

# Highly Concentrated Acetic Acid Poisoning: 400 Cases Reviewed

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## Abstract

**Background:** Caustic substance ingestion is known for causing a wide array of gastrointestinal and systemic complications. In Russia, ingestion of acetic acid is a major problem which annually affects 11.2 per 100,000 individuals. The objective of this study was to report and analyze main complications and outcomes of patients with 70% concentrated acetic acid poisoning.

**Methods:** This was a retrospective study of patients with acetic acid ingestion who were treated at Sverdlovsk Regional Poisoning Treatment Center during 2006 to 2012. GI mucosal injury of each patient was assessed with endoscopy according to Zargar's scale. Data analysis was performed to analyze the predictors of stricture formation and mortality.

**Results:** A total of 400 patients with median age of 47 yr were included. GI injury grade I was found in 66 cases (16.5%), IIa in 117 (29.3%), IIb in 120 (30%), IIIa in 27 (16.7%) and IIIb in 70 (17.5%). 11% of patients developed strictures and overall mortality rate was 21%. Main complications were hemolysis (55%), renal injury (35%), pneumonia (27%) and bleeding during the first 3 days (27%). Predictors of mortality were age 60 to 79 years, grade IIIa and IIIb of GI injury, pneumonia, stages "I", "F" and "L" of kidney damage according to the RIFLE scale and administration of prednisolone. Predictors of stricture formation were ingestion of over 100 mL of acetic acid and grade IIb and IIIa of GI injury.

**Conclusion:** Highly concentrated acetic acid is still frequently ingested in Russia with a high mortality rate. Patients with higher grades of GI injury, pneumonia, renal injury and higher amount of acid ingested should be more carefully monitored as they are more susceptible to develop fatal consequences.

**Keywords:** Poisoning; Acetic acid; Gastrointestinal injury; Stricture; Mortality

## INTRODUCTION

Acetic acid is an organic acid that is available in concentrations up to 70%. It has been used in Russia for homemade vegetable preserves for decades and is available in almost every grocery store. Due to its easy availability and the tradition to keep 70% acetic acid close with food (keeping acetic acid bottles in the refrigerator), poisoning by ingestion is common. The average rate of acetic acid poisoning for the last 8 years in Sverdlovsk region was 11.2 per 100,000 people with the mortality rate of 1.05 per 100,000 (1). A recent annual study of poisonings in Yekaterinburg through 2008 to 2009 revealed that hospital mortality rate of acetic acid poisoning was 13.6%, expressing that it is the leading cause of hospital mortality among patients with acute poisoning (2).

Glacial acetic acid ingestion is associated with more complications and higher mortality rate than alkaline ingestion (3). Patients who ingested highly concentrated acetic acid suffered from esophageal and stomach mucosal burns, respiratory and renal insufficiency and hemolysis (1,4). In addition, enhanced enzymatic activities and disseminated intravascular coagulation (DIC) have been reported following acetic acid ingestion (5,6).

Although acetic acid ingestion is a common problem in

Russia, there are few published reports of highly concentrated acetic acid poisoning elsewhere, because type of caustic substance ingested varies depending on ethnicity (7). The aim of this study was to determine main complications and outcomes of human exposure to 70% concentrated acetic acid.

## METHODS

This was a retrospective study of 400 patients with 70% acetic acid poisoning who have been treated at Sverdlovsk Regional Poisoning Treatment Center during January 2006 to June 2012. All data were obtained from patient records and entered into a single database. Demographic features including age, gender, intention of poisoning, quantity of acetic acid ingested, time from the ingestion until treatment, place of residence (e.g., Yekaterinburg city or outside of Yekaterinburg) and main clinical complications including laryngeal edema, hemolysis, anuria, pneumonia, coma, the presence and duration of shock (defined as systolic blood pressure lower than 100 mm Hg), and bleeding were recorded. Pneumonia was reported as unilateral or bilateral. Renal damage was characterized with the RIFLE criteria (Risk of renal dysfunction; Injury to the kidney; Failure of kidney function, Loss of kidney function and End-stage kidney disease) (8), duration of anuria, number of

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hemodialysis sessions received, and glomerular filtration rate (GFR) at discharge. Cause of death was defined in each case by an experienced pathologist.

