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### Correlation of Clinical Manifestations and C - reactive protein Level to Patients' Outcome in Acute Corrosive Poisoning

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#### ABSTRACT

#### KEYWORDS

Corrosive  
Outcome  
Poisoning  
C-reactive protein  
Prediction

Caustic injury remains an important public health problem despite various regulatory efforts to reduce its occurrence. The aim of this study was to assess the correlation of initial clinical manifestations with patient outcomes and to evaluate the role of initial C-reactive protein (CRP) level as a prognostic marker in patients with acute corrosive poisoning. This prospective study was conducted on fifty-two patients with history of acute corrosives exposure in Tanta Toxicological Unit. Personal, toxicological data and vital signs were recorded, general and local examination (lips, tongue, palate and skin), routine laboratory investigation (arterial blood gases, liver & kidney function tests, C-reactive protein) and chest X-rays were performed to all patients on hospital admission. Patients were divided to either improved or complicated (chest complication or surgical interference). Most of the patients (53.8%) were in the age group (1-5 years), from rural regions and poisoning occurred accidentally (94.2%) by oral route (78.8%) with sodium hypochlorite (clor) (36.5%). The majority of patients suffered from gastrointestinal (GIT) symptoms. Higher number of patients who suffered from dysphagia and chest crepitation had significant complications. Statistically significant association was evident between patients' outcome and each of modes of exposure, pre-hospitalization period, vomiting, respiratory manifestations, CRP level and X- ray finding. Additionally, the prediction of complication by a percentage of 72.2% may occur if CRP level increased more than 11.5 mg/dl. However, exclusion of complication in 80.5% of cases may occur when its level is less than 11.5 mg/dl. Hence, it may be useful as a prognostic marker for acute corrosive poisoning complications.

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#### Introduction

Corrosive poisoning occurs after ingestion of caustic substances, such as acids (acetic, hydrochloric, sulfuric, lactic, oxalic, ect...), alkalis (sodium and potassium agents, soaps, detergents, ect...), heavy metal salts, formalin, and many other chemical agents (Ramasamy and Gumaste, 2003). Caustic ingestion in children is usually accidental especially in developing countries (Baskin et al., 2004). However, ingestion by adults is

often due to suicidal intent, and injuries tend to be more severe (Satar et al., 2004).

According to the annual report of American Center for Poisonings Control in 2011, about 200,000 caustic intoxications occur per year (Bronstein et al., 2011). Additionally, corrosive poisoning was the first common pediatric poison among pediatric poisoned patients under seven years in Ain Shams Poison Control Center during the year 2011 (El Masry and Tawfik, 2013).

There was dramatic decrease of caustic injuries in developed countries due to many factors such as the presence of childproof containers and improved labeling of caustics. On the other hand, the problem is greater in developing countries as a result of widespread

availability of these agents (Lupa et al., 2007; Kluger et al., 2015).

Depending on the exposure routes, caustics can be the cause of gastrointestinal (GIT), dermal and ocular burns. The severity of the damage depends on the type, physical form, quantity and concentration of the ingested substance (Arici et al., 2012).

The ingestion of caustic substances induces a wide range of injuries to the gastrointestinal tract, which can be mild or fatal, or lead to chronic disease (Chibishev et al., 2013). Alkaline substances generally cause more severe injuries than acidic substances in the esophagus by rapidly penetrating and producing liquefactive necrosis (Gautam et al., 2013).

C-reactive protein (CRP) is an acute-phase protein, which increases rapidly in response to infection, trauma, inflammation, ischemia and burns. It is available in all laboratories and relatively cheap (Chan et al., 2002; Póvoa, 2002). Currently, many studies demonstrated that CRP is able to predict prognosis and mortality of various bacterial diseases and poisoning agents (Póvoa, 2002; Sawiniec et al., 2004). However, the value of CRP in outcome prediction of acute corrosive poisoning has not been evaluated.

