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Evaluation of Esophageal Motility in Children with Esophageal Damage Due to Corrosive Substance Ingestion

Koroziv Madde İçilmesi Sonucu Özofagus Hasarı Gelişen Çocuklarda Özofagus Motilitesinin Değerlendirilmesi

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Abstract: Ingestion of corrosive substances is a significant problem causing serious morbidity and mortality among household accidents. Esophageal burns after caustic substance ingestion are a common complication in children and can lead to serious motor dysfunction in the long term. The aim of this study is to evaluate changes in esophageal motility and permanent functional impairments years after the acute phase in children who have suffered severe esophageal burns and developed strictures due to corrosive substance ingestion. Twelve patients aged 5-18 years who were treated for corrosive substance injury at the Department of Pediatric Surgery, Eskişehir Osmangazi University Faculty of Medicine between 2005 and 2010 underwent esophageal manometric studies. Peristaltic activities were evaluated by measuring the amplitude, duration, and speed of esophageal contractions. In severe corrosive burns, peristaltic contractions in the esophagus were found to be significantly weaker than normal, contraction amplitudes were significantly lower ($p<0.001$), contraction durations were significantly shortened ($p<0.01$), there was no significant difference in contraction velocities ($p>0.05$), and the percentage of non-conducted contractions was significantly increased compared to reference value ($p<0.001$). In severe corrosive burns, permanent impairments in esophageal motility develop due to damage to the muscle layer and myenteric plexus. This should be considered in the management of complications such as gastroesophageal reflux and resistance to dilation therapy that may develop in the long term.

Keywords: Dysphagia, Corrosive esophagitis, Esophageal motility, Manometry, Esophageal stricture

Ethics Committee Approval: Approval for the study was obtained from the local ethics committee (approval no: 11, dated 28.02.2011).

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Özet: Koroziv madde alımı, ev kazaları içinde ciddi morbidite ve mortaliteye neden olan önemli bir sorundur. Çocuklarda kostik madde alımı sonrası özofagus yanığı sık görülen bir komplikasyondur. Uzun dönemde ciddi motor disfonksiyona yol açabilmektedir. Bu çalışmanın amacı, koroziv madde içimine bağlı ciddi özofagus yanığı geçiren ve striktür gelişen çocuklarda, akut dönemden yıllar sonra özofagus motilitesindeki değişiklikleri ve kalıcı fonksiyonel bozuklukları değerlendirmektir. 2005-2010 yılları arasında Eskişehir Osmangazi Üniversitesi Tıp Fakültesi Çocuk Cerrahisi Anabilim Dalı'nda koroziv madde hasarlanması nedeniyle tedavi gören 5-18 yaş arası 12 hasta özofageal manometrik çalışmaya tabi tutulmuştur. Özofageal kontraksiyonların amplitüdü, süresi ve hızı ölçülerek peristaltik aktiviteler değerlendirilmiştir. Özofagusta peristaltik kontraksiyonların referans değerlerine göre ileri düzeyde zayıfladığı, kontraksiyon amplitüdlerinin ileri derecede düşük olduğu ($p<0.001$), kontraksiyon sürelerinin belirgin olarak kısaldığı ($p<0.01$), kontraksiyon hızlarında anlamlı farklılık oluşmadığı ($p>0.05$) ve iletilmeyen kontraksiyon yüzdelerinin referans değerlerine göre ileri derecede arttığı ($p<0.001$) saptanmıştır. Ciddi koroziv yanıklarda, kas tabakası ve miyenterik pleksus hasarına bağlı olarak özofagus motilitesinde kalıcı bozukluklar gelişmektedir. Bu durum, uzun dönemde gelişebilecek gastroözofageal reflü ve dilatasyon tedavisine direnç gibi komplikasyonların yönetiminde dikkate alınmalıdır.

Anahtar Kelimeler: Yutma güçlüğü, Koroziv özofajit, Özofagus motilitesi, Manometri, Özofagus striktürü

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1. Introduction

Accidental ingestion of corrosive substances is a common occurrence in pediatric surgery practice. Esophageal damage develops in approximately 20–40% of corrosive substance ingestions. These injuries, which occur accidentally in children and frequently as a result of suicide attempts in adolescents, can lead to life-threatening complications in the acute phase and serious sequelae that impair quality of life in the chronic phase (1).

Various changes in motor function can occur following esophageal damage. Esophageal manometry is the gold standard for evaluating esophageal motor activity (2). This method allows for the quantitative assessment of esophageal pressures, peristaltic coordination, and motility (2).

