Management of Button Battery–induced Hemorrhage in Children

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ABSTRACT

Objectives: Button battery ingestions are potentially life threatening for children. Catastrophic and fatal injuries can occur when the battery becomes lodged in the esophagus, where battery-induced injury can extend beyond the esophagus to the trachea or aorta. Increased production of larger, more powerful button batteries has coincided with more frequent reporting of fatal hemorrhage secondary to esophageal battery impaction, but no recommendations exist for the management of button battery–induced hemorrhage in children.

Materials and Methods: We reviewed all of the reported pediatric fatalities due to button battery–associated hemorrhage. Our institution engaged subspecialists from a wide range of disciplines to develop an institutional plan for the management of complicated button battery ingestions.

Results: Ten fatal cases of button battery–associated hemorrhage were identified. Seven of the 10 cases have occurred since 2004. Seventy percent of cases presented with a sentinel bleeding event. Fatal hemorrhage can occur up to 18 days after endoscopic removal of the battery. Guidelines for the management of button battery–associated hemorrhage were developed.

Conclusions: Pediatric care facilities must be prepared to act quickly and concertedly in the case of button battery–associated esophageal hemorrhage, which is most likely to present as a ''sentinel bleed'' in a toddler.

Key Words: aortoesophageal fistula, bleeding, button battery, hemorrhage

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Button batteries are an ever-increasing part of the environment in which our children live. Ingestion of these batteries is a potentially life-threatening event for children. Catastrophic injuries are possible when the battery becomes lodged in the esophagus, where battery-induced injury can extend beyond the esophagus to the trachea or aorta (1). There is evidence supporting an escalation in the frequency and severity of esophageal button battery injuries. In 1992, Litovitz and Schmitz (2) from the National Battery Ingestion Hotline reported the first large series of 2320 children who ingested button batteries. Of these cases, there were no deaths and no injuries requiring life saving interventions. Of the morbidity reported, there were only 2 severe injuries, both resulting in esophageal strictures requiring repeated dilations (2). Since that landmark publication, there have been multiple case reports of fatal hemorrhage or tracheal damage caused by button battery ingestions (3–9).

Within the last year at our institution, 2 children exsanguinated and died of esophageal hemorrhage caused by a button battery impaction, with 1 death occurring more than 2 weeks following battery removal. As we sought to improve our readiness for future cases, we recognized that no formal recommendations exist for managing children with button battery–induced hemorrhage. These 2 cases dramatically underscored the need for the development of an institutional plan for the management of complicated button battery ingestions. With this goal, our institution undertook a deliberative protocol development effort that engaged subspecialists from a wide range of disciplines. The process and outcome of that effort, which has led to a new management strategy for suspected vascular injury caused by button battery ingestion, are reported here.

MATERIALS AND METHODS

Cases of fatal hemorrhage caused by button battery ingestion were identified from our own institution; a MEDLINE search using the key words ''battery,'' ''hemorrhage,'' and ''aortoesophageal fistula''; and the National Battery Ingestion Hotline.

In-depth interviews at our institution were conducted with pediatric subspecialists representing physicians who would potentially be involved in the immediate care of a child with esophageal hemorrhage secondary to button battery ingestion. Pediatric subspecialists in gastroenterology, surgery, cardiothoracic surgery, cardiology, otolaryngology, emergency medicine, critical care, and radiology were engaged in a deliberative process to gain insight on possible strategies for diagnosis and management. After individual interviews were performed, a protocol was created for the care of children with suspected severe esophageal injury caused by button battery ingestion.

RESULTS

Patient 1

A previously healthy 2-year-old girl presented with chest pain, cough, and nonbloody emesis. A chest x-ray revealed a button battery in the distal esophagus. A 20-mm lithium button battery (CR 2032, 3 V) was removed about 10 hours postingestion using flexible endoscopy, at which time corrosive injury to the esophageal mucosa

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was noted. An immediate postendoscopy chest x-ray did not show pneumomediastinum, and a contrast esophagram, obtained the following morning, did not show extravasation of contrast. The child appeared well and was slowly advanced on feedings until she was ready for discharge 4 days later.

