Effect of perioperative infusion of lidocaine vs. dexmedetomidine on reduced consumption of postoperative analgesics after laparoscopic cholecystectomy

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Background: Postoperative pain is the most common complaint of patients following laparoscopic cholecystectomy (LC). Intravenous lidocaine has analgesic, anti-hyperalgesic, and anti-inflammatory effects, and dexmedetomidine has anti-nociceptive and analgesic sparing effects. We evaluated the effects of perioperative intravenous infusion of lidocaine and dexmedetomidine on postoperative pain control and analgesic consumption after LC.

Methods: Eighty-four patients, aged 20–60 years, who were undergoing elective LC were assigned randomly to three groups (n = 28 in each). The patients in group L received an intravenous lidocaine bolus of 1.5 mg/kg and then continuous infusion of 2 mg/kg/hr. The group D received an intravenous dexmedetomidine bolus of 1 µg/kg, followed by continuous infusion of 0.4 µg/kg/hr. The group N received saline as described for group L. Bolus doses were given during the 10 minutes before the induction of anesthesia, followed by continuous infusion until end of the surgery. Visual analogue scale (VAS) score and postoperative analgesics consumption were evaluated during 24 hours after the surgery.

Results: No significant difference was observed in VAS score among the groups during the first 24 hr after LC. The amount of fentanyl consumption in the post-anesthesia care unit was significantly less in groups L and D compared to group N.

Conclusions: Both perioperative intravenous infusion of dexmedetomidine and lidocaine reduced postoperative requirements of fentanyl in the early post-operative period after LC. However, there was no significant difference between dexmedetomidine and lidocaine in the analgesic sparing effect. (Anesth Pain Med 2014; 9: 185-192)

Key Words: Dexmedetomidine, Laparoscopic cholecystectomy, Lidocaine, Postoperative pain.

INTRODUCTION

Postoperative pain of laparoscopic cholecystectomy (LC) is less intense than that of open cholecystectomy, and LC is typically performed as ambulatory surgery, although the approach is not pain-free [1,2]. Especially, some patients complain of severe pain during the first 24 hours after LC. The origin of pain after LC is complicated. Thus, a combination of inflammatory, incisional somatic, and visceral components, multimodal analgesic regimens and various treatments are suggested, which include opioids, non-steroidal anti-inflammatory drugs (NSAIDs), dexamethasone, injection of local anesthetics into the surgical wound, and removal of residual carbon dioxide [3-5].

Intravenous lidocaine has analgesic, anti-hyperalgesic, and anti-inflammatory effects due to sodium channel blockade and inhibition of N-methyl-D-aspartate (NMDA) receptors [6-8]. Intravenous lidocaine provides pain relief after open abdominal surgery and laparoscopic surgery including LC [9-16].

Dexmedetomidine is a specific α2-adrenergic receptor agonist that has anti-nociceptive and sedative properties. Intravenous dexmedetomidine has a role as postoperative analgesia, which leads to the reduced requirement for opioids [17-20]. Also, Park et al. [21] reported that the administration of dexmedetomidine during LC with multimodal analgesics reduced the postoperative pain score and the amount of ketorolac supplied during the first 24 hr postoperatively; the authors opined that dexmedetomidine had benefits on postoperative pain control.

Pain after LC is multifactorial and various managements have been recommended to relieve postoperative pain. Multimodal analgesia is now proposed to prevent and treat posto-
operative pain of LC. In this study, we thought that lidocaine and dexmedetomidine might be helpful with multimodal analgesia to control postoperative pain after LC. Accordingly, the aim of the study is to compare the analgesic efficacy and side effects of perioperative infusion of dexmedetomidine to those of lidocaine; it relieves postoperative pain and reduces requirements for analgesics after LC.

**MATERIALS AND METHODS**

This study was approved by the Institutional Review Board of our hospital and written informed consent was obtained from 91 patients. In five patients the operation was converted to an open procedure and two patients were lost during follow up because of early discharge on the day of surgery.

The remaining 84 patients satisfied the physical status I and II criteria of the American Society of Anesthesiologists, were 20–60 years of age, and were undergoing elective LC under general anesthesia. The exclusion criteria were: body mass index >30 kg/m², history of cardiovascular diseases, renal or hepatic insufficiency, neurological or psychiatric diseases, gastritis, gastric ulcer, allergy to any medication, previous treatment with anti-platelet agents, antihypertensive medication with clonidine or other α₂ adrenergic agonist, chronic use of opioids, steroids, or inability to comprehend the following pain assessment scale. During the pre-operative visit, patients were instructed about the use of a 10-point visual analog scale (VAS), where 0 corresponded to no pain and 10 corresponded to the worst pain imaginable, to assess pain including abdominal pain 1 hr before the surgery.