Alkaline caustic injury to the esophagus proceeds through three overlapping phases. In the acute phase (first 1–5 days), liquefactive necrosis destroys the mucosa and submucosa, with inflammation extending into the muscle layer in proportion to burn severity, accompanied by vascular thrombosis and, in severe cases, bacterial contamination. A reparative phase then begins around the fifth day, during which tissue edema resolves and neovascularization begins; mucosal re-epithelialization is typically complete within three weeks, but the smooth muscle layer, once destroyed, does not regenerate. Continuity of the esophageal wall is instead restored by fibroblastic proliferation and cross-linking of collagen molecules. Over the following months, progressive collagen deposition produces a retracting, fibrotic stricture and longitudinal shortening of the esophagus; collagen deposition peaks within the first three months and gradually declines toward normal tissue levels over approximately two years, after which the scar is considered stabilized (3). Although the treatment and outcomes of strictures developing after corrosive esophageal injury are well known, the degree and characteristics of long-term motility disorders in these patients are not fully understood.

Because the myenteric plexus is anatomically located between the circular and longitudinal muscle layers, transmural caustic injury damages not only the contractile muscle mass but also the intramural neural network responsible for initiating and coordinating peristaltic contractions; permanent damage to this esophageal plexus in severe, full-thickness caustic burns has been specifically described in the literature. Where injury is severe and circumferential, this combined loss of muscle and myenteric neurons is thought to underlie the persistence of motility impairment even after anatomically successful dilation therapy. Assessment of esophageal motility in children is technically demanding, and normative pediatric manometric data remain comparatively limited. Early studies established normal esophageal motor values in infants and young children (4,5), and subsequent work specifically examined motility changes after caustic esophageal burns in children, describing both acute near-complete loss of motility and, in severe cases, its persistence into the late period (6). More recently, standardized protocols for pediatric high-resolution manometry (HRM) have been developed and endorsed in a joint consensus document by the American Neurogastroenterology and Motility Society and the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition (7) reflecting an evolving standard for esophageal motility assessment in children, although its adoption of Chicago Classification criteria in pediatric populations still requires cautious interpretation (8).

This study aimed to evaluate the changes in esophageal contraction amplitude, duration, and rate in the long term following corrosive substance ingestion.

2. Materials and Methods

This study was designed as a descriptive cross-sectional study aimed at evaluating esophageal motility in children who developed esophageal injury following caustic substance ingestion. Ethical approval was obtained from the Ethics Committee of Eskişehir Osmangazi University Faculty of Medicine (decision dated 28.02.2011, No: 11). Prior to participation, all patients and their parents were informed about the study, and written informed consent was obtained.

Patients treated for caustic esophageal injury between 2005 and 2010 were screened for eligibility. Pediatric patients who had undergone dilation therapy for caustic esophageal injury within the previous five years were included in the study. Only patients who had completed their dilation program and had no active swallowing or feeding complaints at the time of evaluation were enrolled. Manometric measurements could not be performed in three patients due to poor cooperation during catheter placement; therefore, these patients were excluded from the study. As a result, 12 patients were included in the final analysis.

Clinical data were retrospectively obtained from medical records. Clinical characteristics of the patients were evaluated based on the available retrospective data. All patients had a history of severe corrosive esophageal injury leading to stricture formation requiring dilatation therapy. Although exact burn grading was not consistently documented, the need for repeated dilatation was considered indicative of clinically significant injury. Dilatation procedures were performed according to a standardized protocol, typically initiated at approximately 3-week intervals and extended up to 9 weeks depending on clinical response. Detailed patient-specific data regarding stricture length, exact localization, number of dilatations, and interval between sessions were not uniformly available. No standardized record of surgical intervention was present in all cases. Esophageal manometry was performed in the late period following injury, ranging approximately between 2 to 5 years after the corrosive event.

Patients were invited to the outpatient clinic one week prior to the procedure and were informed about the examination. They were instructed to fast for at least 8 hours before the procedure. The study was performed using the MMS Urodynamics System (Enschede, The Netherlands) and Version 8.17 Medical Measurement Systems (MMS) database. Pressure channels were calibrated against a known reference pressure immediately before each study, and catheter patency was confirmed by occluding each channel individually and observing a pressure rise above 300 mmHg, with care taken to ensure no residual air remained in the perfusion tubing. The water-perfusion system was operated at an infusion rate of 0.1–0.25 mL/min per channel. An 8 Fr esophageal motility catheter (GIM 6000E00) was then inserted transnasally to a depth of approximately 45–50 cm, and patients were placed in the supine position. Using intragastric pressure as a reference, the lower esophageal sphincter (LES) was localized and the LES pressure was measured three times as part of the sphincter localization protocol. A total of 10 wet swallows were performed using 5 mL of room-temperature water at 30-second intervals, and the esophageal body response was evaluated. The catheter was then gradually withdrawn to identify the upper esophageal sphincter (UES), and the resting pressure was measured. LES and UES pressures were used to confirm correct catheter positioning during the study; as these values were not systematically tabulated for statistical analysis in the original dataset, they are not reported as outcome parameters in this manuscript.

