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Review / Derleme

Surgical esophageal diseases in children

Çocuklarda cerrahi özofagus hastalıkları

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ABSTRACT

Pediatric age esophageal diseases are rare and complex clinical conditions. Treatment options should be individually determined for the patient. The advances in the follow-up and treatment process is the most important reason for the increase in survival time, particularly for congenital pediatric surgical diseases. This study aimed to evaluate the general characteristics of pediatric surgical esophageal diseases in light of the literature.

Keywords: Esophagus, pediatric, surgery.

ÖZ

Pediatrik çağ özofagus hastalıkları nadir ve kompleks klinik durumlardır. Tedavi seçeneği hastaya özel olarak belirlenmelidir. Günümüzde özellikle konjenital pediatrik cerrahi hastalıkların takip ve tedavi sürecindeki gelişmeler, hayatta kalış süresinde artışın en önemli sebebidir. Bu çalışmada, çocuklarda cerrahi özofagus hastalıklarının genel özelliklerinin literatür ışığında değerlendirilmesi amaçlandı.

Anahtar sözcükler: Özofagus, pediatrik, cerrahi.

Pediatric age esophageal diseases are rare and complex clinical conditions. Treatment options should be individually determined for the patient based on diagnosis, severity of the condition, and other concurrent medical conditions. One of the most important stages of the process is informing the patient and the patient's relatives about the possibility of multistage treatment and its duration, depending on the severity of the condition, the needs of the individual, and the characteristics of the patient. Another important point is patient follow-up, which is carried out by a multidisciplinary team specialized in this field.

Today, the advances in the follow-up and treatment process is the most important reason for the increase in survival time, particularly for congenital pediatric surgical diseases. However, this situation results in the follow-up of an increasing number of patients with morbidity in the clinic. Therefore, in recent years, the focus has shifted from reducing mortality to preventing morbidity.[1]

The most common pediatric surgical diseases in clinical practice are esophageal atresia (EA), tracheoesophageal fistulas (TEFs), esophageal duplications (EDs), esophageal strictures, achalasia, and gastroesophageal reflux (GER).

1. ESOPHAGEAL ATRESIA AND TRACHEOESOPHAGEAL FISTULAS

Both EA and TEF are the most frequently encountered pathologies among pediatric esophageal diseases. Esophageal atresia and TEF are rare conditions with an incidence of 1/2,500 to 4,500 live births.[2] The most common anomaly in babies with EA is congenital heart disease, with a frequency of 30%. Urinary system anomalies, gastrointestinal system anomalies, and neurological and skeletal system anomalies may also be seen. The most commonly used classification is made according to anatomical types made by Ashcraft and Holder,[3] who created five subgroups according to anatomical features (Table 1, Figure 1).

During the neonatal period, babies most frequently present with difficulty in feeding or respiratory distress and excessive mucus and mucus coming out from the mouth and nose, cough, choking, and regurgitation after the first feeding. In addition, in some patients,

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Table 1. Ashcraft and Holder's[3] Classification in EA and TEF

Type	Frequency %
EA and distal TEF	85.8
Isolated EA	7.8
EA and proximal TEF	0.8
Proximal EA and distal TEF	1.4
Isolated TEF	4.2

EA: Esophageal atresia; TEF: Tracheoesophageal fistulas.

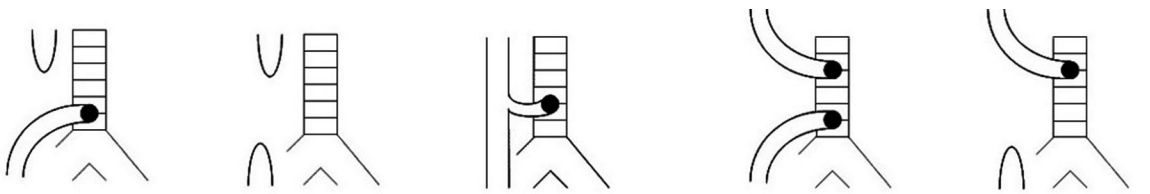
EA or TEF may be incidentally diagnosed when oral feeding is stopped for other reasons and a nasogastric tube is inserted for nutritional purposes. In cases with TEF with concomitant EA, abdominal distension may develop due to air passing into the stomach and intestines through the existing fistula. Moreover, stomach secretions use the existing fistula tract to reach the trachea and lungs, causing chemical pneumonia.[4]