Patients were randomized by computer generated randomized numbers into three groups of 28 patients each. Group L patients received intravenous lidocaine (Lidicaine HCl, lidocaine 2% 400 mg/20 ml; Jeil Pharm, Korea). Group D patients received intravenous dexmedetomidine (Precedex® 200 μg/2 ml; Hospira, USA). Group N patients received intravenous saline.

For group L, lidocaine was diluted to 50 ml volume by adding 30 ml normal saline (8 mg/ml lidocaine). Group L received a bolus of 1.5 mg/kg lidocaine and continuous intravenous infusion of 2 mg/kg/h lidocaine. For group D, dexmedetomidine was diluted to a 50 ml volume by adding 48 ml normal saline (4 μg/ml dexmedetomidine). Group D patients received a bolus of 1 μg/kg dexmedetomidine and continuous intravenous infusion of 0.4 μg/kg/h dexmedetomidine. For group N, 50 ml normal saline was prepared and group N received saline by the same method as group L. Bolus doses were given over 10 min before initiation of anesthesia, followed by infusion until the removal of trocars. Heart rate (HR), systolic (SBP), diastolic blood pressure (DBP), oxygen saturation and Bispectral Index (BIS™, A-2000 monitor; Aspect medical system, USA) were recorded every 5 min after injection of each drug and just before the initiation of anesthesia.

To ensure proper double blind study, study medications were prepared by an anesthesiologist who was blinded to the study and the label-free drug was administered in operating room. Participating nurses and the anesthesiologist knew how to control the infusion rate. All persons involved in the surgery, including patients, surgeon, anesthesiologist, and nurses, were not aware of patient group assignment.

All patients were premedicated glycopyrrolate 0.2 mg by intramuscular injection 1 hr before the induction of general anesthesia. General anesthesia was induced with thiopental 5 mg/kg intravenously and sevoflurane 3-5% was given until the absence of eye-opening in response to verbal command and BIS score under 60. Endotracheal intubation was facilitated with intravenous rocuronium 0.6 mg/kg. Anesthesia was maintained with 50% inspired nitrous oxide (N₂O) combined with sevoflurane at an end-tidal (Et) concentration adjusted to maintain BIS values between 30 and 60, and HR and SBP within ± 20% of respective baseline values. The patients’ lungs were mechanically ventilated with 50% N₂O in oxygen, with minute ventilation adjusted to maintain normocarbia (EtCO₂ at 35 ± 5 mmHg). Intravenous boluses of 10-20 mg esmolol were administered if HR exceeded 100 beats/min. If, intraoperative hypotension was evident (mean arterial blood pressure [MAP] < 60 mmHg), intravenous boluses of ephedrine 5-10 mg were administered. If MAP increased > 20% above baseline, intravenous boluses of nicardipine 0.5 mg were administered.

The infusion of each drug was stopped at the time of removal of trocars and ketorolac 30 mg and tramadol 25 mg were administered intravenously for postoperative pain control. Residual neuromuscular block was antagonized with intravenous pyridostigmine 0.2 mg/kg and glycopyrrolate 0.008 mg/kg. Sevoflurane was discontinued after the last skin suture and the ventilation was controlled with O₂ (6 L/min) until extubation. The endotracheal tube was extubated when adequate spontaneous ventilation (tidal volume > 4 ml/kg), ability to open the eyes, and patient response to anesthesiologist’s verbal commands were established. Patients were transferred to the post-anesthesia care unit (PACU).
All operations were performed by one surgeon who was very experienced with LC. Pneumoperitoneum was achieved with carbon dioxide, and intraperitoneal pressure was maintained below 14 mmHg during surgery. Patients were positioned in a 30° reverse-Trendelenburg position, and were rotated towards the left side to facilitate exposure of gall bladder. At the end of surgery, patients were returned to a supine position, and residual carbon dioxide in the peritoneal cavity was carefully evacuated through the open trocars by manual abdominal compression.