The aim of this study was to assess the correlation of initial clinical manifestations with patient outcomes and to evaluate the role of initial CRP level as a prognostic marker in patients with acute corrosive poisoning.

## Patients and Methods

This prospective study was conducted in Tanta Toxicological Unit. All admitted patients with history of acute corrosives exposure in the period between September 2014 and September 2015 were included in the study. Patients were excluded if they had chronic diseases (heart, renal, liver diseases, skin diseases and cancer),

co-ingestion with other drugs and being asymptomatic.

The study was approved by Research Ethics Committee of Tanta Faculty of Medicine and a written informed consent was obtained from each patient or his guardian. Confidentiality of data was considered by making code number for each patient.

### *Study design and methods:*

All patients were subjected to the following;

1. Personal data (patient code, age, sex, residence, occupation and date of admission).
2. Toxicological data: nature of corrosive substance (acidic, alkali, sodium hypochlorite (clor) or others); mode and route of exposure and time elapsed between poisoning and admission "pre-hospitalization period" and hospital stay (hours to one day, 2-6 days and more than 6 days).
3. Vital data, physical examination, local examination (lips, tongue, palate and skin), laboratory investigations including (arterial blood gases (ABG), liver & kidney function tests and CRP) and chest X-ray radiographs were also routinely performed to all patients on hospital admission. Quantitation of CRP was performed by immunoturbidimetric assay using AVITEX® CRP latex agglutination test kit (Omega Diagnostics Ltd, Scotland, UK) according to manufacturer instruction (positive CRP value was considered if its level above 6mg/dl) (Singer et al., 1957).
4. Patients were divided according to outcomes to either improved or complicated (chest complication or surgical interference).

### *Statistical analysis*

The collected data were organized and statistically analyzed using SPSS software

statistical computer package version 16. Data were presented as description of frequency and percentage. Correlation between demographic data, symptoms, signs, laboratory, radiological findings and patients outcomes were analyzed using Chi-square test. The receiver operating characteristic (ROC) curve was used to calculate cut-off value, sensitivity and specificity of CRP level. The area under ROC curve is graded as follows: 0.90-1 = excellent; 0.80-0.90 = good; 0.70-0.80 = fair; and 0.60-0.70 = poor. ROC curve analysis was carried out according to DeLong et al. (1988). Significance was adopted at  $p < 0.05$  for interpretation of results of tests of significance.

## Results

Fifty-two patients with acute corrosive poisoning were included in this study. The age of subjects ranged from 1.2 to 61 years, more than half of them (53.8%) were between 1-5 years and the least percentage (9.6%) of cases was in old age group (>50 years). The number of males was equal to females' patients. Most of studied subjects (59.6%) were from rural regions. Oral route represented the main route of exposure (78.8%) and the majority of poisoning (94.2%) occurred accidentally. The most frequently ingested substance was clor (36.5%) and the majority of cases (92.4%) were admitted to hospital within 4 hours after ingestion of the caustic substance. There was significant association between patients' outcome and each of; mode of exposure, pre-hospitalization period and hospital stay as the majority of accidentally intoxicated and early-admitted cases were improved. Patients with prolonged hospital stay had significant complications (Table1).

All patients in the present study were fully conscious, while agitation was observed in 13 patients (25%). The greater part of them had normal blood pressure, temperature and respiratory rate (92.3%, 69.2% and 76.9% respectively) while more than half of patients had tachycardia (53.8%). There was no

significant association between patients' outcome and vital data (Table 2).

Concerning clinical manifestations, 32.7% of patients had vomiting, 42.3% had drooling of saliva and 48.1% of patients suffered from dysphagia. Additionally, hyperemia in the lips, tongue and buccal mucosa were observed in more than half of patients. Furthermore, wheezes and chest crepitation were observed in 42.3% and 36.5% of patients respectively. There was no significant association between patients' outcome and clinical presentations of lips, tongue, palate and buccal mucosa. However, higher number of patients with dysphagia, vomiting and chest crepitation had significant complications. Patients with no signs of respiratory distress were significantly improved as shown in table 3.

