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Review Article

Pediatric esophageal emergency: practical algorithms for the diagnosis and treatment from thoracic surgery perspective

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ABSTRACT

Pediatric esophageal emergency pathologies such as esophageal perforation, corrosive esophagitis and esophageal foreign bodies are complex clinical conditions. Most important point of these pathologies are taking a careful history, focused physical examination, and screening laboratory tests. The mortality and morbidity rates are depending on the time between diagnosis and treatment. The aim of the study is to develop practical algorithms in diagnosis and treatment from the perspective of thoracic surgery that guides preclinical and specialized clinical physicians in the treatment of pediatric esophageal emergency pathologies.

Keywords: esophagus, perforation, emergency, foreign body, corrosive

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Introduction

Pediatric esophageal emergency pathologies, consisting of esophageal perforation, corrosive esophagitis, and esophageal foreign bodies, are conditions that have extremely dangerous consequences in terms of both morbidity and mortality. Treatment options should be determined and applied specifically to the patient, diagnosis, severity of the condition, and other medical conditions [1]. The Most important point of these pathologies are taking a careful history, focused physical examination, and screening laboratory tests. And also, the most important point determining the mortality and morbidity rate is the time between diagnosis and treatment [1,2].

The review aims to develop an algorithm that guides preclinical and specialized clinical physicians in the treatment of pediatric esophageal emergency pathologies. We believe that this will enable the effective and prompt identification and treatment of rare conditions associated with pediatric esophageal issues.

Corrosive esophagitis

Corrosive injuries result from the ingestion of caustic or corrosive substances through the oral route and can lead to erosive esophagitis with strictures in the chronic phase. Although acknowledging a continuous decrease in developed nations, the annual incidence is estimated to be between 5,000 and 15,000 cases globally. The clinical manifestations of caustic ingestion in children can range from no injury to fatal outcomes. It's important to note that nearly all corrosive injuries in children are accidental [1].

Caustic injuries are more commonly observed in countries where preventive measures are lacking, often due to social, economic, and educational factors. Approximately half to 80% of these injuries occur in children. While preventive measures have successfully reduced caustic injuries in many countries, there is still a need for the realization of this goal in numerous developing nations. The ingestion of highly alkaline or acidic substances remains a significant cause of morbidity and mortality globally, particularly in developing regions [1,2].

Accidental caustic ingestion poses the highest risk for children under 5 years old due to their curiosity and well-developed skills to find and drink potentially toxic substances. Risk factors for corrosive injuries in this group include male gender, attention deficit hyperactivity disorder, lower parental education, young maternal age, low

socioeconomic status, and living in rural areas (Figure 1).

Approximately 20-40% of individuals who ingest caustic substances develop erosive esophagitis. The severity of corrosive esophagitis depends on factors such as the amount, concentration, pH, and duration of contact with the caustic agents [2]. Risk factors of caustic ingestion in children are shown in table 1.

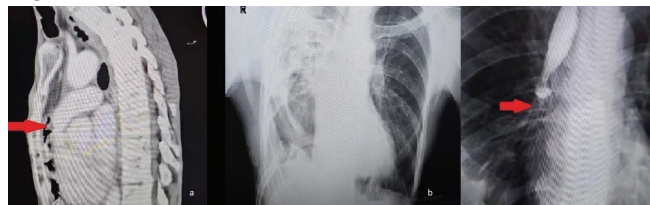


Figure 1. Air images belonging to the colon in the retrosternal area on thoracic CT of the patient who underwent esophagocolostomy (A), volume loss and pleural thickening in the right hemithorax on Chest X-ray (B), leakage of contrast material from the intrathoracic esophagopleural fistula on esophagography (C).

Table 1. Risk factors of caustic ingestion in children [3-11].

Risk factors
• Male gender
• Attention-deficit/ hyperactivity disorders symptoms
• Lower parental education status
• Young maternal age
• Lack of parental supervision
• Rural abode.

Both alkali (pH; 4-11.5) and acid (pH; less than 2) substances can cause significant burns to the cheeks, mouth, oropharynx, esophagus, and stomach, and rarely to the duodenum, as well as the airway. Serious long-term gastrointestinal morbidity occurs in 7-25% of cases, whether or not a stricture is present. Esophageal injury, with or without stricture, results in fibrosis, and abnormal esophageal peristalsis may lead to dysphagia [12-14].

