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CLINICAL REPORT

A case of serious laryngeal edema unpredictably detected during laryngoscopy for orotracheal intubation following induction of anesthesia

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Abstract We report a case of unpredictable and serious laryngeal edema probably caused by preoperative esophagogastroduodenoscopy (EGD). A 54-year-old man with type 2 diabetes mellitus was scheduled to undergo coronary artery bypass grafting (CABG). Two days before surgery, EGD was performed to explore the cause of occult bleeding, resulting in a slightly sore throat and an increased white blood cell count (18,300/μl). Without premedication, general anesthesia was uneventfully induced with intravenous midazolam (10 mg) and fentanyl (50 μg), followed by inhalation of sevoflurane (3%) and intravenous rocuronium (50 mg). Thereafter, manual ventilation was easily performed with a bag and mask. However, on laryngoscopy for orotracheal intubation, serious swelling with rubor and light pus in the epiglottis extending to the arytenoid cartilage was detected, leading to the cancellation of surgery. Immediately following intravenous drip of hydrocortisone (300 mg) and bolus of sugammadex (200 mg), the patient recovered smoothly from anesthesia without complications such as dyspnea, but his sore throat persisted. He was diagnosed with acute epiglottitis. Treatment consisted of intravenous cefazolin (2 g/day) and hydrocortisone (300 mg/day tapered to 100 mg/day) for 9 consecutive days. Consequently, the patient recovered gradually from the inflammation and underwent CABG as scheduled 28 days later. Anesthesiologists should be aware that EGD performed just before anesthesia could unpredictably cause acute epiglottitis, especially in immunocompromised patients, such as those with diabetes.

Keywords Laryngeal edema · Esophagogastroduodenoscopy · Complications · Acute epiglottitis

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Introduction

Esophagogastroduodenoscopy (EGD) is very commonly performed to examine the upper digestive organs. Because the morbidity associated with EGD is low, the risks of complications are prone to be underestimated. Here we report a case of serious acute epiglottitis probably caused by EGD in a surgical patient with diabetes mellitus (DM).

Case description

A 54-year-old man, 173 cm in height and 86 kg in weight, had demonstrated hypertension, hyperlipidemia, and persistent affective disorders for several years. Furthermore, the patient also had a 2-year history of type 2 DM. He had complained of chest compression 3 months earlier and had undergone coronary angiography, which demonstrated obstruction of the right coronary artery and stenosis of the left anterior descending branch. Accordingly, he was scheduled to undergo coronary artery bypass grafting (CABG) under general anesthesia. Preoperatively, he had been taking the following internal medicines (in mg/day): furosemide (20), atorvastatin (10), aspirin (100), nicorandil (15), isosorbide mononitrate (40), and losartan (50) for hypertension, hyperlipidemia, and ischemic heart disease; alprazolam (1.2), zopiclone (7.5), triazolam (0.25),

nitrazepam (5), sulpiride (150), mianserin (10), for persistent affective disorders; and a proton pump inhibitor, rabeprazole (10). In addition, both genetically recombinant human insulin (9 U/day) and insulin glargine (5 U/day) were injected for DM, resulting in a blood glucose circadian rhythm ranging between 89 and 306 mg/dl. Laboratory examination of samples obtained in the early morning 2 days preoperatively yielded normal findings including leukocyte count (6,700/μl), except for aspartate aminotransferase (38 IU/l), alanine aminotransferase (57 IU/l), and hemoglobin A_{1C} (7.1%). In the late morning 2 days preoperatively, to examine the cause of occult bleeding, EGD was uneventfully performed under sedation with 4 mg intravenous midazolam. Consequently, slight gastroesophageal reflux disease and chronic gastritis were demonstrated. During the preanesthetic round on the day before surgery, the patient did not demonstrate hoarseness or any particular complaints, and there was no fever (36.0°C axillary temperature), although leukocytosis (18,300/μl) was observed with 88.1% neutrophils on white blood cell (WBC) differentiation. Regrettably, we did not ask the patient whether he had a sore throat.

On the day of surgery, no premedication was administered. Under standard monitoring, including noninvasive arterial blood pressure measurement, electrocardiography, pulse oximetry, and capnometry, general anesthesia was satisfactorily induced with intravenous midazolam (10 mg) and fentanyl (50 μg), under inhaled oxygen (6 l/min) and 3% sevoflurane. Thereafter, manual ventilation with a bag and mask was easily accomplished. Approximately 1 min following administration of intravenous rocuronium (50 mg), the larynx was observed using a Macintosh laryngoscope for orotracheal intubation. At that time, surprisingly, marked swelling of the epiglottis was detected. An immediate bronchofiberscopic examination demonstrated the presence of serious edema with rubor and a light purulent secretion in the epiglottis extending to the arytenoid cartilage (Fig. 1a). Shortly thereafter, 300 mg intravenous hydrocortisone was infused. Although the vocal cords appeared to be normal and tracheal intubation seemed to be possible, it was decided to cancel surgery, considering the risk of exacerbating laryngeal edema consequent to endotracheal tube insertion. Following intravenous dosage of sugammadex (200 mg), given about 15 min after the administration of rocuronium, he recovered smoothly from anesthesia without signs of airway obstruction, but his sore throat persisted. After waking, the patient reported that he had felt a sore throat continuously since EGD. No urticaria, facial or labial swelling, stridor, or hypotension was observed throughout the course.