During the prenatal period, polyhydramnios, the stomach with no fluid present, a small abdomen, a fetus with lower weight than expected, and an enlarged esophageal pouch in ultrasonographic evaluation should alert the clinician for EA and TEF. In the postnatal period, if the nasogastric tube cannot be advanced into the stomach and the catheter cannot be observed on the radiograph, the gas does not reach the stomach, the presence of the compression and deviation of the trachea, and the absence of gastric gas from being visible strengthens the diagnosis. The diagnosis is confirmed with contrast-enhanced radiographs using low-density radiocontrast materials. The absence of an abdominal gas shadow on radiographs indicates that there is no TEF and that the case is isolated EA. The presence of a gas shadow indicates that there is EA accompanying TEF.[5,6]

Esophageal atresia and TEF are diagnosed immediately after birth and should be urgently treated. Taking the patients to surgery under the most optimal conditions is crucial. The preoperative preparation period is as vital as the perioperative and postoperative periods. During this period, patients should be positioned by elevating the head to prevent reflux, aspiration, and chemical pneumonia. Detailed monitoring and investigation for accompanying pathologies of patients are essential. A detailed echocardiographic evaluation is necessary to assess the presence of possible heart and major vessel deformities. Additionally, a detailed renal ultrasonographic evaluation should be performed to rule out urinary system anomalies. This is important not only for potential postoperative complications but also for determining the surgical approach. Whether the patient defecates the meconium is crucial. In the presence of accompanying intestinal atresia, synchronous abdominal surgery is the best approach.[4]

In recent years, the multidisciplinary management of the pathology at every stage, advancements in intensive care conditions, developments in parenteral and enteral nutrition management, and the use of accurate and targeted antibiotic therapies have been the primary factors for reducing morbidity and mortality during and after treatment.[7,8]

Despite all accompanying anomalies, the posttreatment survival rate is reported to be 95% nowadays in EA/TEF cases. The key to successful surgical treatment in EA/TEF is performing it under the most favorable conditions. If surgery is delayed due to low birth weight, pneumonia, or major cardiac pathologies, the patient's nutrition during this period should be provided through a gastrostomy, and the proximal esophageal pouch must be drained. Surgery should be delayed until the most optimal conditions are ensured.[8]



1. Proximal atresia distal fistula 84% 2. EA without TEF 8% 3. Isolated TEF 4 4. EA double TEF 5. Distal EA proximal TEF 1%

Figure 1. Five subgroups in EA + TEF[6]
EA: Esophageal atresia; TEF: Tracheoesophageal fistulas.

The surgical approach involves closing the TEF and creating an anastomosis of the esophageal segments. This can be done through both thoracotomy and thoracoscopic methods. In recent years, thoracoscopic treatments have been prominent.[9] The first successful thoracoscopic treatments were reported in the early 2000s.[9] In 2005, the literature presented the most extensive multicenter series, where 104 infants with EA/TEF were treated thoracoscopically.[9] In this series, mortality and morbidity rates were remarkably low.

The open surgical method is applied with right posterolateral thoracotomy. The TEF is cut, primarily sutured, and closed. The proximal atretic esophageal pouch is visualized with the aid of a nasogastric tube and opened from the base. A catheter is advanced from the distal end to the stomach, and the stomach is aspirated to prepare for anastomosis. If there is a distal esophageal stricture, it is dilated at this stage. If the proximal and distal ends approach each other safely without tension, the anastomosis is completed with individually absorbable 4/0 or 5/0 sutures.[10]

In cases of isolated EA, the esophageal diverticulum at the diaphragm level often remains, and anastomosis cannot be safely performed without tension. In this case, providing parenteral nutrition through gastrostomy and waiting for three to four months solves the problem in many cases. Otherwise, anastomosis is achieved by placing weighted or unweighted bougies in the upper pouch to bring the proximal and distal ends closer together mechanically. Another method involves opening a proximal esophagostomy and moving the esophagostomy site distally every two to three weeks until the proximal and distal ends are sufficiently close to perform the anastomosis.[10-12]

In minimal invasive approach (MIA), preoperative preparation, anesthesia preparation, and patient positioning are the same as in the open method. Generally, three ports are used in MIA (the camera port [posterior axillary line of the eighth intercostal artery], two working ports [mid-axillary line of the fourth and fifth or the fourth and sixth intercostal artery]). Afterward, all surgical procedures to be performed are similar to open surgery.

