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CASE REPORT

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Successful management of an aorto-esophageal fistula following button battery ingestion: A case report and review of the literature

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Abstract

Foreign body ingestion is a common event among pediatric patients, especially in children less than 6 years of age. Although most cases are relatively benign, with the foreign body passing spontaneously or requiring a brief endoscopic procedure for removal, button battery ingestion is known to cause significant morbidity with the potential for mortality. Although aorto-esophageal fistula (AEF) is a rare complication following button battery ingestion, its clinical manifestations are significant and outcomes are poor. Early diagnosis and aggressive treatment are key in preventing fatal complications. We describe the successful management of an AEF which presented with hematemesis 8 days after removal of a button battery in a 17-month-old female. The literature regarding button battery ingestion and AEF is reviewed and treatment options including intraoperative anesthetic care discussed.

Keywords: Aorto-esophageal fistulae; button battery ingestion; pediatric anesthesia

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Introduction



The ingestion of foreign bodies is a relatively common event among pediatric patients. The ingested items generally pass spontaneously or require brief anesthetic care during endoscopic removal. Rarely, severe complications may occur related to tissue damage from the foreign body that is ingested. The most recent data from the National Capital Poison Center reported 3,244 cases of button battery ingestions in 2017, 1986 (61%) of which involved children less than 6 years of age.^[1] Although the incidence of button battery ingestion has remained unchanged for the past 30 years, the incidence of moderate, major, or fatal complications has risen dramatically with an almost ten-fold increase as compared with 1985. This change is due to the introduction of a more powerful battery (20 mm, 3-volt) to the household market.^[2] All fatalities and 98% of major adverse effects occurred in children less than 6 years of age. The incidence of major morbidity or death in this age group has been reported to be as high as 12.6% compared to a lower incidence of major complication or death in all ages (0.3-1%).^{[1],[2]} Button battery ingestion can result in significant morbidity and mortality including aorto-esophageal fistula (AEF) formation or fistula formation between major blood vessels. We describe the successful management of a life-threatening AEF which formed after button battery ingestion. The literature regarding button battery ingestion and AEF is reviewed and treatment options including intraoperative anesthetic care discussed.

Case Report



Review of this case report and presentation in this format was in accordance with the guidelines of the Institutional Review Board of Nationwide Children's Hospital (Columbus, Ohio). The patient was a previously healthy 17-month-old female infant who presented to the emergency department (ED) with hematemesis and anemia. The patient's past medical history revealed 2 contacts with the ED over the past 10 days for non-specific symptoms including vomiting, diarrhea, congestion, cough, fever, and appetite loss. Discharge diagnoses included gastroesophageal reflux disease and upper respiratory infection. During the current admission, a chest radiograph demonstrated a round, opaque foreign body (23.5 mm), which was presumed to be a button battery [\[Figure 1\]](#). The patient was immediately scheduled for foreign body removal in the operating room (OR). At the time of pre-operative assessment, the patient was tachycardic (heart rate of 154 beats/minute) and hypertensive (non-invasive blood pressure 108/67 mmHg). The hemoglobin and hematocrit were 8 gm/dL and 21%, respectively, and hence a type and cross was obtained. After pre-oxygenation, rapid sequence induction (RSI) was performed with propofol (3 mg/kg) and rocuronium (1.2 mg/kg) and the trachea was intubated on the first attempt. On endoscopic examination of the esophagus, deep ulcers were noted in the upper third of esophagus; however, no active bleeding or perforation was noted. In addition, the foreign body had passed into the small intestine and no attempt was made to remove it. Given persistent tachycardia in the presence of anemia, the patient was transfused at the completion of the procedure. The patient's trachea was extubated and after an uneventful recovery period in the post-anesthesia care unit and she was transferred to the inpatient ward.

