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Risk Factors Predicting Stricture Formation and Death in Patients with Caustic Ingestion

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Abstract: **Background:** Caustic ingestion is still a difficult medical problem, and the outcome is often unexpected. **Objectives:** This study assessed the usefulness of intoxication data, clinical data and some laboratory parameters in predicting the outcome following caustic ingestion, and test the applicability of DROOL Score (DS) as a prognostic indicator of oesophageal stricture. **Methods:** Ninety-two patients with caustic ingestion admitted to Poison Control Centre Ain Shams University (PCCASU), during the period from 1st June to November 30, 2015 were prospectively studied. Diagnoses of stricture made via barium study. **Results:** Sixty-six percentage of cases were children under 5 years. In 48.9% of cases, the caustic agent was alkaline. Symptoms and signs can adequately predict the outcome of corrosive patients. DROOL Score and Total Leucocytic Count (TLC) ≥ 20,000 are useful tools for predicting both stricture formation and mortality, in addition blood pH < 7.2 can also predict mortality. A new value of DS ≤ 5.5 to predict stricture formation and mortality of corrosive patients was proposed. **Conclusion:** Clinical manifestations can adequately predict the outcome also; DS and TLC ≥ 20,000 have predictive value for outcome prediction of corrosive patients. Moreover, blood pH < 7.2 can predict mortality. A new value of DS ≤ 5.5 was identified as a prognostic indicator of outcome in caustic patients.

Key words Corrosive, outcome, poison centres, stricture, prognostic factors.

Introduction

Caustic ingestion is a serious, life-threatening problem in spite of legislation limiting the concentrations and availability of these substances (kaya et al, 2010). The management of caustic ingestion remains a difficult problem because of lack of clear relationship between signs and symptoms and the severity of the gastro-oesophageal injury (Uygun et al, 2012).

Oesophageal stricture is a major complication following caustic substance ingestion. Its incidence nearly 7-15% but may be up 50 % (Riffat and Cheng, 2009). A DROOL Score (DS) is a simple new prognostic non-invasive score, based mainly on initial symptoms and signs to predict stricture formation (Uygun et al., 2012).

This study aimed to evaluate role of intoxication and clinical data in addition to laboratory parameters in predicting the outcome following caustic ingestion, and test the applicability of DS as a prognostic indicator of stricture formation.

Patients and methods

Patients

This is a prospective clinical study that was carried out on all patients with corrosive ingestion within first 48 hours who were admitted to Poison Control Centre Ain Shams University (PCCASU), during the period from 1st June to November 30, 2015, 92 patients were included. Patients with fever, respiratory or gastrointestinal disease and patients exposed to corrosive agent by any route rather than ingestion, or those with delay time more than 48 hours and patients who did not need admission were excluded. The diagnosis was established through history of ingestion of caustic agent and clinical examination. Patients were scored using the DROOL Score **(Table1)**

Methods

Data were collected from a designed patient sheet, including demographic data (age, sex, residence, occupation, and educational level), detailed feature of caustic agent and data regarding intention, place of exposure and delay time, clinical data and laboratory data (TLC and ABG).Stricture was identified by barium swallowing(3-6 weeks post corrosive ingestion). DROOL Score (DS) results and the outcome was also recorded.

Ethical considerations

Full informed consent was taken from the patients or guardians for participating in the study. Institutional ethical committee and PCCASU manger approvals were obtained. Confidentiality of records was kept.

Statistical analysis

Correlations between clinical and laboratory data and groups were analysed using chi-square. Logistic regression was used to assess the set of variables that were independent risk factors of oesophageal stricture and death. Receiver Operating Characteristic (ROC) curve for the risk of either oesophageal stricture or death was drawn and the area under the ROC curve (AUC) was taken to be a measure of the ability of DS to distinguish between patient with and without oesophageal stricture and also to distinguish survivors from non survivors. A value of P less than 0.05 was considered statistically significant. All statistical analyses were performed using SPSS for Windows 10.