Patients transferred to the PACU received humidified O₂ via facemask where BP, HR, and oxygen saturation were monitored by nurses who were blinded to the group assignments. Immediately after arrival at the PACU, sedation status was assessed with sedation score as follows: 1, awake; 2, sleepy but arousable; and 3, sleepy difficult to awake. Also patients were instructed to use a VAS score for pain measurement once more and pain was assessed at 15 min, 30 min, 1 hr, 2 hr, 4 hr, 8 hr, 12 hr, and 24 hr postoperatively by an anesthesiologist or nurse who was not involved in the study. According to the study protocol, when a patient requested analgesics due to pain or when the VAS score exceeded 5, 25 mg of tramadol was injected intravenously. If the VAS score remained > 5 after 15 min, patients received fentanyl 20 µg intravenously, and 15 min later, if the VAS score remained > 5, 30 µg fentanyl was given repeatedly until the VAS score decreased to < 5. Metoclopramide 10 mg was intravenous administered to patients who complained of postoperative nausea and vomiting (PONV). Patients, who satisfied the PACU discharge criteria, were discharged to the ward. The discharge criteria included level of consciousness, respiratory stability, oxygen saturation status, hemodynamic stability, physical activity, postoperative pain (VAS score) and PONV.

**Statistical analysis**

Sample size calculation was based on mean value of a previous study [20]. The authors reported that mean ± SD of VAS score in the control group was 7 ± 2 and dexmedetomidine group was 5 ± 3. Twenty-five subjects in each group were required with α value of 0.05 and a power of 80%, but, considering drop out, the sample size was determined to be 28 subjects in each group.

All data are expressed as mean ± SD or as number unless otherwise indicated. Statistical analyses were performed with SAS 9.2 (SAS Institute, USA). The level of significance was set at P < 0.05. A repeated measures analysis of variance (ANOVA) was performed to compare VAS pain scores, HR, SBP, DBP, and recovery profiles, and Scheffe test was used for post hoc analysis. One-way analysis of variance was used to compare quantitative parameters and Levene’s test was used for equality of variances, while Fischer’s exact test was used to compare qualitative parameters.

## RESULTS

There were no significant differences among the three groups with respect to age, body weight, height, ratio of men-to-women, and duration of surgery (Table 1).

### Hemodynamic effects

The three groups were comparable in pre-infusion HR, SBP and DBP. However, after the infusion of dexmedetomidine, the HR of group D patients decreased gradually and it was significantly lower than other groups at all-time points (P < 0.05, Fig. 1). Immediately before endotracheal intubation, the SBP and DBP of group D patients seemed to be higher than other groups pictorially, but no significant differences were evident statistically among the three groups. Furthermore, at all-time points there were no significant differences in the SBP and DBP (Fig. 2).

### Pain score

There was no significant difference in VAS pain score among the three groups preoperatively or for the first 24 hr after LC (Table 2).
Analgesics consumption

In the PACU, the amount of fentanyl requirements in group D and group L patients was significantly less than group N patients. The amounts of analgesics received in the ward during the first 24 hr after surgery did not differ among the three groups (Table 3).

Recovery profile

There were no significant differences among the groups in the time taken to complete tracheal extubation from discontinuation of infusion of each drug and from turning off the vaporizer. Upon arrival at PACU, 23 patients (82.14%) in group D were not alert as estimated by their sedation scores. However, the duration of the PACU stay was markedly shorter

Table 2. Visual Analogue Scale Score of Pain

<table>
<thead>
<tr>
<th></th>
<th>Group N (n = 28)</th>
<th>Group L (n = 28)</th>
<th>Group D (n = 28)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-op.</td>
<td>0.3 ± 0.7</td>
<td>0.8 ± 1.5</td>
<td>0.5 ± 1.0</td>
</tr>
<tr>
<td>Post-op. 0 min</td>
<td>5.3 ± 2.5</td>
<td>4.1 ± 2.3</td>
<td>3.6 ± 3.0</td>
</tr>
<tr>
<td>Post-op. 15 min</td>
<td>5.3 ± 2.2</td>
<td>4.4 ± 2.5</td>
<td>3.9 ± 2.4</td>
</tr>
<tr>
<td>Post-op. 30 min</td>
<td>5.1 ± 1.8</td>
<td>4.5 ± 2.4</td>
<td>4.3 ± 1.9</td>
</tr>
<tr>
<td>Post-op. 1 hr</td>
<td>4.8 ± 2.0</td>
<td>3.8 ± 2.2</td>
<td>4.1 ± 1.7</td>
</tr>
<tr>
<td>Post-op. 2 hr</td>
<td>4.0 ± 1.8</td>
<td>3.5 ± 1.9</td>
<td>3.5 ± 1.5</td>
</tr>
<tr>
<td>Post-op. 4 hr</td>
<td>3.5 ± 1.4</td>
<td>3.2 ± 1.6</td>
<td>3.2 ± 1.0</td>
</tr>
<tr>
<td>Post-op. 6 hr</td>
<td>3.7 ± 1.7</td>
<td>3.0 ± 2.0</td>
<td>3.2 ± 1.4</td>
</tr>
<tr>
<td>Post-op. 12 hr</td>
<td>3.3 ± 1.6</td>
<td>2.8 ± 2.2</td>
<td>3.3 ± 1.3</td>
</tr>
<tr>
<td>Post-op. 24 hr</td>
<td>2.5 ± 1.9</td>
<td>2.4 ± 1.5</td>
<td>2.6 ± 1.3</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD. There are no significant statistical differences among the three groups. Group N normal saline infusion during perioperative period, Group L: lidocaine infusion during perioperative period, Group D: dexmedetomidine infusion during perioperative period. Pre-op.: pre-operative at 1 hr before the operation, Post-op. 0 min: on arrival in PACU, Post-op. 15 min, 30 min, 1 hr, 2 hr, 4 hr, 6 hr, 12 hr and 24 hr at postoperative 15 min, 30 min, 1 hr, 2 hr, 4 hr, 6 hr, 12 hr and 24 hr.