As no concurrent healthy pediatric control group was studied, reference values were obtained from Genç and Mutaf (2002), which most closely matched the present cohort in terms of patient age range, etiology (caustic esophageal injury), population, and conventional water-perfused manometry methodology (6).

Statistical Analysis: All statistical analyses were performed using SPSS version 15.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as numbers and percentages. Normally distributed parameters were compared against the reference value using a one-sample t-test, and non-normally distributed parameters were compared using a nonparametric test, consistent with the original thesis on which this manuscript is based. Because raw individual-level data from the reference studies were not available to the present authors, the exact nonparametric procedure could not be independently re-verified.

3. Results

The mean age of the patients included in the study was 11.33 ± 3.34 years. The following manometric parameters were assessed: esophageal contraction amplitude (ECA), esophageal contraction duration (ECD), esophageal contraction velocity (ECV), peristaltic contractions (PC), ineffective contractions (IC), and failed contractions (FC) (Table 1 and Table 2).

The mean ECA value was 67.00 ± 21.35 mmHg, the mean ECD value was 2.94 ± 0.83 s, and the mean ECV value was 3.36 ± 1.67 cm/s. The percentage of PC was 50.00 ± 29.21%, the percentage of IC was 4.50 ± 12.77%, and the percentage of FC was 25.92 ± 23.54% (Table 3).

When ECA and FC values were compared with those of the reference values, a statistically significant difference was observed ($p < 0.001$). A significant difference was also found in ECD values ($p < 0.01$). PC values showed a statistically significant difference among patients ($p < 0.001$). There was no statistically significant difference between the study group and the reference value in terms of IC and ECV values ($p > 0.05$).

Table 1. Patient ECA, ECD and ECV values and reference ranges (reference values for ECA and ECD from Castell & Richter (9); ECV reference value from Genç and Mutaf (2002) (6).

Patient	ECA (mmHg)	Reference value	ECD (s)	Reference value	ECV (cm/s)	Reference value
H1	43	30-160	2.8	2-6	5.1	3.4±0.5
H2	70	30-160	2.7	2-6	2.4	3.4±0.5
H3	108	30-160	4.1	2-6	2.8	3.4±0.5
H4	50	30-160	2.5	2-6	4.9	3.4±0.5
H5	73	30-160	3.5	2-6	3.8	3.4±0.5
H6	99	30-160	3.7	2-6	5.7	3.4±0.5
H7	75	30-160	2.3	2-6	2.1	3.4±0.5
H8	47	30-160	3.3	2-6	1.2	3.4±0.5
H9	44	30-160	2.3	2-6	1.8	3.4±0.5
H10	51	30-160	1.1	2-6	1.3	3.4±0.5
H11	79	30-160	3.8	2-6	5.7	3.4±0.5
H12	65	30-160	3.2	2-6	3.5	3.4±0.5

ECA: Esophageal contraction amplitude; ECD: Esophageal contraction duration; ECV: Esophageal contraction velocity

Table 2. Patient PC, IC and FC values and reference values (6).

Patient	PC (%)	Reference value (%)	IC (%)	Reference value (%)	FC (%)	Reference value (%)
H1	22	80-100	0	0	56	0-20
H2	67	80-100	0	0	11	0-20
H3	30	80-100	0	0	70	0-20
H4	30	80-100	0	0	60	0-20
H5	64	80-100	0	0	27	0-20
H6	88	80-100	0	0	12	0-20
H7	70	80-100	0	0	20	0-20
H8	25	80-100	0	0	25	0-20
H9	40	80-100	10	0	20	0-20
H10	0	80-100	44	0	0	0-20
H11	92	80-100	0	0	0	0-20
H12	72	80-100	0	0	10	0-20

PC: Peristaltic contractions; IC: Contractions with progressively deteriorating amplitude across sequential swallows (a conventional-manometry descriptive category, distinct from the Chicago Classification's diagnostic term "ineffective esophageal motility"); FC: Failed contractions.