In children ingesting caustic substances, the development of strictures ranges from 10% to 75%, depending on the concentration involved. Alkaline ingestion is more prevalent in Africa, the Americas, and Oceania, while acid ingestion is more common in Europe and Asia for children. Despite the higher prevalence of acid ingestion in certain regions, stricture formation is more commonly associated with alkaline ingestion. The incidence of stricture formation after acid ingestion in children is reported to be in the range of 2.9% to 15.3% [15].

The mechanism of tissue damage caused by caustic substances depends on their pH. Acidic substances cause coagulation necrosis due to protein denaturation,

while alkaline substances cause liquefaction necrosis by destroying cell architecture. As a result, acids typically cause superficial injuries, whereas alkalis cause deeper injuries. While it's not an absolute rule, acidic substances often lead to long-segment gastrointestinal injuries, while alkaline substances tend to cause more limited injuries in the esophagus [16,17].

In the first week after caustic ingestion, mucosal damage and bacterial invasion become apparent. Full-thickness injuries during this period can lead to perforation. Esophageal remodeling begins around the start of the second week, and as a result, endoscopy is typically not performed between 5 and 15 days after the injury. Scar retraction starts by the third week, driven by fibroblast proliferation. Mucosal re-epithelialization begins during this period and is usually completed by the sixth week after the injury. This timeline highlights the dynamic process of tissue response and repair following caustic ingestion [2].

Presentation and emergency department treatment

Patients typically present to the emergency department following the ingestion of a caustic substance. If the ingested substance is solid, it often causes oropharyngeal injuries, while liquids can result in burns to the esophagus. The presence of oral injury generally indicates esophageal injury as well. However, the absence of oral injury does not rule out esophageal injury. In patients with caustic injury but no oral lesions, approximately 1% may develop esophageal strictures in the chronic period [13].

Many patients are initially asymptomatic, but despite this, esophageal injury can be detected in a significant number through endoscopy. In 6-18% of patients, oropharyngeal and laryngeal injuries may lead to symptoms such as hoarseness, stridor, dyspnea, tachypnea, or wheezing. Severe injuries can result in transmural necrosis, perforation, and mediastinitis, although this is very rare. If a patient develops fever, tachycardia, severe retrosternal or abdominal pain, hemorrhage, sepsis, or organ dysfunction develop in a patient, urgent surgical intervention may be necessary.

The presence of symptoms like drooling, dysphagia, and epigastric pain may indicate a possible gastrointestinal injury. Severe injuries are suggested by vomiting and hematemesis. If a patient shows signs of gastrointestinal injury, such as drooling, dysphagia, and epigastric pain, or if severe symptoms like vomiting and hematemesis are present, it indicates a more severe gastrointestinal injury, and prompt medical attention is crucial.

Treatment

Firstly, it is necessary to determine the type and quantity of the ingested substance. The patient should never be induced to vomit. Neutral liquid such as water or milk can't be given due to concerns regarding potential further harm from heat injury during the neutralization process, diluting agents should be avoided, as they pose safety risks. During this process, it is crucial to start intravenous fluid infusion for the patient [18].

If there is an airway injury, respiratory stabilization (intubation and/or tracheostomy) should be prioritized, followed by an evaluation of the esophagus. Approximately 18% of patients may require emergency tracheostomy, and 1% may need chronic tracheostomy [19].

Although there is no evidence in the literature, broad-spectrum antibiotic therapy can be initiated or prophylaxis can be applied before endoscopy due to the potential for perforation and other bacterial complications [20].

The first diagnostic test should be a posteroanterior chest X-ray. The X-ray is used to evaluate mediastinal widening, mediastinal emphysema, intra-abdominal free air, and pneumothorax. The gold standard for detecting esophageal injury is esophagogastroduodenoscopy. Endoscopic evaluation should be performed within the first 24-48 hours and under general anesthesia, as the risk of perforation is lowest during this period. Additionally, to minimize the risk of possible perforation, air insufflation during endoscopy should be minimal or avoided altogether. The endoscope should not be blindly advanced, and the procedure should be terminated if injury is observed. In cases of potential severe injury, careful nasogastric tube placement with endoscopic visualization should be performed to facilitate enteral nutrition and prevent gastric content treatment at the site of injury [21-23].

The Zargar et al classification system proposed in 1994 is commonly used to grade esophageal injuries in clinical practice [20,24] (Table 2). In the acute phase, the presence of perforation, its location, and dimensions, along with accompanying complications such as mediastinitis, pneumonia, etc., can be assessed using contrast swallow studies with water-soluble contrast (X-ray and/or CT). In the chronic phase (3-6 months later), potential stricture development, pyloric obstruction, or the evaluation of treatment response after dilation following stricture development can also be assessed using these imaging techniques [25,26].