Results

Of the ninety two patients, 50 were males and 42 females. Sixty-one patients (66.3%) were less than 5 years of age. The characters of the corrosive agents and the features of the caustic accident are detailed in table (2). The most frequent ingested caustic was alkaline agent (48.9%). The corrosive substances were frequently stored in non-original containers (81.5%). Most of the patients consumed the liquid form (88%) and the amount of ingested corrosive agent could not be assessed in almost all cases (97.8%).Most ingestions were unintentional (85.9%) and majority of cases exposed to the caustic agents at domestic environment (91.3%). During the six months period of this study, the highest incidence of accidents occurred in summer (80.2%).

In this study, the median delay time was 6 hours (ranged 0.5 – 48 hours) and there was significant relation between the delay time and stricture formation, but delay time had no significant relation with mortality (Table 3, 4).

There were no significant difference in caustic agent type between patients with and those without oesophageal stricture, also patient mortality was independent of type of corrosive substances. No significant relation was found between mode of exposure to the caustic agents and the occurrence of post corrosive stricture, but it was significantly related to the mortality where the outcome was worse in patients with suicidal ingestion **(Table 3, 4).** .

The most frequent clinical manifestations were dysphagia (98.9%), vomiting (92.4%), drooling saliva (85.8%) and oral lesions (75%). Symptoms of drooling saliva, abdominal pain, dysphagia, oral lesions, and hoarseness of voice, chest infection and persistent fever were significant predictors of stricture formation. While the presence of drooling saliva, hematemesis/melena, dyspnoea, tachypnea or respiratory distress and cough showed significant difference between survivors and non-survivors. (Table 5,6).

The most common complication was stricture formation **(Figure1)** developed in 18.5% of patients while mortality rate was 6.5% of cases.

The relationship between outcome and laboratory data (WBCs count at admission and blood pH) is shown on **Table 7, 8**. Elevation of TLC above 20,000 cells/mm3 had a significant correlation with both stricture formation and death. There was a significant relation between the blood pH and the morality, but it cannot predict stricture formation as a late complication of corrosive patients.

Clinical manifestations of odynophagia, drooling saliva, abdominal pain, dysphagia, oral lesions and hoarseness of voice, and the presence of chest infection and persistent fever in addition to Drool score were independent risk factors for stricture formation **(Table 9)**. DS ≤ 5.5, blood pH < 7.2 and leucocytosis ≥ 20,000 were found to be independent risk factors for patient mortality as shown on **Table 10**.

In the present study, the Drool Score was easy to be applied and was a significant predictor of the occurrence of stricture and mortality **(Table 9, 10)**. In addition, this study found that the best cut off point for Drool Score to predict stricture formation and mortality in corrosive patients was 5.5a new value of DS ≤ 5.5, which was more sensitive and specific.

The cut off level of ≤ 5.5 achieved 88 % sensitivity with 69.1 % specificity to predict the development of stricture and achieved 83 % sensitivity with 61.6 % specificity to predict the mortality of corrosive patients in the current study as shown in figure (2, 3).

Table (1): The five criteria of The DROOL Score for the assessment of patients with caustic ingestion (Uygun et al., 2012).

Component of DROOL Score	Signs and symptoms	Score 0	Score 1	Score 2
Drooling	Drooling saliva	≥12 hours	<12 hours	No
Reluctance	Reluctance to eat or dysphagia or food intolerance	≥24 hours	<24 hours	No
Oropharynx	Oral and Oropharyngeal lesions	*Severe lesion	Edema, hyper erythema	No
Others	No. of other symptoms /sign: persistent fever, hematemesis, abdominal tenderness, dyspnea	≥2	1	No
Leukocytosis	High TLC	≥20,000 cells/mcl	≤ 20,000 cells/mcl	No

**Friability, hemorrhage, erosion, blisters, whitish exudate, ulcer or necrosis*

Table (2): Distribution of intoxication data of the studied patients with corrosive ingestion.

Type of corrosive agent	**number (percentage)**
Alkalis	44(48.9%)
Acids	33(36.7%)
Mixed	12(13.3%)
Unknown	3(3.26%)
Stored container	
Non Original	75(81.5%)
Original	17(18.5%)
Form	
Liquid	81(88.0%)
Solid	11(12.0%)
Amount	
Unknown	90(97.8%)
Known	2(2.1%)
Mode of exposure	
Accidental	79(85.9%)
Suicidal	12(13.0%)
Homicidal	1(1.1%)
Place of toxicity	
Home	68(73.9%)
Work	8(8.7%)
Others	16(17.4%)
Monthly variation	
June	27 (29.3%)
July	19(20.6%)
August	19(20.6%)
September	9(9.7%)
October	16(17.3%)
November	2(2.1%)e

Table (3): Chi- square statistical analysis showing the relation between intoxication data and the incidence of stricture in the studied patients with corrosive ingestion.