Gastrointestinal endoscopy was performed during the first week (median: 5th day post-ingestion) and repeated before discharge if ulceration was found on initial examination. Patients with a severe degree of GI injury underwent endoscopic examination 3 or 4 times to verify the risk of GI bleeding and strictures formation. Autopsy data were obtained from 23 subjects who died during the first few days and were not undergone GI endoscopy. Zargar's scale was used for grading of GI tract injuries (9,10). All patients were asked to come for follow up endoscopic and laboratory examinations, 3 and 6 months post-discharge but only 7 patients agreed. Strictures that developed during the first 6 weeks were confirmed by endoscopy and contrast roentgenography. Outcomes were defined as "survival without strictures", "survival with strictures" and "death".

The data were presented as median and range for continuous variables and in frequency (percentage) for categorical variables. Data analysis was performed with Statistical Package of Social Sciences (SPSS Inc., Chicago, IL, USA) and p-values less than 0.05 were considered as statistically significant. The differences between groups according to patient's intention of poisoning were analyzed with the student's t-test. When necessary, non-parametric analyses were used. The differences among groups according to GI injury grade were compared with the Chi-square test. Stepwise logistic regression analysis was performed to analyze predictors of stricture formation and mortality.

## RESULTS

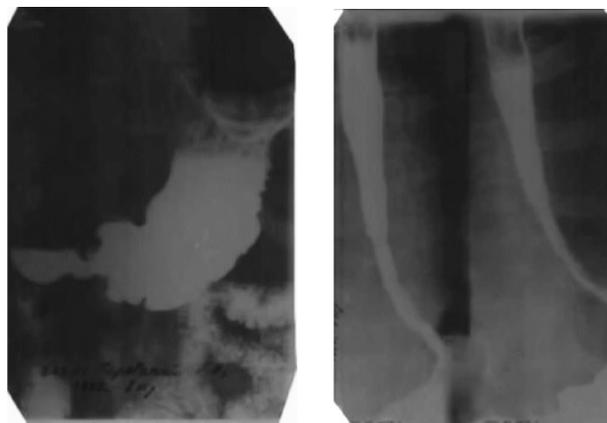
### General findings

Four hundred patients were studied with median age of 47 years (range 14-89) of which 178 cases (44.5%) were males. 393 patients (98%) were admitted to ICU. The volume of acetic acid ingested was reported in 375 cases and the median was 60 ml (range 10-250). The median time from ingestion until treatment was 2 hours (range 0.5-96). 209 patients (52%) were transferred to the poisoning treatment center from local hospitals outside Yekaterinburg. Median hospital stay was 18 days (range 3-136).

Clinical complications on admission included coma in 40 patients (10%), shock in 60 (15%), hemolysis in 221 (55%) and anuria in 71 (18%). The main respiratory disorder on admission was laryngeal edema in 80 cases (20%), which required orotracheal intubation in 47 and tracheostomies in 3. Pneumonia was found in 106 cases (27%) of which 65 were unilateral and 41 bilateral. Seventy two patients (18%) developed psychosis mostly between the second and the fourth day post-ingestion.

Gastrointestinal complications included initial bleeding in 109 cases (27%), repeating bleeding in 68 (17%) and perforation in 13 (3%). Strictures developed in 45 cases of which 28 cases (62%) were in the esophagus, 11 cases (24%) in the stomach, and 5 cases (11%) in both positions (Figure 1). One patient had strictures in the esophagus and duodenum. Stricture formations developed in a median of 3 weeks after acetic acid ingestion (range 1-6).