Regarding investigations, the current study demonstrated that most cases had normal arterial blood gases, liver function tests (84.6% and 92 % respectively), and all patients had normal kidney function.

Table (4) shows elevated level of C-reactive protein (CRP) in 42.3% of cases. The majority of patients with positive CRP and abnormal chest X-ray had significant complications. The greater part of patients 41 (78.8%) improved and 11 (21.2%) had complications (seven cases had chest complications and four cases needed surgical interference).

Figure (1) shows the results of analysis of receiver operating characteristics (ROC) curve of CRP for predicting outcome of acute corrosive poisoning. The CRP had 72.2% sensitivity and 80.5% specificity at cut-off value 11.5 mg/dl and its AUC shows a good result (0.831,  $p < 0.001$ ). Based on this, the prediction of complication by a percentage of 72.2% may occur if CRP level increase more than 11.5 mg/dl. However, exclusion of complication in 80.5% of cases may occur when its level is less than 11.5 mg/dl.

**Table (1):** Association between patients' outcome and sociodemographic data (n: 52).

Sociodemographic data		Outcome		Total(52)	X <sup>2</sup>	p value
		Improve (41) n (%)	Complicated (11) n (%)	n (%)		
Age (years)	1-5	22 (53.6)	6 (54.5)	28 (53.8)	1.55	0.67
	6-18	9 (22)	1 (9.1)	10 (19.2)		
	19-25	6 (14.6)	3 (27.3)	9 (17.3)		
	>50	4 (9.8)	1 (9.1)	5 (9.6)		
Gender	Male	22 (53.7)	4 (36.4)	26 (50)	5.50	0.06
	Female	19 (46.3)	7 (63.6)	26 (50)		
Residence	Rural	24 (58.5)	7 (63.6)	31 (59.6)	0.09	0.76
	Urban	17 (41.5)	4 (36.4)	21 (40.4)		
Substance	Acid	10 (24.4)	6 (54.5)	16 (30.8)	5.68	0.128
	Alkali	8 (19.5)	2 (18.2)	10 (19.2)		
	Clor	18 (43.9)	1 (9.1)	19 (36.5)		
	Others	5 (12.2)	2 (18.2)	7 (13.5)		
Route of exposure	Oral	31 (75.6)	10 (90.9)	41 (78.8)	1.22	0.27
	Inhalation	10 (24.4)	1 (9.1)	11 (21.2)		
Mode of exposure	Accidental	40 (97.6)	9 (81.8)	49 (94.2)	3.95	0.04*
	Suicidal	1 (2.4)	2 (18.2)	3 (5.8)		
Delay time (hours)	0-4	39 (95.1)	9 (81.8)	48 (92.4)	8.16	0.01*
	4-12	2 (4.9)	0 (0)	2 (3.8)		
	>12	0 (0)	2 (18.2)	2 (3.8)		
Hospital stay	Hours-1 day	26 (63.4)	2 (18.2)	28 (53.8)	13.98	0.001*
	2-6 days	14 (34.1)	5 (45.4)	19 (36.6)		
	>6 days	1 (2.4)	4 (36.4)	5 (9.6)		

X<sup>2</sup> chi-square test; n: number; \* significant if p<0.05

**Table (2):** Association between patients' outcome and vital data (n: 52).

Vital data		Outcome		Total	P value	X <sup>2</sup>
		Improve (41) n (%)	Complicated (11) n (%)	n (%)		
Pulse	Normal	21 (51.2)	3 (27.3)	24 (46.2)	0.33	0.96
	Tachycardia	20 (48.8)	8 (72.7)	28 (53.8)		
Blood pressure	Normal	39 (95.1)	9 (81.8)	48 (92.3)	0.14	2.16
	Hypotension	2 (4.9)	2 (18.2)	4 (7.7)		
Temperature	Normal	31 (75.6)	5 (45.5)	36 (69.2)	0.05	3.70
	Fever	10 (24.4)	6 (54.5)	16 (30.8)		
Respiratory rate	Normal	33 (80.5)	7 (63.6)	40 (76.9)	0.22	2.99
	Tachypnea	6 (14.6)	4 (36.4)	10 (19.2)		
	Bradypnea	2 (4.9)	0 (0)	2 (3.9)		