Table 2. Classification in caustic injuries.

Grade	Endoscopic appearance	Risk of perforation (%)	Incidence %	Treatment
0	No evidence of injury	0	11–57	Observed in hospital until full oral feeds are tolerated
I	Mucosal erythema and oedema	0	11–88	
II				2-4 weeks hospitalization A liquid diet, either orally or via nasogastric tubes
A	Superficial non-circumferential erosion, ulcers, haemorrhage or exudate	<5	7–26	
B	Deep or circumferential ulceration	71	13.6–28	
III				
A	Multiple scattered ulcerations with patchy necrosis	100	0.5–12	
B	Extensive necrosis		0–1	

Preventive treatment

PPIs or histamine H₂-receptor antagonists: To reduce stomach acid.

Oral nystatin suspension: To prevent endogenous fungal overgrowth, exacerbation of stricture formation, and colonization of the nasogastric tube. It should be used for 3 weeks.

Nasogastric tube placement: For early enteral feeding.

Steroids: The use of high-dose methylprednisolone (1 g/1.73m² daily for 3 days) decreases stricture formation. However, the use of steroids in caustic esophagitis is controversial. It has been determined that patients with first or third-degree esophageal injuries do not benefit from steroid use. Still, steroids can prevent stricture formation in patients with second-degree caustic esophagitis [1,28-30].

Treatment of long-term complications

Esophageal strictures

Esophageal strictures in children are most commonly caused by caustic injuries. Strictures are most commonly determined in the cervical and middle thoracic esophagus, with an incidence ranging between 2% and 49%. It has been demonstrated in the study conducted by Nunes et al that severe endoscopic lesions, involvement of the entire length of the esophagus, hematemesis, and increased serum lactic dehydrogenase represent risk factors for the development of fibrotic strictures induced by caustic ingestion. Serial stricture dilation is the mainstay of therapy for esophageal strictures. However, esophageal replacement is the most suitable treatment

choice for long and/or refractory strictures [1,27-29].

If patients experience symptoms of dysphagia, and a stricture is identified endoscopically, a dilation program can be initiated 3 weeks after the injury. The success rate of dilation is very high for non-lye strictures, strictures under 5 cm in length, strictures in the upper third of the esophagus, and in younger patients (under 8 years of age). Weekly to bi-weekly dilations over 3 months, with an average of 12 dilations, are required for strictures due to corrosive injuries. The perforation rate of dilation ranges from 0.4% to 17.4% [1,5,8].

Esophageal replacement surgery is required in 18% of patients who experience perforation during dilation, and the mortality rate increases to 18% in such cases [9]. Some reports suggest that the frequency and necessity of dilatation may decrease with the injection of steroids and/or mitomycin into the stricture area, although there are limited pediatric reports on this approach [9].

Stenting of esophageal strictures with double-tube polyamide and silicone, self-expanding covered metal (nitinol) stents, or polydioxanone absorbable stents may be considered as a good alternative to serial/multiple dilations or surgery. However, there are challenges associated with stenting in pediatric cases, including the lack of stents suitable for children, high costs, stent migration, and the potential for secondary stenosis due to granulation tissue in the proximal and distal parts of the stent [1,30]. The diagnosis and treatment algorithm for caustic injuries is prepared based on the literature data and our clinical experience (Figure 2).

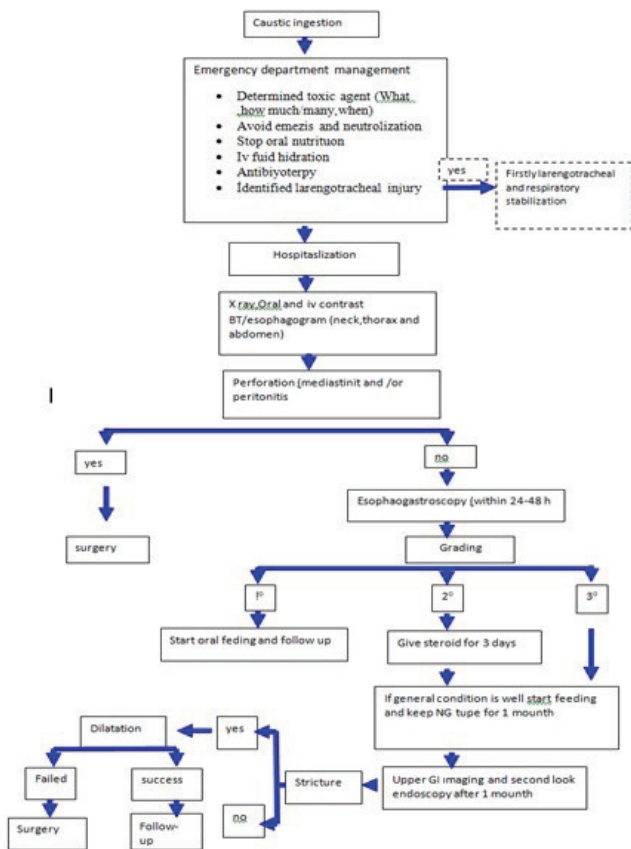


Figure 2. Algoritim in diagnosis and treatment of caustic esophagitis.