The closure of TEF can be done with a metal clip or Hemolok (Teleflex Medical Research, Triangle Park, NC, USA). It is closed primarily with two Vicryl sutures (Ethicon Inc., Somerville, NJ, USA), and then the esophageal proximal and distal ends are anastomosed after they are released. During the release, maximum attention should be paid to avoid damage to the membranous trachea and other adjacent tissues. The entire anastomosis can be done with continuous sutures or the posterior wall can be completed continuously, and the anterior wall can be completed with individual sutures (4/0-5/0 polydioxanone sutures). After completing the anastomosis, a 6-French nasogastric tube is placed into the stomach, and the procedure is completed. To prevent recurrent fistulas, pleural tissue or retropleural tissues can be placed as a support between the two tissues. Anesthesia is concluded, and the baby is taken to the intensive care unit. After the surgical procedure, the patient is monitored in the intensive care unit until respiratory stabilization is achieved.[13,14]

Complications after surgical treatment are areanastomotic leaks (16%), esophageal stricture (5%), recurrent TEF (3-10%), GER (30-70%), and tracheomalacia (40%).[14-18]

2. ESOPHAGEAL DUPLICATIONS AND CONGENITAL ESOPHAGEAL STENOSIS

Gastrointestinal duplications occur with an incidence of 1 in 4,500 live births, with EDs accounting for 20% of these.[19] Esophageal duplications are often classified into three clinical morphological variants: cystic (most common), tubular, and diverticular. To be defined as a duplication clinically, the pathological structure must possess three characteristics: *(i)* a well-developed smooth muscle layer, *(ii)* an epithelial lining representing a portion of the digestive tract (with some respiratory components), and *(iii)* a connection to the esophagus. Congenital esophageal stenosis (CES) can present in three different forms in clinical practice: tracheobronchial remnants, fibromuscular stenosis, and membranous webs. Congenital stenoses due to a mural process should be distinguished from acquired esophageal stenosis, originating from causes such as GER.[19-22]

Esophageal duplications most commonly occur in the distal esophagus. Histologically, 50% exhibit an epithelium lining the interior, drawing attention to respiratory and ectopic gastric mucosa.[23] While these lesions are generally benign, malignant transformation can occur.[23]

The majority of patients with EDs are asymptomatic and are diagnosed incidentally. Symptomatic cases typically present before the age of two. Clinical manifestations of EDs vary based on anatomical localization, the compressive effect of the lesion, complications related to aspiration, and infection

of the cyst. The most common reason for seeking medical attention is obstruction secondary to the mass effect, resulting in aspiration and subsequent respiratory symptoms. Rarely, patients may present with pain, perforation, distention, hemoptysis, and gastric bleeding associated with ectopic gastric tissue that is a part of the lining epithelium.[24]

Thorax computed tomography (CT) may reveal a characteristic posterior mediastinal mass, spherical in shape, 2-10 cm in size, and an inner hyperechoic muscular layer, with hemorrhage or intestinal debris. Additionally, CT can identify relationships with adjacent organs and tissues and detect vertebral deformities if present. In children with vertebral anomalies, the presence of a neurogenic cyst should be ruled out with magnetic resonance imaging before surgery.[20,21] For asymptomatic and small-sized EDs, Technetium-99m pertechnetate scintigraphy (Meckel's scan) can be used to decide on surgery. Scintigraphy can show the ectopic gastric mucosa.

Endoscopically, understanding the relationship between the duplication, the esophageal lumen, and the gastroesophageal junction is crucial before surgery. In some cases of tubular duplications, esophagography can also determine the length of the common wall and help assess the feasibility of endoscopic treatment. A prenatal diagnosis can be made through detailed ultrasonographic evaluations. Typically, prenatal ultrasound shows a thick-walled cystic structure as a characteristic finding.[21]

For ED, indications for treatment include erosion, perforation, bleeding, the risk of cyst infection, and a small risk for malignant degeneration. Surgical intervention is the primary approach to treat ED. In asymptomatic and small-sized cysts, the most significant surgical indication is the possibility of malignant transformation.[23,24]

In recent years, the most frequently applied surgical methods are minimally invasive techniques. Thoracoscopic procedures provide excellent visualization of the cyst, ensuring a safe and easier excision. For cysts located in the cervical region, a cervical incision is used. The recommended approach is the total excision of the cyst. Marsupialization is an alternative method, but the main concerns here are the risk of recurrence and the continuation of the risk of malignant transformation.[24]