The following day, an abdominal radiography demonstrated that the foreign body had passed into the sigmoid colon [Figure 2]. Oral intake was started and advanced without incidence and the patient was discharged home on hospital day 2 with a hemoglobin and hematocrit of 10 gm/dL and 30%, respectively. Three days after discharge, the patient presented to the ED with 2 episodes of hematemesis. On arrival, the patient appeared pale with a heart rate of 163 beats/minute, a blood pressure of 96/42 mmHg, and respiratory rate of 40 breaths/minute. Her hemoglobin and hematocrit were 7 gm/dL and 21%, respectively. Computed tomography of the chest showed a diverticulum at the distal aortic arch immediately distal to the origin of the left subclavian artery, which was presumed to be an AEF. After a multi-disciplinary discussion involving anesthesiology, radiology, general surgery, and cardiothoracic surgery, it was deemed that emergent surgical intervention was necessary. In preparation, two peripheral intravenous cannulas were placed followed by the administration of blood products to correct the existing anemia as the patient transitioned to the operating room. RSI was performed upon arrival to the OR with etomidate (0.4 mg/kg), rocuronium (1.5 mg/kg), fentanyl (5 µg/kg) and the trachea was intubated uneventfully. Anesthesia was maintained with a dexmedetomidine infusion and inhaled isoflurane with bolus doses of fentanyl and rocuronium. A right radial arterial catheter and an internal jugular catheter were placed. Following median sternotomy, cardiopulmonary bypass was instituted and the patient was actively cooled to 20°C. After surgical exposure, a 5 mm ulcerative defect was found on the posterior wall of the descending aorta, with the esophagus effaced to the vessel. Patch angioplasty of the ulcerative defect in the aorta was performed followed by rewarming of the patient and uneventful separation from cardiopulmonary bypass. The patient's trachea was extubated in the OR and she was transferred to the PICU. After 3 days, the patient was returned to the OR for planned repair of the AEF by intercostal muscle flap advancement. Anesthesia included inhaled sevoflurane, a dexmedetomidine infusion, and intermittent bolus doses of fentanyl. Direct laryngoscopy with bronchoscopy was followed by rigid esophagoscopy to fully evaluate the extent of the airway and upper esophageal injuries. Left vocal cord paresis was noted, but there was no evidence of tracheal injury. The cervical esophagus was normal, but the mid-esophagus was injured anteriorly with healing granulation tissue noted, consistent with the negative pole of the button battery facing anteriorly. Rocuronium (1.5 mg/kg) was administered and the patient's trachea intubated. A median sternotomy was performed to establish cardiopulmonary bypass with cooling to 25°C. The patient was then positioned in left lateral decubitus for a posterior thoracotomy incision. The aorta was dissected off the esophagus revealing an 8 mm injury on the anterior wall of the esophagus. An intercostal muscle flap was opposed to the esophageal fistula site. The patient was warmed and then separated from bypass uneventfully. A gastrostomy was placed to allow for enteral nutrition during a prolonged period of *nil per os*. Her trachea was extubated and she was returned to the PICU. Oral intake was started 1 month after the last procedure and the patient was discharged home on hospital day 43. Approximately 1.5 years after the ingestion, the patient had only subtle left vocal cord paresis and has had her gastrostomy tube removed.



Figure 1: Admission chest radiograph showing radio-opaque foreign body suggestive of a button battery

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Figure 2: Abdominal radiograph on hospital day #2 showing radio-opaque foreign body has moved into the distal bowel

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Discussion



Previous work has outlined the mechanisms of tissue injury caused by a button battery.^{[3],[4]} A button battery generates an electric current with hydroxide ions at the negative pole of the battery, which results in liquefaction and necrosis of the adjacent tissue. Tissue damage is observed within 15 minutes of direct contact between the button battery and tissues. As the extent of tissue injury after ingestion is dependent on the length of time that the battery directly contacts the tissues, the most recent guidelines from the National Capital Poison Center recommend no more than a 2 hour window of time from diagnosis to removal of the button battery.^{[4],[5]} Ongoing alkali damage may continue for days to weeks after removal of the button battery, therefore extension of the injury to surrounding tissues, including the trachea and esophagus, may occur, resulting in delayed fatal complications including AEF, TEF, perforation, abscess formation, mediastinitis, and esophageal stricture.^{[4],[5],[6]} Although many of these complications present acutely, delayed presentations are also common even for fatal complications such as AEF. In our review of the literature, the longest time from ingestion to a lethal hematemesis event was 32 days. Scarring and stricture formation generally occurs within a month of the initial event, but delayed stricture formation may occur up to several weeks after the ingestion.^[3]

According to National Capital Poison Center, a total of 307 fatal or severe cases (62 fatal and 245 severe cases) of

button battery ingestions have been reported since 1977. Among the 62 fatal cases, 25 (40%) cases were attributed to AEF, 12 (20%) to esophageal perforation or rupture, 11 (18%) to TEF, and 10 (16%) to bleeding, while no specific cause of death was noted in 4 (6%) cases. Of these 307 fatal and severe cases, 30 (9.8%) involved AEF, with only 5 reports of survival [Table 1] and [Table 2]. All of the reported patients with AEF were younger than 4 years of age with an average age of 26 months. More than half of the patients ingested a button battery that was larger than 20 mm and in the majority of cases, the battery lodged in the esophagus. Age younger than 4 years and size of the battery (diameter of 20–25 mm) are the most important predictors of a clinically poor outcome, with an odds ratio of 3.2 and 24.6 respectively.[2] Merely based on size, larger batteries are more likely to lodge in the esophagus, especially in younger and smaller patients thereby increasing the duration of time that there is direct contact between the battery and the surrounding tissues. Although less likely to lodge in the esophagus, our review demonstrates that batteries smaller than 20 mm in diameter can also be associated with severe or fatal outcomes. Therefore, as noted by the most recent guidelines from the National Capital Poison Center, a button battery that lodges in the esophagus, regardless of its size and the age of the patient, should be urgently removed.[3],[4] Given the high risk of morbidity and mortality, specific guidelines have been developed with recommendations for both home care prior to arrival to the hospital and for pathways to facilitate the rapid transport of these patients to the operating room [Table 3].