Eighty-five patients (21%) died on median of 10th day (range 1-71). Twenty patients (5%) died on the first day, primarily from shock and gastrointestinal hemorrhage. The



**Figure 1.** Contrast roentrography imaging of a 25 years old patient with suicidal acetic acid poisoning on 20th day post-exposure. He ingested 100 ml of 70% acetic acid and was admitted in local hospital 8 hours after ingestion with hemolysis. The images are showing the narrowing (stricture) of pyloric valve and third part of esophagus.

median age of the deceased patients was 59 years (range 23-88), and 44 of them (52%) were females. The main documented causes of death were bleeding in 43 patients (51%), shock in 17 (20%), perforation in 11 (13%), pancreatic necrosis in 11 (13%), and pneumonia in 7 (8%). Myocardial infarction and hepatic necrosis were revealed postmortem in 2 cases. The minimum amount of acetic acid that caused death was 20 ml.

### Intention of ingestion

Suicidal intention was implicated in 252 cases (63%). In the remaining cases, the ingestion was unintentional including 76 patients who confused acetic acid with vodka. Suicidal intention was more common in females (70%). Unintentional poisonings predominated among males (70%). Demographic and clinical characteristics of patients according to intention of poisoning are shown and compared in table 1.

Patients with suicidal poisonings were significantly younger ( $P < 0.001$ ), ingested higher amounts of poison ( $P < 0.001$ ) and more frequently developed hemolysis ( $P = 0.006$ ), and initial bleeding on admission ( $P = 0.012$ ). Unintentionally poisoned patients developed psychosis more often ( $P = 0.008$ ). GI tract strictures were more common in suicidal poisonings ( $P = 0.019$ ). The mortality rate was also higher in suicidal poisonings but the difference was not significant ( $P = 0.21$ ).

### Clinical outcomes according to GI and renal injury

Zargar's grade I of GI tract injury was found in 66 patients (16.5%), IIa in 117 (29.3%), IIb in 120 (30%), IIIa in 27 (16.7%) and IIIb in 70 (17.5%) (Figure 2). Frequency of clinical complications, volume of acetic acid ingested and duration of ICU care, according to GI injury grading are shown in table 2. Outcomes were worse in grades IIb, IIIa and IIIb. Only 18% of patients with grade IIIa survived without stricture while all survived patients with grade IIIb developed strictures.

Renal injury was found in 141 cases (35%) and graded according to the RIFLE scale. Mortality rate was 5% in cases without kidney damage and increased to 27% in cases with "Risk" stage, 40% with "Injury", 58% with "Failure" and 64% with "Lost".

**Table 1.** Demographic and clinical characteristics according to intention of poisoning.

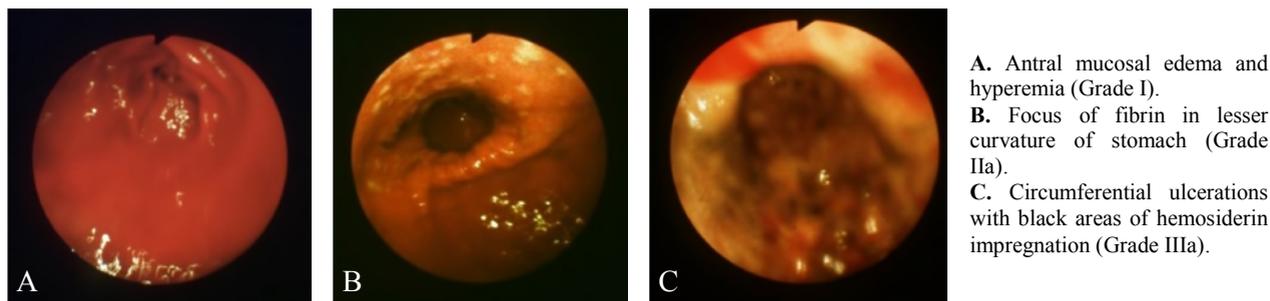
Variables	Suicidal, n=252	Accidental, n=148	P value
Age (median (range))	39 (14-87)	53 (16-86)	<0.001
Acetic acid amount (median (range))	70 (10-250)	50 (10-200)	<0.001
Time until medical care (median (range))	2 (0.5-96)	4 (0.5-96)	0.004
Death (%)	23.4	17.6	0.21
Stricture (%)	14.3	6.1	0.019
Hemolysis (%)	60.7	45.9	0.006
Psychosis (%)	13.9	25	0.008
Initial bleeding (%)	31.7	19.6	0.012
Repeated bleeding (%)	17.1	16.9	0.931

