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Research Article

Effect of ketamine on intraoperative nausea and vomiting during elective caesarean section under spinal anaesthesia: A placebo-controlled prospective randomized double blinded study

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KEYWORDS

Caesarean section;
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Abstract *Purpose:* The well documented maternal and fetal safety following spinal anaesthesia in caesarean section (CS) makes it the preferred anaesthetic technique. Intraoperative nausea and vomiting in parturients subjected to CS under spinal anaesthesia is a major drawback of the technique. Post-spinal hypotension, the sympathetic blockade and associated relative vagal hyperactivity in addition to intraoperative visceral pain are the most important underlying factors behind the high rate of IONV during spinal anaesthesia. Ketamine has a unique sympathomimetic and vagolytic criteria that may help in reducing the incidence of IONV secondary to spinal-induced hypotension. This study was an attempt to evaluate the effect of ketamine on the IONV in parturients subjected to elective CS under spinal anaesthesia.

Patients and methods: Two hundred twenty-nine patients were randomly allocated into two equal groups: the ketamine group; in which 0.5 mg/kg was infused intravenously in 20 min and the placebo group; in which normal saline was infused. The two groups were given subarachnoid block with local anaesthetic hyperbaric 0.5% bupivacaine and intrathecal fentanyl.

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Results: IV infusion of ketamine was associated with significant reduction in the incidence of intraoperative nausea and hypotensive episodes.
Conclusion: This study demonstrated a beneficial effect of IV infusion of ketamine on IONV in parturients subjected to elective CS under spinal anaesthesia.

1. Introduction

Intraoperative nausea and vomiting (IONV) during caesarean section (CS) under spinal anaesthesia is a common complication with an incidence ranging from 40% to 60% [1–4]. Many factors may contribute to this high rate of IONV during CS; sympathetic block and the resultant hypotension secondary to spinal anaesthesia, visceral pain and vagal stimulation during CS are probably the most important factors [5]. Ketamine has unique central sympatho-mimetic, vagolytic and analgesic properties [6]. These properties of ketamine are assumed to reduce the incidence of spinal induced hypotension. In this study, we hypothesized that an IV infusion of ketamine would lower the incidence of intraoperative nausea and vomiting secondary to spinal-induced hypotension. This placebo-controlled prospective randomized double-blinded study was designed to evaluate the impact of ketamine on prevention of intra-operative nausea and vomiting as a primary outcome in parturients subjected to elective CS under spinal anaesthesia. Maternal and fetal side effects were considered as a secondary outcome.

2. Patients and methods

After approval by an ethical committee at Mansoura University Hospitals and obtaining a written informed consent from eligible parturients scheduled for elective caesarean section under spinal anaesthesia, ASA1 and 2 parturients having singleton pregnancy were included. Exclusion criteria included history of motion sickness, post-operative nausea and vomiting, gastrointestinal disease, allergy to (bupivacaine, fentanyl or metoclopropamide), pregnancy induced hypertension, history of non-gestational diabetes and history of smoking. Obese patients (body weight >90 kg), epileptic patients, patients given antiemetics or corticosteroids within 24 h before CS, patients who was heavy sedated (with Ramsy sedation score more than 3), and patients having any absolute contraindications to spinal anaesthesia were excluded. Included patients were randomly (using computer generated randomization table) allocated into two equal groups: the ketamine group ($n = 110$) and the control group ($n = 110$).

In the ketamine group, ketamine was diluted to a total volume of 20 ml via normal saline. Using an infusion pump (Graseby 3100-waltford, Herts-UK), ketamine was given at a dose of 0.5 mg kg^{-1} over 20 min started while patient back was being cleaned and scrubbed for spinal anaesthesia before intrathecal injection. In the control group, a similar volume of normal saline was given using the same technique used in the ketamine group. In order to achieve blindness of the study, one researcher was involved only in drug preparation according to patient's randomization and group assignment. The other researcher (anaesthesia provider) gave spinal block, managed the patient according to the study protocol and recorded the intraoperative data.

Patients started NPO midnight of the surgery, Ringer lactate infusion in a rate of 1.5 ml/kg^{-1} h^{-1} commenced at 6 am on the day of surgery through an 18-G peripheral IV cannula, a 20 ml of 0.3 M sodium citrate given orally 15 min before shifting the patient to the main operating theatre.

In the waiting area of the operating room, patients were monitored with ECG, automated noninvasive arterial blood pressure and pulse oximetry. An average of two readings of the mean arterial blood pressure (MABP) and heart rate (HR) were taken 5 min apart and considered the basal readings. Preloading with 10 ml Kg^{-1} of hydoxyethyl starch solution (6% HES 200/0.5 Braun Melsungen AG-Germany) was completed within 15–20 min, after which patient was shifted to the operating room (OR) where the standard monitoring of ECG, HR, BP and SpO2 was extended.

In the OR, we performed spinal anaesthesia in the sitting position under complete aseptic technique and skin infiltration with 0.5 ml of 1% lidocaine at site (L3-4 or L4-5) of spinal needle insertion. We used a spinal needle 25-G pencil point type, once free flow of clear CSF was obtained a 15 μg fentanyl loaded in 2 ml syringe was injected intrathecally followed by a hyperbaric bupivacaine 0.5%. The dose of bupivacaine was adjusted according to parturients height, 2 ml (10 mg) was given to those having a height <155 cm and 2.2 ml (12 mg) was given if the height was $\geqslant 155$ cm. After intrathecal injection, patients were immediately returned supine with left uterine displacement and supplemented with oxygen 4 L min^{-1} via clear facemask. Dermatomal level of sensory block was assessed by pinprick method; T5 was the minimum acceptable level before surgical incision. Both groups were given the same anaesthetic management by well-trained anaesthetists having an experience more than 10 years in the anaesthesia practice.

Mean arterial blood pressure (MABP) was measured every 1 min for the first 10 min then every 2 min until the end of ketamine or placebo infusion. Intraoperative hypotension-defined by MABP less than 20% of the basal reading-was managed by increasing the infusion rate of Ringer lactate solution concomitant with administration increments of 3 mg ephedrine hydrochloride. Arterial blood pressure was measured 1 min after ephedrine injection and if hypotension persists another ephedrine bolus was given, atropine sulphate (0.5 mg) was given when hypotension was associated with bradycardia (HR <50 beats min^{-1}). At the end of CS the incidence of hypotension (percentage per each group), hypotensive episodes (number of hypotensive episodes per each group) and increments of ephedrine hydrochloride in mg. were recorded.

Ramsay Sedation Scale (RSS; 1 = anxious and agitated, 2 = co-operative and tranquil, 3 = drowsy but responsive to command, 4 = asleep but responsive to glabellar tap, 5 = asleep with a sluggish response to tactile stimulation, 6 = asleep and no response). It was used to measure sedation level at 5, 10, 15, 20, 25, and 30 min after surgical incision, patients having RSS 4 or more were rejected.