Fig. 1. There is no significant difference in the heart rate measured before infusion of each drug among the three groups. However, except at T0, at all times, heart rate of the dexmedetomidine group was significantly lower than the other two groups. *P < 0.05 compared with control group and lidocaine group. T0: before infusion of each drug, T1: 5 min after infusion of each drug, T2: 10 min after infusion of each drug, T3: before intubation, T4: after intubation, T5: before surgical skin incision, T6: after surgical skin incision, T7: stop of infusion of each drug, T8: at the time of extubation, T9: upon arrival at PACU, T10: 15 min after arrival at PACU, T11: discharge from PACU.

Fig. 2. Changes in (A) systolic blood pressure and (B) diastolic blood pressure during operation and in PACU. There was no significant difference among three groups. T0: before infusion of each drug, T1: 5 min after infusion of each drug, T2: 10 min after infusion of each drug, T3: before intubation, T4: after intubation, T5: before surgical skin incision, T6: after surgical skin incision, T7: stop of infusion of each drug, T8: at the time of extubation, T9: upon arrival at PACU, T10: 15 min after arrival at PACU, T11: discharge from PACU.
channels, and inhibits transmission of the peripheral nociceptive
stimulus, peripheral hyperexcitability associated with neuropathic pain, and the development of postoperative central hyperalgesia. When lidocaine reaches the cerebrospinal fluid and the spinal cord, it activates the inhibitory descending pain pathways and blocks the NMDA and neurokinin receptors. In addition, lidocaine has anti-inflammatory activity for tissue ischemia and damage through the release of adenosine triphosphate and K⁺ channels [6-9].

Perioperative intravenous lidocaine infusion decreased postoperative pain score and/or opioid consumption [10,13-15,23]. Another study reported that perioperative intravenous lidocaine infusion had no effect on reduction in the postoperative pain score, and only reduced postoperative consumption of analgesics [16]. Presently, intravenous lidocaine infusion only reduced the requirements of fentanyl in PACU, and had no difference in postoperative pain score. Lauwick et al. [16] reported that in LC, patients who received 1.5 mg/kg lidocaine bolus and 2 mg/kg/hr lidocaine infusion intravenously until the end of surgery showed 36% reduction in fentanyl consumption in PACU, but no difference in postoperative VAS pain score.

Many factors, such as the time of lidocaine infusion, dosage of lidocaine, tissue damage from the operation, and degree of inflammatory response caused these variable results. We thought that the duration of lidocaine infusion might be the most influential factor. Kaba et al. [10] reported maintaining the lidocaine infusion for 24 hr postoperatively, because some actions of lidocaine, such as leukocyte inhibition, were clearly time-dependent. Further study will be needed, to confirm the

### DISCUSSION

This study demonstrates that both perioperative intravenous infusion of dexmedetomidine and lidocaine reduces the postoperative requirements of fentanyl in PACU after LC. However, no significant difference was observed between dexmedetomidine and lidocaine.

LC usually produces less postoperative pain compared with open cholecystectomy and it can be performed on an ambulatory basis [1,2]. However, some patients still experience severe pain that requires strong analgesia during the first 24 hr after LC [22]. Although opioids and NSAIDs are generally used, extensive use of opioids may cause unexpected postoperative complications, such as sedation, PONV, and respiratory depression, and NSAIDs alone have insufficient analgesic effects [4,5].