X<sup>2</sup> chi-square test; n: number

**Table (3):** Association between patients' outcome and both gastrointestinal and respiratory manifestations (n: 52).

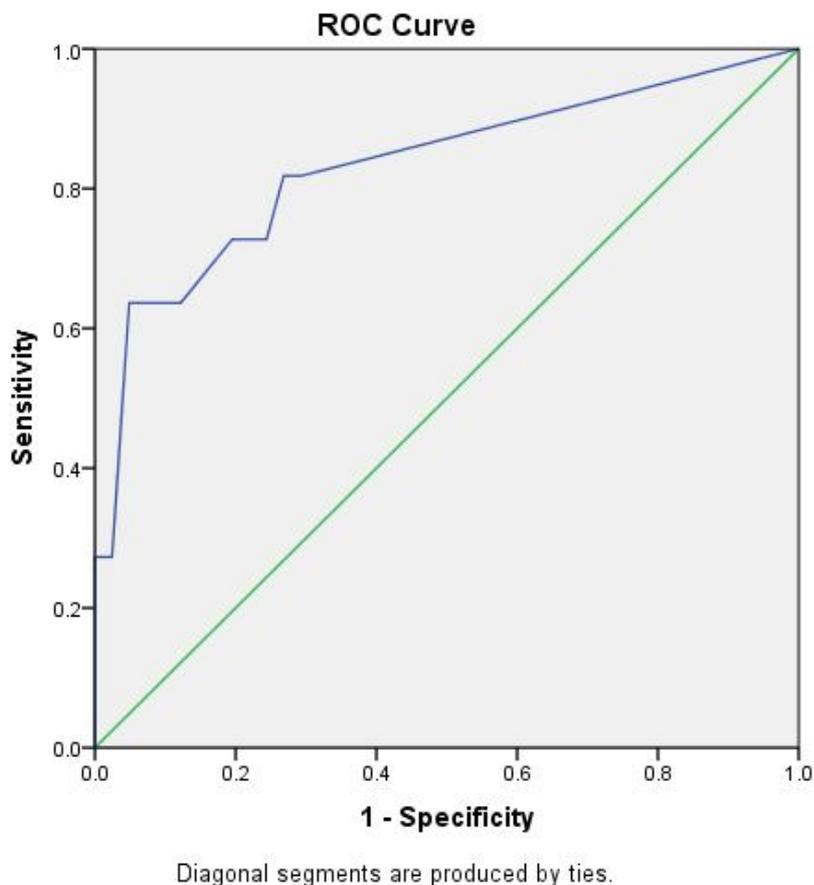
Manifestations		Outcome		Total	X <sup>2</sup>	P value
		Improve (41) n (%)	Complicated (11) n (%)	n (%)		
Dysphagia	No	27 (65.9)	0	27 (51.9)	15.07	0.000*
	Yes	14 (34.1)	11 (100)	25 (48.1)		
Salivation	No	26 (63.4)	4 (36.4)	30 (57.7)	2.60	0.10
	Yes	15 (36.6)	7 (63.6)	22 (42.3)		
Oral lesions	No	18 (43.9)	1(9.1)	19 (36.5)	2.88	0.072
	Yes	23 (56.1)	10 (90.9)	33 (63.5)		
Vomiting	No	25 (61)	3 (27.2)	28 (53.8)	7.32	0.026*
	Yes	13 (31.7)	4 (36.4)	17 (32.7)		
	blood	3 (7.3)	4 (36.4)	7 (13.5)		
Wheeze	No	24 (58.5)	6 (54.5)	30 (57.7)	0.06	0.81
	Yes	17 (41.5)	5 (45.5)	22 (42.3)		
Crepitation	No	30 (73.2)	3 (27.3)	33 (63.5)	8.61	0.003*
	Yes	11 (26.8)	8 (72.7)	19 (36.5)		
Respiratory distress	No	35 (85.4)	6 (54.5)	41(78.85)	9.30	0.026*
	Tachypnea	6 (14.6)	3 (27.3)	9 (17.4)		
	Retraction	0 (0)	1 (9.1)	1 (1.9)		
	Grunting	0 (0)	1 (9.1)	1 (1.9)		

