Administration of Metoclopramide for Prevention of Nausea and Vomiting during Epidural Anesthesia for Elective Cesarean Section

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Nausea and vomiting during regional anesthesia for cesarean section are distressing to the patient, and may interfere with performance of surgery. Santos and Datta1 reported that 2.5 mg of droperidol reduced the incidence of intraoperative nausea in patients undergoing cesarean section with spinal anesthesia. However, droperidol may result in prolonged, unpleasant sedation.2–4 Further, Cohen and associates5 reported the occurrence of early respiratory depression in a patient who had received both 1.25 mg of intravenous droperidol and 1.25 mg of epidural hydromorphone during cesarean section. They concluded that the droperidol may have been "partially or completely responsible for the respiratory depression."5

In contrast, metoclopramide has little or no sedative effect.4,6–9 The antiemetic property of metoclopramide probably results from antagonism of dopamine receptors in the chemoreceptor trigger zone. Further, metoclopramide increases the resting tone of the lower esophageal sphincter, increases the amplitude of gastric contractions, relaxes the pyloric sphincter and duodenal bulb during gastric contractions, and increases peristalsis of the proximal small bowel. This results in accelerated gastric emptying and shortened transit time through the small bowel.10,11 The purpose of the present study was to evaluate the efficacy of giving metoclopramide for prevention of nausea and vomiting during epidural anesthesia for elective cesarean section.

METHODS

The protocol was approved by our review board for research involving human subjects. Each patient gave written informed consent. The study included ASA Class I or II patients receiving lumbar epidural anesthesia for elective cesarean section. A patient was excluded if she had experienced nausea or vomiting during the 24 h before induction of anesthesia, or if she required adjunctive surgery other than tubal ligation (e.g., open renal biopsy, retroperitoneal exploration).

Preeanesthetic medication was limited to 30 ml of orally administered 0.3 M sodium citrate. Each patient received: 1) 20 ml/kg of lactated Ringer’s solution before induction of anesthesia; 2) 18–25 ml of epidural lidocaine (2% solution, with 1:200,000 epinephrine) to achieve a bilateral cephalad dermatomal level of at least T-4; 3) oxygen via a face mask at a flow rate of 5 l/min; and 4) iv ephedrine as soon as any decrease in arterial blood pressure was detected.12 Aortocaval compression was avoided by placing a single folded blanket beneath the right buttock, and then tilting the table to the left. Arterial blood pressure was measured by an automated blood pressure monitor every minute from induction of anesthesia until 5 min after delivery; subsequently, arterial blood pressure was measured every 2.5 min until the patient was transferred to the recovery room. Hypotension was defined as a decrease in systolic arterial blood pressure of ≥20%, or a systolic blood pressure of <100 mmHg.

Immediately after the umbilical cord was clamped, each patient received fentanyl 50 μg and the study solution iv over 30–60 s. The study solution was freshly prepared by the pharmacist according to a table of random numbers and was administered in a double blind manner. The patient, anesthesiologist, obstetrician, and nursing staff were unaware of the identity of the study solution. Women in one group received metoclopramide, 0.15 mg/kg, diluted in saline to a total volume of 5 ml; women in the other group received 5 ml of saline.

The anesthesiologist recorded whether the uterus was exteriorized during closure of the uterine incision, and administered additional fentanyl as needed for surgical discomfort. Epidural morphine, 4.0–5.0 mg, or epidural hydromorphone, 1.0–1.2 mg, was administered during closure of the rectus fascia.

Each patient was observed by the anesthesiologist for the intraoperative occurrence of nausea and vomiting. If a patient experienced nausea and vomiting, the anesthesiologist: 1) administered crystalloid and/or ephedrine iv

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as indicated; 2) requested that the surgeon decrease traction on the uterus and/or peritoneum; 3) made certain that there remained a bilateral cephalad dermatomal level of at least T-4; and 4) reassured the patient.

Immediately after transfer to the recovery room, each patient was asked to indicate, on unmarked 100 mm visual analogue scales: 1) her intraoperative, post-delivery nausea score (0 = no nausea, 100 = worst possible nausea); 2) her intraoperative, post-delivery anxiety score (0 = no anxiety, 100 = worst possible anxiety); and 3) her present sedation score (0 = wide awake, 100 = almost asleep). Subsequently, each patient remained in the recovery room for 4 h and was observed by the nursing staff for the postoperative occurrence of nausea and vomiting. The nursing staff monitored the respiratory rate of each patient who had received epidural morphine or epidural hydromorphone (every 15 min for 1 h, every 30 min for 15 h, and then every hour for 8 h).