Congenital esophageal stenosis

The majority of patients with CES are often occult, asymptomatic, and well-compensated, making the incidence unclear but reported to be between 1 in 25,000 to 1 in 50,000 live births.[25,26] Based on this structural classification, CES can be categorized into three groups: fibromuscular hypertrophy, tracheobronchial remnant, and mucosal webs. MW primarily occurs in the upper or middle third of the esophagus, FMS occurs in the middle or lower third, and TBR mainly occurs in the lower third of the esophagus (Table 2).[26,27]

Patients with CES typically present with symptoms such as dysphagia and vomiting due to esophageal obstruction, often between four to 10 months of age. Other symptoms may include excess salivation, respiratory distress, regurgitation, recurrent aspiration pneumonia, the presence of an impacted foreign body, or failure to thrive.[21]

Diagnosing CES can be challenging, primarily because it needs to be differentiated from strictures secondary to conditions such as peptic stricture, caustic injury, infection, neoplasia, extrinsic compression, and achalasia. Contrast esophagography is a valuable diagnostic tool. Endoscopy allows visualization of the stricture and can exclude the presence of esophagitis through biopsies. Monitoring pH is another diagnostic method to evaluate chronic reflux and associated stricture development. Endoscopic ultrasound provides insights into the morphological structure of the stricture, aiding in treatment decisions.[21]

Table 2. Congenital esophageal stenosis classification[27]

Type	Morphologia	%
Type 1: Isolated CES-segmental type	a. Tracheobronchial remnants (TBR) b. Fibromuscular stenosis (FMS)	29.9 53.8
Type 2: Isolated CES-diaphragm type	Membranous web (MW)	16.2
Type 3: Combined lesions	a. Segmental stenosis occurring distal to EA/TEF b. Segmental stenosis occurring distal to a MW	

CES: Congenital esophageal stenosis; EA: Esophageal atresia; TEF: Tracheoesophageal fistulas.

The primary treatment for CES involves dilation or surgery, depending on the morphology of the stricture. Treatment options may vary based on CES morphology. Some experts prefer surgery for tracheobronchial remnants with cartilaginous structures, suggesting that dilation may not effectively widen these structures and could lead to a higher perforation rate. Conversely, there are publications advocating for endoscopic dilation (bougie and/or balloon), regardless of the morphological structure. Generally, dilation is considered the initial treatment option, with surgery being applied in case of complications or ineffectiveness.[25,26,28] However, balloon dilation, with radial force application, is considered both more effective and safer. In the literature, the perforation rate after dilation for CES is reported to be approximately 10%.[28]

The standard surgical procedure for CES is segmental esophagectomy and end-to-end anastomosis. Fundoplication can be added to strengthen the anastomotic site and reconstruct the lower esophageal sphincter (LES). Subsequently, the circular layer is opened, and myomectomy is applied to repair the opened muscular layer.[29,30]

As the stenotic area is typically localized in the distal one-third of the esophagus, a left thoracotomy is often performed. The procedure can also be done thoracoscopically, which has become increasingly common in recent years. Depending on how distal the lesion is, the procedure can also be performed transabdominally.

Possible complications after surgical treatments include anastomotic leakage (treated operatively and nonoperatively), anastomotic stenosis, hiatal hernia, and reflux esophagitis.[31]

3. ACHALASIA

Achalasia is a primary motility disorder characterized by the inability of the LES to relax during both swallowing and rest, accompanied by aperistalsis in the esophageal wall. Patients often present with symptoms such as dysphagia, GER, regurgitation, and chest pain.[32] The primary pathophysiology involves dysmotility resulting from a decreased number of inhibitory neurons in the esophageal myenteric plexus in the distal esophageal segment, leading to LES dysfunction.[32,33]

Approximately half of the patients have been followed for GER until an achalasia diagnosis is established, with some even undergoing surgical treatment for this reason. Upper gastrointestinal series, endoscopy, and esophageal manometry are the gold-standard diagnostic methods for achalasia. A typical "bird-beak" appearance with a dilated esophagus and retained contrast is observed on contrast esophagography (Figure 2).[33]

Lower esophageal sphincter pressure is significantly elevated at rest (>45 mmHg), and there is no decrease in LES pressure during swallowing, leading to sphincter nonrelaxation. Manometry objectively measures LES and esophageal body pressures to confirm the diagnosis. During endoscopy for achalasia diagnosis, an evaluation is also conducted for other potential esophageal pathologies. Typically, food retention is shown in the lumen without any obstructive pathology.

