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Benign paroxysmal positional vertigo presenting as persistent vomiting in a parturient using epidural analgesia



A healthy 26-year-old woman, G1P0, was admitted in labor and an epidural catheter was inserted uneventfully. Maintenance of epidural analgesia was with levobupivacaine 1.25 mg/ml and fentanyl 1.25 µg/ml at a rate of 6 mL/h, using patient-controlled epidural analgesia (6 mL on demand at a 15 min lockout interval). This proved effective and without significant complication.

Nausea and vomiting were noted two hours after the insertion and these symptoms persisted for 10 hours despite intravenous fluid administration (normal saline 1 L) and anti-emetic treatment (intravenous metoclopramide 10 mg). No other gastro-intestinal symptoms, such as abdominal pain, tenderness or diarrhea, were noted. Her blood pressure was within normal limits and she had a mild tachycardia. We considered it unusual for a parturient to have persistent nausea and vomiting so did a brief neurological examination, in order to rule out serious causes such as increased intracranial pressure induced by pre-eclampsia. Horizontal rotatory nystagmus was a significant finding and the woman volunteered that vertigo, nausea and vomiting were induced by changing position.

Benign paroxysmal positional vertigo (BPPV) was our provisional diagnosis and the Dix-Hallpike test was performed. With the patient sitting upright, we held her head in 45° rotated to the left. She was then instructed to keep her eyes open while she was taken backward to lie down with a pillow under her shoulders to extend her head 20°. Up-beating, left horizontal rotatory nystagmus with a five-second latency and lasting for nearly 30 seconds was induced, and the patient experienced vertigo and vomiting again after the maneuver. Left posterior canal BPPV was diagnosed due to the positive result of the left-sided Dix-Hallpike test. Following the diagnosis, we carried out the Epley maneuver to treat the patient (Fig. 1). The initial maneuver was the same as the Dix-Hallpike test, holding the woman's head with 45° rotation to the left when she sat upright, before taking her backward to a supine position with her head rotated 45° to the left and extended 20° by the pillow. This position was maintained for 30 seconds before turning her head 90° to the right for 30 seconds. Next, we had the patient roll on her right shoulder and turn her head a further 90° to the right, making her head facing down at a 45° angle for 30 seconds. Finally, we brought her to the upright sitting position to complete the maneuver. Her symptoms were relieved after the treatment. She delivered her infant 18 hours after commencing epidural analgesia and was discharged three days later without a recurrence of BPPV.

Benign paroxysmal positional vertigo may be caused by cupulolithiasis. It is the most common cause of dizziness or vertigo, and some studies suggest an association with estrogen change and pregnancy.^{1–3} Prolonged bed rest may be another cause of BPPV in pregnant women, especially if they are usually advised to sleep on their left side to decrease vena caval compression. The gold standard to diagnose BPPV is the Dix-Hallpike test and treatment is by the Epley maneuver or with antihistamines and benzodiazepines. In our opinion, the Epley maneuver is the first-line treatment, not only because of cost-effectiveness but because it is a physical rather



Fig. 1 Dix-Hallpike test and Epley maneuver. Step 1: Let the patient sit upright, with her head rotated 45° to the left. Step 2: Take the patient backward to a supine position with her head rotated 45° to the left. Use a pillow under her shoulders to keep her head in 20° of extension. Step 3: Ask the patient to turn her head to the right until it is rotated 45° to the right. Step 4: Move the patient to the right lateral decubitus position, with her head still rotated 45° to the right. Step 5: Help the patient to sit upright, with her head in the neutral position. Dix-Hallpike test for left posterior canal BPPV: Step 1 to 2. Epley maneuver for left posterior canal BPPV: Step 1 to 5, maintaining each step for 30 seconds. For diagnosing and treating right posterior canal BPPV, use the same steps but reverse the left and right sequence. BPPV: benign paroxysmal positional vertigo.

than pharmacological treatment. In conclusion, detailed history taking and focused physical examination are the keys to the correct diagnosis. In this case, they helped us find the nystagmus which led us to the diagnosis of BPPV.

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Spinal anaesthesia for elective caesarean section in a patient with hereditary neuropathy with liability to pressure palsies



Hereditary Neuropathy with Liability to Pressure Palsies (HNPP) is a rare (0.4% prevalence), autosomal dominant neuromuscular disorder with variable penetrance.¹ It is characterised by susceptibility to recurrent sensory or motor neuropathy caused by pressure, stretch or repetitive use, and most commonly diagnosed in the second or third decade of life. It is caused by a micro-deletion on chromosome 17 containing the gene for peripheral myelin protein (PMP-22).² Injury to the nerve leads to numbness and weakness, caused by areas of demyelination of varying severity and duration, that are most commonly seen in the upper limbs.³ A seemingly trivial insult can cause a palsy, with full recovery only achieved in approximately 50% of cases over a time period of days to months.¹ We describe a case of symp-

tomatic HNPP where caesarean section (CS) was safely performed under spinal anaesthesia.

A 35-year-old primiparous woman presented to the anaesthetic antenatal clinic at 35 weeks' gestation. The diagnosis of HNPP had been made following previous transient hemiparesis following immobility. A previous general anaesthetic (GA) had caused tongue paraesthesia and the use of non-invasive blood pressure measurement (NIBP) resulted in a temporary loss of arm sensation and function lasting two days. The patient had, therefore, declined antenatal NIBP measurement, and urinalysis for proteinuria was used in lieu to screen for pre-eclampsia. At 36 weeks' gestation the patient developed obstetric cholestasis. After multidisciplinary discussion, CS to reduce the likelihood of emergency intervention associated with induction of labour was planned. A review of the English language literature revealed a case report of safely using epidural analgesia for labour, but no reports of HNPP management for CS.

We proceeded with spinal anaesthesia for CS. A 16-gauge intravenous cannula was placed using gentle manual compression rather than tourniquet, and an arterial line placed for blood pressure monitoring and sampling of blood peri-operatively. Following discussion of venous-thromboembolic (VTE) risk the patient declined compression stockings and pneumatic calf compression devices peri-operatively, being concerned by the risk of pressure injury. Agreement for postoperative pharmacological prophylaxis alone with low molecular weight heparin was made. Spinal anaesthesia was established, with subarachnoid block achieved using 2.5 mL hyperbaric bupivacaine and 300 µg of diamorphine (standard at our institution). This was uncomplicated, resulting in adequate anaesthesia. Meticulous attention was paid to positioning and peripheral nerve pressure areas using pillows and gel-pads and padding. An air mattress was utilised postoperatively, with repositioning every 10 minutes until motor function had returned. An uncomplicated CS was performed, spinal anaesthesia regressed over a standard time period, and no new palsies developed.

There is a paucity of literature regarding the anaesthetic management during labour and delivery in HNPP mothers,⁴ with one case report on the successful use of low-dose labour epidural analgesia.⁵ However, several reports of both spinal and general anaesthesia in non-obstetric patients with HNPP supported our understanding for this case.^{6–8} There remains uncertainty regarding the most appropriate dosing of neuraxial anaesthesia, and whether the incidence and risk of permanent neuropraxia is higher than in the general obstetric population. The decision to proceed with spinal anaesthesia was made, as besides being the routine anaesthetic we provide we felt it allowed self positioning