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Review Article

Pediatric Caustic Ingestion: A Review Article

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Abstract

Caustic ingestion injuries represent a significant morbidity and even mortality producing aerodigestive tract burns. Regarded as a major public health issue, caustic injuries may be produced by various chemicals including alkali and acid agents, phenols and oxidising substances such as peroxides or chlorine bleaches. Public awareness including preventive measures is important in avoiding these injuries. The consequences of caustic ingestion may pose great challenge for both the patients, their faimilies together with clinicians dealing with these children. In this review article it is aimed to discuss the clinical presentations, treatment modalities of these children under the light of relevant literature.

Key words: caustic ingestion; children; treatment; pediatric

Introduction

Although preventive measures have made significant impact on reducing caustic injuries in many countries, caustic ingestion continues to be a serious medical and social issue [1]. It has been stated that half to 80% of the injuries are seen in children and these are typically accidental in nature [2, 3]. On the other hand ingestion of caustic materials by adults and teenagers is often suicidal and frequently life threatening. Although the true prevalance of caustic injuries is not known accurately, this clinical entity continues to be a major public health issue.

In this review article it is aimed to give information about this subject and discuss the presentation, short and long-term sequale together with treatment modalities of children with caustic ingestion under the light of relevant literature.

Epidemiology

As the lye became commercially available for household use in the late 19th and early 20th centruies, with an increase in number, injuries due to caustic ingestion were found to be a major cause of morbidity and mortality worldwide [4]. It has been suggested that half to 80% of the injuries are seen in the children [2]. As previously reported, there is a bimodal age distribution in children with caustic ingestion [5]. Victims are generally preschool children [2, 3]. There are risk factors for caustic ingestion in children. These are namely male gender, attention-deficit/hyperactivity disorder symptoms, lower status of parental education, young maternal age, lack of parental supervision and living in rural areas [6-11]. Most of these children are injured by unintentional and accidental ingestion of caustic substances. On the other hand mechanism of caustic injuries in older group of children and adults differ compared to preschool age children. Intentional ingestions as part of suicide attempts have been reported in these late teenagers and adults and consequently higher complication rates due to more ingestion of caustic substance have been reported [5].

Esophageal injury may be seen in 20-40% of patients following ingestion of caustic substances [12, 13]. Alkali substances with pH value of >11.5 and acid substances with pH value of <2 may cause burns to cheeks, mouth, oropharynx, esophagus and stomach as well as airway [14]. Most common agents responsible for caustic injuries are depicted in table 1 [17]. It has generally been stated that deep burns due to strong alkalis result in strictures followed by acids [7, 8, 15].

Type	Caustic agent	Chemical formula
Strong alkalis	Sodium hydroxide	NaOH
	Potassium hydroxide	KOH
	Lithium hydroxide	LiOH
	Calcium hydroxide	$Ca(OH)_2$
	Trisodium phosphate	Na_3PO_4
	Disodium carbonate	Na_2CO_3
Strong acids	Acetic acid	$C_2H_4O_2$
	Citric acid	$C_6H_8O_7$
	Phosphoric acid	H_3PO_4

	Hydrochloric acid	HCl
Oxidising agents	Hydrogen peroxide Sodium hypochlorite Calcium hypochlorite Potassium permanganete	H_2O_2 NaClO $Ca(ClO)_2$ $KMnO_4$
Phenols	Phenol Salicyclic acid	C_6H_5OH $C_6H_6O_3$

Table 1. *Common agents implicated in pediatric caustic ingestion injury [17].*

Factors that are responsible for the establishment of degree of caustic injury include pH value of offending agent, amount of substance ingested, physical state of the agent and duration of exposure [16]. With regard to acid substances their sour tastes may limit accidental intake of these agents while alkali agents with their uncertain tastes may cause serious tissue destruction. Producing protein coagulation called, coagulation necrosis, acid substances do not cause deeper tissue penetration whereas alkali agents with a process konown as liquefactive necrosis, disrupt both proteins and fats destroying cell architecture, destroys tissues from mucosa through muscle wall layers until alkali is neutralised [17].