After the diagnosis is confirmed, the severity of the disease is assessed using the Eckardt scoring system, guiding the choice of treatment. The scoring system ranges from 0 to 12, based on four symptoms

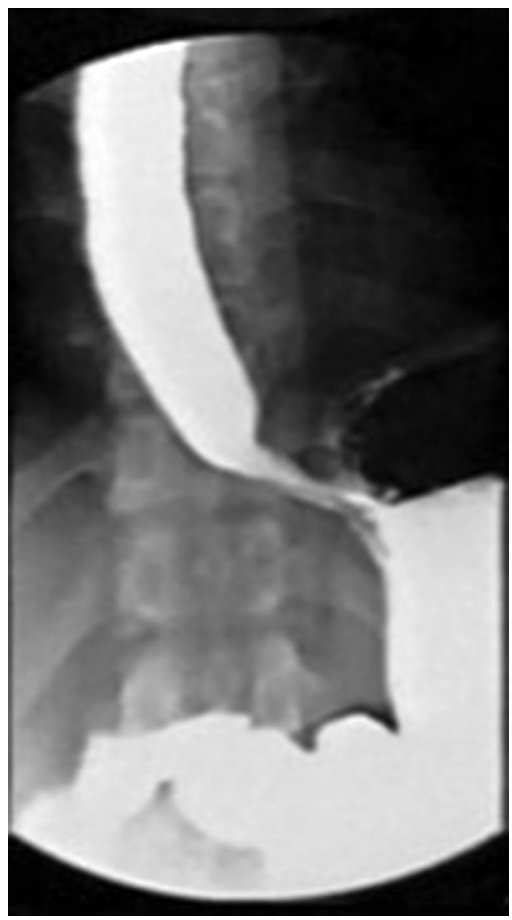


Figure 2. Typical "bird beak" appearance in achalasia on contrast esophagography.

Table 3. Eckardt scoring system for acalasia[34]

Symptom	0	1	2	3
Dysphagia	None	Occasional	Daily	With every meal
Regurgitation	None	Occasional	Daily	With every meal
Chest pain	None	Occasional	Daily	Several times a day
Weight loss (kg)	0	<5	5-10	>10

(dysphagia, regurgitation, chest pain, and weight loss; Table 3).[34]

Medical treatment for achalasia is not widely utilized due to its limited efficacy. However, in cases where endoscopic or surgical treatments cannot be applied, calcium channel blockers, nitrates, and phosphodiesterase inhibitors may be used to alleviate symptoms. The literature shows improvements in manometric and clinical findings in patients treated with these agents.[35,36]

In endoscopic approaches, the primary goal is to relax the contracted LES. This can be achieved through dilation or injection of botulinum toxin into the sphincter area. Pneumatic dilation is the preferred initial treatment method for achalasia patients due to its low complication rates and ease of application. However, the need for multiple interventions, particularly in cases requiring surgery, is a significant disadvantage. Additionally, the success rate in patients requiring multiple interventions is lower than that of surgical myotomy.[32]

Botulinum toxin injection is applied in patients for whom dilation or surgery is not suitable or surgical intervention is not preferred by the patient. Botulinum toxin A injection into the LES blocks the release of acetylcholine, a stimulating neurotransmitter, from visceral motor efferent nerve terminals. This reduces pressure in the LES and allows for esophageal emptying. Effective LES relaxation can be achieved with a minimal percentage of complications. The main disadvantage of this intervention is the need for repetition approximately every six months and the absence of a standardized dose procedure in the pediatric patient population.[33]

Peroral endoscopic myotomy (POEM), initially described by Inoue et al. in 2010, has become a frequent alternative to surgery in pediatric patients after its effectiveness in adults was published.[32] Transluminal endoscopic surgery using natural orifices and POEM have become crucial alternatives to laparoscopic Heller myotomy in recent years. In POEM, a submucosal tunnel is created endoscopically in the gastroesophageal junction, and a longitudinal myotomy is performed. A large series of pediatric achalasia patients treated with POEM report low complication rates, low recurrence, success rates exceeding 90%, and approximately a 7-point decrease in Eckardt scores. Additionally, only 10% of patients require additional intervention after POEM, and symptoms can usually be resolved with dilation.[32]

