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Tension Pneumothorax during Esophagogastroduodenoscopy in a Toddler with a Caustic Ingestion: A Case Report

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Abstract

Accidental caustic ingestions most commonly occur in toddlers and young children, may be alkali or acidic and are concerning for oral, esophageal or gastric mucosal injury; however, esophageal perforation and tension pneumothorax (PTX) during esophagogastroduodenoscopy (EGD) are rare. [1] We present a 2-year-old boy who developed tension PTX during diagnostic EGD for a caustic lye ingestion. Oxygen desaturation, decreased tidal volume and increased peak inspiratory pressure occurred, but differential diagnosis was complicated by possible aspiration event prior to mild hypotension developing. Upon endoscope withdrawal, a grade 4 esophageal injury was noted. PTX identified by chest radiograph was managed with emergent chest tube placement. Tension PTX, while rare, may be more common during early EGD for caustic ingestion. This case highlights the greater risk of evolving esophageal perforation and resultant tension pneumothorax from diagnostic EGD in caustic ingestion, and the need for vigilance, timely recognition, and prompt treatment.

Glossary of Terms: *EGD: Esophagogastroduodenoscopy; ETT: Endotracheal tube; MLB: Microlaryngoscopy and bronchoscopy; PTX: Pneumothorax*

Introduction

Caustic ingestion is a serious medical condition with varied clinical presentation and complications based on pH, volume and form of corrosive substance. Alkali ingestion, such as lye, more commonly occur in developed countries. Accidental caustic ingestion peaks in toddlers and young children whereas suicidal ingestion peaks in adolescents. [1,2] Supportive treatment and esophagogastroduodenoscopy (EGD) are the cornerstones of management. EGD plays diagnostic, prognostic and therapeutic roles for caustic ingestions; however, risks include perforation, hemorrhage, infection and cardiopulmonary events. [1] Perforation can occur at the esophagus, stomach and/or duodenum leading to pneumomediastinum or pneumothorax (PTX).

Complications (i.e. bleeding, perforation, mucosal tears) during pediatric diagnostic EGD have a low incidence (0.06%-0.5%), likely because the most common presentation is nonspecific abdominal pain. But injuries are more frequent with more complex procedures. [3,4] Kramer et al reported perforation occurred in 0.13% of EGD without mention of subsequent PTX; noting all were after interventional EGD (and none were secondary to caustic ingestion). [5]

Tension PTX clinically picture includes varying degrees of hypoxia, hypotension, absent breath sounds, tracheal deviation and increased airway pressure and requires decompression for resolution of symptoms. The following is a rare case of a toddler who developed tension PTX during diagnostic EGD performed for granular lye ingestion. This manuscript adheres to the applicable EQUATOR guidelines including written HIPAA authorization from parent.

Case Description

A 2-year-old, ASA PS 1E, 12.6 kg boy who was intubated and sedated, presented for rigid microlaryngoscopy and bronchoscopy (MLB) and EGD secondary to granular lye ingestion 21 hours prior. Maintaining spontaneous respirations, the child was extubated for the MLB which identified multiple tracheal eschars without tracheoesophageal fistula formation. At the conclusion of the MLB a cuffed endotracheal tube (ETT) was placed. Dark brown emesis ensued, followed by oropharyngeal suctioning without a change in oxygen saturation (SpO_2) of 99%. During EGD, SpO_2 decreased to 90%, tidal volume decreased to 70 ml, and peak inspiratory pressure increased by 2 mmHg with wheezing unresponsive to albuterol, ETT suctioning or recruitment maneuvers. Shortly thereafter, mild non-sustained hypotension and distant breath sounds were noted (Table 1). While 2b circumferential esophageal injuries were seen on EGD insertion, upon withdrawal, a grade 4 esophageal perforation was identified (Table 2). Emergent chest radiograph showed large right tension PTX and a chest tube was placed with normalization of SpO_2 and blood pressure.

	Baseline	EGD	13 min into EGD	Chest tube insertion	End case
HR	135	140	130	138	138
SpO_2 %	98	99	90	95	100
SBP	85	78	70	100	90
MAP	47	44	40	50	51

EGD: Esophagogastroduodenoscopy HR: Heart rate; MAP: Mean arterial pressure; SBP: Systolic blood pressure; SpO_2: Oxygen saturation.

Table 1: *Timeline of cardiopulmonary parameters.*

Subsequent flexible bronchoscopy found minimal gastric aspiration and no fistula. A brief repeat EGD was performed to place a critical nasogastric tube under direct vision for esophageal stenting and gastric decompression. The boy returned to the pediatric intensive care unit intubated and sedated. One week post ingestion he had a normal tracheal exam by MLB and was extubated. MLB 1 month later identified a posterior pharyngeal scar band not requiring intervention; however 2 months post ingestion multiple esophageal strictures were found requiring dilation with complete resolution after 9 months.