Intoxication data		**Patients without stricture** (67 patients)	**Patients with stricture** (17 patients)	**P-value**
		No. (%)	**No. (%)**	
Type of corrosive agent	Acids	21 (31.3%)	10 (58.8%)	0.220
	Alkalis	35 (52.2%)	5 (29.4%)	
	Mixed	8 (11.9%)	2 (11.8%)	
	Unknown	1(1.5%)	0 (0.0%)	
Mode of exposure	Suicidal	9 (13.4%)	2 (11.8%)	1.000
	Accidental	58 (86.6%)	15 (88.2%)	
	Homicidal	0 (0.0%)	0 (0.0%)	
Form	Liquid	56 (83.6%)	17 (100%)	0.110
	Solid	11 (16.4%)	0 (0.0%)	
Delay time (hours) Mean ± SD		3.69 ± 4.79	10.52 ± 12.17	0.036*

SD: standard deviation - P>0.05: Non-significant - P<0.05: Significant.*

Table (4): Chi- square statistical analysis showing the relation between intoxication data and the mortality rate in the studied patients with corrosive ingestion.

Intoxication data		**Survivors** (86 patients)	**Non survivors** (6 Patients)	**P-value**
		No.(%)	**No. (%)**	
Type of corrosive agent	**Acids**	29 (33.7%)	4 (66.7%)	0.419
	Alkalis	42 (48.8%)	2 (33.3%)	
	Mixed	12 (13.9%)	0 (0.0%)	
	Unknown	3 (3.5%)	0 (0.0%)	
Mode of exposure	**Suicidal**	11 (12.8%)	1 (16.7%)	0.001*
	Accidental	75 (87.2%)	4 (66.7%)	
	Homicidal	0 (0.0%)	1 (16.7%)	
Form	**Liquid**	76 (88.3%)	5 (83.3%)	0.545
	Solid	10 (11.6%)	1 (16.7%)	
Delay time(hours) Mean ± SD		5.25 ± 7.57	5.83 ± 9.10	0.857

SD: standard deviation - P>0.05: Non-significant - P<0.05: Significant.*

Table (5): Chi- square statistical analysis showing the relation between signs and symptoms and the incidence of stricture in the studied patients with corrosive ingestion.

Signs and symptoms	Patients without stricture (67 patients)	Patients with stricture (17 patients)	P-value
	No. (%)	No. (%)	
Vomiting	61 (91%)	16 (94.1%)	1.000
Drooling of saliva	56 (83.6%)	16 (94.1%)	0.001*
Abdominal Pain	18 (26.9%)	9 (52.9%)	0.040*
Dysphagia	66 (98.5%)	17 (100 %)	0.000*
Refusal of eating	2 (2.9%)	0 (0.0%)	1.00
Oral Lesions	51 (76.1%)	14 (82.4%)	0.041*
Hematemesis / Melena	12 (17.9%)	5 (29.4%)	0.069
Signs and symptoms of perforation	1 (1.5%)	0 (0.0%)	1.000
Dyspnea	2 (2.9%)	0 (0.0%)	1.000
Tachypnea	2 (2.9%)	1 (5.9%)	0.497
Respiratory distress	1 (1.5%)	0 (0.0%)	1.000
Wheezes	13 (19.4%)	6 (35.3%)	0.197
Chest Crepitations	14 (20.9%)	7 (41.1%)	0.116
Hoarseness of voice	9 (13.4%)	8 (47%)	0.005*
Cough	5 (7.5%)	3 (17.4%)	0.200
Persistent fever	12 (17.9 %)	4 (23.5%)	0.730
Chest infection	2 (2.9%)	4 (23.5 %)	0.014*
Shock	8 (11.9 %)	0 (0.0%)	0.199

P>0.05: Non-significant - P<0.05: Significant.*

Table (6): Chi- square statistical analysis showing the relation between signs and symptoms and mortality in the studied patients with corrosive ingestion.

