extradural procedure was performed. Disposable pack and needles were used, and the anaesthetist scrubbed and wore a sterile gown and gloves. The skin was prepared with unstained chlorhexidine in alcohol from a receptacle on the extradural trolley, dried with a sterile towel and then infiltrated with 2% lignocaine. The extradural space was located at L2-3 with a standard Tuohy needle using loss of resistance to saline. An 11.9 cm, 27-gauge Whiteacre spinal needle was passed through the Tuohy needle and after free flow of clear fluid, 0.25% bupivacaine 1 ml, fentanyl 25 µg and normal saline 0.5 ml (total volume 2 ml) were given into the subarachnoid space. The spinal needle was withdrawn and the extradural catheter inserted. Immediate analgesia was achieved with a sensory level to T8 bilaterally. The solution for extradural top-ups comprised 0.5% bupivacaine 10 ml, fentanyl 100 µg and normal saline 38 ml in a 50-ml syringe. Three top-up doses of this solution were given during subsequent labour: 2% lignocaine 10 ml was also given before a lift-out forceps delivery which was performed for failure to progress in the second stage. A healthy baby girl was delivered 6.5 h after performing the combined spinal–extradural block.

The patient was well the following morning on the anaesthetic ward round, but at 13:00, 21 h after spinal injection, she developed a severe throbbing headache and complained of feeling faint and with shortness of breath. On examination she was apyrexial, heart rate was 68 beat min⁻¹, arterial pressure 90/60 mm Hg and ventilatory frequency 20 b.p.m., but she was not cyanosed and had no neck stiffness. Four hours later she was unable to pass urine and required catheterization. At 18:00 the headache became more severe and she developed an expressive and receptive dysphasia and tingling in the right side of the face and right arm. We were concerned that she had suffered a subarachnoid haemorrhage or a stroke and she was transferred to a neurological unit. After transfer she remained apyrexial but was then noted to have developed neck stiffness, a positive Kernig's sign and global aphasia. Neurological examination of the cranial nerves was normal and reflexes of the limbs were present and symmetrical. One hour later she had a temperature of 38 °C. A CT scan was performed which showed no abnormality. Lumbar puncture demonstrated CSF pressure of 12 cm H₂O, WBC 725 x 10⁹ litre⁻¹ (74% polymorphs, 12% monocytes, 14% lymphocytes) and RBC 27 x 10⁹ litre⁻¹. Increased protein (1.82 g litre⁻¹) and normal glucose (2.3 mmol litre⁻¹) concentrations were seen and blood glucose concentration was 5.0 mmol litre⁻¹. No organisms were seen or subsequently cultured in CSF or blood. A full blood count showed a haemoglobin concentration at 10 g dl⁻¹, WBC 16.9 x 10⁹ litre⁻¹, granulocytes 10.8 x 10⁹ litre⁻¹ and platelets 196 x 10⁹ litre⁻¹. A provisional diagnosis of either bacterial or aseptic meningitis was made and she was given i.v. chloramphenicol, benzylpenicillin, ampicillin, flucloxacillin and metronidazole. The antibiotics were chosen to cover both the common organisms known to cause meningitis and, in view of the history of a recent breach of the dura, more unusual pathogens. The following day, within 10 h of commencing antibiotic treatment, the headache and neck stiffness had improved and aphasia had resolved. The antibiotics were continued for 5 days and she was discharged on day 5 having made a complete recovery.

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recovery. In view of the rapid recovery and absence of positive cultures, despite no prior antibiotic treatment, this illness was attributed to acute aseptic (chemical) meningitis.

CASE 2
A 38-yr-old primiparous woman was admitted in early labour with a known low-lying placenta. A regional block was requested for analgesia before controlled rupture of membranes. A combined spinal– extradural block was performed in the operating theatre. After preparation of the skin with unstained chlorhexidine in alcohol, the extradural space was located, a 27-gauge Whitacre spinal needle was passed through a Tuohy needle and a spinal injection containing fentanyl 25 μg and bupivacaine 2.5 mg was given. The extradural catheter was sited with 3 cm remaining in the extradural space. The membranes were ruptured successfully and she was transferred to a labour ward room for continuation of labour.