Esophageal perforation

Esophageal perforation (EP) is rare in the neonatal population, with an incidence reported as 0.006% in large series [31]. The first spontaneous esophageal perforation due to the esophageal web was reported by James Fryfogel in 1952. Additionally, the first iatrogenic esophageal perforation was reported during stiff rubber catheter suctioning in a 28-day-old infant in 1961 [32].

The esophagus is highly susceptible to trauma and injury due to the absence of supporting tissue around its serosal layer. Once an injury occurs, bacterial spread and the inflammatory response can rapidly and easily lead to complications. Polymicrobial flora dominated by anaerobes, along with gastrointestinal content, can spread into the mediastinum, resulting in conditions such as mediastinitis, empyema, abscesses, sepsis, and multiple organ dysfunction syndrome, all of which have high mortality and morbidity rates [33].

Despite improvements in post-operative intensive care unit management, antibiotic therapy, and advances in enteral and parenteral nutrition, esophageal perforation remains a life-threatening condition. The mortality rate has recently been reported as 28%. It's crucial to note

that after the diagnosis of perforation, mortality doubles for every 24 hours that pass without treatment [34].

Etiology

Esophageal perforation can occur due to various reasons, with endoscopic stricture dilation and nasogastric tube insertion. The table given below outlines the most common causes of esophageal perforation (Table 3). In children, the most frequent causes include iatrogenic traumas, lye burns or chemical burns, direct and indirect trauma, ingestion of foreign objects (Figure 3), operative trauma, and idiopathic causes.

Table 3. Reasons of esophageal perforation.

Reasons of esophageal perforation	
Intraluminal	Extraluminal
1-Endoscopically	1-Penetrant
>Esophagoscopy	>Gunshot wound
>Dilatation	>Stab wound
>Intraesophageal tube insertion	>Foreign bodies erosion
>Biopsy	
>Skleroterapy	2-Blunt injury
>Endotracheal tube	>Traumas
	>Resustation
2-Nonendoscopic injuries	
>Barotrauma	3-Operative trauma
>Coustic injuries	
3-Foreign bodies	

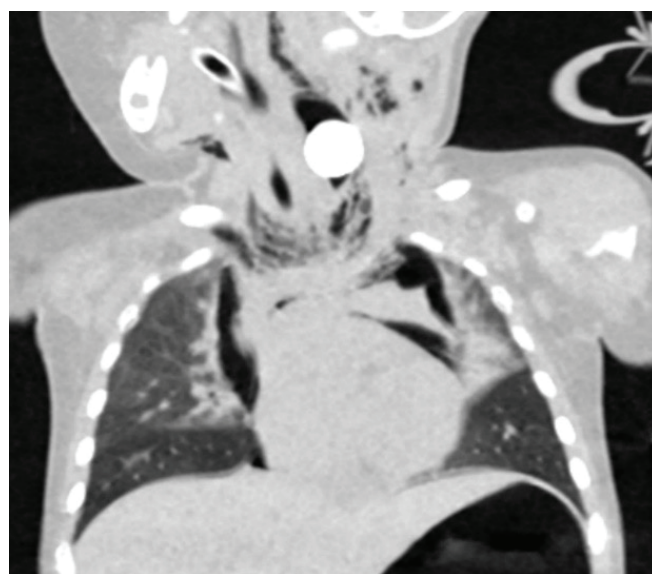


Figure 3. A 3-year-old male patient who swallowed a coin. On thoracic CT, there is a coin in the cervical esophagus, esophageal perforation, and mediastinal emphysema (From the archive of the Department of Thoracic Surgery, Erzurum Atatürk University Faculty of Medicine with permission).