The origin of pain after LC is complicated in nature, mainly involving visceral rather than somatic nerve endings, and being associated with abdominal wall and intra-abdominal trauma, abdominal distension with pneumoperitoneum, and an inflammatory response [3-5,22]. Since postoperative pain is the most common complaint of patients after LC, adequate postoperative pain control would be expected to speed recovery and bolster the use of LC as an ambulatory surgery.

Lidocaine has been used systemically as analgesia for neuropathic pain and central pain, and postoperative pain due to its analgesic, anti-hyperalgesic, and anti-inflammatory effects. Intravenous lidocaine blocks peripheral and central voltage-gated Na⁺ channels, and inhibits transmission of the peripheral nociceptive

### Table 3. Total Postoperative Analgesics Consumption

<table>
<thead>
<tr>
<th></th>
<th>Group N (n = 28)</th>
<th>Group L (n = 28)</th>
<th>Group D (n = 28)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>In PACU</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tramadol (mg)</td>
<td>20.5 ± 9.8</td>
<td>13.4 ± 12.7</td>
<td>15.2 ± 12.4</td>
</tr>
<tr>
<td>Fentanyl (µg)</td>
<td>28.6 ± 30.0</td>
<td>11.4 ± 18.0⁰</td>
<td>5.7 ± 14.0⁰</td>
</tr>
<tr>
<td><strong>In the ward</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tramadol (mg)</td>
<td>13.4 ± 14.4</td>
<td>15.2 ± 17.1</td>
<td>17.0 ± 16.7</td>
</tr>
<tr>
<td>Fentanyl (µg)</td>
<td>18.2 ± 19.5</td>
<td>16.1 ± 18.5</td>
<td>15.7 ± 17.1</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD. Group N: normal saline infusion during perioperative period, Group L: lidocaine infusion during perioperative period, Group D: dexmedetomidine infusion during perioperative period. PACU: postanesthesia care unit. *P < 0.05 compared with Group N.

in group D patients than group N patients (Table 4).

### Table 4. Recovery Profiles

<table>
<thead>
<tr>
<th></th>
<th>Group N (n = 28)</th>
<th>Group L (n = 28)</th>
<th>Group D (n = 28)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time 1 (min)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sedation score</td>
<td>16/12</td>
<td>20/8</td>
<td>5/23³</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD and the number of patients. Group N: normal saline infusion during perioperative period, Group L: lidocaine infusion during perioperative period, Group D: dexmedetomidine infusion during perioperative period. Time 1 time taken to tracheal extubation from discontinuation of infusion of each drug. Time 2 time taken to tracheal extubation from turning off the sevoflurane. Time 3 length of the post-anesthesia care unit stay. Sedation score: estimated upon arrival at post-anesthesia care unit 1, awake: 2, sleepy but arousable: 3, sleepy difficult to awake. *P < 0.05 compared with Group N. †P < 0.001 different from other groups. |
relationship between the time of lidocaine infusion and analgesic effect and response to dose.

Although lidocaine exhibits time-dependent or nonlinear pharmacokinetics, if the time of infusion is shorter than 12 hr, the half-life of lidocaine is about 100 min [12]. In our study, the reduction in fentanyl requirements was observed only in the PACU and not in the ward, in which lidocaine infusion was discontinued at the removal of trocars, its short half-life could support this result.

The primary concern of intravenous lidocaine infusion is to achieve therapeutic steady-state concentrations without systemic toxicity. The toxic concentration of lidocaine is a plasma concentration exceed 5 μg/ml [6]. Based on the results from previous studies, the dose of lidocaine of this study seemed to be well below the toxic levels, and there were no complications related with lidocaine infusion [6,10,12]. Also, the bolus dose of lidocaine we used was in the range for treatment or prophylaxis of ventricular arrhythmias, and corroborated previous reports of reductions in pain score and postoperative analgesic consumption following lidocaine bolus administered prior to intravenous infusion of lidocaine [6,10].

Dexmedetomidine is a new generation highly selective α2-adrenergic receptor (α2-AR) agonist. It binds to α2-AR found in the central, peripheral, and autonomic nervous systems, and in vital organs and blood vessels. Dexmedetomidine reduces transmission of the nociceptive signal by activating α2-AR in the spinal cord, which has an opioid sparing effect [17-20,24].

