Hypertension and tachycardia following high spinal anaesthesia during lower segment caesarean section: An unusual presentation

Sir,

We describe a case of hypertension following high spinal anaesthesia in a parturient undergoing emergency lower segment caesarean section (LSCS). A 26-year-old primigravida without any comorbidities was brought for emergency LSCS in view of non-progression of labour. Her baseline vitals were as follows: heart rate (HR) 86/min; non-invasive blood pressure (NIBP) 116/72 mmHg; peripheral oxygen saturation (SpO_2) 99% on room air. Subarachnoid block (SAB) was performed with 1.8 ml of hyperbaric bupivacaine (0.5%). Initial sensory level of blockade (checked with pin-prick) was T4. Immediately after administration of SAB, NIBP fell to 78/40 mmHg for which intravenous (IV) mephentermine 3 mg was administered and the rate of administration of IV fluids was increased. After a minute, NIBP increased to 106/56 mmHg. After 5 minutes of administration of the SAB, the patient complained of breathlessness, though the SpO_2 was 98%, for which 100% oxygen was administered with a closed breathing circuit. It was observed that the patient was generating 40–60 ml of tidal volume (VT) and minute ventilation (MV) of 1.4 l/min^{-1} with respiratory rate (RR) of 35 min^{-1}. There was almost no visible chest excursion, and she was using her accessory muscles of respiration. High spinal anaesthesia was suspected. A sensory level of blockade up to T1 was noted at that time along with good hand grip strength. Her vitals were: HR 106 min^{-1}, NIBP 170/102 mmHg, RR 38 min^{-1}, SpO_2 98% with fraction of inspired oxygen concentration (FiO_2) of 1.0 and end-tidal carbon dioxide ($EtCO_2$) of 6–8 mmHg. Airway equipment and drugs were kept ready for intubation in case the need arose. However, assuming that diaphragmatic excursion may improve after delivery of the baby, intubation was not attempted immediately. Difficulty in breathing lasted for 3 minutes and by that time the baby was delivered. The patient started generating a VT of 160–200 ml which improved MV to 5.4 l/min^{-1} with a RR of 30 min^{-1}. The $EtCO_2$ increased to 24–30 mmHg. Over the next 5 minutes, her VT improved to 400 ml and RR fell to 20 min^{-1}. Here vitals were: HR 90 min^{-1}, NIBP 130/72 mmHg and SpO_2 99% with FiO_2 of 0.4. The higher HR and BP despite the high level of spinal anaesthesia in this patient were probably due to hypercarbia leading to central sympathetic stimulation.

Hypertension following SAB has been described in literature.[1,2] However, no cases of hypertension following high spinal anaesthesia have been reported so far. In our case, the other possible causes of hypertension such as distended urinary bladder, patient anxiety, inappropriate sized non-invasive blood pressure cuff and pre-existing hypertensive disorders were ruled out. The patient had a sensory level of T1 with absent chest excursion suggesting paralysis of the intercostal muscles, with intact accessory muscles of respiration. The resultant hypoventilation may have resulted in carbon dioxide

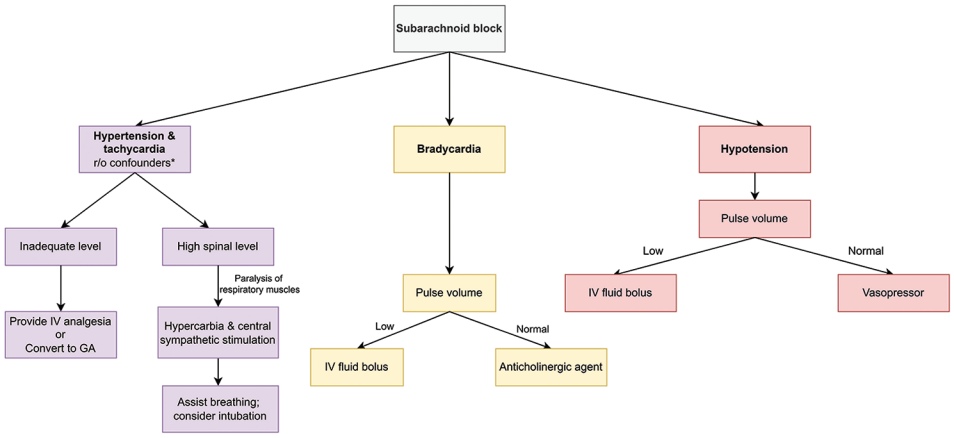

Figure 1: Management of various haemodynamic perturbations during spinal anaesthesia. *Rule out confounders: distended urinary bladder, patient anxiety, inappropriate sized non-invasive blood pressure cuff, pre-existing hypertensive disorders

retention leading to hypertension and tachycardia from central sympathetic stimulation.[3] Following delivery of the baby, improved diaphragmatic excursion resulted in an increase in her minute ventilation, which aided in carbon dioxide washout. A guide to managing the various haemodynamic perturbations in spinal anaesthesia is provided in Figure 1.

The external intercostal muscles of the upper rib cage are inspiratory in function and are innervated by T1–T6 intercostal nerves. During pregnancy, the relative contribution by the diaphragm and the inspiratory intercostal muscles remains unchanged.[4] The accessory muscles of inspiration such as the sternocleidomastoid and scalene are innervated from cervical segments, and hence were preserved in our patient. Diaphragm, which is the principal inspiratory muscle contributing ~65% to the vital capacity, is innervated by C3–C5 cervical roots and was also preserved.[5] However, the cephalad displacement of the diaphragm due to the enlarged uterus and the supine position for surgery resulted in reduced excursion of the diaphragm in the patient. The minute ventilation significantly improved after delivery resulting in gradual correction of the hypertension and tachycardia.

Thus, although hypotension and bradycardia are commonly observed after high spinal anaesthesia, hypoventilation secondary to respiratory muscle paralysis may present as hypertension and tachycardia and this needs to be kept in mind.

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Conflicts of interest
There are no conflicts of interest.

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