Table 3. Statistical analysis results

Parameter	Mean ± SD	p-value
ECA (mmHg)	67.00±21.35	p<0.001
ECD(s)	2.94±0.83	p<0.01
ECV (cm/s)	3.36±1.67	p>0.05
PC (%)	50.00±29.21	p<0.001
IC (%)	4.50±12.77	p>0.05
FC (%)	25.92±23.54	p<0.001

ECA: Esophageal contraction amplitude; ECD: Esophageal contraction duration; ECV: Esophageal contraction velocity; PC: Peristaltic contractions; IC: Ineffective contractions; FC: Failed contractions; SD: Standard deviation

4. Discussion and Conclusion

Esophageal burns resulting from caustic substance ingestion in the pediatric age group remain an important cause of morbidity, mortality, and healthcare expenditure. In our country, esophageal burns due to caustic substance ingestion are encountered more frequently than in developed countries (10).

Studies indicate that quality of life after caustic injury should be evaluated in a multidimensional manner. In addition to the degree of stricture contributing to post-burn morbidity, the severity of the associated motility disorder has also gained importance. Reviews emphasize that caustic strictures are more resistant to treatment than other

benign strictures (for example, anastomotic strictures), and that one of the main mechanisms underlying this resistance is the loss of motility (11).

In children who ingest caustic substances, the swallowing reflex is usually initiated before the child recognizes the caustic taste of the substance in the mouth, allowing several milliliters of the corrosive liquid to pass into the esophagus. Due to the strong muscular structure of the pharynx and cervical esophagus, the ingested substance moves more rapidly through these segments; therefore, the first deep burn typically occurs at the beginning of the thoracic esophagus, at the level of the sternal notch (12).

In caustic injuries, the inflammatory and fibrotic processes may lead to structural alterations in the esophageal wall, resulting in motility disorders. This condition is particularly characterized by a reduction or loss of esophageal peristalsis (6). In our study, a 50% reduction in peristaltic activity suggests a marked decrease in the active peristaltic function of the esophagus. This condition may contribute to the development of resistant strictures, particularly in patients undergoing dilation therapy. In the literature, it is emphasized that, in cases with resistant stricture formation, careful evaluation of patient characteristics and response to dilation is essential in determining the optimal treatment strategy (13).

In our study, the 12 patients examined had ingested NaOH at varying concentrations. Following acute management, the patients who underwent dilation therapy completed their treatment and were able to maintain their daily activities. The time elapsed since the completion of acute treatment ranged from 2 to 5 years. One of the notable features of this study is the long-term manometric evaluation of esophageal motility following corrosive esophageal injury.

In the manometric study conducted by Genç and Mutaf in pediatric patients, it was reported that significant impairments in esophageal motility occur both in the acute and late phases following corrosive esophageal injury, and that these impairments may be associated with the prognosis of the disease (6). In their study, a decrease and irregularity in peristaltic activity were particularly notable, which was thought to be related to inflammation and fibrosis developing in the esophageal wall. Similarly, in our study, peristaltic activity was found to be reduced by approximately 50%, supporting the notion that corrosive injury may have lasting effects on esophageal motor functions in the long term.

In the acute phase of caustic esophageal burns, almost complete loss of motility occurs. During the recovery period, motility impairment resolves in cases with mild burns, whereas in patients with severe burns, normal esophageal motility does not return despite a regular and successful dilation program (6). Impairment of esophageal motility may result from the development of fibrotic structures, shortening of the esophagus, and a reduction in the number of neurons in the myenteric plexus (6). The mechanism underlying persistent motility impairment in severe caustic injury is thought to involve both the smooth muscle layer and the myenteric plexus. As the muscle undergoes necrosis and is replaced by non-contractile fibrotic tissue, the intramural neurons of the myenteric plexus, positioned between the circular and longitudinal muscle layers, are damaged concurrently, since they lie within the same zone of transmural injury; permanent damage to this esophageal plexus has been specifically described in severe caustic injury. This combined loss of contractile tissue and coordinating neural circuitry offers a plausible explanation for why motility failed to normalize in our cohort despite years of follow-up and successful anatomical dilation: dilation restores luminal caliber but does not restore destroyed muscle or regenerate lost enteric neurons (14). Excessive dilation may also lead to tissue damage and dysmotility (11). In our cases, the detection of long-term motility impairment is consistent with these findings.