Clinical presentation

Symptoms of esophageal perforation can vary based on the cause, location of the perforation, and the time between diagnosis and treatment. However, the most common symptoms include dysphagia (60%), dyspnea, and fever. In cervical perforations, symptoms tend to be less severe as the spread of contamination from the retroesophageal space to the mediastinum is slow. On the other hand, thoracic perforations lead to rapid contamination of the mediastinum, resulting in symptoms of systemic sepsis and shock within 24 hours. Gastric contents and bacterial flora from the gastrointestinal tract infiltrate the mediastinum, causing severe necrotizing mediastinitis. Negative intrathoracic pressure can worsen pleural rupture, leading to pleural effusion and pneumothorax. In intra-abdominal esophageal perforations, peritonitis is a common presentation, and symptoms may appear within a few hours [35,36].

In neonates, symptoms of esophageal perforation may include hypersalivation with choking, coughing, or cyanosis during feeding. Neonates might also present with a pneumothorax. Fever is often observed in the first 24 hours after challenging endotracheal intubation or nasogastric tube insertion [34]. Symptoms according to the location of the perforation are shown in table 4.

Table 4. Symptoms according to the location of the perforation.

Localization of perforation	Symptoms
Cervical	Neck pain Cervical dysphagia Dysphonia Bloody regurgitation Subcutaneous emphysema Leukocytosis Swelling Redness in neck
Thoracic	Chest pain Tachycardia Tachypnea Fever Leukocytosis Subcutaneous emphysema
Abdominal	Abdominal pain Shoulder pain Dysphagia Drooling Local tenderness

Diagnosis

The diagnosis of esophageal perforation can be made through various methods. Although it is normal in 12-33% of patients, neck and chest X-rays should be the first diagnostic test in patients with suspected esophageal perforation. A plain X-ray can demonstrate the localization of the enterogastric tube, pneumothorax, pleural effusion, pneumomediastinum, and mediastinal enlargement. In a patient with findings suggestive of esophageal perforation on X-ray, the diagnosis is confirmed through a contrast-enhanced esophagogram and endoscopy. These examinations help to determine the location and size of the perforation, with a reported sensitivity of 100% in the literature [37].

In clinical practice, especially in infants, endoscopy is not recommended due to the difficulty of application and the risk of increasing the size of the perforation [34,36].

Esophagography findings in esophageal perforation include;

- A collection of contrast in a retropharyngeal pocket from a localized cervical leak.
- A submucosal perforation, appearing as a tract parallel and posterior to the esophageal column.
- Free perforation into the pleural space [32,35,36].

Chest computed tomography (CT) can be very useful in diagnosing esophageal perforation. In patients with esophageal perforation, CT may reveal air and fluid in the extraesophageal area in the mediastinum, esophageal wall thickening, and mediastinal or para mediastinal air-fluid collection, abscess cavities in the pleural space or mediastinum [37-40].

Treatment

Surgical treatment

Surgical indications in esophageal perforation (EP) include clinical deterioration, persistence of a leak despite treatment, massive intrapleural or retropharyngeal spillage, sepsis, mediastinitis, abscess formation in pleural and mediastinal spaces, or even massive or persistent leaks [41].

The most important factor determining post-treatment mortality and morbidity is the time between diagnosis and treatment. The main principles of surgical treatment include the debridement of devitalized tissue from the perforation site, adequate pleural drainage, and elimination of the contamination source. After debriding the devitalized tissue from the perforation site, the

muscular layer and mucosa must be closed with absorbable interrupted sutures. The primary repair site should be reinforced using intercostal, omental, pericardial fat pad, diaphragmatic pedicle graft, rhomboid muscle, latissimus dorsi muscle, and intercostal muscle or pleural flaps. Surgical approaches according to the perforation site are shown in the table 5 [34 -38].

Table 5. Surgical approaches according to the perforation site.

Perforation localization	Surgical approach
Servical esophageal perforation	Left neck along the lower third of the sternocleidomastoid (SCM) muscle
Mid esophageal perforation	Right thoracotomy at the sixth intercostal space
Distal esophageal perforation	Left thoracotomy in the eighth intercostal space
Abdominal esophagus	Laparotomy

Esophagectomy may be necessary in cases of severe necrosis and multiple injuries due to caustic esophagitis, severe mediastinal contamination, and failure of primary repair. Diversion with cervical esophagostomy might be required if the esophagus is too friable, the patient remains unstable, the repair is not possible due to the size of the defect or friability of surrounding tissue, or widespread mediastinitis prevents primary repair during the initial procedure [34-38].

Recently, minimally invasive surgery has been employed in esophageal perforation. However, there are only a small amount of patient series reported in the literature that were treated with thoracoscopic drainage of the mediastinum, and no mortalities were reported in these series [32,34,36,37].

