CASE REPORT

Cerebral venous sinus thrombosis following accidental dural puncture and epidural blood patch

S. Ghatge,* S. Uppugonduri, Z. Kamarzaman
Department of Anaesthesia, University Hospital of North Staffordshire, Stoke-on-Trent, UK

ABSTRACT
We report the case of a woman who developed cerebral venous sinus thrombosis after an attempted epidural. The epidural was complicated by an accidental dural puncture and the ensuing headache was initially treated with an epidural blood patch. Cerebral venous sinus thrombosis is an uncommon condition with varying aetiology and risk factors. We discuss the importance of the differential diagnosis for postpartum headache and explore the relationship between cerebral venous sinus thrombosis and the triad of pregnancy, dural puncture and epidural blood patch.

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* Correspondence to: Satyajeet Ghatge, Department of Anaesthesia, University Hospital of North Staffordshire, Stoke-on-Trent, UK, ST4 7LN. Tel.: + 44 (0)1782 715444.
E-mail address: skghatge@hotmail.com

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Introduction
Epidural analgesia for labour is becoming increasingly popular. Post-dural puncture headache (PDPH) following epidural insertion is a known complication. Cerebral venous sinus thrombosis is an uncommon condition with the incidence in pregnancy quoted as 1:10 0001 to 1: 25 000.2

Headache is the commonest symptom of cerebral venous sinus thrombosis,3 and can sometimes lead to difficulty with diagnosis and delay in treatment especially if accidental dural puncture has occurred. The significance of cerebral venous sinus thrombosis in the peripartum period has been highlighted in the 2000-02 report on confidential enquiries into maternal deaths.4

We describe the case of a woman who had attempted epidural analgesia for labour, complicated by an accidental dural puncture, and who subsequently developed PDPH. The woman was initially treated with an epidural blood patch, with relative and temporary success. The headache recurred together with other neurological symptoms and a diagnosis of cerebral venous sinus thrombosis was made. We discuss the importance of the differential diagnosis for post partum headache and explore the relationship between the diagnosis of cerebral venous sinus thrombosis and the triad of pregnancy, dural puncture and epidural blood patch.

Case report
A 22-year-old Caucasian primigravida (height 157 cm, weight 70 kg) was admitted to the labour ward in active labour at 37 weeks of gestation following an uneventful pregnancy. She had mild asthma for which she occasionally used a salbutamol inhaler.

In labour, an attempt to place an epidural catheter at L3-4 space by a trainee anaesthetist using a 16-gauge Tuohy needle resulted in accidental dural puncture. The procedure was abandoned and the woman was advised to lie flat and increase fluid intake. Three hours later, she requested epidural analgesia again. A different anaesthetist attempted epidural placement following reassessment and reiteration of possible complications. The epidural space was then identified with a loss of resistance to saline using a 16-gauge Tuohy needle and a catheter inserted. There was no obvious dural puncture during the procedure. However, clear cerebrospinal fluid was aspirated from the catheter. A senior anaesthetist was informed, who decided that the catheter should be removed and further attempts stopped. Intravenous remifentanil via patient-controlled analgesia was started with good effect and a live baby was delivered spontaneously shortly after.

Twenty-four hours after delivery the patient complained of headache and was seen by the duty anaesthetist. The headache had a frontal and occipital distribution and was relieved on lying supine. It prevented her from sitting up and nursing the baby. There were no other neurological symptoms or signs. A provisional diagnosis of PDPH was made and she was
treated conservatively with oral analgesia and bed rest and was advised to increase oral fluid intake. The possibility of an epidural blood patch was discussed.

The following day she agreed to have a blood patch; the procedure was performed by a senior anaesthetist. Ten millilitres of autologous blood was placed at the L1-2 space with instantaneous improvement although incomplete relief of the headache. Over the next 48 h the headache improved but had a more frontal and nuchal distribution. There were no associated focal neurological signs and a second blood patch was offered. She declined and continued with conservative treatment. She was reviewed daily to ensure that there was no worsening of symptoms or development of new neurological signs.

On the seventh day after delivery the woman requested to be discharged home as she felt better. She was advised to continue with conservative management and to return to hospital if the headache worsened or if there was any other cause for concern. Three days after discharge, she presented to hospital complaining of headache and exhaustion. The headache had now become mid-frontal and no longer affected by posture. She also reported transient episodes of being unable to feel her legs and difficulty with balance. A neurological examination was conducted by the duty anaesthetist. Her Glasgow Coma Scale was 15/15. There was no evidence of central or peripheral neurological deficit, her gait was normal and no cerebellar problems were identified. Her superficial and deep reflexes were normal, her pupils were unremarkable but fundoscopy was not done. She was reassured and discharged home but was advised to come back if symptoms worsened.

Twelve hours later, the patient presented to the accident and emergency department with confusion, expressive dysphasia, right sided weakness, lethargy and vomiting. She was immediately referred to the neurologists. Examination revealed a right-sided weakness in both upper and lower limbs with an up-going plantar reflex of the right foot. Nystagmus was present in the left eye. CT venography showed a basal ganglion infarct with mass effect. Thrombi were seen in the vein of Galen, straight sinus and posterior third of the superior sagittal sinus (Fig. 1). There was an area of low-density involving the left deep basal ganglion region suggesting a venous infarct. This resulted in the compression of left lateral ventricle causing early hydrocephalus. The radiologist concluded that the appearance was highly suggestive of acute/subacute deep basal ganglion infarct probably secondary to venous thrombosis.

A heparin infusion was started. Further blood investigations including rheumatoid factor and autoantibody screening were negative. Over the next 10 days, the neurological status resolved and the patient was prescribed warfarin. A further CT scan of the brain before discharge showed resolution of the thrombus.