Two weeks after discharge (18 days after the battery was endoscopically removed), she had an episode of hematemesis and hematochezia that prompted readmission. Initially, she was taken to the emergency department, where she appeared well with a temperature of 37.3°C, heart rate of 120 beats/minutes, and blood pressure of 104/63 mmHg. Basic laboratory studies were notable for a white blood cell count of 9.8×10^3 cells per microliter, hematocrit of 29% (decreased from 34% at her previous hospitalization), platelet count of 414×10^3 cells per microliter, and an erythrocyte sedimentation rate of 86 mm/hour. There was no electrolyte imbalance or measured coagulopathy. A chest x-ray and contrast esophagram did not demonstrate evidence of perforation. The child was admitted to the general pediatric ward, made nil per os, and started on intravenous fluids.

Two hours after admission to the floor and 8 hours after initial arrival to the emergency department, she began to have large-volume hematemesis. Shock rendered her unresponsive, and she was intubated before transfer to the pediatric intensive care unit. In an attempt to slow hemorrhaging, she was given a continuous octreotide infusion. She required transfusion of multiple units of packed red blood cells as well as fresh-frozen plasma and activated factor VII for significant coagulopathy. Flexible upper endoscopy was performed at the bedside, revealing a distal esophageal ulceration and copious blood in the stomach. An attempt was made to place a Blakemore tube with esophageal and gastric balloon inflation. The patient continued to have unrelenting hemorrhagic shock that led to her death approximately 14 hours after arrival at the hospital. Postmortem examination revealed an aortoesophageal fistula (AEF).

Patient 2

A 16-month-old girl presented to an outside emergency department with abrupt onset of irritability and a single episode of hematemesis. The child had been evaluated 1 week earlier for an acute illness with fever, vomiting, and an elevated white blood cell count. At that time, she was treated with amoxicillin for a presumed otitis media. There was no history of melena or known foreign body ingestion. An abdominal radiograph revealed the presence of a disk-like foreign body in the stomach.

On transfer to our institution, the patient appeared well and had a temperature of 35.9°C, heart rate of 132 beats/minute, and blood pressure of 105/48 mmHg. Her initial laboratory values revealed a white blood cell count of 20×10^3 cells per microliter, hematocrit of 32%, and platelet count of 800×10^3 cells per microliter.

The patient was made nil per os, started on intravenous fluids and ranitidine, and admitted in stable condition to the general ward with plans for endoscopic foreign body removal the following morning. Two hours after transfer to the ward, the patient was resting comfortably, with a heart rate of 137 beats/minute and blood pressure of 106/67 mmHg. The next morning, while in the preoperative area awaiting endoscopic foreign body removal, she began to vomit copious bright red blood and suffered cardiac arrest. Her acute management in the preoperative area included intubation, massive volume resuscitation, and inotrope administration. She experienced 3 cardiac arrests in the operating room, where emergent abdominal exploration was performed. A button battery along with a large volume of blood was found in the stomach. During laparotomy, she arrested a final time and did not respond to cardiopulmonary resuscitation. She was pronounced dead approximately 9 hours after arrival at our institution. Postmortem examination revealed a large esophageal perforation in the midesophagus. There was significant ecchymosis of the aortic arch, but a fistula was not identified. The clinicopathological diagnosis was of major vessel erosion (unspecified), with fatal hemorrhage into the gastrointestinal tract.

Fatal Cases

Using a PubMed search, our local cases described above, and the assistance of the National Battery Ingestion Hotline, 13 cases of pediatric fatalities secondary to button battery ingestion were identified (3–9). Of these cases, 2 deaths were due to tracheal injury, 1 due to tension pneumothorax, and 10 deaths were secondary to fatal hemorrhage. Seven of the 10 hemorrhage cases occurred since 2004.

Elements of the 10 cases of fatal hemorrhage are presented in Table 1. Autopsy findings demonstrated 7 cases of AEF, 1 case of erosion into thyroid artery, 1 case of erosion into the subclavian artery, and 1 case of suspected injury to a major mediastinal vessel in which definite vascular perforation was not visualized postmortem. All of the cases of fatal hemorrhage occurred in toddlers, ranging from 13 months to 3 years of age. In all but 1 of the cases, the battery ingestion was unwitnessed and the exact duration of battery impaction unknown. Where duration of esophageal impaction could be inferred historically from reported symptoms, we estimated a minimum duration of impaction ranging from 10 hours to 10 days. In 5 cases, the site of injury was proximal esophagus, in 2 cases distal esophagus, in 2 cases midesophagus, and in 1 case the position of the injury was unknown.

