Effect of Diazepam Sedation on Arterial Oxygen Saturation during Esophagogastroduodenoscopy: A Placebo-Controlled Study

K K Gombar, J C Dhall, R P Suri, Baljit Singh, S Gombar

Departments of Anaesthesiology and Surgery, Pt. B D Sharma Postgraduate Institute of Medical Sciences, Rohtak 124 001

Abstract

Objective: To determine the effect of sedation using diazepam on hemoglobin oxygen saturation (SpO2) in patients undergoing esophagogastroduodenoscopy (EGD).

Method: 100 consecutive patients scheduled for EGD were randomly allocated to receive 0.03 mL/Kg of either diazepam (5 mg/mL solution) or normal saline intravenously after topical oropharyngial anesthesia immediately before the procedure. SpO2 was continuously monitored throughout the procedure by an anesthetist who was unaware of the drug received.

Results: Fall in SpO2 exceeding 4% was noted in 78% of patients in the diazepam group and in 38% of patients in the placebo group (p<0.001). Fall in SpO2 to suboptimal level (89%) was seen in 20% of patients in the diazepam group and in 10% patients in the placebo group (p<0.001). The duration of suboptimal SpO2 was similar (means ± SD being 2.47±0.10 min in diazepam group and 2.86±0.32 min in placebo group).

Conclusion: Intravenous diazepam administration before EGD produces a significant fall in SpO2 during the procedure, and so should be avoided; continuous monitoring of SpO2 should be done during EGD.


Key words: Hypoxemia, endoscopy.

Introduction

Esophagogastroduodenoscopy (EGD), though a reasonably safe procedure, is not entirely free of complications; the most serious ones involve the cardiopulmonary system. A potentially dangerous complication is arterial oxygen desaturation during the procedure, which if not corrected can lead to significant hypoxemia. In one study, 52% of patients had significant hypoxemia during endoscopy. Choudhuri and Agarwal suggested that gagging followed by retching and repeated straining leading to hyperventilation probably caused oxygen desaturation. Hypoxemia has also been attributed to aspiration of gastric contents or saliva during the passage of the endoscope causing parasympathetic stimulation resulting in bronchospasm and fall in cardiac output and to the use of sedative drugs during the procedure.

Diazepam, a benzo diazepine, is the most commonly used drug for sedation during endoscopy. Its use achieves better patient compliance and useful amnesia. Diazepam has only a mild respiratory depressant effect; it may, however, aggravate hypoxemia by depressing hypoxic ventilatory drive. The present study was undertaken to determine the effects of sedation with diazepam on hemoglobin oxygen saturation (SpO2) in patients undergoing EGD.

Methods

One hundred consecutive adult patients of either sex scheduled for EGD and having American Society of Anesthesiologists' physical status I or II were studied. All patients were studied in a standard endoscopic set-up; those with unstable hemodynamics, acute hemorrhage or on oxygen therapy were excluded.

The study protocol was approved by the hospital ethics committee. Patients reported to the endoscopy room after an overnight fast; informed consent for the procedure was obtained. An intravenous line was set up and basal SpO2 was recorded using a finger probe of pulse oximeter (Ohmeda Biox 3700, Louisville, USA). A 2% lidocaine (5 mL) viscous gargle for 4-5 minutes was given to all patients, who were then randomly allocated to receive 0.03 mL/Kg of either diazepam (5 mg/mL solution) or a placebo (normal saline) intravenously immediately before the EGD.

EGD was carried out in the left lateral position using a fiberoptic gastrointestinal endoscope (Olympus GIF-P2, Japan) with 9 mm outer diameter. During the procedure, SpO2 was continuously monitored by an anesthetist who was unaware of the patients' drug allocation. After the procedure, SpO2 was monitored till it returned to baseline.
Table: Patient data

