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ALTERED THERMOREGULATORY RESPONSES FOLLOWING SPINAL MORPHINE FOR CAESAREAN DELIVERY: A CASE REPORT

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Abstract

Objective: Spinal anaesthesia interferes with physiological thermoregulatory responses, potentially leading to perioperative hypothermia. Spinal morphine can further compound this by a paradoxical clinical presentation leading to poor patient outcome. Case Report: Following an uneventful caesarean delivery (CD) under spinal anaesthesia with intrathecal morphine for post-operative analgesia, a parturient presented in the recovery room with increasing somnolence, excessive sweating and a sensation of feeling hot. She was haemodynamically stable, but her temperature was 34.5°C. Active warming measures were implemented, and normothermia was achieved in 3 hours. Conclusion: Spinal morphine can alter the clinical presentation of hypothermia by manifesting as excessive sweating and subjective sensation of warmth. Teams involved in the perioperative care of parturients should be aware of (a) the possibility of spinal anaesthesia causing perioperative hypothermia, (b) intrathecal morphine masking the clinical presentation of hypothermia and (c) the importance of monitoring temperature of patients who have received spinal anaesthesia with added morphine.

Keywords

caesarean delivery • spinal morphine • altered thermoregulation • hypothermia

Introduction

Spinal anaesthesia for caesarean delivery (CD) is performed routinely and safely worldwide. Intrathecal morphine for post-operative pain management provides optimal and prolonged analgesia [1]. We report a case of profound post-operative hypothermia (34.5°C) presented in the recovery room as somnolence and paradoxical diaphoresis following elective CD under spinal anaesthesia with intrathecal morphine. Altered mental status following the CD could be attributed to several life-threatening causes. It is critical for patient safety and optimal outcomes that all healthcare teams involved in the perioperative care of the parturient should be aware of altered thermoregulatory responses following spinal morphine.

A 30-year-old, 129.3-kg gravida 2, para 1 at 39 weeks of gestation presented for repeat elective CD. She had no other significant past medical history or allergies; her current pregnancy and previous CD were uncomplicated. Initial vital signs were within normal range with a temperature of 36.7°C measured by sublingual thermometer (Welsh-Allyn SureTemp Plus 690, Skaneateles Falls, NY).

In the operating room (ambient room temperature: 22.2–23.3°C), following a co-load of 750cc of lactated Ringer at room temperature, spinal anaesthesia was performed in the sitting position with a 25G Whitacre needle at the L3–L4 level with 12 mg of 0.75% hyperbaric bupivacaine, 15 µg of fentanyl and 100 µg of preservative-free morphine for post-operative pain control. Monitoring included ECG, pulse oximetry and non-invasive blood pressure. Warmed blankets were wrapped around arms and upper chest to patient comfort. As per institutional protocol, the mean blood pressure was maintained within 20% of baseline with phenylephrine infusion and appropriate antibiotics were administered.

Following the establishment of bilateral T4 sensory anaesthesia, surgery proceeded uneventfully. Gravimetric assessment of blood loss was 628 mL and total intraoperative intravenous fluid was 2.5 L of lactated Ringer's solution administered at room temperature. Total operating room time was 120 min. The patient was transferred to the recovery room with normal vital signs, mentation, no pain or nausea and a sublingual temperature of 36.5°C. She was maintained on an infusion of oxytocin at a dose of 6 U/h and intravenous crystalloids at a dose of 125 mL/h.

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After approximately 90 minutes in the recovery room, the obstetricians were alerted by nursing that the patient was extremely diaphoretic, lethargic and complained of 'feeling hot'. Blankets were removed; cool compresses were applied to the forehead, and extra fans were used for patient comfort. Bleeding was minimal, blood glucose was normal and no additional narcotics or sedating medications had been administered. Vital signs were measured: blood pressure was 107/57 mm Hg, heart rate was 61 bpm and oxygen saturation was 96% on 2-L oxygen via nasal cannula, and the patient had no complaints aside from feeling warm.