Signs and symptoms after corrosive intoxication	Survivors (86 patients)	Non survivors (6 patients)	P-value
	No. (%)	No. (%)	
Vomiting	79 (91.9%)	6 (100%)	1.000
Drooling of saliva	75 (87.2%)	4 (66.7%)	0.010*
Abdominal Pain	26 (30.2%)	3 (50%)	0.375
Dysphagia	85 (98.8%)	6 (100%)	0.005*
Refusal of eating	2 (2.3%)	0 (0.0%)	1.00
Oral Lesions	65 (75.6%)	4 (66.7%)	0.875
Hematemesis/ melena	14 (16.2%)	6 (100%)	0.000*
Signs and symptoms of perforation	0 (0.0%)	1 (16.7%)	0.065
Dyspnea	0 (0.0%)	3 (50%)	0.000*
Tachypnea	1 (1.2%)	3 (50%)	0.001*
Respiratory distress	0 (0.0%)	2 (33.3%)	0.004*
Wheezes	18 (20.9%)	2 (33.3%)	0.608
Chest Crepitations	19 (22%)	3 (50%)	0.146
Cough	6 (6.9%)	3 (50%)	0.011*
Hoarseness of voice	16 (18.6%)	2 (33.3%)	0.334
Persistent fever	15 (17.4 %)	1 (16.7 %)	1.00
Chest infection	7 (8.1%)	0 (0.0 %)	1.00
Shock	6 (6.9%)	2 (33.3%)	0.084

P>0.05: Non-significant - P<0.05: Significant.*

Table (7): Chi- square statistical analysis showing the relation between the blood pH and TLC on admission and stricture formation in the studied patients with corrosive ingestion.

	Patients without stricture (67 patients)	**Patients with stricture** (17 patients)	**P-value**
	No (%)	**No (%)**	
Blood pH			
pH < 7.2	2 (2.9 %)	0 (0.0 %)	1.00
pH > 7.2	63 (94%)	16 (94.1%)	
TLC			
Normal	36 (53.7%)	3 (17.6%)	
11,000- 20,000 cells/mm3	25 (37.3%)	12 (70.6%)	0.026*
> 20,000 cells/mm3	6 (8.9%)	2 (11.8%)	

P>0.05: Non-significant - P<0.05: Significant.*

Table (8): Chi- square statistical analysis showing the relation between the blood pH and TLC on admission and the mortality in the studied patients with corrosive ingestion.

	Survivors (86 patients) No (%)	**Non survivors** (6 patients) No (%)	**P-value**
Blood pH			0.003*
pH < 7.2	0 (0.0%)	2 (33.3 %)	
pH > 7.2	84 (97.7 %)	3(50%)	
TLC			
Normal	43 (50%)	1 (16.7%)	
11,000- 20,000 cells/mm3	38 (44.1%)	2 (83.3%)	0.001*
> 20,000 cells/mm3	5 (5.8%)	3 (50%)	

P>0.05: Non-significant - P<0.05: Significant.*

Table (9): Logistic Regression for estimation of risk factors associated with stricture formation in the studied patients.

	B	**Wald**	**P-value**	**Exp(B)6t5**
Risk factors				
Odynophagia	3.262	2.487	.115	26.094
Drooling of saliva	.538	.084	.771	1.713
Abdominal Pain	-1.855	.354	.552	156
Dysphagia	1.100	.219	.640	3.005
Oral Lesions	2.212	1.219	.269	9.132
Hoarseness of voice	1.550	.510	475	4.713
Chest infection	-4.453	1.912	.167	.012
Tachypnea	.089	.205	.650	1.093
Persistent fever	-4.321	1.443	.230	.013
Drool score	-.325	.147	.701	.722
Constant	-7.808	.336	.562	.000

B: logistic regression coefficient – 95%

Table (10): Logistic Regression analysis for estimation of risk factors influencing mortality in the studied patients.

.	**B**	**Wald**	**P-value**	**Exp(B)**
Risk Factors				
Drool score	-2.243	3.881	.049*	.106
Blood pH	-39.770	.000	.999	.000
TLC	-.549	3.457	.063	.577
Constant	89.780	.000	.999	9.797+038

B: c regression coefficient – 95%

Figure (1): Barium study show post-corrosive stricture formation in this study.