TABLE 2. Operative Management

<table>
<thead>
<tr>
<th></th>
<th>Metoclopramide (N = 34)</th>
<th>Saline (N = 35)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Operative time (min)*</td>
<td>57.2 ± 15.9</td>
<td>58.8 ± 17.8</td>
<td>NS</td>
</tr>
<tr>
<td>Total dosage of fentanyl</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50 μg only</td>
<td>26 (76%)</td>
<td>29 (88%)</td>
<td>NS</td>
</tr>
<tr>
<td>51–100 μg</td>
<td>6 (18%)</td>
<td>3 (9%)</td>
<td></td>
</tr>
<tr>
<td>101–150 μg</td>
<td>2 (6%)</td>
<td>1 (3%)</td>
<td></td>
</tr>
<tr>
<td>Uterus exteriorized</td>
<td>32 (94%)</td>
<td>26 (79%)</td>
<td>NS</td>
</tr>
<tr>
<td>If uterus exteriorized, time of exteriorization (min)*</td>
<td>21.2 ± 9.0</td>
<td>24.9 ± 11.0</td>
<td>NS</td>
</tr>
<tr>
<td>Tubal ligation performed</td>
<td>20 (59%)</td>
<td>18 (55%)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypotension postdelivery</td>
<td>10 (29%)</td>
<td>6 (18%)</td>
<td>NS</td>
</tr>
<tr>
<td>Postoperative analgesia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epidural morphine</td>
<td>24 (71%)</td>
<td>27 (82%)</td>
<td>NS</td>
</tr>
<tr>
<td>Epidural hydromorphone</td>
<td>7 (21%)</td>
<td>5 (15%)</td>
<td></td>
</tr>
<tr>
<td>Intramuscular morphine</td>
<td>3 (9%)</td>
<td>1 (3%)</td>
<td></td>
</tr>
</tbody>
</table>

* Mean ± SD. NS = not significant.

Statistical analysis was by Student’s t test, Wilcoxon test, Chi square, and Fisher exact test, as indicated. P < .05 was considered significant.

RESULTS

There were 34 women in the metoclopramide group and 33 women in the saline group. The two groups were similar with regard to maternal characteristics (table 1) and operative management (table 2). Six women in the metoclopramide group, and seven women in the saline group, complained of nausea after induction of anesthesia, but before delivery. No woman in either group vomited before delivery.

Four (12%) women in the metoclopramide group, versus 12 (36%) women in the saline group, spontaneously complained of intraoperative, post-delivery nausea (P < .05) (fig. 1). No woman in the metoclopramide group, versus five (15%) women in the saline group, experienced intraoperative, post-delivery vomiting (P < .05).

Women in the metoclopramide group had a mean (±SD) nausea score which was significantly less than the mean nausea score in the saline group (9.1 ± 17.6 versus 18.1 ± 24.1; P < .01). However, there was no significant difference between groups in mean anxiety scores (28.2 ± 28.0 versus 24.2 ± 25.7) or mean sedation scores (47.7 ± 29.3 versus 46.8 ± 28.7). No woman in either group had a respiratory rate of 10 or less during the first 24 postoperative hours.

During the first 4 postoperative hours, five (15%) women in the metoclopramide group, versus 12 (36%) women in the saline group, spontaneously complained of nausea (P < .05). Four (12%) women in the metoclopramide group, versus 12 (36%) women in the saline group, experienced postoperative vomiting (P < .05). The postoperative antiemetic efficacy of metoclopramide was most apparent in the period immediately after surgery. During the first 30 min after surgery, no woman in the metoclopramide group, versus six (18%) women in the saline group, spontaneously complained of nausea (P < .05). Similarly, no woman in the metoclopramide group, versus five (15%) women in the saline group, vomited (P < .05).
DISCUSSION

There are two similarities between the methodology of our study and the study of Santos and Datta. First, there were attempts to limit or control the occurrence of factors that may predispose to intraoperative nausea and vomiting. All patients had a sensory level of at least T-4 and received supplemental oxygen via a face mask. Intravenous crystalloid and ephedrine were administered liberally to prevent and/or treat hypotension. Second, all patients received an iv narcotic at the time that the study solution was administered. Patients in the earlier study received 1 mg of butorphanol; patients in our study received 50 μg of fentanyl iv.

We may be criticized for administering fentanyl to each patient. Fentanyl was administered concomitantly with the study solution because: 1) the majority of our patients undergoing cesarean section with epidural anesthesia requested and/or require supplemental analgesia during closure of the uterine incision and peritoneal cavity; and 2) any transient sedation that might have occurred after administration of metoclopramide alone would have negated the double blind study design. Transient drowsiness or restlessness have been reported in 0–20% of patients who have received 10–20 mg of metoclopramide. We did not attempt to evaluate any potential difference between groups in intraoperative sedation, and we acknowledge that our assessment of postoperative sedation was limited to one assessment immediately after transfer of each patient to the recovery room. However, there was no apparent increase in sedation immediately after surgery in the group of women who had received metoclopramide. In contrast, we and others have observed prolonged, unpleasant sedation after administration of droperidol. Although extrapyramidal reactions have been reported with administration of larger dosages of metoclopramide, we observed no side effects after administering 0.15 mg/kg of metoclopramide in the present study.