Non-surgical treatment

In the literature, the total mortality of non-operative treatment in esophageal perforation (EP) is reported to be 18%. This rate is lower than other treatments for EP in childhood compared to adults. Non-operative treatment consists of antibiotics that cover both anaerobes and aerobes, adequate fluid resuscitation, stopping oral intake and starting parenteral nutrition, instituting gastric drainage via gastrostomy or nasogastric tube, and placing a thoracostomy tube. Oral nutritional intake is stopped to decrease further contamination of the wound and allow for esophageal healing [38,39,41,42]. If enteral tubes can be safely passed under fluoroscopic guidance, enteral

nutrition should be continued. If not, total parenteral nutrition (TPN) should be reserved. Oral feeds can be resumed after an esophagram shows no further leak. It is reported that the average interval to the first esophagram after diagnosis is 7 days, and the average interval to the initiation of oral feeds is 11.5 days [38].

Systemic infection and sepsis progression can be prevented by using broad-spectrum antibiotics that generally cover gram-negative and anaerobic spectra. Typically, antibiotic therapy consists of piperacillin/tazobactam, vancomycin, and gentamicin, along with antifungal agents. The treatment is continued for 7–14 days from the time of perforation diagnosis. If the leak due to perforation persists on esophagography, antibiotic therapy must be continued for an additional 7 days. If pneumothorax and/or pleural effusion are present, drainage must be provided by tube thoracostomy. Pneumothorax has been reported in 32% of patients with esophageal perforation. It is usually seen in the right hemithorax and can be treated with a chest tube or small pigtail catheters [42]. The diagnosis and treatment algorithm for esophageal perforation is prepared based on the literature data and our clinical experience is shown in figure 4.

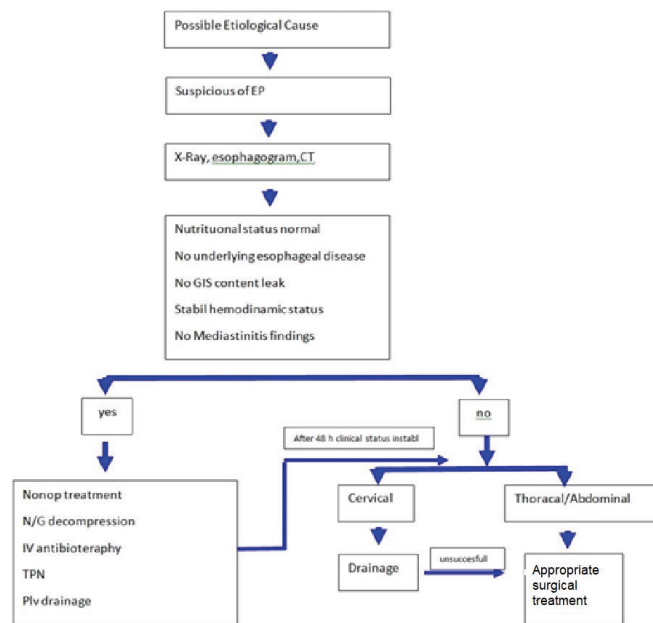


Figure 4. Algorithm in diagnosis and treatment of EP. (Abbrev.; EP:Esophageal perforation, CT:Computed tomography, N/G:Nasogastric tube, IV:Intravenous, TPN:Total parenteral nutrition,Plv: Plevral)

Foreign Bodies

Aerodigestive foreign bodies are one of the most important morbidity and mortality reasons in infants and children. The aspiration of foreign bodies usually cause

a chronic lung infection. and most of the patients admitted to hospital for this reason. Prevalence of foreign body ingestion is reported as 17.9 / 10,000 children who are applied in U.S. emergency departments [43]. The most commonly seen FBs are coins, toys, batteries, and high-powered magnets [44] (Figures 5,6).