In 6 cases, the battery had been removed before the fatal hemorrhage, whereas in 4 cases hemorrhage ensued with the battery still in the digestive tract. For those patients in whom the battery had been removed endoscopically, the duration of time between removal and presentation with fatal hemorrhage ranged from 1 to 18 days. Two of these patients bled to death during their hospitalization after battery removal, whereas 4 patients had appeared well following endoscopy and had been discharged home. In 7 patients, there was evidence of gastrointestinal bleeding, either hematemesis or melena, in the days or hours preceding their fatal hemorrhage.

Aortoesophageal Fistula in Children and Adults

Because the most common cause of death from button battery ingestion is AEF, we sought to thoroughly understand this entity. Beyond button battery–associated cases, AEF secondary to other causes have been described in both adults and children. In children, AEF has been described in the setting of double aortic arch and prolonged nasogastric tube use (10,11), esophageal coin impaction (12,13), and sharp foreign body ingestion such as pins and bones (14–16). Specific to adults, fistulas have been reported in the setting of thoracic aortic aneurysm (17), caustic ingestion (18), esophageal malignancy (19), esophageal ulceration (20), and as a postoperative complication (21,22).

Although there have not been any reported survivors of button battery–induced AEF, children with fistula and hemorrhage for other reasons have been managed with encouraging success. In the setting of double aortic arch, a recent review of published cases revealed a 60% survival rate for infants who hemorrhaged while hospitalized (23). Although prompt resuscitation and immediate surgical repair were the hallmarks of successful care, many authors have reported the use of a Blakemore tube in the acute preoperative management of these double aortic arch patients (24–26).

TABLE 1. Summary of cases of fatal hemorrhage due to button battery ingestion

Year	Bleeding type	Age	Minimum time to battery removal	Sentinel bleed?	Removal to bleed	Intervention?	Esophageal location of injury	Source data
2009	AEF	13 mo	10 d	Yes	2 d	Unknown	Unknown	NBIH
2009	Subclavian artery	29 mo	4–5 d	Yes	N/A	Blakemore/CPR/Endo	Proximal	NBIH
2009	AEF	14 mo	10 d	Yes	N/A	CPR	Mid	Mortensen et al (7)
2009	AEF	29 mo	10 h	Yes	18 d	Endo/Blakemore	Distal	TCH
2008	Indeterminate	16 mo	7–13 d	Yes	N/A	Laparotomy	Mid	TCH
2005	AEF	19 mo	24 h	No	10 d	Thoracotomy	Distal	Hamilton et al (9)
2004	AEF	2½ y	10 d	Yes	N/A	Thoracotomy	Proximal	NBIH
1994	AEF	3 y	Unknown	No	5 d	CPR	Proximal	Sigalet et al (8)
1979	AEF	16 mo	4 d	No	1 d	CPR	Proximal	Shabino and Feinberg (5)
1977	Thyroid artery	2½ y	26 h	Yes	8 d	CPR	Proximal	Blatnik et al (4)

AEF = aortoesophageal fistula; CPR = cardiopulmonary resuscitation; Endo = endoscopy; NBIH = National Battery Ingestion Hotline; TCH = local case at The Children's Hospital, Aurora, CO.

Endovascular intervention has also been used in adults to gain control of a hemorrhaging AEF (22,27), but this technique has not been described in children.

Surgical management of AEF in adults is well described. The surgical approach involves primary repair of the aorta, using synthetic grafts, or more recently, homograft, followed by either primary repair of the esophagus or esophageal resection. In the latter case, a staged reconstruction approach to the esophagus uses techniques such as colonic interposition and gastric pull-up to achieve continuity of the gastrointestinal tract. The principal complication of surgery in the setting of AEF is contamination of the mediastinal space and infection of the aortic graft.

Proposed Management Guideline

A flow diagram was proposed to guide management of ingested button batteries (Fig. 1). Our preference is to remove all esophageal batteries in the operating suite, with pediatric surgery immediately available to use rigid esophagoscopy if significant esophageal edema makes flexible endoscopic battery removal impossible.

With regard to gastric button batteries, a comprehensive triage and treatment guideline is available at *www.poison.org/battery/guideline.asp*. Children with a gastric button battery and pain, vomiting, or evidence of hemorrhage should undergo emergent endoscopy to remove the battery and assess for esophageal injury acquired during battery passage.

