

CASE REPORT

Acute Renal Failure Following Brake Fluid Poisoning

SEYED MOHAMMAD NAVABI, JAFAR NAVABI*, HOSSEIN POLADI, FERESHTA ZAHERI

For author affiliations, see end of text.

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ABSTRACT

Brake fluid is a mixture of different Glycol derivatives which are poisoning and their ingestion triggers significant CNS symptoms, severe metabolic acidosis, cardiac, pulmonary, and renal failure. Here in this study, a case is reported who had been ingested 150mL of brake fluid for suicidal attempt.

Keywords: *Acute renal failure, Brake fluid, Glycol derivatives, Poison*

Brake fluid contains a mixture of different Glycol including Diethylene glycol, ethylene glycol, polyethylene glycol, polypropylene glycol ether. Ethylene glycol poisoning is more common; however, poisoning with brake fluid is very rare. Ethylene glycol (antifreeze) and Diethylene glycol are colorless, sweet-tasting fluids that have many applications in industry [1-5].

Ethylene glycol is metabolized by alcohol dehydrogenase to toxic metabolites including glycol aldehyde, glycolic acid and glyoxalic acid. This toxic metabolite (glycolic acid) appears to be primarily responsible for the metabolic acidosis observed in this condition. Intoxication is characterized by profound CNS symptoms, including seizures and coma, severe metabolic acidosis, cardiac, pulmonary, and renal failure [1-2, 5]. Here in this study, an experience on acute brake fluid poisoning with acute renal failure is reported.

CASE REPORT

A 20-year old man was admitted to hospital with abdominal pain and vomiting. He had been ingested 150mL of brake fluid for suicidal attempt. He was admitted 4 hours after ingestion. Although the patient was alert, he suffered from severe poisoning. The degree of poisoning was assessed by the quantity of brake fluid volume ingested.

On admission, physical examination revealed normal blood pressure (110/70 mm Hg), the temperature

was 36.7, respiratory rate was 18 and there were severe agitation and generalized tenderness in abdominal palpation. The patient had no urine output. Blood analysis showed metabolic acidosis and acute renal failure. Table 1 shows results of the performed renal and liver function tests.

The patient was preliminary treated with normal saline, bicarbonate and furosemide in the emergency ward. After 24 hours, daily hemodialysis was performed. Results of biochemical and other tests of patient during hospitalization are presented in Table 2 shows. The patient was anuric in the beginning, then oliguric and finally he had urine output about 1200 mL in 24-hours. The initial urines' color was bloody and then it became clears. On the 6th day after admission, the patient developed severe pain in limbs, restlessness and dyspnea. As a matter of fact, treatment of pulmonary edema was performed for the patient. On the 7th day, cardiac and pulmonary arrest occurred during dialysis. CPR was performed and the patient was transferred to ICU. The patient was in a deep coma (GCS = 3) in ICU. Meanwhile, he developed recurrent seizures and severe hemorrhage from multiple organs and therefore, diagnosis and treatment of DIC and seizure were conducted. Unfortunately, the patient died suddenly after 24 hours, due to severe bradycardia and cardiac arrest. At autopsy, enlarged kidneys and on slice, bleeding and necrosis of renal cortex were seen. Multiple organ involvement (brain, cerebellum, heart, lung, liver, spleen and gastrointestinal system) were observed at autopsy. In histopat-

Table 1. Renal and liver function tests of patient

Days	Urea	AST	ALT	Cr	ALP	PT	PTT	BIL-T	NA	K	U/A
1	87	326	361	2.7	139	13.6	35	0.8	138	4.0	Anuric
2	130	166	239	6.2	170	18.0	53	1.0	137	3.8	Anuric
3	164	133	279	7.8	179	18.2	120	0.8	137	3.9	Anuric
4	179	126	279	8.1	185	-	-	-	137	3.8	WBC=Many RBC=Many Glucose2+ Albumin=2+
5	190	304	541	9.1	220	17.0	65	1.2	135	3.4	Active
6	240	207	458	7.5	230	14.0	45	1.0	142	5.4	Active
7	231	--		11.5		-	-	-	-	-	-
8	145	218	460	11.8	458	-	-	-	142	5.0	-

hology reporting: Edema, hemorrhage, inflammation and necrosis of the renal cortex were seen. Cerebellar neuronal ischemia, severe edema around the vessels and the deposition of hemosiderin in macrophages were quite inevitable. Also pulmonary edema and alveolar hemorrhage and centrilobular necrosis of liver were observed.

DISCUSSION

Acute renal failure usually occurs after 24 hours of toxin exposure in 73% -84% of cases [2]. However, 4 to 5 hours after ingestion of brake fluid the target patient developed acute renal failure. Half-life of ethylene glycol is 3 hours, therefore if the patient is admitted after 24 to 72 hours, ethanol administration isn't indicated [1-2]. Ethylene glycol poisoning may cause myocarditis without neurological symptoms [6].

In some countries, ethylene glycol is the most common type of poisoning and may be observed along with encephalopathy, coma and brain stem injury [7]. Also, bilateral facial nerve palsy has been reported previously [8]. In one study from Slovakian, in addition to metabolic acidosis and acute renal failure, acute toxic hepatitis was reported [9].