**Discussion**

Headache is common in pregnancy with primary headaches being 20 times more frequent than secondary headaches. Five percent of women are reported to have headache in the postpartum period. For those who have received a regional block with either apparent or possible accidental dural puncture, a postural headache is invariably considered to be the result of dural puncture. With a growing number of women now receiving epidural analgesia during labour, there is an increase in the number of accidental dural punctures. Rates vary between 0.19% and 3.6%. Conservative treatment of PDPH is often inadequate and epidural blood patch is frequently required. The success rate of epidural blood patch has been quoted as 60-70%, with that of a second epidural blood patch reported to be similar giving an overall success rate of >90%. A recent UK survey suggested that, if two successive epidural blood patches were unsuccessful or the headache recurred, further investigations involving diagnostic imaging should be considered to exclude other possible causes for the headache.

The incidence of cerebral venous sinus thrombosis during pregnancy is quoted as 1 in 10 000 to 1 in 25 000. Between 70 and 90% of cases present with headache. Damage to the venous sinuses due to
fluctuations in intracranial pressure during delivery and the hypercoagulable state of pregnancy due to increased platelet adhesion and an increase in clotting factors are the possible explanations for the increased risk of cerebral venous sinus thrombosis in the peripartum period. The incidence of cerebral venous sinus thrombosis and PDPH with or without epidural blood patch is unknown. Our literature search revealed seven case reports of postpartum women who had evident or possible dural puncture in whom the diagnosis of cerebral venous sinus thrombosis was made on radiological investigation.

Dural puncture causes loss of CSF and lowering of CSF pressure. The loss of CSF produces compensatory venodilatation increasing intracerebral blood volume, which may lead to further decrease of blood flow. Measurement of venous blood flow velocity with transcranial Doppler ultrasound immediately before, during and after lumbar puncture has demonstrated a decrease in mean blood flow velocity in the sagittal sinus, which remained for more than 6 h. There is a correlation between the magnitude of the decrease in intracranial pressure induced by lumbar puncture and the decrease in the mean blood flow velocity in the sagittal sinus. It has been suggested that the sudden decrease in CSF pressure after dural puncture could activate brain adenosine receptors, which produce venous and arterial dilatation responsible for PDPH.

While the mechanisms that could contribute to cerebral venous sinus thrombosis in the puerperium following possible dural puncture are highly complex, the use of epidural blood patch to treat PDPH further adds to this complexity. The blood patch occludes the dural puncture, preventing further CSF leak, thus relieving headache. However, conclusions from animal studies suggest that an epidural blood patch relieves headache by cerebral vasoconstriction. This is thought to be caused by subarachnoid spread of injected blood or by the epidural blood patch acting as a signal and antagonizing adenosine receptors in a similar fashion to caffeine and theophylline. The effect of this cerebral vasoconstriction, both arterial and venous, is to restore the cerebral hemodynamics and thus relieve headache. In our case, as in others, cerebral venous sinus thrombosis was diagnosed after epidural blood patch was performed and the headache persisted. In such cases, the role of epidural blood patch and the possible cerebral vasoconstriction caused remains unclear. It is questionable whether the vasoconstriction and its effects on cerebral blood flow contributed to cerebral venous sinus thrombosis.

One of the difficulties with this case was establishing the diagnosis. With a documented dural puncture and postural headache the most likely initial diagnosis was of PDPH. The woman was at first treated conservatively and then received a blood patch. Although symptoms improved the headache persisted and a second blood patch was offered but declined. On day 10 when the patient returned to hospital with headache, exhaustion and neurological symptoms, she was assessed by the duty anaesthetist. Involvement of a senior neurologist at this stage could have helped in diagnosis and treatment. Furthermore the input of a radiologist and the role of diagnostic imaging are important. The definitive investigation for diagnosing cerebral venous sinus thrombosis is a CT angiogram. Magnetic resonance imaging (MRI) is also helpful and may be the investigation of choice when suspicion arises.

The management of the dural puncture could have been different. In retrospect, an intrathecal catheter could have been placed when the first dural puncture occurred. Similarly, a greater volume of blood could have been used at a lower lumbar interspace when performing the epidural blood patch. There is, however, evidence that a lower volume of blood can be effective in the treatment of PDPH.

The aim of treatment of cerebral venous sinus thrombosis is to reduce intracranial pressure and to avoid extension of the thrombus. Our patient was immediately given intravenous heparin followed by warfarin once discharged from hospital. In a reported series of 38 patients with angiographically proven cerebral venous sinus thrombosis, four out of 15 untreated patients died compared with none of the 23 patients treated with anticoagulants. Extremely severe cases, involving seizure and progressive neurological deficit, were treated successfully with urokinase.

There is an associated ten-fold increase in the risk of venous thromboembolism in pregnancy. Women above the age of 35 years with a body mass index greater than 30 kg/m² are at particular risk. Our patient was otherwise fit and well with no known risk factors for venous thromboembolism. She was thus not considered for early thromboprophylaxis. In retrospect, this could have also contributed to the development of cerebral venous sinus thrombosis. With her mobility limited in the early postpartum period, with headache and bed rest, she should have been considered for thromboprophylaxis. We suggest that women who develop PDPH post partum should receive thromboprophylaxis.

This case emphasizes the importance of reviewing the diagnosis thoroughly and including cerebral venous sinus thrombosis as part of the differential diagnosis, when treating persistent PDPH. There is a need for a multidisciplinary approach and early involvement of neurologists and radiologists. Early use of diagnostic imaging could reduce morbidity and mortality associated with cerebral venous sinus thrombosis. We believe that obstetric units should have protocols for the management of PDPH, which include thromboprophylaxis for patients with restricted mobility and guidance on early involvement of neurologist and radiologist for patients with persistent headache after EBP.
References