<table>
<thead>
<tr>
<th></th>
<th>Diazepam group (n=50)</th>
<th>Placebo group (n=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>39.8 ± 9.1</td>
<td>42.5 ± 11.9</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>11.7 ± 1.9</td>
<td>11.4 ± 2.6</td>
</tr>
<tr>
<td>Basal SpO₂</td>
<td>98.3 ± 0.6 (97-100)</td>
<td>97.5 ± 0.6 (96-99)</td>
</tr>
<tr>
<td>Minimum SpO₂ during the procedure</td>
<td>91.7 ± 3.2 (78-98)</td>
<td>93.6 ± 3.5 (84-98)</td>
</tr>
<tr>
<td>Frequency of &gt;4% fall in SpO₂</td>
<td>78%</td>
<td>33%*</td>
</tr>
<tr>
<td>Frequency of SpO₂&lt;89%</td>
<td>20%</td>
<td>10%*</td>
</tr>
<tr>
<td>Duration of fall of SpO₂&lt;89% (min)</td>
<td>2.47 ± 0.18 (1.11-3.73)</td>
<td>2.86 ± 0.32 (1.78-3.76)</td>
</tr>
</tbody>
</table>

Data are shown as mean ± SD (range), except where percentages are shown. *p<0.001 as compared to diazepam group.

The data are expressed as mean±SD. Student’s t test for paired data was used for intragroup comparisons and for unpaired data for intergroup comparisons. Categorical data were analyzed using the χ² test.

Results

The two groups had similar mean age, hemoglobin level and baseline SpO₂ (Table). During EGD, SpO₂ fell significantly from the basal value of 98.3±0.6 to the lowest value of 91.7±3.2 (p<0.001) in the diazepam group and from 97.5±0.6 to 93.6±3.5 (p<0.001) in the placebo group. Fall in SpO₂ of 4% or more during the procedure was observed in 78% of patients in the diazepam group and 38% in the placebo group (p<0.001). Fall in the level of SpO₂ below 89% occurred in 20% of patients in the diazepam group and 10% of patients in the placebo group (p<0.001). Fall in the level of SpO₂ below 89% occurred in 20% of patients in the diazepam group and 10% of patients in the placebo group (p<0.001). Mean duration of fall in SpO₂ below 89% was similar in the two groups, being 2.47±0.18 min in diazepam group. No patient in either group had any morbidity due to arterial oxygen desaturation or any other side-effect.

Discussion

Detection of hypoxemia by clinical signs alone is notoriously unreliable; there is a wide variation in the ability of experienced observers to detect cyanosis at arterial oxygen saturation of 70%-80%. Continuous monitoring of arterial oxygen saturation using pulse oximetry is not a routine and hence the exact frequency of arterial oxygen desaturation during EGD is unknown. If undetected, it may lead to life-threatening complications in critically ill patients.

In the present study, we looked for 4% or greater fall in oxygen saturation by 4% or more because this degree of fall is generally considered to be significant. Further, fall to a level below 89% important because at this level the oxygen tension is 60 mmHg; any drop below this level leads to a steep fall in oxygen tension in blood due to the sigmoid shape of the oxygen dissociation curve.

Several studies have shown significant hypoxemia during endoscopy. Desaturation of arterial blood in patients undergoing EGD has been attributed to various factors: age of the patient, nature or dosage of drug used, and the type of procedure do not accurately predict which patients will develop oxygen desaturation. Topical oropharyngeal anesthesia with lidocaine does not cause hypoxemia by itself. On the contrary, partial pressure of oxygen in the blood was found to be significantly higher in patients who had lidocaine gargles than in those who did not. The presence of the endoscope itself in the oropharynx may partially impede the airflow into the trachea due to partial mechanical obstruction and induce low grade aspiration which may contribute to hypoxemia. However, desaturation during endoscopy does not correlate well with changes in the breathing pattern. In our patients, SpO₂ returned to basal levels in the majority of patients while the EGD was in progress, suggesting that the presence of endoscope does not have an important role in causation of hypoxemia.

Diazepam produces minimal changes in arterial oxygen tension in healthy individuals. It is a mild respiratory depressant, produces a decrease in minute ventilation, and makes the slope of ventilatory response curves to CO₂ become flatter without any shift to right as seen with opioids. Further, it depresses the hypoxic ventilatory drive which is maximal at low arterial oxygen tension. In the present study, there was a fall in SpO₂ in both the groups but the frequency and magnitude of desaturation were significantly higher in patients who received diazepam suggesting that the desaturation was directly related to the administration of diazepam before the procedure suggesting that the desaturation was directly related to the administration of diazepam before the procedure.
In conclusion, sedation with diazepam during endoscopy increases the likelihood of significant arterial oxygen desaturation. Continuous monitoring of \( \text{SpO}_2 \) during EGD is suggested in patients receiving diazepam sedation.

References