Spelina et al. reported no difference in intraoperative nausea and vomiting among patients who had received either 10 mg of metoclopramide or placebo before induction of spinal anesthesia for orthopedic surgery. However, Spelina et al. noted that the highest incidence of nausea and vomiting occurred 70–100 min after induction of spinal anesthesia. Palazzo and Strunin suggested that "metoclopramide should only be considered for immediate prophylaxis" of nausea and emesis. The difference in results between the study of Spelina et al. and our study may be explained, in part, by the consistently brief duration of surgery in the present study (table 2).

During the first 4 postoperative hours, we observed a lower incidence of nausea and vomiting in the group of women who had received metoclopramide. Previous studies of the efficacy of metoclopramide in reducing the incidence of postoperative nausea and vomiting have yielded conflicting results. The usefulness of metoclopramide in prophylaxis of postoperative nausea and vomiting may be limited by its relatively brief duration of action. In our study, the postoperative antiemetic efficacy of metoclopramide was most apparent during the 30-min period immediately after surgery. Other investigators have observed that the incidence of postoperative nausea and vomiting is reduced most consistently when metoclopramide is given at the end of surgery. We concluded that intravenous administration of metoclopramide, 0.15 mg/kg, reduced the incidence of intraoperative nausea and vomiting during epidural anesthesia for elective cesarean section. Further, there was no apparent increase in sedation immediately after surgery in the group of patients who had received metoclopramide.

REFERENCES


Unusual Presentation and Novel Solution for Hemodynamic Compromise during Thoracic Surgery

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Cardiovascular collapse during or following major thoracic surgery requires prompt diagnosis and immediate treatment. On occasion, the "classic" or expected presentation of a life-threatening complication does not occur, and such a situation tests the clinician's diagnostic and problem solving abilities. We describe such a case.

CASE REPORT

A 30-yr-old female with the diagnosis of diffuse large cell lymphoma presented for thoracic surgery. Over a 3-yr period, the patient had received multiple standard and salvage radiation and chemotherapy treatments for lymphomatous involvement of the left lung and left chest wall. Multiple recurrences of tumor in the left anterior chest wall led to the development of a large chest wall ulcer. The left lung exhibited upper lobe consolidation and the presence of a mass. The possibility of sepsis or hemorrhage secondary to the necrotic lesion necessitated excision of a large area of the left chest wall. An exploration of the left thorax and the mediastinum was planned to determine the possibility of resection of the lung mass. A combined thoracic surgery and plastic surgery procedure was planned.

Anesthesia was induced with the iv administration of thiopental 4 mg/kg and pancuronium 0.08 mg/kg, and inhalation of 1.5% isoflurane. The trachea was intubated with a double lumen endotracheal tube. Anesthesia was maintained with 50% N₂O, 50% O₂, and 1.5% isoflurane. Monitoring included a CVP catheter (Hickman Catheter) in the right subclavian vein, right radial arterial cannula, EKG, foley catheter, temperature probe, esophageal stethoscope, O₂ analyzer in the inspiratory limb, and end tidal CO₂ analyzer.

A left thoracotomy was then performed in the right lateral decubitus position. Single lung ventilation and anesthesia were provided with 1.5–2% isoflurane and 100% O₂ while the left thoracic cavity was explored. A resection was performed removing the entire left lung. A segment of pericardium (an intrapericardial approach for pneumonectomy facilitated access to major vessels and enabled a wider dissection), and a large section of the left anterior chest wall (including ribs 2–5), including a part of the left sternum, were resected. The pericardium was closed by approximation of the free edges. A latissimus dorsi muscle flap was used to close the chest wall defect. The patient was then placed in the supine position, and a rectus muscle and skin flap (below the umbilicus) was tunneled cephalad and positioned to reinforce the latissimus flap. Hemodynamic variables had remained stable throughout the surgical procedure (approximately 8 h) with arterial blood pressures ranging from 90/60–120/80 mm Hg, CVP readings from 12–15 cm H₂O, and heart rates from 70–90 bpm. Arterial blood gases with continued one-lung ventilation were excellent (PaO₂ 200–300 mm Hg, PaCO₂ 36–42 mm Hg, pH 7.36–7.42). Blood loss was estimated to be approximately 1000 cc with iv replacement including 2 units of packed red blood cells, 50 cc of 25% albumin, and

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