The patient became increasingly somnolent with time in the recovery room. The anaesthesia team was contacted to rule out an anaesthesia-related aetiology for the patient's clinical presentation. After the assessment by anaesthesia team, the temperature was measured and was 34.5°C, confirmed both rectally and sublingually. Forced air warmers (Bair-Hugger) and warmed lactated Ringer's solution were initiated along with maximum warming of patient's room and use of warm blankets. A Foley catheter with temperature sensor (Level 1®, Smiths Medical ASD, Inc., St. Paul, MN) was inserted and temperature was checked every hour until the achievement of normothermia to 36.0°C. The temperature steadily increased to 35.5°C after 1 h, 35.9°C after 2 h and 36.2°C after 3 h from the initiation of warming measures. The patient became less lethargic and her diaphoresis resolved approximately 90 min after the efforts to rewarm the patient were started. The patient was transferred to floor level care with no further complications and discharged home on post-partum day 3.

Discussion

The incidence of inadvertent mild perioperative hypothermia (<36°C) in women receiving spinal anaesthesia for CD has been reported to be 32% or higher in various studies [2, 3]. We report a case of significant post-operative hypothermia (<34.5°C) in a parturient after spinal anaesthesia; the degree of hypothermia being amplified by altered thermoregulatory responses caused by spinal morphine [4]. It is critical that all healthcare providers involved with the immediate management of patients after CD be aware of this clinical response and presentation in order to initiate appropriate patient care.

Body temperature regulation is a sophisticated process with tight control to within few tenths of a degree. The spinal cord, brain and hypothalamus all serve to integrate signals from peripheral temperature receptors resulting in autonomic output, which induces pre-capillary vasodilation, sweating at higher temperatures and arteriovenous shunt vasoconstriction and shivering at lower temperatures [5].

Majority of central thermoregulation occurs within the hypothalamus and can be significantly impaired after neuraxial (spinal/epidural) anaesthesia via the following mechanisms [5]. First, the major initial contributor to heat loss is redistribution of heat from core to periphery because of vasodilation from the sympathectomy of the spinal block [5]. The short surgical duration of CD would favour redistribution as a significant mechanism for hypothermia. Second, the block also serves to impair physiological defences against heat loss by greatly reducing vasoconstriction and shivering [5]. Third, the spinal anaesthesia also lowers the shivering threshold, thus amplifying the inter-threshold range (temperature range between sweating and shivering thresholds) [5]. Lastly, hypothermia with neuraxial blockade does not cause the conscious feeling of cold discomfort as would be expected, possibly due to the interpretation of lack of tonic cold signals from the legs as relative warmth by the central controller [5].

In our patient, the onset of lethargy, excessive diaphoresis and complaints of feeling warm began 90 min after the arrival to recovery and approximately 3 h after the placement of spinal anaesthesia with morphine. In this crucial post-operative window, a differential diagnosis of lethargy and diaphoresis from an obstetric standpoint might include post-partum haemorrhage, infection, hypoglycaemia or an endocrine-related emergency, whereas the paradoxical symptoms of diaphoresis and hot sensation might guide a clinician away from a diagnosis of hypothermia.

The delay in symptomology in our case was likely due to the slow cephalad spread of intrathecal morphine because of its hydrophilic properties [4]. There have been reports of diaphoresis, cutaneous vasodilation and hypothermia after intrathecal morphine injection. Although the exact mechanism is unknown, one theory is that morphine binds to opioid receptors in the hypothalamus, triggering a decrease in the body's temperature 'set point', thus tricking the body to interpret a normal temperature as fever and consequently causing sweating, vasodilation and evaporative cooling [4]. It is likely that hypothermia in our patient was caused by expected thermoregulatory changes following a spinal anaesthetic, which was further exacerbated by intrathecal morphine's altered thermoregulatory responses to hypothermia. Evaporative cooling from sweating, compounded by cold compresses, decreasing ambient room temperature and adding cool airflow by electric fans likely added to the total heat loss in our patient.

