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CASE REPORT

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

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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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Keywords: Cerebral venous sinus thrombosis; Dural puncture; Epidural blood patch; Pregnancy

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 remifentanyl 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

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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.

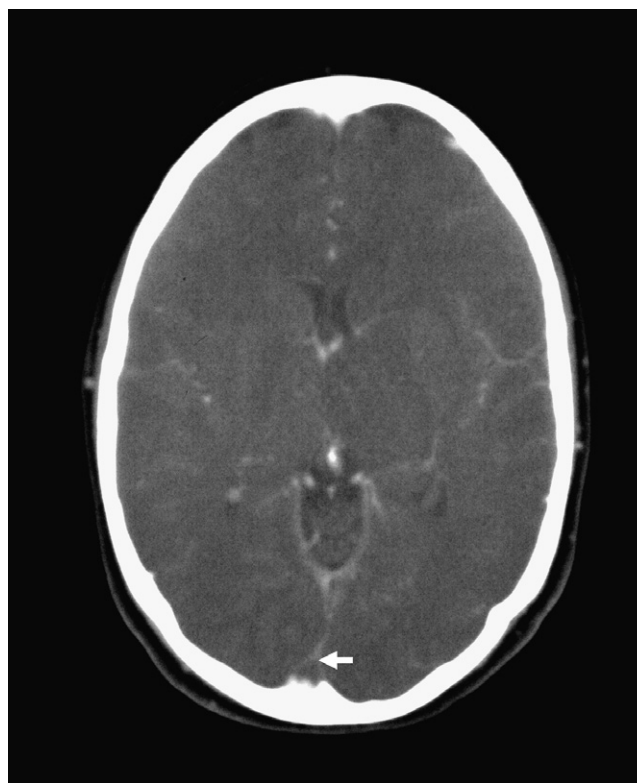


Figure 1 Superior sagittal sinus thrombosis (filling defect shown by arrow).

Discussion

Headache is common in pregnancy with primary headaches being 20 times more frequent than secondary headaches.⁵ Thirty-nine percent of women are reported to have headache in the postpartum period.⁶ 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%.¹² 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%,¹³ with that of a second epidural blood patch reported to be similar giving a overall success rate of >90%.¹⁴ 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.¹⁵

The incidence of cerebral venous sinus thrombosis during pregnancy is quoted as 1 in 10 000¹ to 1 in 25 000.² Between 70 and 90% of cases present with headache.^{3,9-11} Damage to the venous sinuses due to

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