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RESEARCH ARTICLE

PREDICTORS OF SEVERITY OF CORROSIVE INJURY

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ABSTRACT

Objective: To evaluate clinical and biochemical predictor and role of a 6-point EGD classification system of injury in predicting outcomes in patients diagnosed with caustic agent ingestion.

Material and Methods: 91 patients admitted to SMS hospital Jaipur, with definite caustic ingestion between 2010-2012 were evaluated prospectively. Detailed history, physical examination, biochemical, radiological and endoscopic evaluation was done in all patients at the time of admission and during a follow up period of 6 month. Patients were given standard care treatment during this period depending on severity of disease.

Results: Endoscopic grading wast most accurate predictor. Voluntary ingestion, vomiting, drooling, stridor and abdominal tenderness were associated with the severity of injury. Leucocytosis, low pH, low phosphorus and alkaline phosphatase level, and high uric acid were significantly associated with severe injury.

Conclusions: Serious signs and symptoms (vomiting, drooling, and stridor) had serious esophageal injury. Leucocytosis, low pH, low phosphorus and alkaline phosphatase level**,** and high uric acid also predict severity of injury. Best predictor of injury is endoscopic grading.

INTRODUCTION

Ingestion of caustic substances induces an extensive spectrum of injuries to the aerodigestive tract. The suspicion of any ingestion is accepted to mandate an esophageal evaluation. The biochemical alterations that follow caustic ingestion have not been thoroughly evaluated in the literature so a prospective clinical study was planned to evaluate if any biochemical predictors of caustic ingestion and complicating esophageal injury exist. And to evaluate the role of signs and symptoms and a 6-point EGD classification system of injury in predicting outcomes in adult patients diagnosed with caustic agent ingestion.

Aims And Objectives

1. To study clinical profile of patients presenting with acute corrosive injury.
2. To evaluate whether or not any clinical predictor of caustic ingestion and complicating esophageal injury exists
3. To evaluate whether or not any biochemical predictor of caustic ingestion and complicating esophageal injury exists
4. To evaluate the role of a 6-point EGD classification system of injury in predicting outcomes in patients diagnosed with caustic agent ingestion.

MATERIALS AND METHODS

91 patients admitted to SMS hospital Jaipur, with definite caustic ingestion between 2010-2012 were evaluated prospectively. Patients without metabolic, hepatic or renal disease and who were admitted within 24 hours following ingestion were enrolled into the study.

Parameters analysed were age, gender, intent of ingestion, substance ingested and amount, length of hospital stay, complications, and the severity of mucosal injury as assessed by EGD.

All patients and/or their attendants were questioned about the type of ingested substance and complaints. Physical examination was performed.

Chest X-ray was obtained, and blood samples were withdrawn for CBC, ESR, CRP, blood gas estimations and biochemical analyses including SGOT, lactic dehydrogenase (LDH), alkaline phosphatase (ALP), calcium, phosphorus and uric acid determinations, timing of sample.

Radiological investigation Chest and abdomen x ray, barium swallow studies, CECT thorax and abdomen were done whenever indicated.

All patients underwent esophagogastroscopic examination. The status of the esophagus, stomach and pylorus was evaluated. If a burn was encountered, the localization, degree

and extension were noted, mucosal damage was graded using Zargar's modified endoscopic classification scheme.

Patients were treated with a proton pump inhibitor or H2 antagonist and were maintained without oral intake until their condition was considered stable. Patients received parenteral nutrition during this period. If infection was suspected, antibiotics (a 1st generation cepholasporin and metronidazole) were administered after blood cultures were obtained.

If a patient's condition destablized or respiratory difficulty was encountered, they were transferred to the intensive care unit for further evaluation.