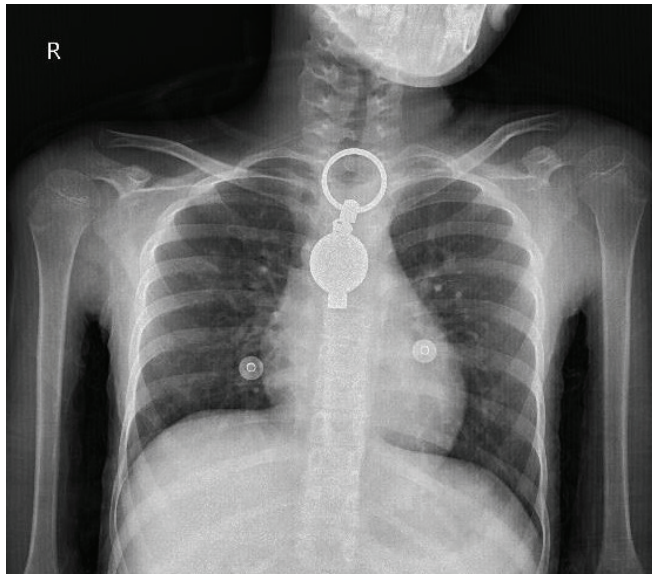


Figure 5. A 12-year-old male patient who swallowed a keychain. On X-ray, a keychain is observed in the upper thoracic esophagus (From the archive of the Department of Thoracic Surgery, Erzurum Atatürk University Faculty of Medicine with permission).

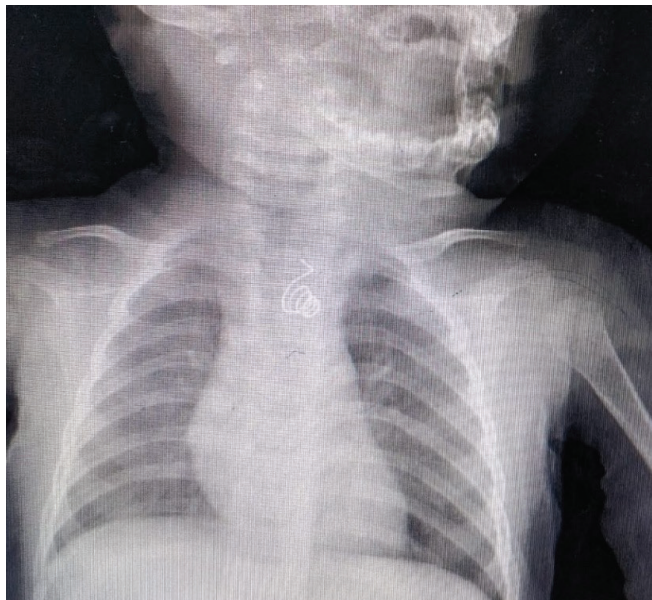


Figure 6. A 5-year-old male patient who swallowed a clothespin spring. On X-ray, a clothespin spring is observed in the upper thoracic esophagus (From the archive of the Department of Thoracic Surgery, Erzurum Atatürk University Faculty of Medicine with permission).

While a lot of GIS foreign bodies pass spontaneously

through the gastrointestinal (GI) tract, 40% of esophageal FBs are asymptomatic. For this reason, diagnosis and treatment are delayed and complications that may be mortal and morbid (such as oesophageal perforation, periesophagitis, and mediastinal infection) may appear [45]. The most common symptoms are dysphagia, vomiting, drooling, feeding refusal, and neck and chest pain. According to the European Society of Pediatric Gastroenterology, Hepatology, and Nutrition guidelines, Large FBs even compressing the airway may present with stridor, wheezing, and respiratory distress [46,47].

Because of cricopharyngeal area in the cervical esophagus is the narrowest part of the GIS tract, foreign bodies most commonly lodge in this area. If the FBs cross the cricopharyngeal area, these often pass through into the lower GIS tract and uneventfully are excreted from the intestine [48]. In radiopaque esophageal FBs, diagnostic procedures are firstly neck and chest PA and lateral X-rays even if, plain radiographs of the neck are normal, if a retained esophageal object is suspected by history, examination, or symptoms, an esophagram is needed [49,50].

Treatment

The most common treatment strategies for EFBs in children are rigid oesophagoscopy and Foley catheter balloon extraction. The use of a Foley catheter is used, while the patient is maintained in the Trendelenburg position. Complications of Foley catheter include perforation, aspiration, and acute airway obstruction. The Foley catheter is a safe, efficient, convenient, and practical method for the treatment of children with coins and other blunt EFBs, and it can significantly reduce the need for endoscopy. Some meta-analyses showed that no significant difference between rigid and flexible endoscopy regarding efficacy and safety for the treatment of esophageal foreign bodies [51,52]. However, rigid oesophageal endoscopy under general anesthesia is recommended in Children with EFBs to protect the airway. Moreover, in literature, it is strongly recommended that rigid endoscopy for children because it allows both the use of optical forceps with a strong grasping ability for EFBs and the positioning of sharp and pointed objects inside the rigid endoscope the removal of oesophageal button is extremely more important than other foreign bodies. Because, these cause chemical corrosion, electrical damage, thermal burns, and physical compression

to the oesophageal mucosa. Oesophageal necrosis and even life-threatening complications can occur within 2 hours after accidentally ingesting a button battery. For this reason, in patients, emergency endoscopy and/or operation should be performed without fasting or pre-surgical preparation to reduce the corrosion of the oesophageal mucosa [50-51].