The introduction of the endoscopic functional lumen imaging probe (EndoFLIP; Medtronic, Minneapolis, MN, USA) device during POEM allows for measurable effectiveness of the procedure. This ensures an adequate myotomy. Thus, POEM has become one of the most important treatment options in pediatric achalasia.[32]

While endoscopic interventions are frequently applied and preferred as the initial choice in achalasia patients due to their effective results and low complications, surgical myotomy remains the gold standard, primarily because of its efficacy and low complication rates.[31-33]

The treatment for adult achalasia is the same as for pediatric achalasia. The myotomy, followed by fundoplication to prevent GER, is still the gold standard in treatment. Myotomy is applied longitudinally, leaving the mucosa intact on the muscle layer above the esophagus and below the cardia towards the stomach. The procedure can be performed through thoracotomy, laparotomy, or MIA. Postsurgical LES pressures of 10 mmHg predict a good long-term clinical response.[32]

Minimally invasive surgery has become the standard treatment method for achalasia in children due to lower pain, shorter length of stay, and earlier return to normal activity compared to open surgeries. As suggested by the International Pediatric Endosurgery Group, an antireflux procedure should be performed in patients after myotomy. Depending on the patient and the situation, Dor, Toupet, or Nissen fundoplication can be selected for this purpose.

Dor fundoplication is the most frequently applied fundoplication for this purpose in the literature.[32-34]

Late-onset achalasia is quite rare in pediatric patients. For these patients, esophageal resection can be performed using gastric and colonic conduits, similar to adults. The mortality rate after this procedure is reported to be 2%.[32]

There is a lack of objective data on long-term outcomes in pediatric patients after surgical treatments. However, some studies report cases of persistent dysphagia due to reasons such as incomplete myotomy, esophageal dysmotility, relative obstruction from a fundoplication, or postsurgical fibrosis in the distal esophagus.[33]

4. GASTROESOPHAGEAL REFLUX AND GASTROESOPHAGEAL REFLUX DISEASE

Gastroesophageal reflux is a physiological event that can occur many times a day without any pathology in many adults and children. It involves the transfer of gastric contents into the esophageal lumen without vomiting or regurgitation. In a study involving 948 infants, it was reported that half of the infants aged 0-3 months experienced regurgitation at least once a day. This rate increased to 67% in infants aged 4-6 months and then sharply decreased to 21% at the age of 7. Gastroesophageal reflux is the most common gastrointestinal complaint in the pediatric age group, with an incidence of approximately 75%. It most commonly occurs in infants around 4-5 months of age. In a study with a large patient series, pyrosis or heartburn was reported in 1.8% of 3-9-year-olds and 3.5% of 10-17-year-olds.[37]

Gastroesophageal reflux disease is a clinical condition that develops due to an underlying pathology. Making the differential diagnosis between these two conditions will save the patient from unnecessary tests and overtreatment. One commonly used tool to differentiate between GER and GERD is Orenstein's Infant Gastroesophageal Reflux Questionnaire (Table 4), which is a symptom-based, 11-point questionnaire with a maximum score of 25. A score >7 has 74% sensitivity and 94% specificity in diagnosing GERD in infants.[38]

Normal conditions involve a LES pressure of 12-15 mmHg during relaxation. The sphincter, which is normally closed at rest, opens in response to esophageal peristaltic waves and can remain open for 5-30 sec. In infants and young children, there are no specific symptoms that can diagnose GERD or

Table 4. Orenstein's infant gastroesophageal reflux questionnaire[38]

1	How often does the baby usually spit up?	
	• 1-3 times per day	1
	• 3-5 times per day	2
	• >5 times/day	3
2	How much does the baby usually spit up?	
	• 1 teaspoonful to 1 tables spoonful	1
	• 1 tables spoonful to 1 ounce	2
	• >1 ounce	3
3	Does the spitting up seem to be uncomfortable for the baby?	2
4	Does the baby refuse feeding even when hungry?	1
5	Does the baby have trouble gaining enough weight?	1
6	Does the baby cry a lot during or after feeding?	3
7	Do you think the baby cries or fusses more than normal?	1
8	How many hours does the baby cry or fuss each day?	
	• 1 to 3 h	1
	• >3 h	2
9	Do you think the baby hiccups more than most babies?	1
10	Does the baby have spells of arching the back?	2
11	Has the baby ever stopped breathing while awake and struggled to breathe or turned blue or purple?	6
	Total score	25