Classification	Description
Grade 0	Normal mucosa
Grade I	Edema and erythema of the mucosa
Grade 2a	Non-circumferential superficial ulcers, blisters, hemorrhage
Grade 2b	Circumferential lesions or deep ulcerations with features of IIa
Grade 3a	Patchy necrosis
Grade 3b	Extensive or circumferential necrosis
Grade 4	Perforation before or during endoscopy

Table 2: *Endoscopic grade of esophageal caustic injury.*

Discussion

Caustic agents can be acidic or alkaline in nature. Common alkali-containing caustic agents are household bleaches, drain openers, toilet bowel cleaners, dishwashing agents and detergents. Acid-containing agents include toilet bowl cleaners, anti-rust compounds, swimming pool cleaners, vinegar, formic acid and other similar acids. [6] More than 200,000 exposures to household and industrial cleaning products occur every year in the United States. [7] Caustic ingestions have a bimodal age occurrence: children <6 years and adolescent-young adults. Ingestion in the former is usually accidental but often related to psychiatric disorders in the latter. [6,7] In 2005, data from American Association of Poison Control Center reported 9.8% of ingestions in children <6 years were from household cleaning substances. Of the 24 fatalities in this group, 3 were from household products. [8]

Alkali ingestions (i.e. lye) cause liquefaction necrosis, deep tissue damage and typically more esophageal injury than acidic ingestions. [1,9] The extent of mucosal injury from caustic ingestion depends on pH, concentration, tissue contact time, location of contact, amount ingested, viscosity, and the ingested form of the agent (liquid, gel, or solid). [2] However, the relationship between symptoms and severity of injury does not correlate. [10] Early complications of caustic ingestion include chemical pneumonitis, atelectasis, aspiration pneumonia, tracheoesophageal fistula, esophageal perforation, aorta-esophageal fistula, gastrointestinal bleeding, and

dysphagia. Late complications include esophageal or pyloric strictures, gastroesophageal reflux disease, hiatal hernia, gastric outlet obstruction and dysmotility of esophagus and stomach. Additionally, there is an increased risk of esophageal carcinoma compared to normal population. [1,2,6,7,10].

Acute treatment for caustic ingestion is immediate resuscitation and early evaluation for extent of damage. After airway and hemodynamic stabilization, EGD for staging (Table 2) should be performed within the first 24-48 hours then after 3 weeks, as symptoms do not predict injury and esophageal strictures are the most common sequelae. [11] Supportive care includes use of proton pump inhibitors, H2- blockers, and/or antibiotics. [9] EGD is usually avoided in cases of hemodynamic instability, severe respiratory compromise and suspected perforation (grade 4 injury). [12] Our patient was initially cardiopulmonary stable, presenting to the operating suite intubated and mechanically ventilated. EGD demonstrated evolution of the esophageal caustic injury. On endoscope insertion, grade 2b injury (i.e.circumferential lesions with deep ulcerations) of the esophagus was seen. Clinical deterioration occurred 13 minutes into the EGD while the stomach was insufflated and inspected. Insufflation was released, the endoscope slowly retracted, finding lesion progression to grade 4 mucosal injury (i.e. perforation), further demonstrating the need for staging to dictate medical care. Gastric decompression should only be performed under direct visualization to minimize further mucosal injury, thus initially increasing the risk of aspiration. Upon placement, it also functions as an esophageal stent due to the increased risk of stricture. [10]

The incidence of esophageal perforation during pediatric diagnostic EGD is quite rare but greater when done at 96 hours to 2 weeks after caustic ingestion or during balloon dilation. [3,13] While, severe complications from EGD are rare in children, caustic ingestion increases the risk of intraoperative complications including aspiration, esophageal perforation and PTX. Pneumoperitoneum, pneumomediastinum and subcutaneous emphysema also occur in rare cases during EGD. [14] In our case, despite performing the EGD during the advised timeframe, insufflation likely caused a perforation of already severely damaged esophageal mucosa leading to PTX.

Esophageal perforation may result in concomitant rupture of the parietal pleura directly or from a delayed breach due to the enzymatic effect of gastric contents ultimately resulting in PTX. [14] However, there is insufficient data to predict side of PTX after esophageal perforation. Regardless, progression to tension PTX is a severe condition that results when air is trapped in the pleural space under positive pressure, displacing mediastinal structures, and compromising cardiopulmonary function. Tension PTX manifests as hypoxia, hypotension, absent breath sounds, tracheal deviation and increased airway pressure. Tension PTX is life-threatening and requires immediate needle decompression and/or chest tube placement. [15] General anesthesia can complicate the diagnosis of tension PTX. Decreased cardiac output and venous return typically result in hypotension and tachycardia. While our patient had a decrease in blood pressure, SpO_2, and mild increase in airway pressure, he had a decrease in heart rate. This could reflect depth of anesthesia or his inability to further compensate. Therefore, one must have a high index of suspicion that any acute deterioration in hemodynamics, pulmonary compliance and saturation suggests tension PTX.

The initial differential diagnosis of hypoxia in our case included bronchospasm, gastric aspiration, endobronchial intubation, and endotracheal obstruction given the emesis. However, progression to mild hemodynamic instability and eventually distant breath sounds suggested PTX. Given the mild degree of hypotension, concurrent concern for aspiration and ample support services, we obtained a portable chest radiograph then placed a chest tube. Acute needle decompression would be indicated for more severe hypotension and desaturation especially if faced with limited resources or support services until definitive assessment and treatment can occur. Subsequent flexible bronchoscopy was performed to assess and treat concomitant aspiration. It should be considered as a diagnostic tool with acute respiratory deterioration in caustic ingestions during EGD as aspiration or fistula formation may evolve.

While severe complications from EGD are rare in children, caustic ingestion increases the risk of intraoperative complications including aspiration, esophageal perforation and PTX. Anesthesia providers must be vigilant, respond quickly and appropriately to these rare complications and utilize additional resources if readily available.

Conclusion

Complications from diagnostic EGD in children are infrequent. Caustic ingestions, which can have evolving mucosal injury, increase the risk of serious complications. This case highlights three rare events in a toddler during diagnostic EGD from caustic lye ingestion: intraoperative aspiration, evolving esophageal

perforation, and resultant tension pneumothorax. Vigilance, timely recognition, and prompt treatment is essential for safe care of patients presenting for EGD from caustic ingestion.

Conflict of Interest

The authors have no conflicts of interest to disclose.

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