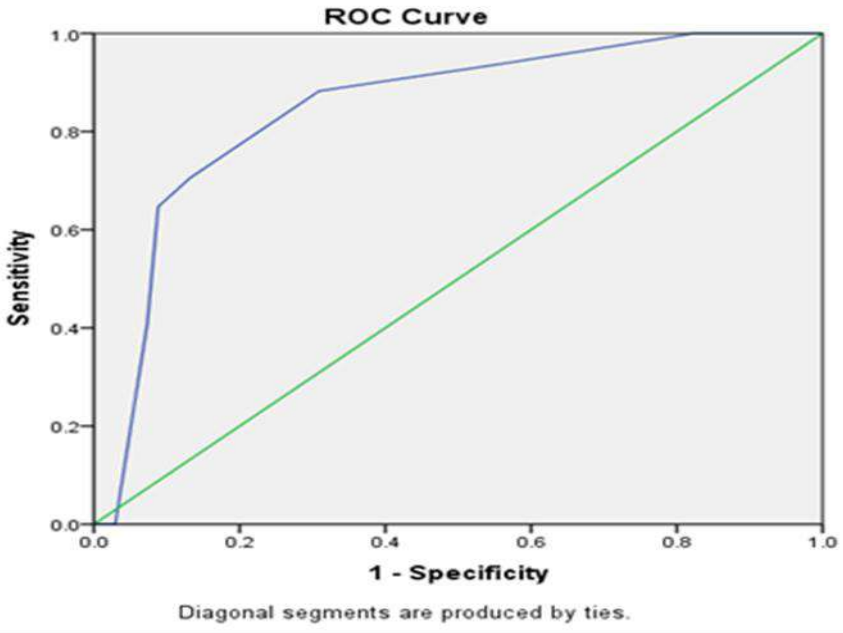

Figure (2): ROC Curve displaying the diagnostic accuracy of Drool score to predict the incidence of stricture in studied patients.

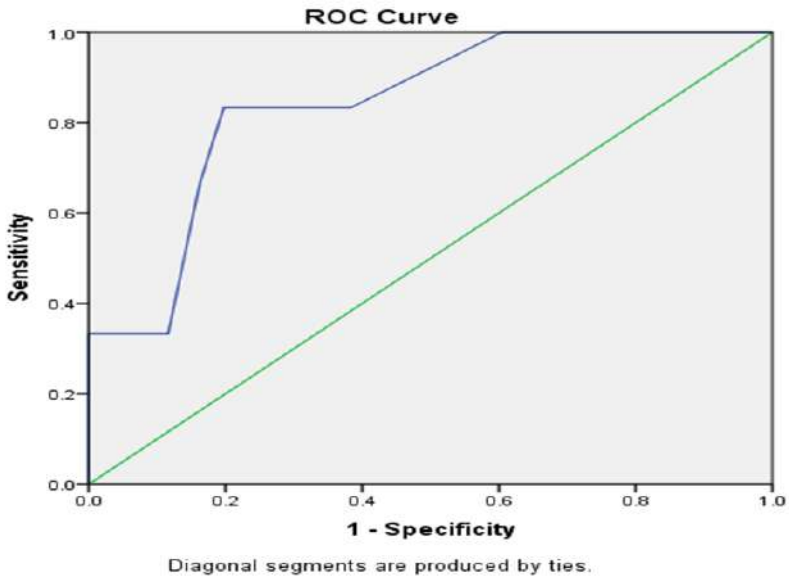

Figure (3): ROC Curve displaying the diagnostic accuracy of Drool score to predict the mortality rate in studied patients.

Discussion

Predication of severity and appropriate management in corrosive ingestion is mandatory to prevent serious and life threatening complications and to get better prognosis (Grace et al., 2013).

In the current study maximum incidence (66.3%) of corrosive poisoning occurred with age less than 5 years, this was similar to the result of study done in Australia by Riffat and Cheng (2009) which concluded that , children younger than 5 years were the highest risk group for caustic ingestion poisoning. This could be explained by the fact that at this age children develop skills of localization, but had poor discrimination between the harmless and harmful substances (Budhathoki et al., 2009).