Any complications observed during follow- up were recorded. Upper GI complications included bleeding, perforation, and stricture formation. Bleeding was defined as melena, hematemasis, and/or coffee ground vomitus. Perforation was diagnosed by the presence of free air on a plain chest radiograph. Stricture was defined as dysphagia, symptoms of regurgitation, or difficulty in swallowing with confirmation by endoscopy, esophagogram, and/or upper GI radiography.

After discharge, patients were followed in the outpatient clinic for at least 6 months. For evaluating the alterations in blood gas and biochemical parameters, the values were compared with reference values (Table 1). Results of blood gas evaluations and biochemical evaluations according to the groups were compared. The values were also compared according to the presence or absence of caustic injuries within the groups.

Table 1 Normal Range Of Biochemical Parameters

Parameter	Normal ranges
pH	7.35-7.45
Bicarbonate Arterial:	19-24 mEq/L
Venous:	22-26 mEq/L
Sodium	138-145 mEq/L
Potassium	3.4-4.7 mEq/L
Chloride	95-110 mEq/L
Uric acid	2.5-7 mg/dl
Creatinine	0-1.3 mg/dl
SGOT	0-40
SGPT	5-36
Lactic dehydrogenase	240-480 U/L
Alkaline phosphatase	25-150 U/L
Calcium	8.8-10.8 mg/dl
Phosphorus	2.5-4.3 mg/dl
Glucose	60-100 mg/dl

Zargar's grading classification of mucosal injury caused by ingestion of caustic substances

Grade 0 Normal examination
Grade 1 edema and hypermia of the mucosa
Grade 2a Superficial ulceration, erosions, friability, blisters, exudates, hemorrhages, whitish membranes
Grade 2b Grade 2a plus deep discrete or circumferential ulcerations
Grade 3a Small scattered areas of multiple ulceration and areas of necrosis with brown-black or greyish discoloration
Grade 3b Extensive necrosis.

RESULTS

Ninety one patients were evaluated during the study period. There were 43 (47.2%) males 48 (52.7%) females The mean age was 23+/_11.6 years (range: 2 to 44 years). There was no sex or age difference between the groups (p>0.05). Acid intake was present in 61(67.0%) and alkali in 30 (32.9%) patients. In 70(76.9%) patients amount of corrosive ingestion was more than 15 ml. Accidental ingestion was seen in 78 (85.7%) patients and suicidal intention in 13(14.2%) patients.

Grade first, second and third degree endoscopic injury was seen in 3(3.2%), 42(46.15%), and 46 (50.5%) patients respectively. Blood pH level was significantly decreased in patients with grade third injury. Blood bicarbonate level did not show any significant difference between groups, and it was not altered according to presence or absence of injury.

Serum uric acid values were significantly increased in patients with severe esophageal injury resulting from either acid or alkali substances .This finding was especially pertinent for esophageal injury due to alkali substance ingestion. Serum phosphorus and ALP levels were significantly decreased patients with esophageal injury The decrease was independent of the acid or alkali nature of the caustic substance.

Serum potassium, chloride, urea nitrogen, creatinine, SGOT, SGPT, LDH, calcium, glucose, protein, albumin and bilirubin levels did not differ between groups, nor between patients in the same group who were with or without esophageal injury.

66(72.5%) patient developed stricture on follow up and majority of these patient had grade 2b and above injury on initial endoscopy. Systemic complications were seen in 44 (48.3%) of patients. 7 (7.6%) patients expired during the period of study.

Table 2 Demographic features of patients

variables	Observation (n=91)
Age	23+/_11.6 years
Male	43(47.2%)
Female	48(52.7%)
Acid	61(67%)
Alkali	30(38.9%)
Amount (<15ml)	31 (34.0%)
Amount (>15 ml)	70(76.9%)
Accidental	78(85.7%)
Suicidal	13(14.2%)