X<sup>2</sup> chi-square test; n: number; \* significant if p<0.05.

**Table (4):** Association between patients' outcome and investigations (n: 52).

Investigations		Outcome		Total	X <sup>2</sup>	P value
		Improve (41) n (%)	Complicated (11) n (%)	n (%)		
HB	Normal	32 (78)	6 (54.5)	38 (73.1)	2.44	0.119
	Anemia	9 (22)	5 (45.5)	14 (26.9)		
WBC	Normal	32 (78)	6 (54.5)	38 (73.1)	2.44	0.119
	Leukocytosis	9 (22)	5 (45.5)	14 (26.9)		
CRP	Negative	29 (70.7)	1 (9.1)	30 (57.7)	13.50	0.00*
	Positive	12 (29.3)	10 (90.9)	22 (42.3)		
Chest X ray	Normal	32 (78.1)	4 (36.4)	36 (69.2)	10.69	0.01*
	Bronchovascular marking	8 (19.5)	4 (36.4)	12 (23.1)		
	Pneumonic patches	1 (2.4)	2 (18.2)	3 (5.8)		
	Pleural effusion	0	1 (9.1)	1 (1.9)		

X<sup>2</sup> chi-square test; n: number; HB: Hemoglobin; WBC: White blood cells;\* significant if p<0.05



**Fig. (1):** The Receiver Operating Characteristic (ROC) curve for C-reactive protein

## Discussion

Caustic injury remains an important public health problem worldwide despite various educational and regulatory efforts to reduce its occurrence. Determination of the risk factors following caustic injuries is an important step to determine the grading of severity, advance treatment protocols and improve prognosis of patients (Quinking et al., 2013).

In the present study, more than half of the studied patients (53.8%) were between 1-5 years and males were equal to females. This result is in line with Arıcı et al. (2012) in Turkey who demonstrated that 54.5% of patients with acute corrosive poisoning were

children. Previous Egyptian studies by Aglan (2007); El Masry and Tawfik; (2013); Hassan and Siam (2014) found that cleaning and disinfectant agents were the most frequent poisoning agents in pediatric patients (29.4%, 19.7% and 17% respectively). Moreover, in the United States, over 5,000 cases consuming caustic substances were reported annually, and most cases occurred in pediatric patients (Riffat and Cheng, 2009). This could be attributed to high mobility and quick self-endangering of the preschoolers as well as their inability to discriminate edible liquids from toxic ones. Inadequate household equipment and insufficient children watchfulness may be additional factors (Al-Binali et al., 2009). This is in disagreement

with Adedeji et al. (2013) who found that children constituted only one third of the studied patients with male predominance. However, Chibishev et al. (2014a) reported that most of the studied patients were female (71.08%).

The majority of the patients in the current study were from rural regions. This could be attributed to overcrowding, illiteracy, poverty and uncontrolled unlabeled cheaper domestic cleaners that are usually sold in rural areas.

Oral route represented the main route of exposure (78.8%) and the majority of cases (94.2%) occurred accidentally in the current study. These results can be attributed to that more than half of the patients were children. Reference wise, Lakshmi et al. (2013) stated that corrosive poisoning in children is usually accidental whereas, in adults, it is due to suicidal attempts. In Nigeria and Republic of Macedonia, Adedeji et al. (2013); Chibishev et al. (2013) and Chibishev et al. (2014a) found that 71.4%, 93.49% and 93.43 % respectively from the total number of patients ingested corrosive agent were due to suicidal attempts.