As regard the gender in this study, cases of males outnumbered females, this result agreed with the result of study done in India by Lakshmi et al., (2013) who reported that, corrosive toxicity was predominant in males. This was explained by curious behaviour of males compared with females.

This study found that most of cases ingested alkaline caustic agents (48.9%). Many studies reported that alkaline agents are the most common and widely spread caustic agents used as household and industrial cleaners and are easily available at home (Lakshmi et al.,2013).While, Chibishev et al. (2012) found that, toxicity was commonly due to acidic agents compared with alkaline agents.

In the current study, most cases of corrosive ingestion (81.5%) were due to storage of caustic substances in non-original containers. This result was similar to the results found by Uygun et al. (2012) who reported that, most cases of caustic ingestion are due to the transfer of caustic substances to smaller, non-original bottles.

Most of the cases in this study were exposed to caustic agents accidentally (85.9%). This result was similar to the results found by Contini et al. (2009) and Meena et al. (2014) who mentioned that, accidental exposure to corrosive agents was more common than suicidal exposure. However, a study by Chowdhury et al. (2013) revealed that majority (88.9%) of the poisoning was suicidal. Increasing the incidence of accidental exposure is explained by keeping these corrosive substances within the reach of children, and these commercial products are sold with cheaper price .They are mainly used as household cleaners and stored in non-original unlabelled containers (Urganci et al.,2014).

Most of the cases in the current study exposed to the caustic agents at home. It was similar to the results of study by Riffat and Cheng (2009) who observed that, the most common place of exposure to caustic agent was the domestic environment, where these agents were available and commonly used daily.

In this study, there was significant relation between the delay time and stricture formation, but delay time had no significant relation with mortality. Similarly Chibishev et al. (2012) reported that, the occurrence of stricture formation depends on the delay time. While, Indira and Rao et al. (2015) found no relation between the delay time and the outcome of corrosive patients.

In the current study, it was observed that, the type of the ingested caustic agents could not be used to predict stricture formation or death in corrosive intoxicated patients. This goes hand in hand with the results of studies by Onotai and Nwogbo (2010) and Chang et al. (2011) who found that, the type of corrosive agents had a poor prognostic value to detect the degree of injury after caustic ingestion.

Raghu and Vadivelan (2012) also reported that, both acids and alkalis caused cicatrisation and stricture with equal percentage. However, Chen et al. (2003) concluded that the alkali character of caustic materials is associated with oesophageal stricture. Some studies considered that acid ingestion associated with higher mortality rates compared with alkaline agents.

In this study, no significant relation was found between mode of exposure to the caustic agents and subsequent stricture formation. However, it was significantly related with the mortality. Gümürdülü et al. (2010) reported that, intentional ingestion is a factor that can affect the degree of severity of corrosive toxicity and associated with a higher risk of complications. Suicidal cases usually ingest large amounts and the patient can overcome the bad taste of the caustic agents. Another study done by Brusin and Krayeva (2012), found no significant difference between mode of exposure and the outcome of corrosive intoxicated patients.

The use of initial symptoms and signs to predict the outcome is controversial. Some studies concluded that initial signs and symptoms were not useful predictors for the severity of oesophageal injury (Ramasamy and Gumaste, 2003 and Chen et al., 2003), Other studies showed that how initial manifestations could be reliable predictors of outcome (Brusin and Krayeva ,2012, and Grace et al., 2013). While, kaya et al., (2010) found that absence of an oropharyngeal burn does not rule out the presence of severe esophagogastric lesions.

The current study found that manifestations of drooling of saliva, abdominal pain, dysphagia, oral lesions and hoarseness of voice, chest infection and persistent fever were significant predictors of stricture formation. While the presence of drooling of saliva, hematemesis/melena, dyspnoea, tachypnea or respiratory distress and cough as showed significant difference between survivors and non-survivors.

Stricture formation is the most severe late post-corrosive complication (Radenkova-Saeva et al., 2016). Oesophageal stricture is a major consequence of corrosive poisoning and develops after caustic ingestion in 7-15% of patients (Betalli et al., 2008). In the current study, stricture formation developed in 18.5% of patients.