In cases of hemorrhage associated with battery ingestion, with suspected vascular injury, multidisciplinary surgical intervention is required because stabilization and repair of vascular injury is paramount, followed by management of esophageal perforation. A child with a ''sentinel bleed'' event after battery ingestion should be managed only in a high-acuity setting such as the emergency department trauma unit or the pediatric intensive care unit. Computed tomography angiogram can be obtained rapidly and is available emergently at most tertiary care institutions 24 hours/day.

Before hospital discharge, in all of the patients with moderate to severe esophageal injury, we suggest endoscopic or radiologic surveillance studies to look for evidence of poor healing or evidence of extraesophageal injury. Because catastrophic hemorrhage has been seen up to 18 days after battery removal, consideration of the timing of hospital discharge must include the proximity of the family to a pediatric facility capable of managing life-threatening bleeding.

On discharge from the hospital, anticipatory guidance should be given to families regarding the range of potential complications of esophageal button battery impaction, which include vascular injury with hemorrhage, tracheoesophageal fistula, mediastinitis, vocal cord injury, esophageal stenosis, and spondylodiscitis.

DISCUSSION

The National Battery Ingestion Hotline has collected data on battery ingestion since 1982, and recently published data clearly demonstrate a temporal trend toward more severe battery ingestion-associated injury (28). This trend is also reflected in the reporting of battery-associated deaths because 7 of 13 fatal cases have occurred since 2004.

The increased severity of button battery–associated injuries during the last 2 decades has paralleled the battery industry's transition to lithium cell production. As production costs of lithium button batteries have fallen, they have become omnipresent in consumer electronics and toys. Its higher voltage of 3 V makes the lithium cell potentially more injurious than traditional 1.5-V manganese dioxide, silver oxide, or zinc-air button batteries. Experimental placement of a 3-V lithium button battery in the