Demographic data of 36 cases	Number
Age (month) (mean±SD)	26±17
Gender (male/female/sex not specified)	20/13/3
Battery diameter (millimeters)	
<20	2
20	10
>20	18
Site ingested	
Esophagus	8
Less than 20 hours	12
More than 20 hours	12
Not reported prior to death	3
Not specified	8
Battery location (one case had two batteries)	
Upper esophagus	1
Mid-esophagus	9
Distal esophagus	2
Esophageal location not specified	6
Stomach	2
Distal esophageal membrane	1
Final outcome	
Reintubated	12
Systemic acid-base disturbances	11
Not specified	1
Outcome	
Fatal	25
Re-treated	5
30 included in review	

Table 1: Demographic data of patients with aorto-esophageal fistula after button battery ingestion

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Case	Year	Age	Sex	Site	Size	Time	Outcome
1	1977	1	M	Esophagus	20	24h	Fatal
2	1978	1	F	Esophagus	20	24h	Fatal
3	1979	1	M	Esophagus	20	24h	Fatal
4	1980	1	F	Esophagus	20	24h	Fatal
5	1981	1	M	Esophagus	20	24h	Fatal
6	1982	1	F	Esophagus	20	24h	Fatal
7	1983	1	M	Esophagus	20	24h	Fatal
8	1984	1	F	Esophagus	20	24h	Fatal
9	1985	1	M	Esophagus	20	24h	Fatal
10	1986	1	F	Esophagus	20	24h	Fatal
11	1987	1	M	Esophagus	20	24h	Fatal
12	1988	1	F	Esophagus	20	24h	Fatal
13	1989	1	M	Esophagus	20	24h	Fatal
14	1990	1	F	Esophagus	20	24h	Fatal
15	1991	1	M	Esophagus	20	24h	Fatal
16	1992	1	F	Esophagus	20	24h	Fatal
17	1993	1	M	Esophagus	20	24h	Fatal
18	1994	1	F	Esophagus	20	24h	Fatal
19	1995	1	M	Esophagus	20	24h	Fatal
20	1996	1	F	Esophagus	20	24h	Fatal
21	1997	1	M	Esophagus	20	24h	Fatal
22	1998	1	F	Esophagus	20	24h	Fatal
23	1999	1	M	Esophagus	20	24h	Fatal
24	2000	1	F	Esophagus	20	24h	Fatal
25	2001	1	M	Esophagus	20	24h	Fatal
26	2002	1	F	Esophagus	20	24h	Fatal
27	2003	1	M	Esophagus	20	24h	Fatal
28	2004	1	F	Esophagus	20	24h	Fatal
29	2005	1	M	Esophagus	20	24h	Fatal
30	2006	1	F	Esophagus	20	24h	Fatal
31	2007	1	M	Esophagus	20	24h	Fatal
32	2008	1	F	Esophagus	20	24h	Fatal
33	2009	1	M	Esophagus	20	24h	Fatal
34	2010	1	F	Esophagus	20	24h	Fatal
35	2011	1	M	Esophagus	20	24h	Fatal
36	2012	1	F	Esophagus	20	24h	Fatal

Table 2: Previous reports of aorto-esophageal fistula formation following button battery ingestion