There was a controversy about the ability of some laboratory parameters as total leucocytic count

(TLC) and blood pH to detect the degree of injury and the mortality of corrosive intoxicated patients, Rigo et al., (2002) concluded that increased TLC ≥20,000 cells/mm3 was a predictive factor for the mortality following corrosive ingestion. Cheng and Kao (2003) reported that arterial pH <7.22 may indicate severe injury in the gastrointestinal tract that may require immediate surgical management.

In the current study, it was found that the presence of leucocytosis ≥ 20,000 cell/mm3 was a good predictor for stricture formation and the mortality in corrosive intoxicated patients and there was significant relation between blood pH < 7.2 and the morality, but it cannot predict the occurrence of stricture formation as late complication. Kluger et al. (2015) also concluded that, increasing TLC ≥20,000 cells/mm3 was significant risk factor that can assess the degree of the severity of post corrosive injury and also can be correlated with the mortality.

Some authors have considered that TLC ≥ 20,000 cell/mm3 at admission an independent predictor of mortality. However, Chen et al. (2003) concluded that white blood cell counts could not accurately predict severity of oesophageal injury or oesophageal stricture.

Havanond and Havanond (2007) explained the increase in the total leucocytic count by the pathophysiology of corrosive injury as in the first few hours oedema with eosinophilic necrosis and mobilization of leucocyte as inflammatory reaction to corrosive injury occur. Another cause of increasing leucocytic count may be bacterial translocation to the necrotic tissues (kaya et al, 2010).

Acidosis and its progression may be also used as an indicator for the prognosis of corrosive intoxicated patients, the accurate cause of blood acidosis following corrosive ingestion is unknown (Cheng and Kao, 2003).

A study by Chou et al. (2010) reported that, patients with pH < 7.2 were associated with a higher risk of mortality. On the contrary, Otçu et al. (2003) concluded that blood pH values had no role in predicting the severity of injury in corrosive intoxicated patients.

The presence of metabolic acidosis can be explained by production of lactate from the damaged tissue or occurrence of shock in patients with severe injury after corrosive ingestion (kaya et al., 2010). Kluger et al. (2015), reported that pH below 7.2 was a highly significant factor that can predict severe injury and was associated with poor prognosis with high risk of mortality. Thus, once detected, emergency surgical intervention is indicated and no need to wait until full correction of acidosis because the main line of treatment of acidosis is to remove the necrotic tissue.

In this study manifestation of odynophagia, drooling of saliva, abdominal pain, dysphagia, oral lesions and hoarseness of voice and the presence of chest infection and persistent fever in addition to Drool score were independent risk factors for stricture formation. While Drool Score ≤ 5.5, blood pH < 7.2 and leucocytosis ≥ 20,000 were found to be independent risk factors for patient mortality.

Grace et al. (2013) concluded that increasing the TLC ≥ 20,000 was the only significant risk factor of severe gastrointestinal injury and can predict the incidence of stricture formation, while clinical manifestations as dysphagia was found to be a weak factor. Havanond and Havanond (2007) found that, hyper-salivation; oral lesions and leucocytosis were independent predictors for high-grade injury.

Rigo et al. (2002) found that age, ingestion of a strong acid and the presence of gastric deep ulcers or gastric necrosis at endoscopy were independent risk factors for mortality in corrosive intoxicated patients. Another study observed some risk factors for mortality after corrosive ingestion, which included age over 65 years, history of other medical illness, presence of peritoneal signs, blood pH below 7.2 and increased total leucocytic count over ≥ 20,000 (Chou et al. ,2010).

Uygun et al. (2012) developed a simple new prognostic non-invasive score, DROOL Score based mainly on initial symptoms and signs to predict oesophageal stricture without endoscopic grading. The presence of stricture can be highly predicted in patients with DS (≤ 4).

Current study tested the applicability of Drool Score in corrosive intoxicated patients and found that it was easy to be applied and confirms its ability to predict the occurrence of stricture. This study found that Drool score could also predict the mortality following corrosive ingestion. In addition, this study proposed a new value Ds ≤ 5.5 that was more sensitive and specific to predict stricture formation and death in corrosive patients.

