Post-tonsillectomy hemorrhage (PTH) is a known complication of tonsillectomy and adenoidectomy, occurring in about 5% of cases. [1] Primary hemorrhage occurs less than 24 hours post-operatively, while secondary hemorrhages, which are less common, occur more than 24 hours post-operatively. Lethal PTH, however, is rare, and occurs suddenly and unpredictably, leaving most clinicians unprepared to deal with this postoperative emergency.

The objectives of this case report are to describe the anesthetic hazards associated with PTH, present ways to address these hazards, and raise awareness of this rare and devastating complication associated with one of the most common ENT surgeries performed.

Introduction

Post-tonsillectomy hemorrhage (PTH) is a known complication of tonsillectomy and adenoidectomy, occurring in about 5% of cases. [1] Primary hemorrhage occurs less than 24 hours post-operatively, while secondary hemorrhages, which are less common, occur more than 24 hours post-operatively. Lethal PTH, however, is rare, and occurs suddenly and unpredictably, leaving most clinicians unprepared to deal with this postoperative emergency.

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Case Report

7 year old female with Down’s syndrome, presented for repeat tonsillectomy and adenoidectomy for severe obstructive sleep apnea. Surgeon noted difficult tonsillar landmarks secondary to scarring from prior tonsillectomy, but otherwise surgery was uneventful. Patient was discharged on post-operative day #4, when she was taking PO, voiding, and pain was controlled.

On POD#6 she presented to the ER with poor PO intake, sore throat, and spitting up blood. She was rehydrated for dehydration. The next day, while attempting to swallow pain medication, she developed acute-onset bleeding from her mouth leading to profuse bleeding and organizing clots in the oropharynx not amenable to rigid suction. Establishing a seal with a laryngeal mask airway is also difficult, though there has been one successful case report of LMA use in PTH. [6] Intubation attempts should be limited, with pediatric anesthesia involved early. A smaller than normal tube may be necessary. Therefore, rigid suction must be available and clots removed as much as possible to locate the site of bleeding and pressure must be applied to limit blood loss. Vascular access is of the highest importance. Intraosseous lines maybe necessary if IV or central line access is difficult. Resuscitation with IV fluids, blood products, and vasopressors should be initiated swiftly. CPR may lead to chest trauma and pneumothorax, which may impair ventilation leading to worsening acidosis. As soon as there is a return of spontaneous circulation, the patient should be taken to the OR emergently. Anesthetic implications for taking a patient immediately after CPR include continued resuscitation and initiating hypothermia to avoid worsening anoxic brain injury. Although breath sounds were bilateral and ETT was patent, ABGs showed respiratory acidosis that was attributed to the small tube. However, a left pneumothorax was identified postop, which was likely due to earlier CPR. Epinephrine drip was weaned off. Fentanyl and versed were used for anesthesia. Bleeding site was determined to be a branch of the carotid artery at the bed of the left tonsil. The bleeding area was saturated and oral cavity packed. The patient was transferred to the PICU for continued care. During her ICU course, patient remained comatose and mechanically ventilated. Brain MRI showed diffuse global hypoxic injury with EEG evidence of refractory seizures. Withdrawal of life support was made 2 weeks later.

Discussion

Risk factors associated with the occurrence of severe PTH are young age (<12 years), secondary hemorrhage, repeated episodes of bleeding with spontaneous cessation, and aberrant course of the internal carotid artery. [2] Two of these risk factors influence the surgeon’s decision for readmission when bleeding occurs. Inpatient observation after a bleeding episode does not eliminate the threat of lethal outcome. [3] This indicates that PTH is so sudden and catastrophic that clinicians and staff are not adequately prepared for its occurrence.

There is a paucity of anesthetic management guidelines for PTH. Fields et al. carried out a retrospective cohort study to determine incidence of PTH at Children’s Hospital of Philadelphia from 1998-2005, and to identify anesthetic complications.

Fortunately, all 475 cases were non-lethal, and the most adverse outcomes reported were transient hypoxia (9.9%) and difficult intubation (2.7%). Anesthetic hazards reported included anemia, hypovolemia, intragastic blood, risk of aspiration, and clogging bleeding leading to difficult intubation. [4] As our case report illustrates, one of the most important anesthetic considerations for lethal PTH is difficult intubation. Difficult intubation is due to profuse bleeding and organizing clots in the oropharynx not amenable to rigid suction. Positive pressure by mask ventilation can push blood and clots further down the trachea causing more airway obstruction and aspiration. [5] Establishing a seal with a laryngeal mask airway is also difficult, though there has been one successful case report of LMA use in PTH. [6] Intubation attempts should be limited, with pediatric anesthesia involved early. A smaller than normal tube may be necessary. The decision for cricothyrotomy or tracheostomy should not be delayed. This case report demonstrates a novel approach to blindly intubate during CPR by following bubbles that indicate the location of the larynx.

The next anesthetic hazard is risk of exsanguination. Most lethal PTHs are arterial bleeds, commonly from superficial branches of the ICA or ECA. This can quickly lead to cardiopulmonary arrest requiring CPR. Therefore, rigid suction must be available and clots removed as much as possible to locate the site of bleeding and pressure must be applied to limit blood loss. Vascular access is of the highest importance. Intraosseous lines maybe necessary if IV or central line access is difficult. Resuscitation with IV fluids, blood products, and vasopressors should be initiated swiftly. CPR may lead to chest trauma and pneumothorax, which may impair ventilation leading to worsening acidosis. As soon as there is a return of spontaneous circulation, the patient should be taken to the OR emergently. Anesthetic implications for taking a patient immediately after CPR include continued resuscitation and initiating hypothermia to avoid worsening anoxic brain injury. Although breath sounds were bilateral and ETT was patent, ABGs showed respiratory acidosis that was attributed to the small tube. However, a left pneumothorax was identified postop, which was likely due to earlier CPR. Epinephrine drip was weaned off. Fentanyl and versed were used for anesthesia. Bleeding site was determined to be a branch of the carotid artery at the bed of the left tonsil. The bleeding area was saturated and oral cavity packed. The patient was transferred to the PICU for continued care. During her ICU course, patient remained comatose and mechanically ventilated. Brain MRI showed diffuse global hypoxic injury with EEG evidence of refractory seizures. Withdrawal of life support was made 2 weeks later.

References