The most frequently ingested substance was clor (36.5%) and the majority of cases (92.3%) were admitted to hospital within 4 hours after ingestion of the caustic substance. This is comparable with the findings of Chibishev et al. (2013) who reported that many patients ingested hydrochloric acid (51.95 %) and all of them were admitted in the first 24 h after ingestion. In discordance with this, Adedeji et al. (2013) revealed that 78.6% of the patients ingested alkali and about two third of the patients were admitted 24h after ingestion.

Regarding the hospitalization period, the current study revealed that, the greater part of patients (53.8%) was admitted for hours to one day. This can be attributed to that the majority of the patient had ingested clor. Schaffer and Hebert (2000) found that bleaches such as

chloride and peroxide, are usually neutral in pH and of low concentration in commercial preparations. Therefore, they do not cause extensive esophageal damage. In disagreement with Chibishev et al. (2013) showed that the mean length of hospital stay ranged between 19 and 39 days as the majority of cases ingested acid and had severe post-corrosive injuries.

The current study demonstrated that, the majority of accidental cases were improved. This can be explained by the fact that most patients of the present study were children. Myers (1997) illustrated that unlike adults, most children swallow caustic materials by accident and in small amount resulting in rapid improvement. Moreover, only seven cases had chest complication and four cases needed surgical interference. These results disagree with Chibishev et al. (2014b) who demonstrated that 52.04% of patients had respiratory complication after intentional ingestion of hydrochloric acid. The good prognosis and the lower complication incidence in the present study could be explained by accidental mode of exposure and the early admission of the majority of patients.

The majority of the patients in this study suffered from GIT symptoms. These findings partially coincide with Arıcı et al. (2012) in Nigeria and Taromsari et al. (2013) in Iran who found that the most common findings in corrosive exposure were vomiting (35.7% and 47% of patients respectively) and Kaya et al. (2010) in Turkey who demonstrated that 39% of patients suffered from oral lesions.

Higher number of patients with dysphagia, chest crepitation and respiratory distress had significant complications in this study. Consistent with that, Rigo et al. (2002) and Kaya et al. (2010) mentioned that presence of symptoms as dyspnea, dysphagia, excessive salivation, hematemesis or hoarseness suggests severe injury. Additionally, Friedman, (2006) reported that caustics are irritating, which may

lead to a patient's choking and gagging that may predispose them to aspirate the caustic material, with subsequent airway compromise.

In the present study, more than half of complicated cases were feverish. Zargar et al. (1989) and Cibisev et al. (2007) attributed high temperature early to metabolic changes, and late to secondary bacterial infections in the damaged tissues.

Chest radiographs showed exaggerated broncho-vascular markings in 23.1% of patients of the present study. This goes with the results of Contini et al. (2009), who proved that corrosives induced bronchospasm and chemical bronchitis are the main causes of chest x ray changes.

C-reactive protein is a marker of inflammation, which increased in cases of tissue injury (Pan and Zhang, 2001; Rey et al., 2007). Moreover, Sawiniac et al. (2004) suggested that CRP levels can be used to evaluate the severity of acute poisoning cases.

The current study revealed that the majority of patients with positive CRP had significant complications. Similarly, Katzka (2001) and Chen et al. (2003) stated that elevated CRP correlate with the extent and severity of acute caustic injury. Furthermore, the results of ROC curve analysis demonstrated that the AUC of CRP level reach 0.831 with 72.2% sensitivity and 80.5% specificity and patient whose CRP level over 11.5mg/L had complications. These results show that the level of CRP at admission has a good prognostic value in prediction of patients' outcome. To the best of authors' knowledge, no other studies evaluated the value of CRP as a predictor of patients' outcome in corrosive poisoning. However, Ning et al. (2015) found that patients with CRP level over 18 mg/L had a more poor prognosis in acute paraquat poisoning. Furthermore, bad outcome of acute organophosphorus poisoning was associated

with elevated CRP level above 10.2 mg/L (Wu et al., 2016).