Conclusions

Symptoms and signs can adequately predict the outcome of patients with corrosive ingestion, the present study also clarified that some blood tests (TLC and blood PH) were very useful in prediction of the outcome following corrosive ingestion. Drool Score, blood pH < 7.2 and leucocytosis ≥ 20,000 were identified as risk factors associated with mortality. Stricture formation can be predicted by using the simple DS instead of late barium study. Moreover, this study found that DS can also predict the mortality of corrosive patients and proposed a new value of DS ≤ 5.5 that was more sensitive and specific to predict stricture formation and mortality in corrosive patients.

Recommendations

This study highlights that storage corrosives in original childproof containers and parent education are very important to decrease corrosives accidents. The present study supposed that a better assessment of outcome after corrosive ingestion could be achieved by integrating clinical features and laboratory data (ABG and TLC). In addition, this study recommends the use of simple new prognostic DS instead of late classical barium swallow for earlier diagnosis and treatment of stricture. Also the study recommends using the DS for predicting mortality. Further prospective studies are needed involving larger sample size, to verify the findings of the present study and to evaluate the

usefulness of these prognostic indices toward adverse outcome.

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الملخص العربى

عوامل التنبؤ بحدوث ضيق بالجهاز الهضمي وحدوث الوفاة للمرضى الذين تناولوا المواد الكاوية

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المقدمة: يمثل تناول المواد الكاوية خطرا صحيا كبيرا في البلاد المتقدمة والنامية وهناك عددا من المرضى تحدث لهم إصابات كيميائية شديدة ومضاعفات في الجهاز الهضمي العلوي ونسبة عالية من الوفيات. يعتبر التنبؤ بوجود اصابه بالمريء ودرجه شدتها بعد ابتلاع المادة الكاوية تحديا كبيرا ويعد استخدام الأعراض والعلامات الإكلينيكية أو الفحص المعملي للتنبؤ بنتائج هذه الإصابة مثيرا للجدل.

الهدف من هذه الدراسة: تقيم اهميه بيانات التسمم والعلامات الإكلينيكية و بعض الاختبارات المعملية للتنبؤ المبكر بنتائج المرضي بعد ابتلاع المواد الكاوية واختبار إمكانية تطبيق معيار درول كمؤشر لنتائج المرضي.

طريقة البحث:اجريت دراسة مستقبليه على ٩٢مريض الذين تم حجزهم في مركز علاج التسمم بمستشفيات جامعه عين شمس بسبب تناول مواد كاويه خلال الفترة من اول يونيو حتى اخر نوفمبر٢٠١٥.

النتائج:اوضحت النتائج ان اغلبيه الحالات أطفال أقل من ٥ سنوات (٢. ٦٦%)، وأغلبهم من الذكور (٣. ٥٤%). وكان التعرض العرضي هو الأكثر حدوثاً (٨٥/٩%) كما ان تناول المواد القلوية كان هو الأكثر شيوعاً (٤٨,٩%).

وكان حدوث ضيق في الجهاز الهضمي هو المضاعفة الأكثر شيوعاً بعد تناول المواد الكاوية (٤. ١٨%). وكان معدل الوفيات٦,٥% وقد تبين أن زمن التأخر عقب التعرض للمواد الكاوية يمكنه التنبؤ بحدوث ضيق في الجهاز الهضمي كما تبين أن الأعراض والعلامات يمكنها التنبؤ بحدوث ضيق بالجهاز الهضمي وبحدوث الوفاة بعد تناول المواد الكاوية.

يمكن ايضا التنبؤ بنتائج المرضى الذين تناولوا المواد الكاوية ببعض الاختبارات المعملية مثل عدد كرات الدم البيضاء الكلية وحمضيه الدم فقد وجد في الدراسة الحالية ان زيادة عدد كرات الدم البيضاء كان مؤشرا للتنبؤ بحدوث ضيق بالجهاز الهضمي وحدوث الوفاة بينما انخفاض حمضيه الدم كان مؤشرا فقط للتنبؤ بالوفاة وقد تبين ايضا أن معيار درول يمكنه التنبؤ بحدوث ضيق بالجهاز الهضمي وبحدوث الوفاة وتم استنتاج قيمة جديده لمعيار درول أكثر حساسية وتحديدا للتنبؤ بنتائج المرضى الذين تناولوا المواد الكاوية.

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