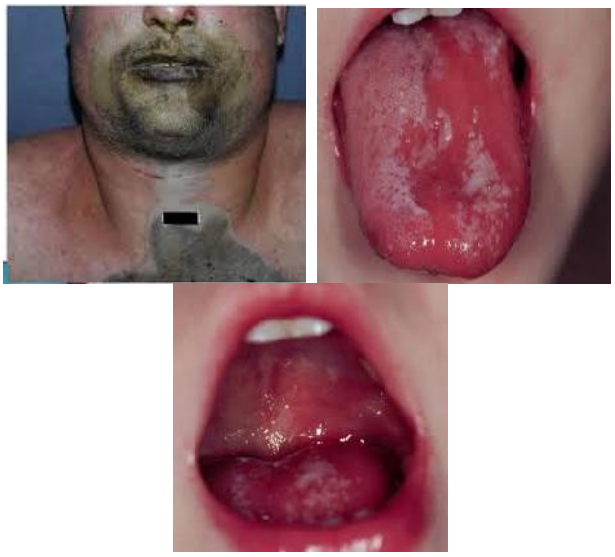

Figure 1 External injury signs of corrosive injury

Table 3 Clinical features and investigations in relation to Endoscopic grading of injury

VARIABLE (n=91)	Drooling of saliva (n=71)	Oral mucosal burn (n=66)	BLEED (n=49)	pH (mean value)	TLC (mean value)	Phosphorus (mean value)	ALP (mean value)	Uric acid (mean value)
GR 1(n=3)	0	1	0	7.35	7400	3.2	101	5.1
GR 2a(n=11)	6	4	6	7.31	8300	2.9	95	6.3
Gr 2b(n=31)	24	25	9	7.29	9100	2.6	90	9.1
Gr 3a(n=33)	29	25	16	7.21	11500	2.1	61	12.4
Gr3b(n=13)	12	11	18	7.12	13700	1.9	55	16

Table 4 Mortality and morbidity in relation to Endoscopic grading of injury

Variables	Endoscopic Grading				
	1	2a	2b	3a	3b
Median Hospital Stay (days)	2	5	11	15	21
Icu admission (n=19)	0	0	2	7	10
Expired (n=7)	0	0	0	1	6
Systemic Complication (n=44)	0	5	8	11	20
Gastrointestinal Complication Overall(n=117)					
Stricture (n=66) (72.5%)	0	2	11	22	31
Bleeding(n=41)	0	1	6	12	22
Perforation(n=6)	0	0	0	1	5
Fistula(n=4)	0	0	0	0	4

