

Case Report

Postpartum Headache and Convulsions as Complications of Dural Puncture: A Case Report and Review of Intracranial Hypotension

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Abstract: We report the case of a 30-year-old female patient, G2P1001, presenting initially with headache, followed by tonic-clonic seizures tonic-clonic seizures on postoperative day 5 of an elective caesarean section indicated for transverse position, and performed after epidural anaesthesia. A cerebral revealed engorgement of the sagittal venous plexuses, and suggested the diagnosis of the diagnosis of intracranial hypotension. Management was multidisciplinary, including anti-epileptic treatment, analgesics and an epidural blood patch, leading to a favourable outcome.

Keywords: Intracranial hypotension; epidural puncture; postpartum; caesarean section; CT scanner.

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INTRODUCTION

Post-dural puncture headaches associated with convulsive seizures in the post-partum period may be related to intracranial hypotension, which should be considered after ruling out other causes, particularly those related to pregnancy [1]. We report here the case of a 30-year-old patient whose diagnosis of intracranial hypotension due to dural rupture was evoked on CT scan, leading to appropriate treatment and a favourable outcome.

CASE REPORT

This was a 29-year-old woman, G2P2001, with no contributory history, who had given birth by caesarean section to a full-term newborn (39 weeks' amenorrhoea and 2 days), male, weighing 3,350 grams, with a good Apgar score (7/8/9). The Caesarean section, which had been indicated for transverse presentation, was performed at the Douala Gynaeco-Obstetric and Paediatric Hospital, after epidural anaesthesia. At 2 days post-operatively, the patient complained of diffuse pulsatile headache of intensity 7/10, exacerbated by

sitting, which was initially treated as post-dural puncture headache (PDPH) with oral analgesics (paracetamol followed by tramadol). Three days later, the patient presented with several episodes of tonic-clonic convulsions of sudden onset, with ocular revulsion, with no signs of focalization, in an afebrile context. Blood pressure was normal (109/85 mmHg), as was heart rate (91 beats/min). There was no sign of meningeal irritation on clinical examination, and the diagnosis of de novo epilepsy was retained (an intracranial expansive process to be excluded). She was put on diazepam (1 ampoule intramuscularly), with 2nd tier analgesics, pending the results of her additional work-up. A blood ionogram showed moderate hypocalcaemia (83.86 mg/l), and a urine dipstick was negative. A cerebral tomodensitometry was carried out using a HITACHI Supria 16 multibarette scanner, without and after injection of contrast medium. The scan spontaneously revealed excessive visibility of the sagittal sinus (Figure 1A), associated with engorgement of the other venous plexuses (Figure 2), particularly the sagittal sinuses (Figure 1B). The diagnosis of intracranial hypotension due to dural rupture was accepted. Hospital management

was adjusted with anti-epileptic drugs (phenobarbital 200 mg/day intramuscularly), analgesics (nefopam electric syringe pump) and rehydration (approximately 3 litres/24 hours). An epidural blood patch was then placed

over the L4-L5 interbody space, after 20 ml of blood had been drawn from the cephalic vein. The outcome was favourable, and the patient was discharged home at 11 days post-partum.