Surgical intervention is indicated in cases of large, sharp-edged foreign bodies embedded in the mucosa that cannot be removed endoscopically and in the presence of perforation. In these situations, employing a surgical method (such as VATS and/or thoracotomy) for the removal of the foreign body would be the safest approach [50-51].

Complications

The complication rate of rigid endoscopy for either airway or esophageal FB is low (0.2%-5%) and mortality is rare (<0.1%). Postoperative complications increase for children who present with a delay in diagnosis greater than 24 hours. Delayed treatment or failed and/or improper treatment can cause complications [51]. The factors of complications in EFB patients are complex. It was shown that sharp foreign bodies and long durations have a higher incidence of complications due to mechanical damage to the mucosa. Risk factors for complications in EFBs are the younger the child, the long implication time of the foreign body, the sharp foreign body, the location of the foreign body, and the presence of fever or cough [49,50].

In young children, the lumen at the entrance of the esophagus is narrow and the oesophageal muscle layer is weak. For this reason, this patient's esophagus is more vulnerable. Sharp foreign body may cause edema and inflammation of the oesophageal mucosa, and long-term compression of the oesophageal mucosa will cause ischaemic necrosis. Esophageal endoscopy performed in a narrow and oedematous oesophageal cavity also increases the risk of mechanical damage and mucosal tearing injury. After 24 h, complication risk significantly increases regardless of the type of foreign body and other factors. All of the esophageal foreign bodies must be removed within 24 h [47-52]. The diagnosis and treatment algorithm for esophageal foreign bodies is prepared based on the literature data and our clinical experience is shown in figure 7.

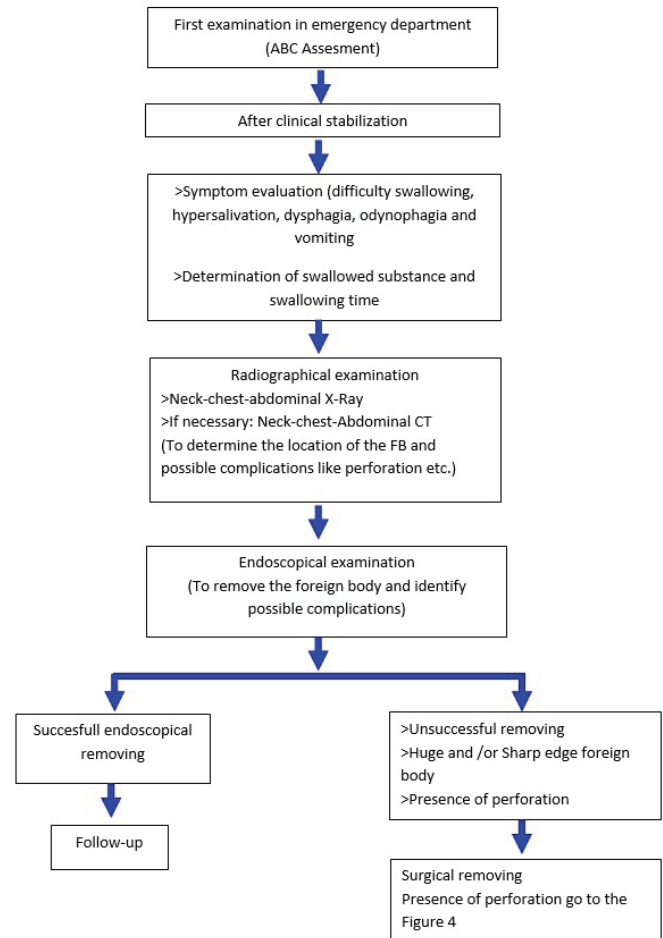


Figure 7. Diagnosis and treatment algorithm of esophageal foreign bodies.

In conclusion, pediatric esophageal emergency pathologies are conditions that require long-term multidisciplinary follow-up due to their chronic complications. The most important aspects of managing these pathologies are taking a careful history, conducting a focused physical examination, and performing screening laboratory tests. By doing so, the development of chronic complications, which make treatment most challenging, can be prevented.

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Authors' contribution

MGP,SG,Cİ,ATŞ,YB: conceived and designed the review, co-wrote the paper, collected the clinical data.

The authors discussed the paper under the literature data together and constituted the final manuscript.

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