Calcium oxalate crystals in the urine proposed as a diagnostic test [10]. Treatment of poisoning with ethylene glycol is based on estimate of the bicarbonate deficit. When an osmolar gap exists, competitive inhibition of alcohol dehydrogenase should be initiated with fomepizole or ethanol. Hemodialysis is required in severe cases. If the diagnosis is made promptly and appropriate therapy is initiated, outcomes are favorable. Renal failure may be reversible [11].

Table 2. Results of biochemical and other tests of patient during hospitalization

Days	CPK	TroponinI	Uric acid	Ca/p	Mg	BS	Amylase/Lipase	WBC/Hgb/plt	ABG
1	129	2	-	9/3.4	2.2	128	116/35	19.600/14.4/160000	PH=7.25 PCO2=41 PO2=72 Hco3=18
2	-	-	-	-	-	-	-	-	PH=7.15 PCO2=43 PO2=58 Hco3=14
3	54	-	-	-	-	-	15/54	-	-
4	63	-	7.5	9/3.4	-	-	-	-	-
5	-	-	-	9/7.6	2.1	135	-	22.700/11/92000	-
6	-	-	8	8/7	-	202	-	-	PH=7.32 PCO2=39 PO2=49 Hco3=18
7	-	-	-	-	-	-	-	-	-
8	-	-	-	9/7.9	2	136	-	20000/10/81000	-

Based on animal studies, ingestion of ethylene glycol 1-1.5ml/kg or 100 ml is sufficient for an adult poisoning [12]. Ingestion of 100 ml of ethylene glycol (1.8 mole, equivalent of about 110 grams) kills an adult and should be treated with ethanol and if renal failure occurs, hemodialysis or peritoneal dialysis should be administered as soon as possible [13].

Brake fluid poisoning should be treated similar to poisoning with ethylene glycol or methanol. This treatment includes administration of bicarbonate, fomepizole or ethanol and immediate hemodialysis if necessary.

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REFERENCES

- Sharma N, Jain S. Toxicity of brake oil. *Emerg Med J* 2002; 19:267-268.
- Turk J, Morrell L, Avoili LV. Ethylene glycol intoxication. *Arch Intern Med* 1986; 146:1601-1603.
- Megges G. Analytical techniques and results in a case of fatal poisoning by brake fluid. *ZRechtsmed* 1979; 83(1):81-85.
- Nahir S, Sinha S, Siddiqui KA. Brake fluid toxicity feigning brain death. *BMJ* 2012. doi: 10.1136/bcr-02-2012-5926.
- Vaphiades MS. Visual and hearing loss from percutaneous brake fluid toxicity. *Am Orthopt J* 2005; 55:136-138.
- Denning DW, Berendt A, Chia Y, Chia Y, Morgan S. Myocarditis complicating ethylene glycol poisoning in the absence of neurological features. *Postgrad Med J* 1988; 64: 867-870.
- Sienkiewicz J, Kwieciński H. Acute encephalopathy in ethylene glycol poisoning. *Wiad Lek* 1992; 45(13-14):536-539.
- Marczyńska-Wolańska H, Hasięc T, Ksiazek A. Bilateral peripheral facial nerve palsy in acute poisoning with ethylene glycol. *Pol Tyg Lek* 1989; 44(46-48):965-976.
- Mydlík M, Derzsiová K, Mizla P, Böör A, Macingová E, Slosarčíková L. Diagnosis and therapy of ethylene glycol poisoning--analysis of 20 patients. *Vnitr Lek* 2002; 48(11):1054-1059.
- Andreelli F, Blin P, Codet MP, Fohrer P, Lambrey G, Massy Z. Diagnostic and therapeutic management of ethylene glycol poisoning. Importance of crystalluria. Apropos of a case. *Nephrologie* 1993; 14(5):221-225.
- JULIAN SEIFTER, Acid-Base Disorders, In: GOLDMANS CECIL MEDICINE. LEE GOLDMAN& ANDREW I. SCHAFER, 24th ed. Philadelphia:SAUNDERS, ELSEVIER ; 2012.P,741-748.
- Barceloux DG, Krenzelok EP, Olson K, Watson W. American Academy of Clinical Toxicology practice guidelines on the treatment of ethylene glycol poisoning. Ad Hoc Committee. *J Toxicol Clin Toxicol* 1999; 37:537-560.
- Vale JA, Prior JG, O'hare JP, Flanagan RJ. Treatment of ethylene glycol poisoning with peritoneal dialysis. *BMJ FEB* 1982; 284(20): 557

CURRENT AUTHOR ADDRESSES

SEYED MOHAMMAD NAVABI, Kermanshah University of Medical Sciences, Kermanshah, Iran.

JAFAR NAVABI, Department of Internal Medicine, Imam Khomeini Hospital, Kermanshah University of Medical Sciences, Kermanshah, Iran. E-mail: jafarnavabi@kums.ac.ir (Corresponding author).

HOSSEIN POLADI, Legal Medicine Research Center, Kermanshah Province-General Office of Legal Medicine, Kermanshah, Iran.

FERESHTE ZAHERI, Legal Medicine Research Center, Kermanshah Province-General Office of Legal Medicine, Kermanshah, Iran.