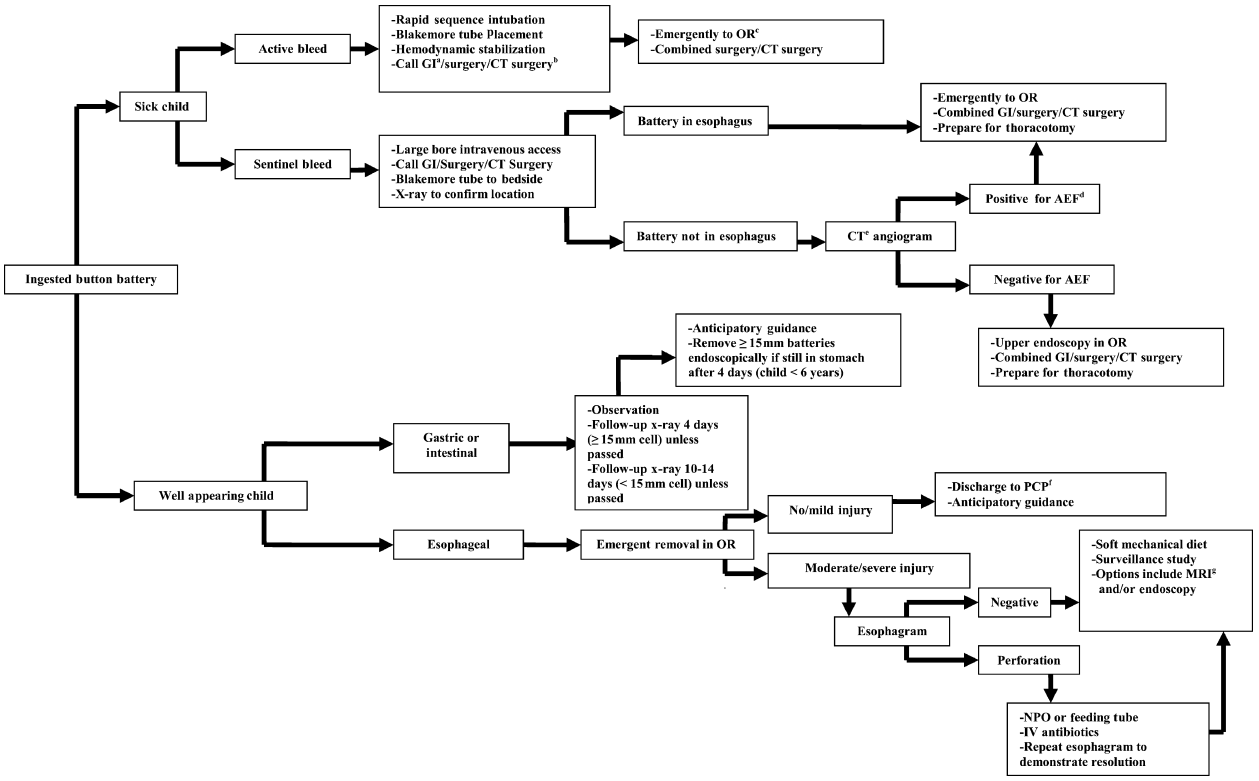

FIGURE 1. Proposed button battery management algorithm. (a) Gastroenterology, (b) cardiothoracic surgery, (c) operating room, (d) aortoesophageal fistula, (e) computed tomography, (f) primary care provider, (g) magnetic resonance imaging.

esophagus of a dog leads to necrosis of paraesophageal tissue after 1 hour of impaction (29). Several of the most common lithium button batteries are of larger diameter (20 mm) than nonlithium button batteries, and these larger batteries are more likely to become lodged in the esophagus of a toddler.

The mechanism of injury of esophageal battery impaction is electrochemical. Esophageal tissue traverses the positive and negative electrodes, which lie in proximity. The flow of electricity then leads to pH changes in surrounding tissue. Experimental models have clearly demonstrated more severe injury in esophageal tissue approximating the negative pole of the battery, where pH changes are alkaline (1). The orientation of the battery within the esophagus may then be helpful in predicting the anatomic direction of tissue necrosis and thus the extraesophageal structures at highest risk of injury.

From our review of fatal cases of hemorrhage caused by button battery ingestion, 2 critical themes emerged. First, the majority of deaths took place after button battery removal, which suggests that the process of tissue injury, healing, and remodeling can lead to fistula development even several weeks after the battery is removed. We endorse active surveillance of children with moderate to severe esophageal injury noted at the time of button battery removal. At present, the ideal strategy for monitoring children after battery removal is uncertain. Both radiologic and/or endoscopic approaches may allow for earlier diagnosis of battery-induced vascular complications. Computed tomography angiogram has been used for diagnosis of aortoenteric fistulas in adults but may not be helpful unless the fistula has actively bled. We have recently used magnetic resonance imaging to evaluate for extraesophageal injury in cases in which significant esophageal ulceration was noted at the time of battery removal, but the sensitivity of magnetic resonance imaging for predicting vascular injury is not established. Finally, endoscopic surveillance of the esophagus may reveal concerning findings, such as a nonhealing ulceration, active bleeding, or pulsatile mass/impression, that require additional investigation.

The second major theme to emerge from our review is that 70% of fatal cases of hemorrhage presented with a history of mild bleeding preceding their exsanguination. If these ''sentinel bleeds'' are recognized in the stable patient, a time window exists in which surgical intervention can be accomplished.

The Blakemore tube has been successfully reported as a means of slowing active hemorrhage from an AEF, but esophageal balloon pressures were not specified. The child-sized Blakemore tube is designed to carry a maximum esophageal balloon pressure of 35 mmHg, which, although sufficient to tamponade venous hemorrhage, may be inadequate in the setting of an arterial bleed. We hypothesize that a substantially higher esophageal balloon pressure would be required to tamponade arterial bleeding but should be used only as a bridge to more definitive intraoperative management because risks of increasing esophageal balloon pressure include the development of pressure necrosis or esophageal perforation.

Button battery–associated hemorrhage is still a relatively rare event and is therefore not amenable to purely quantitative research methods. The present study used a mixed-methods approach involving a retrospective case series and in-depth expert interviews. The strengths of this protocol-development process include its inclusion of a wide range of subspecialist inputs, its use of a deliberative process for the development of recommendations, and its emphasis on multidisciplinary teamwork in managing patients. The present study is limited by the lack of high-quality evidence to guide our recommendations. These recommendations were developed at a tertiary care pediatric facility and may not be applicable to all of the hospitals that care for children.

CONCLUSIONS

Increased production of larger, more powerful button batteries has coincided with more frequent reporting of fatalities secondary to esophageal battery impaction. The most common lesion associated with fatal hemorrhage is a battery-induced AEF. Pediatric primary care providers, emergency practitioners, endoscopists, and surgeons must be prepared to act quickly and concertedly in the case of button battery–associated esophageal hemorrhage, which is most likely to present as a ''sentinel bleed'' in a well-appearing toddler. Fatal hemorrhage can still occur up to 18 days after endoscopic removal from the esophagus.

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