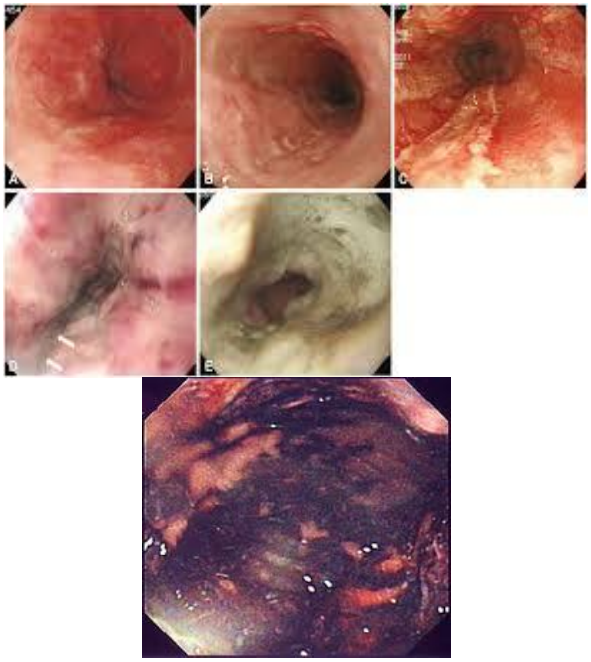

Figure 2 Endoscopic grades of corrosive injury

DISCUSSION

Since many caustics are easily accessible ingestion is a common worldwide problem[2]. The extent of the injury depends on the type of agent, its concentration, quantity and physical state, the duration of exposure and the presence of food particles in the stomach[3].Early stratification of severe corrosive injury is important as treatment plan depends on it [4,5]. Endoscopic grading of injury is well established and correlates with severity and future complication in corrosive intake. A number of other clinical and biochemical variables have been studied in few studies with variable results. The biochemical alterations that follow caustic ingestion have not been thoroughly evaluated in the literature so a prospective clinical study was planned to evaluate if any biochemical predictors of caustic ingestion and complicating esophageal injury exist. And to evaluate the role of signs and symptoms and a 6-point EGD classification system of injury in predicting outcomes in adult patients diagnosed with caustic agent ingestion.

Hypernatremia and hyperchloremia in association with metabolic acidosis have been reported following ingestion of a large amount (500 ml) of bleach in few patients [6]. Increased sodium and chloride concentrations were also encountered due to large loads of those elements in this case. Studies on acid or alkali substance ingestion revealed an increase in serum uric acid levels, they contrarily revealed decreases in phosphorus and alkaline phosphatase levels. Uric acid is the end product of dietary and endogenous purine metabolism in humans. It is formed from the purines of tissue and dietary nucleoproteins and nucleotides [7].

An increase in uric acid results from increased production or inefficient clearance.Increased production is usually due to increased destruction of nucleoproteins in conditions such as lymphoproliferative disorders, disseminated neoplasms and leukemia[8]. Acutely damaged esophagogastric mucosa and/or deep layers may increase the cellular turnover and result in increased serum uric acid level following caustic injury. Approximately 75% of the daily urate production is excreted by the kidneys. The remainder is eliminated through the gastrointestinal tract via biliary, gastric, and intestinal secretions[8].

Since esophageal and/or gastric mucosa is destroyed by caustic substances, this may have resulted in a deficiency in the clearance of uric acid and caused an increase in the serum uric acid level. Additionally, hypovolemia due to disturbed oral fluid intake and loss of fluid through the burned surfaces in patients with caustic injury may have provoked a relative increase in serum uric acid concentrations.

Hypophosphatemia is known to result from intracellular shift of phosphate, increased loss via kidney or intestine, or decreased intestinal absorption[8]Acidosis induces a shift of phosphorus from intracellular to extracellular fluid and causes a rise in serum phosphate levels. Adversely, alkalosis may be expected to cause hypophosphatemia. Intravenous glucose administration causes a decrease in serum phosphorus levels. Similarly, serum glucose levels did not differ between patients with or without caustic injury. Therefore, phosphorus appears to have decreased due to loss from injured alimentary tract and/ or inappropriate intake following caustic injury.

Alkaline phosphatase is an enzyme that works mainly in the liver and bones. Decrease of this enzyme is associated with an excess of vitamin D ingestion, milk-alkali syndrome, hypophosphatasia or malnutrition. Thus, decrease in serum phosphorus levels may satisfactorily explain the mechanism of decrease in serum ALP levels following caustic injury. While the serum uric acid level appears to reflect the tissue damage, serum phosphorus and ALP levels appear to

indirectly reflect the damage through limitation of oral intake in patients who have had esophageal injuries following caustic ingestion.

On the other hand, serum electrolytes and other biochemical parameters including SGOT, SGPT, LDH, urea nitrogen, creatinine, calcium, glucose, protein, albumin and bilirubin do not appear to be indicative of a caustic substance ingestion and/or predictive of a caustic esophageal injury. Endoscopic grading has been most predictive of degree of mucosal injury and subsequent complications.[9,10]

Larger sample size prospective studies are required to validate these observations.

CONCLUSION

Serious signs and symptoms (vomiting, drooling, and stridor) had serious esophageal injury. Leucocytosis, low pH, low phosphorus and alkaline phosphatase level**,** and high uric acid also predict severity of injury. Best predictor of injury is endoscopic grading.

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