**Table 2.** Demographic and clinical characteristics according to endoscopic GI injury grading.

Variables	Endoscopic grade					
	I	IIa	IIb	IIIa	IIIb	
No. of patients (F/M)	66 (36/30)	117 (68/49)	120 (68/52)	27 (12/15)	70 (38/32)	
Age (median (range))	34 (15-80)	39 (14-89)	49 (16-82)	54 (17-88)	58 (24-82)***	
Acetic acid amount (median (range))	50 (20-150)	50 (10-250)	85 (15-200)	100 (30-170)	100 (50-150)***	
Death (%)	1 (2)**	0	10 (8)	11 (41)	63 (90)***	
Stricture (%)	0	2 (2)***	25 (21)***	11 (41)***	7 (10)***	
Hemolysis (%)	11 (17)***	39 (33)***	82 (68)***	23 (85)***	66 (94)***	
Pneumonia (%)	4 (6)***	7 (6)***	30 (25)	18 (67)	47 (67)***	
Laryngeal edema (%)	7 (11)*	13 (11)**	34 (28)**	10 (37)*	16 (22)***	
Initial bleeding (%)	4 (6)	14 (12)	34 (28)	13 (48)	44 (63)	
Repeated bleeding (%)	2 (3)	2 (2)	19 (16)	13 (48)	32 (46)***	
Kidney damage (RIFLE scale) <sup>a</sup>	R (%)	8 (12)	5 (4)*	10 (8)	2 (7)	8 (11)
	I (%)	2 (3)	0**	10 (8)*	3 (11)	5 (7)
	F (%)	1 (2)*	3 (3)**	11 (9)	3 (11)	20 (29)***
	L (%)	2 (3)**	1 (1)***	12 (10)	11 (41)***	24 (34)***
E (%)	0	0	0	0	0	
Days of ICU admittance of survived patients (median (range))	1 (0-94)	3 (0-70)	7 (1-61)	19 (2-52)	34 (19-63)**	
Days of ICU admittance of deceased patients (median (range))	10	-	18 (1-71)	17 (5-48)	5 (1-47)***	

\* P < 0.05, \*\* P < 0.01, \*\*\* P < 0.001 (Chi-Square test)

<sup>a</sup> RIFLE; Risk of renal dysfunction, Injury to the kidney, Failure of kidney function, Loss of kidney function and End-stage kidney disease



**Figure 2.** Endoscopic examinations of patients with 70 percent acetic acid poisoning.

**Table 3.** Treatments administered for patients with acetic acid ingestion

Treatment	Patients, n (%)
Gastric lavage	249 (62)
Enteral nutrition (NG tube)	119 (30)
Partial parenteral nutrition	89 (22)
Epidural anesthesia	43 (11)
Erythrocyte transfusion	86 (21.5)
Fresh frozen plasma (FFP)	90 (23)
Artificial ventilation	98 (24.5)
Antacids (H2 blockers, PPIs)	400 (100)
Prednisolone (IV)	235 (59)

### Treatments

Treatments which were administered for the patients during hospital admission are summarized in table 3. Although gastric lavage was implemented for 249 cases (62%), only 30 of them were during the first hour post-ingestion. Respiratory failure in 98 cases (24.5%) necessitated artificial ventilation with a median duration of 7 days (1– 81days). IV prednisolone 1-2 mg/kg was administered for 235 patients (59%) for a median of 3 days (range 1-32). Although, for enhancing the post-inflammation repair process and achieving the steroid-mediated arrest of fibroblast proliferation, a minimum of 10-day course of steroid therapy has been found to be essential (11,12), 44 patients received IV prednisolone for 1 day, 80 patients for 2-3 days, and only 43 patients for 10 days and more.

