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Research Article

**ESOPHAGEAL INJURIES AND STRICTURE FORMATION
FOLLOWING CORROSIVE INTAKE**¹Dr. Khadija, ²Dr. Seher Anwar, ³Dr. Hunza Malik¹Nawaz Shareef Hospital, ² Holy Family Hospital, ³Jinnah Hospital.**Article Received:** December 2018**Accepted:** February 2019**Published:** March 2019**Abstract:**

This study is conducted to discuss the effect of corrosive intake lead to the esophageal injuries and strictures.

Method: *This is the cross-sectional study, where 192 patients with history of corrosive intake lead to esophageal injuries and strictures were selected. These patients were examined during first 48 hours of corrosive intake by the upper GI endoscopy and the same procedure was carried out after 6 weeks in order to check the strictures formation.*

Result: *In the concerned data 101 patients have severe esophageal injuries following the corrosive intake; on the other hand, 91 patients have mild injuries. Among the 101 severely injured patients 31 developed the strictures in the esophagus due to corrosive intake in comparison to the 9 patients of mildly injured esophagus developed the strictures. This difference was statistically significant.*

Conclusion: *This study concluded that the patients with lethal esophageal injuries have higher frequency of developing strictures as compared to patient with mild esophageal injury.*

Keywords: *Corrosive, alkali, acid, stricture.*

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INTRODUCTION:

Corrosive intake is the lethal medical problem which is associated with serious complications i.e. perforation, bleeding, strictures and systemic complications (hepatic failure, renal insufficiency and DIC) gastric outlet obstruction, fistulas and cancer [3]. In the children of age below than 10years intake happened accidentally whereas in the case of adult intake occurred intentionally. [3]

The alkali results in the liquefaction necrosis which penetrates deeply in the body tissues [4]. The injury caused by the alkali lasts for three or four days and this injury is associated with the ulceration, vascular thrombosis, and mucosal inflammation and sloughing of the tissues [5]. The injury caused by alkali is far more lethal as compare to the acid [6]. The acid results in the superficial coagulation and vascular thrombosis of the effective area of body. Acid injury results in the formation of protective Escher which prevents the further penetration of the acid deep into the tissues. Moreover, the acid produces severe pain in the pharynx so this result in the reduction in the consumption amount of an acid [7,8].

The single most important test to diagnose the extent and level of injury occurred by the ingestion of acid and alkali is the upper gastrointestinal endoscopy [9]. This should be carried out in the first 24 hours to evaluate the nature of injury and to predict the clinical outcome and the prognosis in this case. Moreover, it's also predict the medication should be used to reduce the hyperemia and swelling in the tract [10].

The grading system is also used to evaluate the extend of the injury, stricture formation and the level of malnutrition following the corrosive intake injury [11].

PATIENTS AND METHODS:

This study was conducted in the gastroentology department of holy family hospital from July 2016 to august 2016. During the study 192 patients with the history of corrosive intake was selected. All the patients had positive clinical finding i.e. severe muscol edema, vomiting, drooling, hematemesis, oropharyngeal fibrosis, hyperemia and respiratory distress. Moreover these cases had undergone the upper GI endoscopy within first 48hours of intake in order to grade and evaluate the nature of injury. The patients who were clinically unstable due to severe laryngeal edema, hemorrhage, respiratory distress, necrosis and perforation were excluded from the study. The patients who were asymptomatic or in

which upper GI endoscopy was contraindicated were also no selected for this study. All the patients were fully informed about the procedure and written consent had been taken from them. Upper GI endoscopy was done during the first 48hours of intake in order to access the magnitude and extent of injury according to the grading system of Di Costanza. This grading system is as follow: grade 0 = normal, grade 1 = muscol edema and hyperemia, grade 2A = bleeding and superficial ulcer, grade 2B = deep ulcers, grade 3= scattered necrotic area with black or brown mucosa.

The upper GI endoscopy was done under the local anesthesia with the fiberoptic Pentax LH-150PC (Japan) endoscope under the supervision of senior consultants. Injection Co-amoxiclav and ranitidine were given before the procedure to all the patients. Intravenous antibiotic and H2 receptor blockers were stopped in the patients with grade 0 and grade 1 injuries. These patients were discharged on the oral medication after the endoscopy had been done. The patients with grade 2 and grade 3 injuries were given the injectable antibiotics and H2 receptor blocker for 2 weeks. All the patients with grade 2 and grade 3 esophageal injuries without severe gastritis were fed through nasogastric tube. Whereas the patients with severe gastritis were kept on total parenteral nutrition for the period of 2 weeks. The upper GI endoscopy was repeated after 6 weeks to rule out any stricture formation.

RESULTS:

More female patients were reported as compare to the male. 42 were male and remaining 150 were female. Mean age of the patients were 26.09 years. About 31 patients had normal esophagus on the upper GI endoscopy. In remaining 161 patients some degree of esophageal damage were noted on endoscopy. Grade 1 esophageal injury was the commonest. Grade 2a, 2b and 3 evident on endoscopy done within first 48 hours regarded as a severe esophageal injury. In 31 patients with grade 0 esophageal injury not a single case of strictures was observed. While in 69 patients with grade 1 esophageal injuries, 9 patients developed esophageal strictures. Among 51 patients with grade 2a injury, 15 cases of esophageal strictures were noted. Among 25 patients with grade 2b esophageal injury, 16 patients developed strictures. While in remaining 16 patients with grade 3 injury, 12 cases of strictures were noted. Hence, the strictures formation was directly proportion to the grade of esophageal injuries.