The extradural was topped up by the midwife on four occasions, each time with 15 ml of a solution containing 0.1% bupivacaine and 0.0002% fentanyl. After 6 h the block was noted to have become unilateral and the extradural was resited. After another 3 h the decision was made to perform emergency Caesarean section after fetal blood sampling recorded a pH of 7.16. The anaesthetist judged the extradural to be inadequate for Caesarean section and performed a further single shot spinal using heavy bupivacaine 1.5 ml via a 27-gauge Whitacre spinal needle. A satisfactory block was obtained and Caesarean section resulted in the delivery of a live male infant at 14:50.

The following day the patient was seen by the anaesthetist when she was well. On the third day after operation the anaesthetist was requested to review her in the morning because she had developed a postural headache. This was typical of a post-dural puncture headache; she had been afebrile for the preceding 48 h and was advised to have an extradural blood patch which was performed at 18:15. During the procedure she developed transient pain radiating into the legs. Later that evening both the leg pain and headache had resolved. At 01:30 she vomited and at 02:15 she again vomited and was noted to have a temperature of 38.2°C, heart rate of 82 beat min⁻¹ and arterial pressure of 110/70 mm Hg. At 08:00 she complained that the headache was worse, she had severe neck stiffness and was again pyrexial with a temperature of 38°C. A full blood count was obtained and Caesarean section resulted in the delivery of a live male infant at 14:50.

No organisms were seen but Staphylococcus epidermidis typical of a community skin commensal, was isolated from CSF after 48 h. The patient was treated with i.v. vancomycin and cefotaxime and made an uneventful and complete recovery.

Discussion
We have described two cases of meningitis after central block for obstetric analgesia. Such a serious complication is rare; none was reported in a retrospective postal survey involving 505,000 extradural blocks over a 5-yr period in the UK [1]. However, there have been several reports of meningitis after spinal and extradural anaesthesia. Roberts and Petts [2] described a case developing 18 h after spinal anaesthetic for manual removal of placenta and questioned if this type of anaesthesia is safe in the presence of bacteraemia or for potentially bacteraemic manipulations [2]. However, diagnostic lumbar puncture is considered safe in the investigation of pyrexial patients. Lee and Parry reported a case of meningitis occurring 24 h after urgent Caesarean section [3]. In neither case was any organism seen on gram staining of CSF or subsequently cultured, but the clinical features combined with low CSF glucose concentration suggested bacterial meningitis. Both patients were given i.v. antibiotics and made a full recovery. Sansome, Barnes and Barrett reported a case of meningitis occurring on the third day after an accidental dural tap [4] and Berga and Trierweiler described a parturient who received extradural blood patch on two occasions after a dural tap with the extradural catheter and developed bacterial meningitis on the third day postpartum [5]. CSF cultures grew Streptococcus sanguis which is a commensal mouth organism. Kilpatrick and Girgis reviewed a series of 17 non-obstetric patients admitted to a meningitis ward in Cairo who had a recent spinal anaesthetic [6]. Four of the patients died. Ten of the 17 had positive CSF cultures; eight were Pseudomonas aeruginosa, one was Staphylococcus aureus and one was Streptococcus mitis. The data suggested strongly that meningitis in patients with recent spinal anaesthesia was commonly caused by unusual or nosocomial organisms.

Bacterial meningitis associated with extradural anaesthesia where no breach of the dura has occurred is less common but has been reported in two cases [7], the causative organisms being Streptococcus faecalis and an alpha haemolytic streptococcus, Streptococcus uberis. Davis, Hargreaves and Robinson described a parturient who developed meningitis with group B beta haemolytic streptococcus 40 h after uncomplicated vaginal delivery with extradural analgesia [8]. The organism was also isolated from a high vaginal swab and blood cultures. While carriage of this organism is not uncommon, maternal meningitis is extremely rare.