Table 5. Predisposing factors for GERD in childhood

1.	Obesity
2.	Neurological impairment, e.g. cerebral palsy
3.	Neuromuscular disease, e.g. congenital myopathy
4.	Genetic conditions, e.g. Down syndrome
5.	Repaired tracheoesophageal fistula
6.	Repaired oesophageal atresia
7.	Congenital diaphragmatic hernia
8.	Chronic lung disease, e.g. bronchopulmonary dysplasia, bronchiectasis, asthma
9.	Cystic fibrosis
10.	Scleroderma
11.	Previous oesophageal caustic injury
12.	Significant prematurity
13.	Strong family history of GERD, Barrett esophagus or esophageal adenocarcinoma

GERD: Gastroesophageal reflux disease.

evaluate the response to treatment. In older children, symptoms such as vomiting, epigastric pain and dyspepsia, anemia, failure to thrive, and strictures may occur. Airway problems, such as laryngeal irritation, chronic cough, or recurrent pneumonia, can also be observed. Patients with GERD may frequently experience reactive airway disease. Asthma attacks may even occur in these patients due to reflux.[39]

In studies, some factors that could be predisposing for GERD in childhood were identified (Table 5).[39]

According to the recommendations of the North American and European societies, contrast esophagography should not be used for GERD diagnosis due to its high rate of false positives.[40] Continuous pH monitoring is considered the most accurate test for GERD. A scoring method is used in pH monitoring, including the number of pH drops (<4 and lasting >15 sec), the time required for pH to return to normal (clearance), and the number of reflux episodes. If the clearance time is more than 5 min, GERD is considered.

Clinical symptoms and pH monitoring results often do not parallel each other. Monitoring pH in children requires hospitalization, is invasive, and can be challenging, particularly in complex cases; pH studies cannot detect alkaline or neutral reflux. However, these limitations can be addressed by adding multichannel intraluminal impedance studies to the procedure. Combined multichannel intraluminal impedance/pH studies correlate better with clinical symptoms and provide better information about the prognosis of the disease. Combined multichannel intraluminal impedance/pH studies are more commonly used and have higher sensitivity and specificity in the diagnosis and monitoring of the disease.[40-42]

According to the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition and the European Society for Pediatric Gastroenterology consensus,[43] diagnostic methods include history and physical examination, esophageal pH monitoring, combined motility studies, endoscopy and biopsy, barium contrast radiography, nuclear scintigraphy, esophageal and gastric ultrasonography, tests on fluids from the ear, lung, and esophagus, and empiric trials of acid suppression. Considered treatment modalities include lifestyle changes, pharmacologic therapies (histamine type-2 (H2) receptor antagonists, proton pump inhibitors, and prokinetic agents), and surgical therapy. This guideline emphasizes starting the evaluation of patients from the most accurate point, avoiding unnecessary tests, using appropriate diagnostic methods, and ultimately selecting the correct treatment method.[39] In 14 randomized controlled trials reported in the literature, this dietary approach has been shown to prevent regurgitation, significantly reduce vomiting attacks during the day, and contribute to weight gain in patients.[44]

Pharmacological treatment includes prokinetic and motility-regulating agents, as well as antiacid therapy. The most commonly used agent is the H2 receptor blocker ranitidine (6-8 mg/kg/day). A Cochrane review published in 2014 supports the use of proton pump inhibitors (PPIs; omeprazole at 0.7-3.5 mg/kg/day).[45] Furthermore, these studies indicate that PPIs and histamine antagonists moderately improve symptoms, pH indices, and endoscopic/histological evaluations.[45]

The use of these agents, particularly ranitidine for 12 months, has demonstrated a 60-70% improvement, while omeprazole has shown a 90-100% improvement in both symptomatic and endoscopic findings (e.g., erosive esophagitis).[46,47]

In cases where medical and conservative treatments fail to improve symptoms, surgical fundoplication is indicated. Recent studies have shown a decrease in interest in fundoplication due to the significant improvement of symptoms with PPIs. A meta-analysis published in 2014 and another in 2015 found that laparoscopic fundoplication and long-term PPI therapy were equally successful.[48,49] Algoritm of GERD diagnosis and treatment shown in Figure 3.