**Table 4.** Predictors of hospital mortality in acetic acid ingestion

Variables	Survival, n	Death, n	Crude		Adjusted		
			OR	95% CI	OR	95%CI	
Age, yr	16-19	27	0	-	-	-	
	20-29	66	3	Ref. <sup>a</sup>		Ref.	
	30-39	63	10	3.5	0.9-13.3	3.6	0.3 – 55.4
	40-49	51	11	4.8*	1.3-17.9	0.9	0.07 – 11.0
	50-59	54	19	7.7***	2.2-27.6	0.9	0.08 – 10.8
	60-69	21	18	18.9***	5.1-70.4	17.3*	1.1 – 270.7
	70-79	30	19	13.9***	3.8-50.7	15.2*	1.3 – 181.3
	80-89	6	5	18.3***	3.5-96.2	2.3	0.1 – 51.5
Acetic acid dose, mL	10-59	165	24	Ref.		Ref.	
	60-100	113	35	2.1**	1.2 – 3.8	0.7	0.2 – 2.7
	>100	24	16	4.6***	2.1– 9.8	0.7	0.1 – 3.5
Pneumonia	Absent	273	25	Ref.		Ref.	
	Unilateral	34	28	9.0***	4.8 – 17.2	4.4*	1.1 – 17.2
	Bilateral	11	32	31.8***	14.3 – 70.6	6.9**	1.6 – 30.1
Kidney injury	No injury	248	14	Ref.		Ref.	
	R	24	9	6.6***	2.6 – 16.9	2.5	0.4 – 14.7
	I	12	8	11.8***	4.2 – 33.6	15.5***	2.0 – 121.9
	F	16	22	24.4***	10.5 – 56.4	6.2*	1.1 – 36.3
	L	18	32	31.5***	14.3 – 69.4	7.1*	1.5 – 34.4
Zargar's grading classification	I	69	1	Ref.		Ref.	
	IIa	116	0	-		-	
	IIb	110	10	6.3	0.8 – 50.1	1.6	0.2 – 17.2
	IIIa	16	11	47.4***	5.7 – 394.5	22.5*	1.8 – 385.9
	IIIb	7	63	621.0***	74.3 – 5188.9	206.8***	16.2 – 2647.7
Prednisolone use (days)	Not administered	148	19	Ref.		Ref.	
	1-3	90	35	3.0***	1.6 – 5.6	4.4*	1.6 – 18.4
	>3	80	31	3.0***	1.6 – 5.7	7.8***	1.7 – 36.5

<sup>a</sup> For each variable, outcomes were compared to the reference group (Ref.) which was the largest group within the variable.

\* P < 0.05, \*\* P < 0.01, \*\*\* P < 0.001 (logistic regression)

Cells with no data are related to the groups with small sample size encompassing a low statistical power.

Sixty-seven patients underwent hemodialysis due to acute renal failure. Instead of heparin, anticoagulation was achieved with a solution of 4% sodium citrate, as it has been previously shown that using sodium citrate for hemodialysis prevents bleeding aggravation in patients with acetic acid poisoning (13). Nevertheless, 48 patients (72%) developed bleeding on a median of tenth day (range 3-19), and this was the main cause of death in half of the deceased patients with acute kidney failure.