Lastly, our patient had the sensation of 'feeling hot' in the setting of hypothermia and residual spinal blockade, which was another distracting symptom for the diagnosis of hypothermia. Although explanation of this phenomenon is mostly unclear, it has been theorised that a lack of tonic cold signals from the blocked lower extremities causes the central controller to process relative warmth [5].

Prevention strategies for perioperative hypothermia focuses around achieving normothermia with active warming measures such as forced air warming, radiant heaters and warmed fluid administration [5]. Pre-warming and intraoperative forced air warming during CD has demonstrated conflicting results to prevent intraoperative hypothermia [6, 7, 8]. Passive warming with blankets and increasing ambient room temperature should only be used for patient comfort and not as treatment [9]. Another strategy to decrease the intraoperative redistribution hypothermia is the administration of phenylephrine. The thermoprotection from redistribution hypothermia is due to vasoconstriction of the pre-capillary vasculature, mediated by α_1 receptor activation [10]. The pharmacological treatment of opioid-induced altered thermoregulatory responses has largely focused on the improvement of patient comfort. One case report successfully used naloxone, a μ-receptor antagonist, for reversing symptoms of nausea, vomiting, pruritus and sedation whilst decreasing diaphoresis [4]. Atropine, an anti-cholinergic, has been shown to reverse opioid-induced sweating because it is an acetylcholine-mediated process [11]. Lorazepam has shown success in reversing the intrathecal morphine-induced hypothermia when active warming measures have proven unsuccessful after a CD [12].

Continuous monitoring of core temperature (nasopharyngeal and oesophageal) during CD under spinal anaesthesia is not practical as patients are awake. Bladder temperature will reflect inaccurate temperatures because of proximity to surgical site. The most suitable estimate of core temperature in this setting is a sublingual or aural canal measurement because it is non-invasive; however, controversy exists regarding accuracy [5,13].

The detrimental consequences of perioperative hypothermia include coagulopathy, increased blood loss, higher surgical site infection rates, cardiac rhythm disturbances, myocardial ischaemia, prolonged hospital duration, increased costs, prolonged post-anaesthesia recovery, shivering and patient discomfort [5,13,14]. In the obstetric setting, neonatal outcomes can be impacted by maternal hypothermia. For the neonate, respiratory distress syndrome, hypoglycaemia, lower APGAR scores reflecting neonatal distress and umbilical pH changes have been reported because maternal hypothermia could result in neonatal hypothermia [13,14]. Skin-to-skin bonding and feeding may be interrupted by maternal discomfort and rewarming measures because hypothermia is often detected and treated post-operatively [13]. Maintenance of normothermia before, during and after CD has significant downstream effects, and further studies are needed to assess how best to use active warming techniques for the prevention of hypothermia that continues well into the post-operative recovery period.

Conclusions

Our case report highlights an overlooked and major issue of inadvertent perioperative hypothermia that commonly affects parturients undergoing CD with spinal anaesthesia. The concomitant administration of spinal morphine can further alter the normal thermoregulatory responses and paradoxically present as sweating and sensation of feeling hot in the face of on-going hypothermia. This can lead to incorrect diagnosis and management with detrimental outcome. Although the best prevention strategy is unknown at this time, awareness, prompt diagnosis and treatment are invaluable. It is important for the perioperative team to be aware of the mechanism, surprisingly high incidence, diagnosis and treatment of perioperative hypothermia in patients receiving spinal anaesthesia especially with added intrathecal morphine for pain management as its variable presentation; symptom and treatment duration can far exceed the length of the spinal blockade. By raising awareness of this issue within the obstetric community, we hope to reduce the maternal morbidity, increase satisfaction, reduce costs and promote safety.

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