Acute chemical or aseptic meningitis after spinal anaesthesia was a well recognized complication until the 1940s. In a large series it was reported as occurring in 1 in 400 spinal anaesthetics [9]. Since then cases have been reported sporadically [10, 11] and the reasons for this decline are discussed by Bert.
and Hans Laasberg [12]. Chemical meningitis is a clinical syndrome, characterized by fever, headache, neck stiffness and photophobia. When associated with spinal anaesthesia, it has an acute onset within 24 h of dural puncture and a self-limiting and benign course. Lumbar puncture reveals cloudy CSF under increased pressure. A raised CSF white cell count is usually caused by polymorphonuclear cells but occasionally lymphocytes. No organism is seen on microscopy and none is grown from culture. Protein concentration is increased and CSF glucose concentration is normal. Symptoms resolve rapidly without specific treatment and lumbar puncture is normal within a week.

Chemical meningitis has been attributed most often to a contaminant, usually a disinfectant or detergent used in sterilizing the spinal needles and syringes [9, 12–14]. Other cases are believed to be caused by pyrogens contained in the anaesthetic solution, blood or other body protein introduced at the time of lumbar puncture. Awareness of the problem led to improvements in hospital sterilizing and rinsing procedures and a move towards single use needles and syringes, and prepackaged sterile spinal trays. Traumatic taps are less likely when smaller short bevel needles with well fitting stylets are used.

We believe that our first patient suffered from chemical meningitis because of the rapid course of the illness, the CSF biochemistry and the lack of organisms and positive cultures. The decline in the incidence of acute aseptic or chemical meningitis has led to a reduced awareness of the syndrome and the possibility that similar patients may be labelled as partially treated bacterial meningitis. The failure to culture an organism does not necessarily exclude bacterial meningitis and patients will frequently have received i.v. antibiotics before lumbar puncture is performed and this reduces the culture rate. Even when the clinical picture suggests acute chemical meningitis, it is imperative that antibiotics are given to prevent the possible disastrous consequences of untreated bacterial meningitis. In our view the most likely source of contaminant was chlorhexidine spirit solution used for skin preparation. There were no problems with drugs from the same batches used in other patients. Meticulous attention must be exercised to prevent contamination of gloves or unused Tuohy or spinal needles. A change in our standard practice has resulted following this case, whereby skin preparation is applied remote from the extradural trolley and is not allowed to contaminate needles or catheters.

Our second patient suffered from bacterial meningitis which was caused probably by a skin commensal organism. Bacterial meningitis after spinal and extradural anaesthesia and extradural blood patching is rare. Many unusual nosocomial organisms have been isolated and this has important implications for antibiotic therapy. Broad spectrum antibiotics covering both gram-positive and gram-negative organisms should be started before culture results become available. In view of the time course of the illness we feel that this may have been introduced at the time of the extradural blood patch.

Blood patching is accepted as an effective treatment for post-dural puncture headache and several authors have reported a lack of serious complications [15, 16]. A recent review from Birmingham, however, reported that only 64% of patients gained complete relief from a single blood patch [17]. Another 5% obtained relief after a second blood patch. Severe immobilizing back pain has been reported after extradural blood patch [18, 19] and this, together with the possibility of introducing infection into the CNS, should be weighed carefully against the severity of the patient's symptoms.

These cases illustrate the importance of meticulous technique in performing central neural block to prevent both infection and contamination which could cause aseptic meningitis. Headache and meningism in a patient who has had recent spinal or extradural anaesthesia is often attributed to post-dural puncture headache. It is essential that full evaluation of the patient takes place and that meningitis is considered as a differential diagnosis.

References