Case Report of a Parturient with Post Dural Puncture Headache (PDPH) and Coincidental Late-Onset Preeclampsia

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Abstract

Preeclampsia has several subtypes. Of those, early-onset (EO-PE) and late onset (LO-PE) are the best known. These are classified by the time of delivery. EO-PE is Pregnancy Induced Hypertension (PIH) before 34 weeks and LO – PE after 37 weeks while the intermediate (34-37 weeks) is a mixture of both types. Late onset preeclampsia has been reported as late as 3 months after delivery.

We described a case of a 30-year-old lady who had inadvertent dural puncture and also coincidentally late onset preeclampsia presenting a clinical dilemma. These complications were recognized, diagnosed and the patient made full recovery. This paper presents case details, reviews the literature and management of late onset preeclampsia.

CASE PRESENTATION

A 36-year-old, 126 kg woman was admitted to labour suite and was in active labour. She had completed 39 weeks of gestation. She had a previous caesarean section 6 years ago under spinal analgesia and had an uneventful post-operative course. She had been irregular with antenatal visits. She was keen to have a normal delivery.

On admission, her blood pressure was 130/76mm Hg, pulse 90/min and respiratory rate of 16-18/minute.

Labour analgesia was requested by the patient and ordered by the obstetrician. During the first attempt, there was an inadvertent dural tap at L3-L4 interspace with 18G Tuohy needle. Spinal blockade due to the inadvertent dural tap and intrathecal placement of the catheter was recognised and a spinal dose of 2.5 ml of 0.25% bupivacaine was given through the epidural catheter.

The patient was explained that there had been a dural tap and counselling for the P.D.P.H. along with the explanation of signs and symptoms of PDPH. Due to non-progress, it was decided to have a caesarean section. Her blood pressure remained normal during caesarean section and immediate postpartum period. On the first postpartum period, she

complained of headache but had a normal blood pressure. She refused an epidural blood patch and conservative treatment with oral fluids, analgesics, caffeine and bed rest was started.

By early morning, she complained of photophobia followed by an episode of generalized tonic-clonic convulsions. The patient maintained her airway during the attack. Oxygen was administered during the convulsion. The patient was shifted to intensive care unit and started with magnesium SO4 prophylaxis. On examination her blood pressure was 140/102 mm Hg, respiratory rate 14-16/minute and pulse rate of 90/minute.

Simultaneously she was also started treatment with hydralazine infusion at the rate of 5 mg/hr and was increased every 15-20 minutes by 1 to 2 mg/hr to obtain a 20% reduction in the mean arterial blood pressure along with MgSO4 prophylaxis. A neurological consultation was called and the neurologist concurred with the diagnosis. The CT scan showed occipital lobe involvement and diffuse brain edema showing a clinical correlation of visual disturbance and headache respectively.

Over the next two days, the patient started with the

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symptomatic improvement and the headache improved. She was subsequently discharged with oral nifedipine (20 mg) B.D. There was no protein in the urine (checked twice daily) and the coagulation profile and serum electrolytes were normal.

Follow up at 2-4 weeks showed continued improvement of her blood pressures with the values returning to the pre – delivery baseline values.

DISCUSSION & CONCLUSION

Confusion can be associated with late onset postpartum eclampsia because it can occur in women who are not previously diagnosed by preeclampsia. Postpartum, preeclampsia is a rare condition usually occurring within the first 48 hours after birth but it can occur as late as six weeks postpartum.

Women with gestational hypertension or preeclampsia are able to stop all the antihypertensives within six weeks postpartum. Preeclampsia can cause headaches, visual disturbances, nausea and vomiting and other unpleasant symptoms. Occasionally, preeclampsia presents for the first time up to four weeks after birth.

Wintson1 reviewed 152 cases of eclampsia and found that 37 (27%) occurred in postpartum period and of these, 17 (47%) occurred at least 48 hours postpartum.

Sibai B.M2 et al reviewed six cases of eclampsia occurring 3 or more days and concluded that seizures which occur after delivery, even after 48 hours, should be considered as eclampsia until proven to the contrary. A full neurological and metabolic evaluation to rule out other causes of seizures should be performed.

Lubarsky3 et al reported that in a study including 334 women who developed eclampsia, 16% were diagnosed with late onset pre-eclampsia and of these, 44% had not been previously diagnosed with pre-eclampsia.

Brown CE4 et el observed and provide strong support for

waiving the 24-hour rule at least when convulsions from no other apparent cause and accompanied by hypertension and proteinuria occur in a primipara as late as 10 days postpartum. Late postpartum eclampsia seems an appropriate term for this very uncommon condition.

There is a decreased incidence of PDPH after unintentional dural puncture in parturients with an increased BMI even after controlling for pushing during labour.

PDPH and PIH can be difficult to differentiate because of the common features such as photophobia, headache and convulsions. In our case, hypertension was attributed to the anxiety and headache and visual changes were attributed to the Dural puncture.

When a dural puncture occurs and a PDPH is suspected, the epidural blood patch should be a definitive treatment and would rule out the PDPH as a cause of headache. So, the differential diagnosis of PDPH should be also the late onset postpartum preeclampsia along with the other differential diagnosis for headache and treatment should be initiated immediately so as to avoid morbidity and mortality.

LIST OF ABBREVIATIONS

EO PE - Early onset preeclampsia.

LO PE - Late onset preeclampsia.

PDPH - Post dural puncture headache.

PIH - Pregnancy induced hypertension.

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