Whatever the inciting agent, 3-7 days after ingestion mucosal sloughing and bacterial invasion becomes evident [17]. As rhe esophageal wall becomes weakened between 1-3 weeks, fibroblast proliferation and collagen synthesis begins and lastly fibrosis and stricture phase results at around 4-6 weeks [18]. This process called scar formation may lead to shortening of the esophagus together with luminal strictures producing vomiting or inability to swallow.

Symptoms in acute phase following caustic ingestion include hoarseness, stridor and dyspnea if there is concomittent airway injury [3]. Odynophagia, drooling and refusal of food may be observed in severe cases with caustic ingestion. In more severe cases with esophageal or stomach perforation chest or abdominal pain and rigidity may be detected.

Direct x-rays of neck and chest should be taken in these cases and there is no necessity to obtain radioopaque esophagography in acute phase following caustic ingestion. A technetium-labelled sucralfate scan with a positive predictive value of 47% has been recommended in the diagnosis of these cases in acute phase [17]. But the gold standard in diagnosing these cases during acute phase is esophagogastroduodenoscopy under general anesthesia which should be performed after 48 hours ingestion of caustic substance. There are numerous grading systems identifying the lesion in esophagus and one of the suggested grading systems is depicted in table 2 [8].

Grade 0	No detectable mucosal change
Grade 1	Erythema of mucosa
Grade 2	Erythema, sloughing, ulceration and non-circumferential exudates
Grade 3	Deep mucosal ulceration and circumferential mucosal sloughing
Grade 4	Eschar, full thickness changes and perforation

Table 2. *Endoscopic grading of esophageal injury [8].*

Keeping in mind that nearly all pediatric injuries (86-90%) are due to accidental ingestion of caustic substances occurring in the home environment primary prevention is all that is needed [8, 10]. For example large amounts of detergent must not be kept at home, chemical substances should be placed in the upper shelves, and not be stored in food containers, child prof bottle taps etc. should be used. Furthermore cleaning agents involving sodium hydroxide should be banned or in domestic preparations concentration of the agents should be limited.

Treatment

Most patients have mild injuries with grade 0-2 lesions and are observed in hospital until full oral feeds are tolerated. First liners of medical providers should keep in their minds that induced emesis and gastric lavage and also usage of neutralization agents such as vinegar are strictly contraindicated and must not be attempted. Patients with severe injuries like grade 3-4 esophagitis should be managed in order to avoid stricture development once their acute management is complete. Intravenous fluid resuscitation including total parenteral nutrition (TPN) if needed, until oral feed is commenced should be preferred in these children. There are other treatment modalities for these children for the aim of prevention or modulation of stricture formation. These are proton pump inhibitors, oral nystatin suspension if indwelling nasogastric catheter becomes colonized. There is no concensus for using of steroids in the management of these children but if there is severe burn with grades of 3-4 at the time of diagnosis, a nasogastric tube may be inserted into stomach under direct vision for early enteral feeding and to avoid complications of TPN or undernutrition.

Management of long term sequelae includes treatment of strictures. It has been suggested that there are stricture rates varying from 2% to 49% [19, 20]. Dilatations may be performed antegrade or retrograde in fashion starting at 3 weeks post injury [7, 21, 22]. Balloon dilators can also be used in managing these children with the help of radial force of balloon dilatation itself. Local steroids and mitomycine application can be added to dilatation programs for decreasing stricture rates. As an alternative to serial dilatation, long term stenting of esophageal strictures has also been reported with good results [23, 24]. Both medical and surgical management of gastroesophageal reflux should also be kept in mind.

Other morbidities facing these children include esophageal cancer development and psychosocial impact of prolonged dilatation programs. Development of squamous cell carcinoma of the esophagus and adenocarcinoma has been reported in these cases during follow up [25, 26]. According to previous reports, the time interval between the caustic injury and development of carcinoma may be as high as 45 years [27]. Reported mortality rates related to caustic injuries in children ranges form 0-0.6% [7, 28].

Conclusion

In conclusion; accidental caustic injuries continue to be a major public health issue. Primary prevention including informing community of these severe injuries and prevention is all that is nede. Otherwise long term management of these children with esophageal strictures should be perfomed in order to avoid future complications and to gain future growth of the child with an acceptable quality of life that that limits both

economical and human resources. It is concluded that the community must be reminded of these potential hazards accordingly.

Conflicts of interest:

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