Postoperative examination, 3 h after the event, showed leukocytosis (16,700/μl) with 90.8% neutrophils in the differential WBC and an increased serum concentration of C-reactive protein (CRP 8.81 mg/dl). Taking all these findings into consideration, the patient was diagnosed with acute epiglottitis or supraglottitis. Treatment consisted of an intravenous antibiotic (cefazolin 2 g/day) and steroid (hydrocortisone 300 mg/day tapered to 100 mg/day) for 9 consecutive days, followed by oral antibiotic (garenoxacin 400 mg/day), anti-inflammatory enzyme preparation (serrapeptase 30 mg/day), and expectorant (L-carbocisteine 1,500 mg/day) for the subsequent 2 days. Consequently, the patient recovered gradually from epiglottitis, demonstrating some measure of improvement in supraglottic findings on laryngeal endoscopy performed the next day (Fig. 1b) and marked improvement 9 days later (Fig. 1c). WBC and serum concentration of CRP returned to normal 2 and 9 days later, respectively. Throughout the postoperative course, fasting blood glucose ranged between 154 and 250 mg/dl, and axillary temperature remained normal. Twenty-eight days later, the patient underwent CABG uneventfully as scheduled, using the same agents, such as rocuronium, as administered when the initial incident occurred.

Discussion

We reported a case of serious laryngeal edema associated with acute epiglottitis, which was unpredictably detected on direct laryngoscopy for tracheal intubation following the induction of general anesthesia in a diabetic patient. The patient underwent EGD 2 days preoperatively and subsequently developed a persistent sore throat. It was speculated that bacterial infection in the pharynx, which might have been introduced by mechanical injury associated with EGD manipulation, spread to the supraglottic tissue, which was vulnerable to infection because of DM, resulting in acute epiglottitis. In general, supraglottic laryngeal edema occurs in the loose connective tissue on the anterior surface of the epiglottis and on the aryepiglottic folds. Treatment of acute epiglottitis includes administration of antibiotics in combination with steroid, if edema is severe. In our case, although the bacteria causing epiglottitis were not identified, a broad-spectrum antibiotic and glucocorticoid were administered for 9 days, resolving the inflammation. Glucocorticoid is well known to have anti-inflammatory and antiedema effects by inhibiting the activities of phospholipase A_2 and the arachidonic acid cascade. However, the usefulness of systemic steroid administration to manage laryngeal edema is less clear. Several studies in both children and adults indicated that dexamethasone was ineffective in the prevention of laryngeal edema after tracheal extubation [1, 2].

In general, perioperative laryngeal edema will occur in relationship to tracheal intubation and extubation by

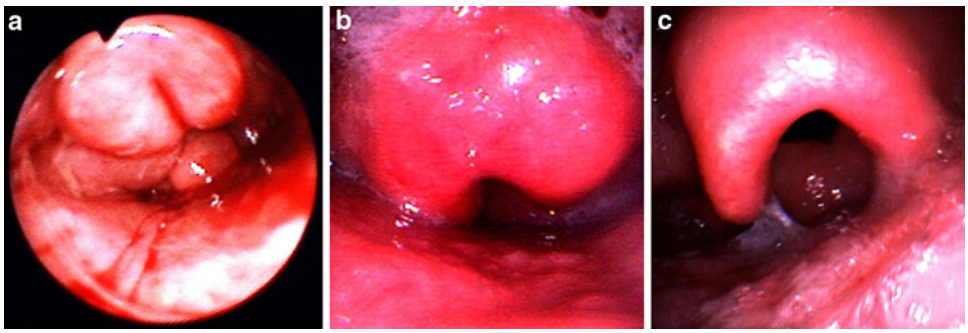

Fig. 1 Endoscopic findings of the larynx, showing the seriously edematous epiglottis and arytenoid cartilage (**a**). The edema was somewhat improved the next day (**b**), and markedly improved 9 days later (**c**)

mechanical injury to the mucosa around the glottis. Factors contributing to the development of laryngeal edema are the use of an inappropriately large tracheal tube, occurrence of trauma at tracheal intubation, prolonged intubation, coughing on the tracheal tube, and a change in position of the patient's head and neck during surgery [3]. Furthermore, so-called nasogastric tube syndrome (NGTS) has been noted as one of the causes of perioperative laryngeal edema [4–9]. Its pathophysiological mechanism is proposed to involve rubbing of the mobile laryngeal structures against the fixed nasogastric tube, compression of the tube against the spine by the cricoid bone in a supine position, and pulling the tube against the posterior cricoid mucosa by contraction of the cricopharyngeus muscle. Although NGTS is rare, laryngeal involvement is common and occasionally fatal. Awareness of NGTS is indispensable to anesthesiologists. It should also be clearly kept in mind that NGTS can occur in patients in whom a probe for transesophageal echocardiography or an ileus tube is inserted.

It is well known that anaphylaxis often induces prompt and intense laryngeal edema [10–12]. The overall incidence of perioperative anaphylaxis is estimated to occur in 1 of 10,000–20,000 anesthetic procedures and in 1 of 6,500 administrations of muscle relaxants [12]. In our case, laryngeal edema was discovered immediately after the administration of rocuronium. However, anaphylaxis as a cause of laryngeal edema might conceivably be deniable, because he did not show any clinical features of anaphylaxis such as urticaria, bronchospasm, or hemodynamic collapse. Moreover, similar anesthetic agents were administered during surgery in the first incident and subsequent uneventful procedure.

In many hospitals, EGD is routinely performed to examine whether patients undergoing cardiac surgery have hemorrhagic lesions in the digestive organs, such as peptic ulcer. Laryngeal edema as a complication of EGD is very rare [13], and during routine preoperative rounds, anesthesiologists observe the oral cavity but do not examine as far as the larynx. However, it should be emphasized that EGD complicated by sore throat could cause laryngeal edema, especially in immunocompromised patients such as those with diabetes.

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