#### Predictors of mortality and stricture formation

In order to identify potential factors of hospital mortality and stricture formation in patients with acetic acid ingestion, stepwise logistic regression analysis was done. Pneumonia (unilateral (OR=4.4, 95% CI) and bilateral (OR=6.9, 95% CI), stage "Injury" (OR=15.5, 95% CI), stage "Failure" (OR=6.2, 95% CI) and stage "Lost" (OR=7.1, 95% CI) of kidney injury, and grade IIIa (OR=22.5, 95% CI) and IIIb (OR=206.8, 95% CI) of GI injury were found to be significantly associated with mortality (Table 4). Moreover, it was found older ages (60-79 years) and prednisolone administration both in short and long courses were significant mortality predictors.

In addition, multiple logistic regression analysis revealed that ingestion of over 100 ml acetic acid (OR=5.21, 95% CI), grades IIb (OR=14.62, 95% CI) and IIIa (OR=138.6, 95% CI) as significant predictors of stricture formation (Table 5).

Besides, it should be noted that because all survived patients with grade IIIb GI injury developed strictures, logistic regression analysis for stricture formation was not possible for this subgroup.

## DISCUSSION

Acetic acid ingestion in concentrations exceeding 12% causes corrosive effects and can also be absorbed systemically inducing shock, acidosis, hemolysis and renal injury (1,4). In addition, acid aspiration may cause lung injury and pneumonia (14). In our study, we found that systemic complications such as hemolysis occurred more frequently than GI complications. Conversely, Cheng et al. showed that ingestion of caustic substances in general, can cause less systemic complications (20.5%) compare to GI injuries (27.8%) (10).

Suicidal intention in caustic poisonings is reported from 61 to 71% in different studies (10, 15, 16). Christesen in a 16-year retrospective study showed that caustic substance ingestion with suicidal intention was more common among females (61%), while in case of unintentional poisonings, the number of males and females were equal (15). In contrast, in our study 70% of unintentional poisonings were males. This can be explained by the predominance of alcohol drinking behavior in men and the common tradition of keeping an acetic acid bottle (which looks similar to a small bottle of

**Table 5.** Predictors of stricture formation in acetic acid ingestion

Variables	Survival without stricture, n	Survival with stricture, n	Crude		Adjusted		
			OR	95% CI	OR	95% CI	
Age, yr	16-19	25	2	0.98	0.18 – 5.37	2.02	0.23 – 17.83
	20-29	61	5	Ref. <sup>a</sup>		Ref.	
	30-39	55	8	1.78	0.55 – 5.75	1.59	0.35 – 7.25
	40-49	43	8	2.27	0.7 – 7.41	1.71	0.37 – 7.92
	50-59	37	17	5.61**	1.91 – 16.47	3.23	0.79 – 13.24
	60-69	18	3	2.03	0.44 – 9.34	0.96	0.12 – 7.68
	70-79	28	2	0.87	0.16 – 4.77	0.40	0.04 – 4.20
	80-89	5	1	2.44	0.24 – 25.14	2.41	0.08 – 70.10
Acetic acid dose, mL	10-59	154	11	Ref.		Ref.	
	60-100	91	22	3.39**	1.57 – 7.30	1.88	0.68 – 5.23
	>100	13	11	11.85***	4.31 – 32.50	5.21*	1.25 – 21.73
Zargar's grading classification	I	69	0	-		-	
	IIa	114	2	Ref.		Ref.	
	IIb	84	26	17.08***	4.075 – 76.394	14.62***	3.24 – 65.91
	IIIa	5	11	125.4***	21.73 – 723.58	138.6***	18.79 – 1022.6
	IIIb	0	7	-			
Prednisolone use (days)	Not administered	127	21	Ref.		Ref.	
	1-3	78	12	0.93	0.43 – 2.0	0.87	0.3 – 2.56
	>3	67	13	1.17	0.55 – 2.49	0.68	0.23 – 2.03

<sup>a</sup> For each variable, outcomes were compared to the reference group (Ref.) which was the largest group within the variable.