Table 1: Grade of injuries

Grade	No
Grade 0	31
Grade 1	69
Grade 2a	51
Grade 2b	25
Grade 3	16

Table 2: Esophageal stricture among different grade of injury

Grade	Stricture
Grade 0	0
Grade 1	9
Grade 2a	15
Grade 2b	16
Grade 3	12
Total	52

DISCUSSION:

The patients with high grade esophageal injuries are more prone to develop esophageal strictures. In the study by Lucky et al, the incidence of corrosive intake is higher in adult during the third decades of life while in the children; the incidence rate is higher during the first 5 years of life. The most commonly used corrosive is caustic soda (40%). The mean age of patients is 23.9. The most common complication is esophageal strictures. About 50% cases of corrosive intake were due to suicidal attempt which is more common among adult and 9% cases were by the accidental ingestion which is common in children. Most common complication was odynophagia (30%) with the mortality was 10%. The result differs from our studies because acid ingestion is far more common in developing countries, where sulphuric acid is available in toilet cleanser, and it is available in almost every home. In the our study more than 50% ingestion of corrosive was due to suicidal attempt because of domestic stress, marital conflicts, any death in family, mental illness, physical illness and educational stress. In case of children accidental ingestion happened because they have well developed skills to identify and drink the liquids without knowing whether it is harmful or not.

In above mentioned study the esophageal strictures developed in.... whereas in Nigerian study [12] short segment esophageal strictures were evident in the 40% of patients. Results of various studies conducted in different setup showed that rate of esophageal stricture were as high as 60% following the corrosive intake [13]. The formation of esophageal strictures also depends upon the type of corrosives, particularly the acids taste sour which result in choking or vomiting. The acid reduce the rate of stricture formation and lead to develop chemical esophagitis or pneumonitis [14].

In one of the study done by chen et al, in which 32 children with corrosive intake were selected [15]. Out of that 16 children had high grade injury while 16 had low grade injury. Out of the 16 with low grade injury 1 developed esophageal stricture. On the other hand, out of the 16 children with high grade esophageal injury, 10 developed esophageal stricture. This result is in consistent with our study, where 41.5% patients with severe esophageal injury developed strictures.

Suicidal ingestion of corrosive liquid usually results in the marked oral, oropharynx and proximal esophageal injuries due to hesitant sipping of fluid [16]. Whereas incidental ingestion result in the massive intake of liquid due to unknown nature and taste of fluid and incidental cases associated with high grade gastric injuries.

REFERENCES:

1. Temiz A, Oguzkurt P, Ezer SS, Ince E, Hicsonmez A. Longterm management of corrosive esophageal stricture with balloon dilation in children. *Surg Endosc* 2010;24:2287–92.
2. Zagar SA, Kochhar R, Nagar B. Ingestion of corrosive acid. *Gastroenterology*.1989;97:702–07.
3. Spiegel RJ, Sataloff RT. Caustic injuries of the Esophagus. In: Castell DO, Richter J, eds. *The Esophagus*. Philadelphia: Lippincott, Williams and Wilkins;1999:557–64. 5
4. Zargar SA, Kuchhar R, Mehta S. The role of fibroptic endoscopy in the management of corrosive ingestion and modified endoscopic classification of burns. *Gastrointest Endosc*. 1991;37:165–69. 7
5. Mutaf O, Genc A, Herek O. Gastroesophageal reflux: A determinant in the outcome of caustic

- esophageal burns. *J Pediatr Surg.* 1996;31:1494–95.
6. Bautista A, Varela R, Villanueva A. Motor function of the esophagus after caustic burn. *Eur J Pediatr Surg.* 1996;6:204–07
 7. Pace F, Antinori S, Repici A. What is new in esophageal injury (infection, drug-induced, caustic, stricture, perforation)? *Curr Opin Gastroenterol* 2009; 25:372.
 8. Kay M, Wyllie R. Caustic ingestions in children. *Curr Opin Pediatr.* 2009;21:651–54. 12
 9. Javed A, Pal S, Krishnan EK, Sahni P, Chattopadhyay TK. Surgical management and outcomes of severe gastrointestinal injuries due to corrosive ingestion. *World J Gastrointest Surg* 2012;4:121–15
 10. Cabral C, Chirica M, de Chaisemartin C, Gornet JM, MunozBongrand N, Halimi B, et al. Caustic injuries of the upper digestive tract: a population observational study. *Surg Endosc* 2012;26:214–21.
 11. Betalli P, Falchetti D, Giuliani S, Pane A, Dall'Oglio L. Caustic ingestion in children: is endoscopy always indicated? The results of an Italian multicenter observational study. *GastrointestEndosc* 2008;68:434–39.
 12. Onotai LO and Nwogbo AC. Pattern of corrosive ingestion injuries in Port Harcourt: A ten year review. *The Nigerian Health Journal*, 2010;10:1 -2.
 13. Thomas MO, Ogunleye EO, Somefun O. Chemical injuries of the oesophagus: Aetiopathological issues in Nigeria. *J Cardiothorac Surg.* 2009;4:56-59
 14. Zargar SA, Kuchhar R, Mehta S. The role of fiberoptic endoscopy in the management of corrosive ingestion and modified endoscopic classification of burns. *Gastrointest Endosc.*1991;37:165–69.
 15. Chen TY, Ko SF, Chuang JH, Kuo HW, Tiao MM. Predictors of esophageal stricture in children with unintentional ingestion of caustic agents. *Chang Gung Med J.* 2003;26:233-39
 16. Arévalo-Silva C, Eliashar R, Wohlgelernter J, Elidan J. Ingestion of caustic substances: A 15-year experience. *Laryngoscope.* 2006;116:1422–26.