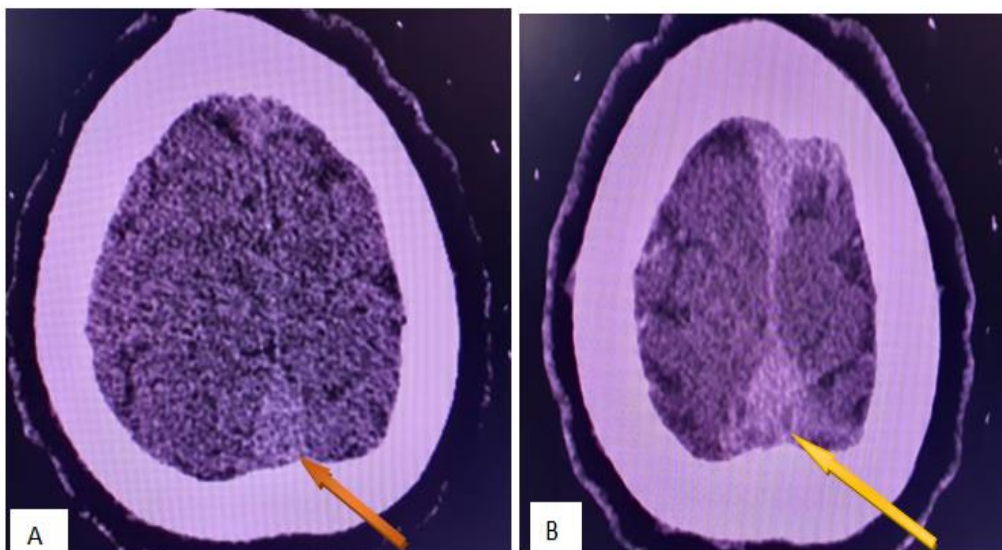


Figure 1: Axial sections of cerebral CT without (A) and with contrast medium, (B), showing a spontaneously sparse superior longitudinal sinus (brown arrow), with engorgement after IV PDC (yellow arrow)

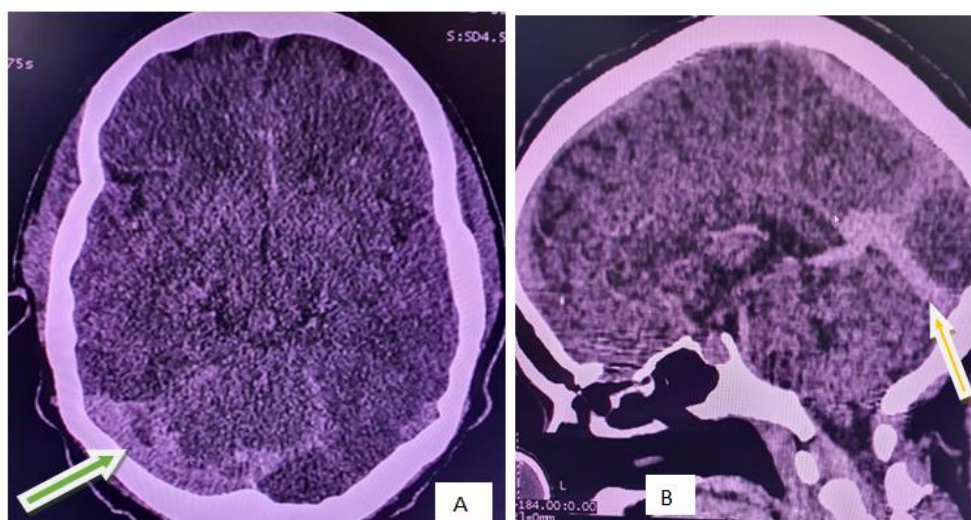


Figure 2: Axial section (A) and sagittal reconstruction (B) of cerebral CT with contrast medium, showing engorgement of the right transverse sinus (green arrow) and right sinus (orange arrow)

DISCUSSION

Intracranial hypotension secondary to a meningeal breach has been known since 1898, when the first spinal anaesthesia was complicated by headaches [2]. More recently, a North American study by Shearer *et al*, which looked at 19,000 obstetric spinal anaesthetics over a period of 10 years, found that 5% of patients suffered from post-dural puncture headaches, some of whom had epileptic seizures (incidence of around 1:100) [3]. It is important to emphasise, as Shearer *et al*, pointed out, that this case report should not be seen as an indictment of spinal anaesthesia [3]. In fact, spinal anaesthesia is used in 76% of vaginal deliveries and 94% of caesarean deliveries in the United States [4]. In our

environment too, elective caesarean sections are generally performed under locoregional anaesthesia via the epidural route [5]. However, this method can present certain complications, such as a meningeal breach, with leakage of cerebrospinal fluid (CSF) from the subarachnoid space, responsible for post-dural puncture syndrome. The main risk factors for dural-arachnoid invasion are the calibre of the needle, the sharpness of its tip, and the orientation of the bevel when it is introduced [6].