\* P < 0.05, \*\* P < 0.01, \*\*\* P < 0.001 (logistic regression)

Cells with no data are related to the groups with small sample size encompassing a low statistical power.

vodka) in the refrigerator. In our study, the median amount of acetic acid ingested was significantly lower in accidental poisonings. In spite of this fact, we did not find any significant difference between accidental and suicidal cases regarding the rate of repeated bleeding and the mortality rate. The high mortality rate in accidental cases in our study can be explained by development of psychosis and respiratory insufficiency in alcoholic patients (17).

Our patients with grade IIb, IIIa and IIIb had worst outcomes as 8%, 41% and 90% of them died and among those who survived 22%, 68% and 100% developed strictures respectively. Similarly, it has been ascertained in some studies that the most complicated grades of GI injury were the grades IIb, IIIa and IIIb (9,10). Nevertheless, the mortality rate of our patients with grade III (a and b) GI injury was much higher than mortality rate of ingestion of other caustic substances with the same GI injury grade (10,16).

It was found in this study that pneumonia, stages "Injury", "Failure" and "Lost" of kidney damage and grades IIIa and IIIb of GI injury according to Zargar's scale were the strongest risk factors of mortality. Likewise, in several studies about caustic substance ingestion, it was found that the grade of GI mucosal injury (assessed with endoscopy or CT scan) was a strong predictive factor for the occurrence of GI and systemic complications and mortality (3,10,15). The higher mortalities among patients with renal injury in our study can be explained by anticoagulation applied during the hemodialysis course (1), which exposes the patients at risk of uncontrollable bleeding from the affected sites of the GI tract. In this study, we also showed that patients with older ages are at higher risk of mortality following acetic acid ingestion. Correspondingly, Chang et al. demonstrated that elderly patients (>65) are at the greatest mortality risk following caustic ingestion (18).

Ingestion of over 100 ml acetic acid, grades IIb and IIIa GI injury were found as significant predictors of stricture formation in this study. Similarly, Cheng et al. and Ryu et al. demonstrated that higher grades of GI injury were at higher risks of developing strictures (10,16).

### LIMITATIONS

In this study, following factors may have limited the validity of findings. The amount of acetic acid ingested was based on the patient or family history that might be unreliable. Since caustic injuries have a rapid onset, the initial therapy may have a significant impact on the ultimate outcome. However, more than half of the patients were transferred from other hospitals and we cannot be sure if gastric lavage and IV infusion were performed correctly. Moreover, the patients received different doses of prednisolone and the duration of prednisolone treatment also varied. Keh et al. (19) considered that follow up endoscopy should be provided within 6 weeks post-discharge but unfortunately such data were not available due to lack of cooperation of our patients.

### CONCLUSION

Highly concentrated acetic acid is still frequently ingested in Russia with a high mortality rate. Patients with higher grades of GI injury, pneumonia, renal injury and higher

amount of acid ingested should be more carefully monitored as they are more susceptible to develop fatal consequences.

