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Drinking Detergents: A Study of Accidental Ingestion of Common Household Liquids

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Abstract

Many readily available household liquids are known to cause significant damage to the upper aerodigestive tract with significant associated morbidity and mortality. Current literature reports long term anecdotal clinical evidence and findings from forensic studies. We performed a novel study on the effects of 3 common household liquids (thin bleach, drain cleaner and toilet bowl cleaner) on sheep oesophageal mucosa and muscle over 24 h. The specimens were examined both macro and microscopically at 0, 4, 14 and 24 h. Macroscopically there was significant weight loss after a 24-h period when compared to a control. Histologically, there were no demonstrable morphological changes; however, paradoxically, both controls demonstrated an increase in average apoptotic count. This study demonstrates that common household liquids have a destructive effect on the upper gastrointestinal tract and proposed mechanisms for this are presented in this study.

Keywords: Oesophageal; Aerodigestive; Injury; Acid; Alkali; Mechanism

Background

Ingestion of caustic substances into the aerodigestive and gastrointestinal tract can result in serious injury and significant morbidity and mortality [1]. Patients present with pain, dysphagia and stridor. The typical population is children who ingest the substances accidentally and adults with a substance abuse or mental health history [1]. These patients often require multidisciplinary management by Otolaryngology, Gastroenterology and General surgeons.

A recent literature review advised initial management involving resuscitation and airway management as indicated, nebulised adrenaline, steroids and urgent surgical opinion in patients suspected of oesophageal perforation [1]. Oesophageogastroduodenoscopy (OGD) is the investigation of choice to assess damage done to oesophageal mucosa. To date, evidence of what occurs to gut parenchyma is anecdotal, cohort reports or forensic studies [2-6]. It has been established that ingestion of caustic household liquids typically results in either liquefactive necrosis with alkali agents or coagulative necrosis with acid agents [1].

Aims and Objectives

The primary objective of this study was to assess the degree of changes that occur over time after exposure to a caustic substance. Furthermore, we sought to explore any possible histological changes that might occur in the gut parenchyma. This information could help clinicians in their patient management in terms of timelines and possibly aid prediction of the deterioration of a patient presenting after caustic liquid ingestion.

Methods

An initial feasibility study was performed using sheep oesophagus with 2 reagents; thin bleach and a control. This was a preliminary study with two primary outcomes: Change in weight (mass) and degree of morphological changes on histopathology. Secondary outcomes were changes in color, character and any further histopathological features. All results were compiled on an excel database and used for study design purposes.

The main study used 3 fresh, "same day kill" sheep oesophaguses, aged six months old, that were procured from a local abattoir. Generic acid and alkali liquid cleaners were purchased from a local grocery store cleaning aisle in the forms of a toilet bowl cleaner and a drain cleaner. The active ingredients were identified as hydrochloric acid in the toilet bowl cleaner and sodium hypochlorite (bleach) and sodium hydroxide (lye) in the drain cleaner. Phosphate-Buffered Saline solution (PBS)

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Figure 1: Administration setup. A funnel is placed in the superior lumen with a clamp securing the lower lumen.

was used as a control and storage solution for the experiment. All specimens were cleaned, weighed and placed in PBS and stored at four degrees centigrade within an hour of retrieval.

All specimens were hung vertically with the inferior aspect clamped and a funnel inserted in the superior lumen (Figure 1). A different solution was poured into each specimen and kept in place for a total of 120 sec each, to mimic several swallows of ingested liquid. The clamp was released, and the remaining solution gently milked through to mimic peristalsis. The lumen was flushed with PBS and transverse sections of the oesophagus were taken at the specified time intervals for histopathology. These were weighed along with the final weight of remaining specimen to evaluate any mass changes.

All sections were place in formalin and routinely processed to paraffin blocks. Transverse sections of the oesophagus were stained with Hematoxylin and Eosin (HE). The sections were reviewed by a consultant head and neck pathologist (KAS), who was blinded to the intervention, for any observable changes in the morphological structure. An apoptotic count (sum of the count measured in 5 high power fields or 1 mm^2 of area) of the squamous mucosa was carried out for each sample.

Results

There was a notable change in mass between each specimen after collating all masses. The alkali had an overall loss of 8.9 g of mass while the acid lost 15.4 g of mass. The control had an increase in mass of 10.6 g. Subjective assessments of the specimens color and character performed at the end of the 24-h time period, suggested the control became paler and oedematous while the experimental specimens retained their color but were a smaller size overall. The same clinician made blinded subjective assessments to avoid any inter-observer variability. The results are summarised in tables 1 and 2, respectively.

Summary of Results

There was a difference in mass between the experimental specimens and control. Both experimental specimens lost mass, with the acid losing proportionally nearly twice the amount (15.4 g) as that of the alkali treated tissue (8.9 g). The control gained mass (10.6 g). This correlated with the macroscopic appearances of the specimens, where the control tissue was notably more oedematous. There was no histopathological evidence of tissue damage or loss, nor evidence of liquefactive or coagulative necrosis. Whilst these occur following injury to organs in-situ, their lack and the absence of inflammation and other vasoreactive changes in our test tissue can be explained by the use of avascularized tissue. While there was a significant change in the gross appearance and weight of the tissue, there were no significant correlative histological alterations, which may be explained by the lack of a vascular tissue response. This suggests that significant damage is sustained *in vivo*, which is further enhanced by vascular response. Hartnet et al. [4] performed a forensic study demonstrating substantial destruction to soft tissue with similar substances but these were submerged over 24 h and do not mimic exposure likely seen in accidental ingestion. It is difficult to say if this mass loss was due to extracellular matrix structure or parenchyma. The increase in mass of the control is likely due to an osmotic influx of PBS fluid. Acid and alkali will cause transmural injury and therefore loss of mass.