Post-dural puncture syndrome, which is characterised by typical symptoms such as orthostatic headaches, vertigo, tinnitus, diplopia, nausea and

epileptic seizures [7]. The major sign is headache, which is thought to be due to traction on the meningeal structures and blood vessels, and generally appears within 3 days [8]. In our patient, too, the headaches started on day 3 and the attacks began on day 5 post epidural puncture. According to the literature, convulsive seizures occur between days 2 and 73, and present a real diagnostic challenge [1].

Other differential diagnoses of post-partum seizures include obstetric causes (eclampsia, Sheehan's syndrome, and amniotic fluid embolism), anaesthetic causes (post-dural puncture headache, and gas embolism), neurological (epilepsy, stroke, posterior reversible encephalopathy), metabolic (withdrawal syndrome, electrolyte abnormalities, hypoglycaemia), infectious (meningitis or encephalitis), and psychiatric (pseudoseizure) [9]. Several authors, such as Song *et al*, recommend that post-partum eclampsia should be considered as a priority [10]. This hypothesis was not accepted in our patient, who had normal blood pressure and a negative urine dipstick. Similarly, infectious causes were ruled out, given the afebrile context and the absence of a biological inflammatory syndrome.

With regard to the paraclinical work-up usually requested for the investigation of post-dural puncture headaches associated with post-partum convulsions, an electroencephalogram is recommended, in order to identify a primary epileptic disorder [11]. Manometry shows a drop in CSF opening pressure of less than 6cm H₂O at lumbar puncture, but is not constant [12], and is not necessary in centres with access to MRI [13]. Biological tests include a blood count, urine test, blood ionogram, coagulation test, liver enzymes and blood creatinine [14].

As far as imaging is concerned, cerebral tomography is the examination of first choice, carried out without and after injection of a contrast product, and analysed using multiplanar reconstructions [15]. It can be used to diagnose intracranial haemorrhage or cerebral ischaemia [14]. It can also reveal meningeal thickening and enhancement, ventricular collapse and engorgement of the dural venous sinus [15]. In our patient, the CT scan showed engorgement of the venous plexuses, particularly the sagittal sinuses.

According to some authors [12], encephalic magnetic resonance imaging (MRI) can be used to confirm the diagnosis. It reveals a diffuse thickening of the intracranial meninges, strongly enhanced after injection of gadolinium, which is pathognomonic of this pathology [6]. This appearance is reversible, disappearing completely a few weeks after the dural breach has been sealed [16]. MRI also reveals subdural fluid collections, an increase in the height of the pituitary gland, and complicating signs such as subdural haematomas and cerebral venous thrombosis [12]. MRI of the lumbar spine (with proton density and T2

sequences) shows retraction of the dural sac, vasodilatation of the epidural plexuses, CSF in the epidural space, and possibly haemosiderosis indicating vascular invasion during puncture [6].

Radioisotopic cisternography can identify sites of CSF leakage in the lumbar region [17]. Ultrasound can be useful for exploring the lumbar spine, looking for breaches of a few millimetres in size caused by Tuohy needles [17]. It can also be used to study the polygon of Willis using the Doppler mode, noting a reduction in the speed of blood flow in the middle cerebral artery, in relation to vasodilatation [3].

The management of intracranial hypotension secondary to a dural tear is multidisciplinary, involving strict decubitus, hyper-hydration, drug treatment, and above all the epidural blood patch, which has proved effective, or at least helps to reduce the intensity and duration of headaches [2, 18].

CONCLUSION

Post-partum intracranial hypotension must be ruled out in the presence of orthostatic headaches and post-dural puncture epileptic seizures. Neuroradiological diagnostic work-up using cross-sectional imaging techniques should be rapid, improving multidisciplinary management, reducing the need for ineffective treatments and avoiding the onset of chronic pain syndromes.

Conflicts of Interest: The authors declare no conflicts of interest.

Authors' Contributions

All authors contributed to the research work. They read and approved the final version of the manuscript.

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