There are some limitations in the present study; this study is a single-center study, which may not be representative of all patients. The overall number of the present study was relatively small. Therefore, we suggest performing a larger prospective multi-center study of acute corrosive poisoned patients to evaluate the role of CRP in prediction of patients' outcomes. Moreover, serial measurement should be performed to verify the best time of sampling for prediction of outcome.

## Conclusion

The current study revealed a significant impact of mode of exposure, delay time, vomiting, dysphagia, respiratory symptom (crepitation and respiratory distress), hospital stay and chest X ray finding on patients' outcome. Patients with increase CRP level above 11.5 mg/L may be associated with complicated outcome; thus, CRP level at admission may be useful for prediction of acute corrosive poisoning.

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## ارتباط العلامات الإكلينيكية ومستوى بروتين سي التفاعلي بمصير مرضى التسمم الحاد بالمواد الأكلة

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لا تزال الإصابة الناتجة من المواد الأكلة مشكلة صحية عامة رغم بذل العديد من الجهود التنظيمية للحد من حدوثها. وكان الهدف من هذه الدراسة هو تقييم العلاقة بين العلامات الإكلينيكية الأولية ومصير المرضى وتقييم مستوى البروتين سي التفاعلي الأولي كمؤشر للتنبؤ بالتسمم الحاد للمواد الأكلة. وقد أجريت هذه الدراسة المستقبلية على اثنين وخمسين من المرضى الذين تعرضوا للمواد الأكلة بوحدة علاج التسمم بطنطا. وقد تم أخذ جميع البيانات الشخصية والسمية و تسجيل العلامات الحيوية وعمل فحص عام وموضعي (للشفاه واللسان و سقف الحلق والجلد)، وإجراء الفحوصات المعملية الروتينية (غازات الدم الشرياني ووظائف الكبد والكلية وقياس بروتين سي التفاعلي) وأيضاً إجراء أشعة على الصدر لجميع المرضى عند دخول المستشفى. وقد تم تقسيم المرضى إلى معافين أو لديهم مضاعفات (إما مضاعفات بالصدر أو تدخل جراحي). وكان معظم المرضى (٥٣,٨٪) في الفئة العمرية من سنة إلى ٥ سنوات، من المناطق الريفية وقد حدث التسمم عرضياً في (٩٤,٢٪) وعن طريق الفم في (٧٨,٨٪) وبمادة الكلور في (٣٦,٥٪) من الحالات. وقد عانت غالبية المرضى من أعراض بالجهاز الهضمي. وحدثت بعض المضاعفات لعدد كبير من المرضى مثل عسر البلع وإفرازات بالصدر. وقد وجدت علاقة واضحة ذات دلالة إحصائية بين مصير المرضى وكلا من طريقة التعرض، فترة ما قبل المجيء للمستشفى، القيء، الأعراض التنفسية، مستوى بروتين سي التفاعلي وأشعة الصدر. بالإضافة إلى ذلك، أظهرت النتائج أنه عند ارتفاع مستوى بروتين سي التفاعلي أكثر من ١١,٥ ملجم / ديسيلتر فإنه يمكن التنبؤ بحدوث المضاعفات بنسبة ٧٢,٢٪ من الحالات بينما يمكن استبعاد حدوث المضاعفات في ٨٠,٥٪ من الحالات عندما يكون مستوى بروتين سي التفاعلي أقل من ١١,٥ ملجم / ديسيلتر. وبالتالي يعتبر بروتين سي التفاعلي مؤشراً جيداً للتنبؤ باحتمال حدوث مضاعفات التسمم الحاد بالمواد الأكلة.