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### REFERENCES

1. Brusin KM, Baygozina OK, Mjachkova LP, Novikova OV, Chekmarev AV, Yentus VA. A Study of Outcomes of Steroid Therapy in Severe Acetic Acid Poisoned Patients. Abstracts of the 2010 International Congress of the European Association of Poisons Centres and Clinical Toxicologists; 2010 May 11-14, Bordeaux, France. *Clin Toxicol (Phila)* 2010; 48(3):247. [Abstract].
2. Brusin KM, Kondrashov DL, Krayeva YV, Sentsov VG, Hovda KE. A One-Year Observational Study of Fatal Poisonings in Yekaterinburg, Russia. Abstracts of the 2011 International Congress of the European Association of Poisons Centres and Clinical Toxicologists; 2011 May 24-27, Dubrovnik, Croatia. *Clin Toxicol (Phila)* 2011; 49(3):237. [Abstract].
3. Poley JW, Steyerberg EW, Kuipers EJ, Dees J, Hartmans R, Tilanus HW, et al. Ingestion of acid and alkaline agents: outcome and prognostic value of early upper endoscopy. *Gastrointest Endosc* 2004 Sep; 60(3):372-7.
4. Davids PH, Bartelsman JF, Tilanus HW, van Lanschot JJ. Consequences of caustic damage of the esophagus. (in Dutch) *Ned Tijdschr Geneesk* 2001 Nov; 145(44):2105-8.
5. Sarmanaev SKh. Diagnostic value of determination of serum enzyme activities in patients with gastrointestinal lesions caused by caustic substances. (in Russian) *Klin Med (Mosk)* 2009; 87(2):32-5.
6. Govorin AV, Vitkovski DluA, Rutschina EA, Solpov AV, Sokolova NA, Bodko EV, et al. Lymphocyte-platelet adhesion and the content of D-dimers in patients with acute acetic acid poisoning. (in Russian) *Anesteziol Reanimatol*. 2008 May-Jun; (3):69-71.
7. Arevalo-Silva C, Eliashar R, Wohgelernter J, Elidan J, Gross M. Ingestion of caustic substances: a 15-year experience. *Laryngoscope* 2006; 116(8):1422-6.
8. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P. Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Crit Care* 2004; 8(4):204-12.
9. Zargar SA, Kochhar R, Mehta S, Mehta SK. The role of fiberoptic endoscopy in the management of corrosive ingestion and modified endoscopic classification of burns. *Gastrointest Endosc* 1991; 37:165-9.
10. Cheng HT, Cheng CL, Lin CH, Tang JH, Chu YY, Liu NJ, et al. Caustic ingestion in adults: the role of endoscopic classification in predicting outcome. *BMC Gastroenterol*. 2008 Jul 25; 8:31.
11. Fulton JA, Hoffman RS. Steroids in second degree caustic burns of the esophagus: a systematic pooled analysis of fifty years of human data: 1956-2006. *Clin Toxicol (Phila)* 2007 May; 45(4):402-8.
12. Howell JM, Dalsey WC, Hartsell FW, Butzin CA. Steroids for the treatment of corrosive esophageal injury. *Am J Emerg Med* 1992; 10:421-5.

13. Sentcov VG, Nazarov AV, Drujinin NV, Brusin KM, Novikova OV, Sukhanov VA, Egorov VM. Bleeding Complications in Acetic Acid Poisoning Patients with Acute Renal Failure are Diminished with Citrate Hemodialysis. Abstracts of the European Association of Poisons Centres and Clinical Toxicologists XXV International Congress. Clin Toxicol (Phila) 2005; 43(5):511. [Abstract].
14. Tseng YL, Wu MH, Lin MY, Lai WW. Outcome of acid ingestion related aspiration pneumonia. Eur J Cardiothorac Surg 2002; 21(4):638-43.
15. Christesen HB. Caustic ingestion in adults--epidemiology and prevention. J Toxicol Clin Toxicol 1994; 32(5):557-68.
16. Ryu HH, Jeung KW, Lee BK, Uhm JH, Park YH, Shin MH, et al. Caustic injury: can CT grading system enable prediction of esophageal stricture? Clin Toxicol (Phila) 2010 Feb; 48(2):137-42.
17. Adrian M, Barry SJ. Physical and mental health problems associated with the use of alcohol and drugs. Subst Use Misuse 2003; 38(11-13):1575-614.
18. Chang JM, Liu NJ, Pai BC, Liu YH, Tsai MH, Lee CS, et al. The role of age in predicting the outcome of caustic ingestion in adults: a retrospective analysis. BMC Gastroenterol 2011 Jun 14; 11:72.
19. Keh SM, Onyekwelu N, McManus K, McGuigan J. Corrosive injury to upper gastrointestinal tract: Still a major surgical dilemma. World J Gastroenterol 2006; 12(32):5223-8.