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.

M.-S. Sun
Department of Anesthesiology, Changhua Christian Hospital, Changhua, Taiwan
E-mail address: mengsheng60@me.com

S.-S. Chen
Department of Otorhinolaryngology-Head and Neck Surgery, Changhua Christian Hospital, Changhua, Taiwan

L.-T. Yeh
Department of Anesthesiology, Changhua Christian Hospital, Changhua, Taiwan

References

1. Ogun OA, Büki B, Cohn ES, Janky KL, Lundberg YW. Menopause and benign paroxysmal positional vertigo. *Menopause* 2014;**21**:886–9.
2. Giacomini PG, Napolitano B, Alessandrini M, Di Girolamo S, Magrini A. Recurrent paroxysmal positional vertigo related to oral contraceptive treatment. *Gynecol Endocrinol* 2006;**22**:5–8.
3. Çoban K, Yiğit N, Aydın E. Benign paroxysmal positional vertigo in pregnancy. *Turk Arch Otorhinolaryngol* 2017;**55**:83–6.

0959-289X/$ - see front matter
Elsevier Ltd
https://doi.org/10.1016/j.ijoa.2019.06.001

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 microdeletion 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 symptomatic 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

of the neck and arms throughout the procedure, additionally avoiding the risk of tongue paraesthesia and standard concerns of obstetric patients undergoing general anaesthesia.

Despite the requirement to deviate from our usual perinatal blood pressure monitoring and VTE prophylaxis protocols, we were able to provide a safe and appropriate means of delivery without development of palsies peri-operatively.

K. Samuel, K. Mead, T. Cominos, N. Weale
Department of Anaesthesia, North Bristol NHS Trust Bristol, UK
E-mail address: katie.samuel@nhs.net

References

1. Bird TD. Hereditary neuropathy with liability to pressure palsies. In: Adam MP, Ardinger HH, Pagon RA, et al., ed. GeneReviews [Internet]. Seattle: University of Washington; 1993–2019. Available at: https://www.ncbi.nlm.nih.gov/books/NBK1392/. Accessed April 12, 2019.
2. Chance PF, Alderson MK, Leppig KA, et al. DNA deletion associated with hereditary neuropathy with liability to pressure palsies. *Cell* 1993;**72**:143–51.
3. Behse F, Buchthal F, Carlsen F, Knappeis GG. Hereditary neuropathy with liability to pressure palsies. Electrophysiological and histopathological aspects. *Brain* 1972;**95**:777–94.
4. Kopp SL, Jacob AK, Hebl JR. Regional anesthesia in patients with pre-existing neurologic disease. *Reg Anesth Pain Med* 2015;**40**:467–78.
5. Lepski GR, Alderson JD. Epidural analgesia in labour for a patient with hereditary neuropathy with liability to pressure palsy. *Int J Obstet Anesth* 2001;**10**:198–201.
6. Wijayasiri L, Batas D, Quiney N. Hereditary neuropathy with liability to pressure palsies and anaesthesia: peri-operative nerve injury. *Anaesthesia* 2006;**61**:1004–6.
7. Carassiti M, Doyle DJ, Cataldo R, Mattei A, Gallì B, Agrò FE. Regional anesthesia for a patient with hereditary neuropathy with liability to pressure palsies. *Can J Anaesth* 2007;**54**:325–6.
8. Kramer M, Ly A, Li J. Phenotype HNPP (Hereditary Neuropathy with Liability to Pressure Palsies) induced by medical procedures. *Am J Orthop* 2016;**45**:27–8.

0959-289X/$ - see front matter

https://doi.org/10.1016/j.ijoa.2019.05.004

Displaced cervical spine hardware and esophageal perforation during labor

Mechanical complications of graft and plate implant in a patient with anterior cervical discectomy and fusion are well described long-term complications.[1,2]

A 35-year-old patient in the 39th week of pregnancy received labor epidural analgesia. Her past surgical history included C6-7 discectomy and fusion 10 years prior, subsequent to neck trauma in a motor vehicle accident. During labor she complained of neck pain which worsened after her vaginal delivery. The neck pain was associated with severe nuchal rigidity but neurologic examination was otherwise unremarkable. Cervical spine computed tomography (CT) revealed a screw at C6 (Fig. 1), which was displaced, and bony non-union. A magnetic resonance image (MRI) showed a soft tissue shadow with an epidural collection of fluid at C5-6 but no significant cord deformation. Gastrografin swallow showed esophageal perforation. On post-delivery day five the patient underwent surgery for removal of displaced hardware, and discectomy and fusion of C6-7, for which she was intubated fiberoptically. Following failed conservative management of the perforation, two weeks after delivery she underwent a sternocleidomastoid pedicled muscle flap for repair of the esophagus.

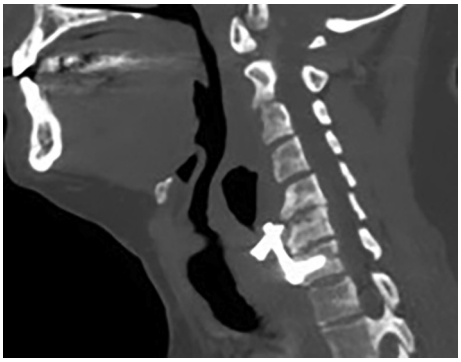

Fig. 1 Cervical spine CT showing a displaced screw at C6.

The physical changes associated with pregnancy may result in difficult endotracheal intubation and in this case the cervical spine fusion added a further challenge. We hypothesize that increased laxity of ligaments and exaggerated movement of the cervical spine during the process of labor and delivery may have resulted in movement of the hardware, causing perforation of the esophagus. In obstetric anesthesia, general anesthesia is most frequently used for emergency indications. If our patient had needed emergency intubation, our lack of knowledge of the hardware displacement might have been catastrophic.

This case emphasizes the need for meticulous clinical examination and a high degree of suspicion for possible hardware displacement in a patient with a history of cervical spine surgery, particularly in one complaining of neck pain. In such a situation having a plan to manage the patient's airway atraumatically, and with the least amount of motion and manipulation, is of paramount importance.