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Intraoperative Intravenous Lidocaine Prevention of Vomiting in Adnexial Mass Operations

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Abstract

Objective: Postoperative Vomiting (POV) is a common complication in intra-abdominal operations. The use of intravenous lidocaine infusion in adult patients who underwent abdominal surgery may prevent POV. We aimed to evaluate the anti-emetic effect of intravenous lidocaine infusion used as an adjuvant to general anesthesia in intra-abdominal operations.

Patients: ASA I-III adult women aged 30 to 70 years scheduled for elective adnexal mass operations were selected.

Intervention: We have standardized the induction and maintenance of anesthesia in our oncological surgery rooms. Patients were randomly administered lidocaine (1.5 $mg.kg^{-1}$ intravenous (i.v) lidocaine followed by 2 $mg.kg^{-1}.h$) or only 0.9% saline (same proportion and volume) for 5 minutes. Infusions were continued until the end of the surgery.

Results: 200 women with adnexal mass were operated. In the lidocaine group, 60 (60%) of the 100 patients had POV and 80 (80%) of the 100 patients had POV in the Saline group. The probability of having POV was 20% less than patients receiving lidocaine in the Saline group. The mean lidocaine plasma concentration was 4.1 $\mu g.ml^{-1}$ (range: 0.87 to 4.88).

Conclusion: The use of intravenous lidocaine infusion as an adjunct to general anesthesia reduced POVN in oncology patients.

Keywords: General anesthesia; Vomiting; Nausea; Lidocaine

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Introduction

Postoperative Vomiting (POV) is a known complication of intraabdominal operations. At least 70% vomiting occurs when anti-emetic prophylaxis is not used. There are many studies examining the use of perioperative intravenous lidocaine infusion to improve postoperative analgesia and improve bowel function improvement [1].

Various pharmacological interventions have been studied to prevent POV. Most existing anti-emetic drugs are costly and don't completely eliminate POV [2]. In addition, side effects such as agitation, extrapyramidal symptoms, bleeding, and cardiac rhythm disturbances have been reported to increase cost [3].

There is some evidence that the use of intravenous lidocaine infusion in patients undergoing abdominal surgery may provide better postoperative pain control with postoperative nausea and vomiting. This effect of lidocaine has been linked to increased bowel motility and/or reduced postoperative pain and reduced opioid use [4]. Other studies suggest that this decrease in POV is not associated with the opioid protective effect of lidocaine [5]. There is little evidence for the use of lidocaine infusion. Our hypothesis is that lidocaine infusion may prevent POV in women undergoing general anesthesia for adnexal mass.

Methods

In 2016-2018 Zekai Tahir Burak Training and Research Hospital Oncology Clinic, the patients who were taken to the operation from the adnexal mass with the approval of EPK (TUYEK) were scanned from archive and data processing. In this study,

anesthesia follow-up forms, wake-up observation papers, and anesthesia procedures were scanned.

We excluded patients with a history of mental impairment, obesity, diabetes mellitus, use of any psychoactive and/or anti-emetic medication, preoperative, known congenital heart conduction disorders, gastroesophageal reflux, liver or kidney failure within 24 hours prior to surgery [6-10].

Intravenous lidocaine [receiving 1.5 $mg.kg^{-1}$ bolus lidocaine Intravenously (IV) for 5 minutes followed by lidocaine infusion (2 $mg.kg^{-1}.h^{-1}$) continued until the end of surgery] (Lidocaine Group) or at the same rate there were patients who received 0.9% saline (Saline Group).

The minimum preoperative starvation time was 6 hours and was not used premedication. Pentothal (5 $mg.kg^{-1}$), Fentanyl (2 $\mu g.kg^{-1}$) and Rocuronium (0.6 $mg.kg^{-1}$) were administered intravenously. Meanwhile, some patients were given intravenous lidocaine (bolus dose 1.5 $mg.kg^{-1}$, infusion dose using a concentration 5$mg.ml^{-1}$ and administered lidocaine infusion from 2 mg. $ml^{-1}.h^{-1}$. Endotracheal intubation was performed using the oral intubation tube (No: 7.5 Fr) and anesthetized with 2% sevoflurane (3 L/min) in a mixture of 50% nitrogen oxide+50% oxygen. Fentanyl (1 $\mu g.kg^{-1}$ bolus) was administered to maintain BP (Blood Pressure) and Heart Rate (HR) at 20% of the baseline. All patients were received i.v ringer lactate from 25-30 ml. kg^{-1} throughout the anesthesia [11-14].

An orogastric tube was used to relax the stomach. All patients were left awake in the operating room when they could open their eyes and then transferred to PACU. The extubation time, defined as the time from postoperative to tracheal extubation, was recorded.

After extubation, each vomiting or retching event was documented by one of the investigators, and a rescue antiemetic was made as described previously (intravenous ondansetron 0.15 mg kg^{-1} or intravenous droperidol 0.015 mg kg^{-1}). Continuous cardiac monitoring was provided by telemetry during their stay in PACU.

Watcha scale 9 (1-4 points) was used to evaluate the resulting delirium. The pain was assessed using a visual analog scale (VAS; 0=no pain, 10=worst possible pain) as a postoperative pain scale.

Our primary outcome was defined as the presence of at least one vomiting (excretion of the stomach from the mouth), retching (non-vomiting exertion) or both (POV) within the first 24 hours postoperatively. Secondary outcomes included lidocaine plasma concentrations and postoperative pain.