Dexmedetomidine shows biphasic cardiovascular changes and dose-dependent hemodynamic effects [24-26]. It has a vasoconstriction effect through the post-synaptic alpha-2 receptor in the vascular smooth muscle, and a vasodilatation effect caused by sympatholysis through the alpha-2 receptor of the sympathetic and central nervous system. Therefore, when a loading dose of dexmedetomidine is administrated rapidly (<10 min), hypertension may occur due to peripheral vasoconstriction and dexmedetomidine is not recommended in patients with advanced heart block and ventricular dysfunction [24-26].

To avoid adverse effects of dexmedetomidine and achieve stability in MAP, we excluded patients with cardiac problems including heart block, arrhythmia, heart failure, and coronary artery disease, and slowly infused dexmedetomidine over 10 min at an adequate dosage for patients with BIS and vital sign monitoring. Similar to previous studies, during loading of 1 μg/kg dexmedetomidine, eight patients experienced bradycardia with HR < 50 beats/min. However, BP was not lower than in other groups and was in an acceptable range, with no medications needed to control vital signs [21,25]. In addition, the HR of the dexmedetomidine group was significantly lower than the other groups at all-time points. Although we could not analyze the variation of HR, SBP, and DBP, dexmedetomidine may be useful to maintain stable hemodynamics in the event of endotracheal intubation and during the operation [25,26].

The present study echoed previous studies [21,24,27], in the intravenous dexmedetomidine infusion reduced the requirement for fentanyl in the PACU and the time of PACU stay. However, VAS scores and analgesic consumption in the ward were not significantly different compared with the control and lidocaine groups. Dexmedetomidine may not have had a sustained opioid-sparing effect in the later post-operative period because of its short elimination half-life of 2 hr. Iirola et al. [28] reported that context-sensitive half times of 40 year old patients were 20, 50, and 55 min after a 1, 2, and 3 hr infusion, respectively, with no further increase in the context-sensitive half time beyond an infusion period of 3 hr. In this study, the average time of dexmedetomidine infusion was 49 min, and the context-sensitive half time was estimated about 20 min according to previously data [20,28].

One of most commonly encountered problems of patients receiving perioperative dexmedetomidine is delayed recovery and longer discharge time in the PACU due to its sedative property [29,30]. Patel et al. [30] reported that the dexmedetomidine group showed significant sedation at 30 min post extubation. However, other studies reported that dexametomidine enabled an earlier emergence and a shorter length of stay in the PACU [20,26]. They demonstrated that these results were due to an anesthetic-sparing effect and decrease in the requirements of opioid analgesics and antiemetic drugs promoted by perioperative dexametomidine infusion. In our study, although the time taken to tracheal extubation in the dexmedetomidine group was no longer compared with the other two groups, 23 patients receiving dexmedetomidine were not completely alert on arrival in the PACU. However, all patients in the dexmedetomidine group became completely alert to arrival in the PACU. However, all patients in the dexmedetomidine group became completely alert by 20 min after arrival in the PACU and the length of the PACU stay was significantly shorter than the other groups. The dexmedetomidine group showed significant sedation. But, due to its short elimination time and unlike opioids, dexmedetomidine is not associated with respiratory depression, and delayed recovery is not a concern. The reduced need for potent opioid analgesics in the dexmedetomidine group contri-
buted to the reduced PACU stay.

One potential limitation of this study is that we did not evaluate the plasma level of each drug. Although decisions concerning the dosage of each drug were based on previous studies [6,10-12], if we would have evaluated plasma level of drug, we could estimate dose to response relationship of the drug’s analgesic effect and, especially, could estimate plasma concentration of lidocaine to the timing of administration. Another limitation is that pain assessment was done only with abdominal pain at rest. Because many patients complain the shoulder pain as well as abdominal pain after LC, it will be necessary to evaluate shoulder pain and postoperative pain on movement.

In summary, perioperative intravenous infusion of both dexmedetomidine and lidocaine had a beneficial effect on reduction in requirement of opioids in the early post-operative period. We found that it could play an important role in postoperative pain control after LC with multimodal analgesia without any significant side effect. Furthermore, we expected that it might be helpful to reduce the complications in early post-operative period reducing the consumption of opioids. In addition, we could perform LC on ambulatory surgery, if we could control postoperative pain and complications effectively. Although both the dexmedetomidine and lidocaine groups had an opioid sparing effect compared to the control group, there was no significant difference between dexmedetomidine group and lidocaine group. However, intravenous dexmedetomidine infusion reduced the PACU stay compared to the control group.

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REFERENCES