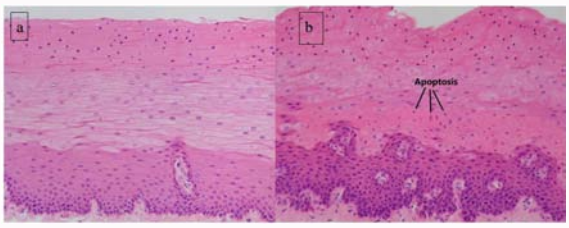

Figure 2: Microscopic analysis of the oesophageal sections at x200 magnification.
2(a): A cross section of healthy oesophageal mucosa with the intra luminal surface superiorly. 2(b): Oesophageal mucosa with apoptotic nuclei highlighted.

Interestingly, apoptotic activity was only noted in control tissue. Apoptosis, or programmed cell death provides a regulatory role in multicellular organisms in effort to maintain cellular integrity. Apoptosis aims to get rid of cellular waste with minimal damage to the underlying tissue, as opposed to necrosis. We are unable to explain the paradox in this study, where no significant apoptotic activity was seen in test tissues.

Limitations of this study are primarily practical in nature. The sample size was small, eliminating the ability to test for statistical significance. Regression slopes for the control, alkali and acid were 0.44, -0.37 and -0.64 respectively. This cannot demonstrate significance, but it can suggest trend. Further, whilst best efforts

Table 2: Macroscopic assessment of the 3 specimens.

	Color	Character
Control	Paler	Oedematous
Alkali	No change	Thinner
Acid	No change	Thinner

Table 1: Change in weight of the 3 specimens at the different time points.

Solution	Initial specimen (Grams, g)	T0 (Grams, g)	T4 (Grams, g)	T14 (Grams, g)	T24 (Grams, g)	Remaining specimen (Grams, g)	End weight (Grams, g)	Change in weight (Grams, g)
Control (PBS)	35 g	2.2 g	4.2 g	2.9 g	3.1 g	33.2 g	45.6 g	10.6 g
Alkali (Bleach/Lye)	45 g	3.6 g	4.3 g	2.9 g	4.3 g	22 g	36.1 g	-8.9 g
Acid (HCl)	46 g	2.5 g	3.1 g	2.7 g	3.5 g	15.7 g	30.6 g	-15.4 g

Table 3: Apoptotic count of the specimens at the different time intervals in the various agents.

AC-0	AC-4	AC-14	A-24	ALK-0	ALK-4	ALK-14	ALK-24	C-0	C -4	C-14	C-24
0	N/A	0	0	0	0	0	0	N/A	14	0	14
0	N/A	0	0	0	0	0	0	N/A	9	0	14
0	N/A	0	0	0	0	0	0	N/A	8	0	5
0	N/A	0	0	0	0	0	0	N/A	5	0	7
0	N/A	0	0	0	0	0	0	N/A	7	0	4
0	N/A	**0**	**0**	**0**	**0**	**0**	**0**	N/A	**43**	**0**	**44**

AC: Acid; ALK: Alkali; C: Control. This table demonstrates the apoptotic count of each specimen at each time interval after exposure to a specific reagent for a certain time. In the case of the specimen in acid at 4 h post exposure and the control at time point zero, it was not possible to perform an apoptotic count due to technical error.

were made to ensure the tissue was as fresh as possible, this is an *in vitro* study which likely affected the results due to a lack of a lymphoproliferative reaction. However, to perform this study in vivo would be neither ethical nor feasible. Further limitations include the length of time of exposure to caustic liquid. This will vary in each case and indeed, resting in PBS is not the same as constant salivary exposure and peristaltic activity, although PBS is a widely recognized control buffer solution.

Further research that would be useful given our findings would be to allow for increased length of exposure to the caustic agent and/or studying the tissue at a later time period after exposure. Additionally, further studies would demonstrate if the apoptotic activity isolated in the control is a pervasive pattern or not. A larger sample size would also allow for testing of statistical significance. Lastly, as we only looked broadly at acids and alkali, it would be interesting to expand this study to include a wide range of various liquid materials found in the household that could be accidentally ingested and use a greater number of specimens in each experimental arm.

Conclusion

This study has demonstrated that there is a significant mass change, specifically, an almost double reduction in weight of the tissue exposed to acid *vs*. alkali. Whilst no histological indication of this was found, this could be an initial osmotic effect with any subsequent necrosis/inflammation being abrogated in our tissue by lack of vascularization. Our study suggests that the initial damage received *in vivo* after ingesting a caustic liquid is compounded by a vascularized inflammatory/immune reaction resulting in tissue necrosis. Further research into pathogenesis of damage and its management could result in an improved outcome for patients presenting with a caustic liquid ingestion.

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