Statistical Analysis

The comparisons between the groups were made according to Shapiro-Wilk test results. Student's t-test or Wilcoxon rank-sum test were used to compare the groups. X^2 test and Fisher's exact test were used for the inferences. Postoperative pain scores were analyzed using Variance Analysis for repeated measures. Descriptive statistics were mean ± SD, median [interquartile range] or ratio ratio (OR) [95% confidence interval (CI)]. A bilateral p-value less than 0.05 was considered significant. STATA/SE v 13.1 (Stata Corp. LP, College Station, Texas, USA) was used for analysis.

Table 1: Demographic, anesthetic characters and postoperative care data.

Woman	Lidocaine (n:100)	Saline (n:100)	p-value
	100	100	1
Age (years)	52 (30-70)	55 (30-70)	0.86
Weight (kg)	82,1 (50-98)	75,6 (55-90)	0.65
Height (meters)	1.68 (1.55-1.70)	1.66 (1.56-1.69)	0.98
Anesthesia Time (minutes)	85 ± 10	82 ± 12	0.64
Operation Time (min)	72 ± 5	70 ± 6	0.66
Extubation Time (min)	10 ± 5	7.5 ± 4	<0.01
Total Fentanyl Consumption ($\mu g.kg^{-1}$)			
Intraoperative	5 (4.2-6)	5 (4.5-6.2)	0.4
Postoperative (PACU)	0.5 (0-1)	0.7 (0.1.2)	0.25
Observation of delirium	35 (35%)	40 (40%)	0.35
Antiemetic treatment	6 (6%)	10 (10%)	0.45

Table 2: Postoperative pain scale.

Group	0 min	15 min	30 min	45 min	60 min	75 min	120 min	24 hour	p
Lidokain (VAS)	0	0	1	2	2	2	2	2	0.75
Saline (VAS)	0	0	1	2	2	2	3	3	
CI: Confidence Interval; VAS: Visual Analogue Scale									

Result

We recorded the data of 200 women. All of them received the standardized study treatment we were assigned. Patient characteristics were compared between the groups (**Table 1**). Extubations of the lidocaine group took longer [10.3 (4.2) and 7.5 (3.3) minutes, p<0.001] (**Table 1**).

Postoperative pain scores were similar between the groups. Antiemetic treatment intake and nausea and vomiting rates were lower in the lidocaine group (**Table 2**). We found that intravenous lidocaine infusion used as an adjunct to general anesthesia is effective in preventing nausea and vomiting in adnexal mass operations.

Discussion and Conclusion

We aimed to show that lidocaine infusion given as adjuvant medication in women with adnexal mass operated under general anesthesia reduces the risk of POVN (postoperative nausea and/or vomiting) compared to placebo treatment. The underlying mechanism of POVN has not been fully elucidated. The central pattern generator for vomiting is located within the lateral reticular structure of the medulla oblongata. This area receives multiple sensory inputs from the heart, the internal organs of the abdomen, the vestibular system, the posterior area of the brain stem (the chemoreceptor triggering site) and higher brain centers [15]. Neurotransmitter receptors among the signals that cause nausea and/or vomiting are dopaminergic (D2),

histaminergic (H1), cholinergic, serotonergic (5-HT3) [16] and neurokinin NK1 systems [17]. Harmful stimuli can stimulate POVN through different mechanisms such as pain (such as surgery), neurotransmitter release (such as serotonin, dopamine), head position (via vestibular nerve stimulation) and opioid use [18].

Multiple mechanisms of action for local anesthetics have been described. Lidocaine prevents Na^+ ions from flowing through channel pores and provides this by connecting to voltage-gated sodium (Na^+) channels [19]. Muscarinic, nicotinic and dopaminergic receptor blockade, stimulation of gamma-aminobutyrinergic pathways, inhibition of opioid receptors, and anti-inflammatory properties have also been reported [20-22].

There is also evidence that local anesthetics inhibit the release of Substance P [16,21,22], a potent NK1 agonist. Lidocaine can realize its anti-emetic properties through one or more of these mechanisms.

In a recent meta-analysis by Weibel et al. [20], intravenous lidocaine infusion showed lower nausea [45/218 patients in the lidocaine group, 66/222 in the control group, relative risk (RR) 0.82 (95% CI (0.70 to 0.97)], but the risk of vomiting was not different between the groups [RR 0.49 (95% CI, 0.16-1.18)].

Kranke et al. [5], suggested that there were fewer POV attacks among patients receiving lidocaine due to opioid protective effect. There was no difference between the groups in terms of opioid consumption during or after surgery.

The extubation time was longer in the lidocaine group (2.5 minutes), which was statistically significant, and we think that this difference wasn't clinically significant. Furthermore, the potential sedative effect of intravenous lidocaine didn't affect the resulting incidence of delirium.

None of the patients had clinical evidence of local anesthetic systemic toxicity, including arrhythmia. All measured lidocaine plasma levels were in the range of 5-1.25 $\mu g.ml^{-1}$ and below the toxicity threshold, suggesting that this method may be a safe alternative for the prevention of POV. The dosing scheme used in this study is similar to that described previously for other use protocols of lidocaine, either as an anti-arrhythmic drug or as an adjunct to general anesthesia to reduce opioid consumption [5,8]. Low lidocaine levels may also be effective in preventing POV. Further studies should be performed to find a minimum effective concentration to achieve this effect.

Finally, although we found a statistically significant decrease in the incidence of POV in the lidocaine group by 21.3% (ITT analysis=19.6%), this was less than the 30% level we chose to use for our power analysis. However, a 30% decrease was at the calculated 95% CI (ITT upper limit of 95% CI 37.2%).

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