A notable feature of our findings is that contraction velocity (ECV) did not differ significantly from reference values, despite markedly reduced amplitude (ECA) and a markedly reduced percentage of peristaltic contractions (PC). Amplitude reflects the contractile force generated by the smooth muscle mass at a given esophageal level, and would therefore be expected to fall in proportion to muscle loss and fibrotic replacement. Propagation velocity, by contrast, is governed largely by the latency gradient of inhibitory innervation along the myenteric plexus, which times the sequential activation of successive esophageal segments, rather than by the force each segment is able to generate. A plausible interpretation is that, in our cohort, all long-term survivors who had completed successful dilation therapy, enough of the neural circuitry responsible for timing peristaltic propagation remained intact to preserve broadly normal

conduction velocity in those contractions that were actually transmitted, even though the contractile mass itself, and hence the proportion of swallows producing an effective, adequately propagated contraction, was substantially diminished. This would be consistent with a pattern in which muscle loss and fibrosis are more extensive, or more consistently present, than complete destruction of the coordinating neural network. This interpretation remains indirect, as conventional manometry does not provide the segmental, topographic detail needed to localize propagation failure precisely; high-resolution manometry would be better placed to test it directly (15,16).

Da-Costa-Pinto and colleagues reported lower amplitude and longer contraction duration in their patients (12). In our study, the ECA values were found to be significantly lower compared to the reference values ($p < 0.001$). A marked difference was also observed in the ECD values ($p < 0.01$).

Torres and colleagues did not find a correlation between radiological and manometric findings in seven patients with chronic caustic esophagitis. In contrast, Cadranel and colleagues reported significant impairment in all clinical, anatomical, and functional parameters during the acute and subacute phases of the disease. The same authors also observed good concordance between esophageal transit, manometric findings, and clinical symptoms throughout the chronic phase, with follow-up periods ranging from 3 months to 7 years (17,18). In our cases, non-transmitted contractions were found to be significantly higher compared to the reference value ($p<0.001$).

In our cases, the percentages of peristaltic contractions in response to swallowing were found to be significantly lower compared to children of the same age in the reference value for children of the same age ($p<0.001$). This finding is consistent with manometric studies conducted in patients with caustic esophageal burns, which have reported near-complete loss of peristalsis and low-amplitude waves in cases with severe burns (19).

Although this study used conventional water-perfused manometry rather than high-resolution manometry (HRM), it is worth relating our findings to current concepts from the Chicago Classification, now in its fourth version (v4.0), which provides a standardized framework for categorizing esophageal motility disorders based on HRM-derived metrics such as the distal contractile integral and integrated relaxation pressure. Within that framework, our findings, reduced contraction amplitude, a reduced proportion of effectively peristaltic swallows, and an increased proportion of non-conducted contractions, broadly parallel the hypomotility spectrum described by Chicago Classification v4.0 as ineffective esophageal motility and absent contractility. However, these are formally HRM-based diagnostic categories defined by specific pressure-topography thresholds, and cannot be directly assigned from conventional manometry data such as ours. Future prospective studies in this population using HRM and Chicago Classification v4.0 criteria would allow more precise, segment-level characterization of the motility pattern described here (8,20).

This study has certain limitations. Although the reference values used were derived from the pediatric study most closely matching our cohort in age, etiology, and methodology, they do not constitute a concurrently studied, matched control group using an identical catheter and software. Throughout the study period, conventional manometry was the standard technique available in our clinic. While high-resolution manometry provides superior topographic assessment, it was not available at our center at that time. The small sample size and the single-center design may limit the generalizability of the results. Additionally, clinical findings and acute-phase data related to corrosive substance ingestion were not recorded in a standardized manner for all patients, which restricts the evaluation. Furthermore, exposure characteristics such as concentration, volume, and contact time were not consistently or systematically documented and could not be reliably retrieved. Similarly, detailed patient-specific clinical parameters (e.g., stricture length, localization, and dilatation characteristics) were not uniformly available due to the retrospective design. Nevertheless, one of the strengths of this study is the long-term manometric assessment of esophageal motility in children who have experienced corrosive esophageal injury.

In this study, long-term (2–5 years) manometric evaluations in children who developed esophageal injury due to ingestion of corrosive substances were found to be consistent with the existing literature. Peristaltic contractions in the esophagus were markedly weakened compared to reference values, contraction amplitudes were significantly reduced, contraction durations were notably shortened, and the percentage of non-transmitted contractions was considerably increased. No significant differences were observed in contraction velocities.

In light of these findings, it is evident that motility disorders that occur in the acute phase of severe esophageal burns persist in the long term and may

even become permanent. Unless preventive measures are implemented to reduce the incidence, caustic esophageal burns will continue to be an important cause of morbidity, mortality, and healthcare expenditure in our country.

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