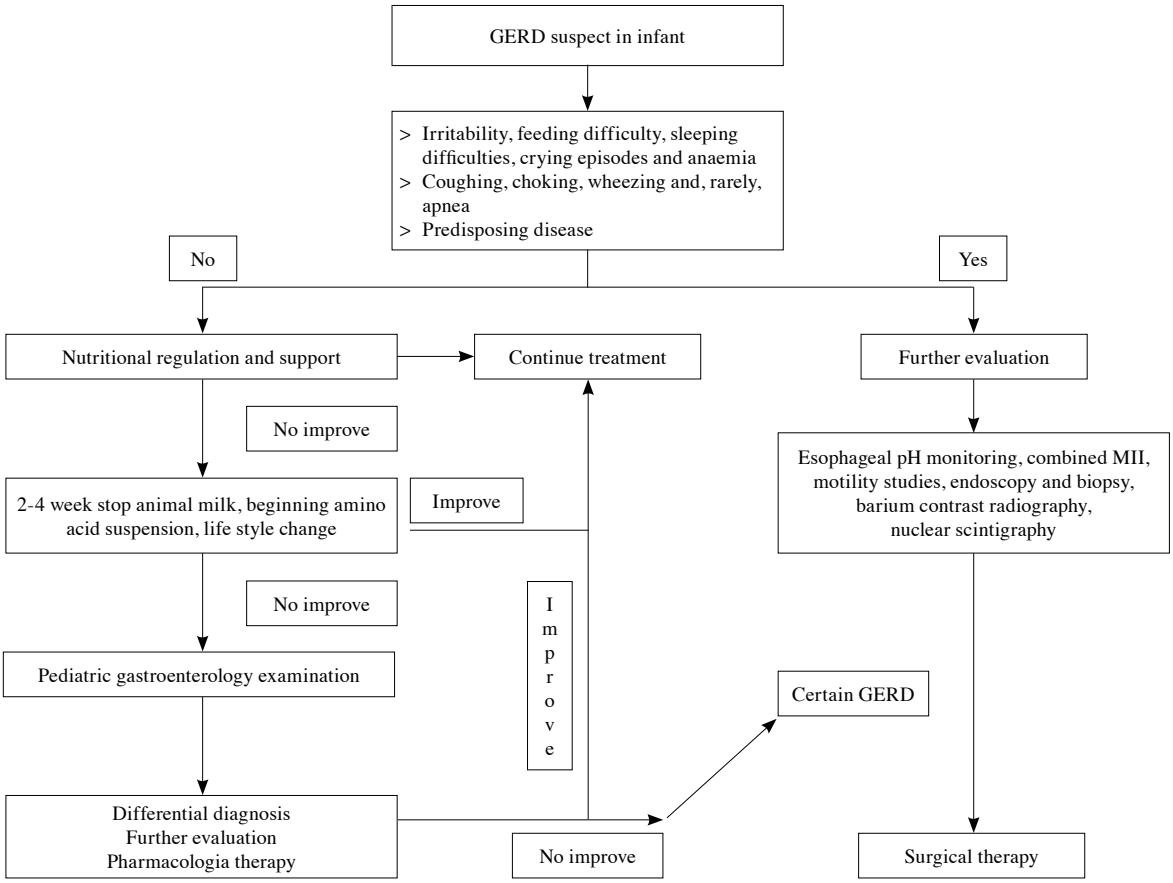


Figure 3. Algoritm of GERD diagnosis and treatment.
GERD: Gastroesophageal reflux disease; MII: Multichannel intraluminal impedance.

In a study comparing 33,355 cases, including 5,392 laparoscopic fundoplications, where laparoscopic and open fundoplications were performed, the open group had a higher incidence of fact decubitus ulcers and sepsis. Hospital mortality, length of stay, and charges were less in the laparoscopic group.[50] In a multicentric meta-analysis comparing 7,083 patients who underwent fundoplication (50% laparoscopic and 50% open), it was reported that the laparoscopic group had lower costs and a decreased rate of infectious and other surgical complications.[51]

A retrospective multicentric meta-analysis with an mean follow-up period of 30 months, focusing on patients who underwent partial and total fundoplications, revealed a higher reoperation rate in the partial fundoplication group. In contrast, patients who underwent total fundoplication had a higher need for postoperative dilatation. Postoperative antireflux treatment needs were found to be equal in both patient groups.[52]

The most common complication after fundoplication is recurrent GERD related to transhiatal herniation, with an incidence reported in the literature ranging from 5-15%. Preventative measures include minimizing esophageal dissection and applying esophagogastric fixation after fundoplication.[53]

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: All authors contributed equally to the article.

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