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Case	Year	Age	Sex	Site	Size	Time	Outcome
1	1977	1	M	Esophagus	20	24h	Fatal
2	1978	1	F	Esophagus	20	24h	Fatal
3	1979	1	M	Esophagus	20	24h	Fatal
4	1980	1	F	Esophagus	20	24h	Fatal
5	1981	1	M	Esophagus	20	24h	Fatal
6	1982	1	F	Esophagus	20	24h	Fatal
7	1983	1	M	Esophagus	20	24h	Fatal
8	1984	1	F	Esophagus	20	24h	Fatal
9	1985	1	M	Esophagus	20	24h	Fatal
10	1986	1	F	Esophagus	20	24h	Fatal
11	1987	1	M	Esophagus	20	24h	Fatal
12	1988	1	F	Esophagus	20	24h	Fatal
13	1989	1	M	Esophagus	20	24h	Fatal
14	1990	1	F	Esophagus	20	24h	Fatal
15	1991	1	M	Esophagus	20	24h	Fatal
16	1992	1	F	Esophagus	20	24h	Fatal
17	1993	1	M	Esophagus	20	24h	Fatal
18	1994	1	F	Esophagus	20	24h	Fatal
19	1995	1	M	Esophagus	20	24h	Fatal
20	1996	1	F	Esophagus	20	24h	Fatal
21	1997	1	M	Esophagus	20	24h	Fatal
22	1998	1	F	Esophagus	20	24h	Fatal
23	1999	1	M	Esophagus	20	24h	Fatal
24	2000	1	F	Esophagus	20	24h	Fatal
25	2001	1	M	Esophagus	20	24h	Fatal
26	2002	1	F	Esophagus	20	24h	Fatal
27	2003	1	M	Esophagus	20	24h	Fatal
28	2004	1	F	Esophagus	20	24h	Fatal
29	2005	1	M	Esophagus	20	24h	Fatal
30	2006	1	F	Esophagus	20	24h	Fatal
31	2007	1	M	Esophagus	20	24h	Fatal
32	2008	1	F	Esophagus	20	24h	Fatal
33	2009	1	M	Esophagus	20	24h	Fatal
34	2010	1	F	Esophagus	20	24h	Fatal
35	2011	1	M	Esophagus	20	24h	Fatal
36	2012	1	F	Esophagus	20	24h	Fatal

Table 3: Summary of triage and treatment guidelines for button battery ingestions

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Following button battery ingestion, children may be asymptomatic or manifest non-specific symptoms, especially if a patient is non-verbal age and the ingestion was unwitnessed. These issues can lead to a delayed diagnosis and treatment. In reported cases, the most common signs and symptoms of AEF included hematemesis, vomiting, and abdominal pain [Figure 3]. Although hematemesis may not be a presenting sign, patients may return to the hospital with the abrupt onset of hematemesis following an apparent asymptomatic period after removal of the button battery. Delayed hematemesis has been noted at 2 to 32 days after battery removal. Patients with a history of button battery ingestion who present with hematemesis should be considered to have an AEF. Stabilization of the patient followed by radiologic imaging and upper endoscopy is needed to either confirm or rule out the diagnosis.[6],[22] Diagnostic imaging may include CT or MR angiography to identify the presence of the AEF and its location. Effective care should include a multidisciplinary team including gastroenterology, radiology, general surgery, cardiac surgery, otolaryngology, and pediatric anesthesiology. Although clinically stable at the time of presentation, hematemesis or other presenting signs can be rapidly followed by massive hemorrhage and death.

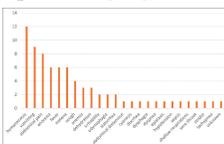


Figure 3: Literature review demonstrating initial symptoms among children who developed aorto-esophageal fistula following button battery ingestion. The x-axis demonstrates the specific symptom with case numbers listed on the Y axis

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Following a rapid and focused preoperative evaluation, preparations should be made to rapidly transport the patient to the operating room. Given the potential for hemorrhage, adequate venous access and blood products should be available in the operating room. Arterial access may be required for hemodynamically unstable patients and to allow for intermittent laboratory analysis as well as continuous blood pressure monitoring. Intraoperative evaluation of hemoglobin, platelet count, coagulation parameters, and acid-base status may be required. Rapid sequence induction is indicated, as these patients are considered to have a full stomach and may be at risk for aspiration during the induction of anesthesia. The choice of anesthetic agents is based on the patient's hemodynamic status. Surgical access for repair of an AEF generally requires a thoracotomy. Cardiopulmonary bypass may be required during repair of an AEF. Although endovascular aortic repair for an AEF is less invasive and has been reported in adults, to date, there remains only one report in a pediatric-aged patient.[23],[20] Postoperatively, the patient should be monitored in a critical care setting as the ongoing alkali damage may continue for days to weeks.

In summary, although the majority of button battery ingestions in children are resolved uneventfully, severe outcomes

including death or stricture formation have been reported. The potential for severe injury is greater in patients less than 4 years of age and with ingestion of larger batteries (diameter greater than 20 mm). Tissue injury continues for days to weeks after removal of the button battery and fistula formation and fatal hemorrhage have been reported. As the majority of patients require anesthetic care during button battery removal, anesthesiologists should be familiar with the current guidelines. The reader is referred to references 3 and 5 for further recommendations and updates.^[3]^[5] Care by a multidisciplinary team and prompt treatment interventions are key to a successful outcome.

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Conflicts of interest

There are no conflicts of interest.

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