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Case Report

Ethylene Glycol Toxicity Following Brake Oil Ingestion

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Abstract

Break oil is used as a lubricant in automobiles. Children and predisposed individuals with a history of mental illness are particularly at risk of intoxication. The wide range of toxic doses and delay in onset made it difficult to categorise the severity of intoxication at presentation. A 17-years-old boy presented to emergency department with excruciating abdominal pain. He was drowsy had a GCS of 12/15 and had been anuric for the previous 24hrs. The Patient disclosed that he ingested about 50ml of break oil as a suicidal attempt. Labs revealed severe metabolic acidosis with acute kidney injury at presentation. Due to late presentation and severe metabolic acidosis with kidney injury, immediate hemodialysis was planned. Three sessions of hemodialysis yielded considerable improvement over course of 3 days but patient died due to cardio toxicity on 4th day of treatment. Break oil poisoning has several clinical evolutions through the deposition of harmful metabolites. After intoxication patient can present in any phase of toxicity. Cardiopulmonary involvement and delayed treatment could be fatal as seen in this case. Early treatment of metabolic acidosis should be done to prevent vital organs from toxicity.

Keywords: Break lubricants, cardio toxicity, Acute kidney injury.

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Introduction

Break oil consists of highly water-soluble compounds like diethylene glycol, ethylene glycol and glycol ethers. Some Break lubricants also contain boric acid in addition. Break oil is used as a lubricant in automobiles, as an oil for clutches and in hydraulic breaks. The major constituent of break oil is ethylene glycol which is water soluble and is readily absorbed from stomach. Its half-life is 7 to 10 hours and it metabolized in liver by alcohol dehydrogenases to more toxic and lethal compounds like glycol aldehyde and oxalic acid[1]. The non-toxic metabolites require thiamine and pyridoxin as a cofactor so in the early phase of intoxication, administration of these vitamins will help to convert ethylene glycol to non-toxic compounds, as liver stores get depleted in acute toxicity[2]. We report a case of ethylene glycol intoxication following brake fluid ingestion presented with severe pain abdomen and altered sensorium complicated with severe metabolic acidosis, acute renal failure and acute liver failure. Cardio toxicity leads to fatal demise of our patient.

Case Presentation

A 17-years-old young boy was transferred from a local hospital with history of excruciating abdominal pain, nausea, anorexia and fever. These symptoms got worse with time and lasted for 48 hours without any improvements. At presentation to emergency department his vitals were: Pulse 120 beats per minute, Blood pressure 110/70, Respiratory rate of 28 cycles per minute, oxygen saturation was 95% at room air, Random sugar was 115mg/dl, anuric from last 24hrs, GCS was 13/15 and bilateral pupils were reactive to light.

Our initial working diagnosis was sepsis with multiorgan failure, treatment was initiated accordingly—blood culture obtained; but we could not explain the reasonable source of septicemia. After some improvement of pain with painkillers (Ketorolac 30mg and Tramadol 50mg) patient disclosed the history of suicidal attempt in which he self-ingested break oil about 40 to 50ml.

On admission his renal functions and liver functions were deranged. His serum creatinine was 11.5 mg/ dL, blood urea of 183 mg/dL, Na+ 124 mmol/l, and K 4.1 mmol/l. Arterial blood gases showed pH 7.29, partial pressure of oxygen (PO2) 100, partial pressure of carbon dioxide (PCO2) 28, bicarbonate (HCO3) 9.3, and base excess−13 mEq per liter. His measured serum osmo-

lality was 304 mOsm with an osmolar gap of 17 mOsm/kg and anion gap was 29 mEq/l. Liver profile showed total bilirubin of 3.1mg/dL, Alanine transferases 551U/L and alkaline phosphatase 223U/L. Although relevant, his urine could not be examined for calcium oxalate crystals because of anuria. Ultrasound abdomen and pelvis was suggestive of multiple hyper echoic bilateral kidneys and small concretions. Full blood count showed hemoglobin of 12.7 g/dl, platelet of 185 × 106 /L, and white cell count of 15.2 × 106 /L. His random blood sugar was 115 mg/dl. Serum calcium was 10.4mg/dl. A chest X-ray did not reveal any abnormalities such as consolidation or pleural effusion. He was started on consecutive daily dialysis which yielded a considerable improvement in renal functions detail shown in Table1, and conscious level, after 3 sessions 1 in 24hrs but patient remained anuric for days. On 4th day of admission (6th day of poisoning) patient suddenly developed shortness of breath and gasping, and became hemodynamically unstable, patient was resuscitated with CPR and ambo bagging with oxygen of 10 Liters, injection Hydrocortisone 100mg intravenous was given. Immediately ECG was done which showed diffused ST elevations in all leads. Patient was loaded with Aspirin 300mg through N/G tube. Troponin I with all base line ordered and patient was shifted to ICU. In ICU patient was intubated for ventilator due to poor respiratory efforts. Lab results revealed massive raised trop I with value of 21ng/ml. After 6 hours patient developed Ventricular fibrillation and wasn't reverted back, on visual autopsy there was no petechia or hemorrhage noted in mucosal membranes. Cardiotoxicity secondary to break oil poisoning lead to death of patient.

Time	RFTs		Electrolytes			LFTs			Arterial Blood gases				CBC		
Since poisoning	Urea	Creatinine	Na		Cl	ALT	ALP	Bilirubin	PH	PCO_2	PO_2	HCO_3	WBC	HB	Platelets
21July(10-12Hr)	84	4.6	-	-	-	-	-	-	-	-	-	-	18.8	13.1	159
22July	No Record Available														
23July	189	11.5	126	4	90	551	223	3.1	7.2	18	100	9.3	23.6	12.1	176
POST HEMODIALYSIS BLOOD INVESTIGATIONS															
24July	200	11	130	4	93	650	200	3.6	7.2	20	100	13	16	12.5	150
25July	150	8.5	132	4	95	950	186	4.1	-	-	-	-	-	-	-
26July	118	6.2	133		97	1443	134	4.6	7.3	28	100	18.3	13.9	11.1	112

Discussion

Ethylene glycol is a toxic alcohol, implicated in both accidental and intentional poisoning. The toxic and lethal dose of ethylene glycol is not fully understood but Robert Hess et al states that lethal dose of ethylene glycol is 1600mg/Kg body weight, while 20 to 500ml intoxication is necessary to produce systemic side effects[3]. Study done in Romania states that 1-time 150ml to 1500ml consumption of pure ethylene glycol causes death in humans, while in animals lethal dose is higher than humans[4]. A Patient can land in any phase irrespective of time[2]. Toxicity is divided in 3 phases: 1) neurologic phase (0.5 to 2hr) patient is drowsy, can have nystagmus, convulsions and depressed conscious level 2) Cardiopulmonary phase (12 to 24hours) patient develops cardiopulmonary signs 3) Renal insufficiency, lasting 24-72hr. There are studies reporting that nephrotoxicity is critical end point[3,5]. In our case, late presentation, nephrotoxicity and severe metabolic derangement at presentation are the main risk factor leading to fatal outcome in offender. Another study linked poor prognosis to metabolic acidosis, coma and hyperkalemia[4]. Myocardial toxicity occurs due to hypocalcemia and sever metabolic acidosis[2,5]. To prevent urinary crystallization the goal of urinary PH greater than 7 is necessary with sodabicarbonate[6]. Acidosis causes depression of myocardial activity and can lead to fatal arrythmias.

Conclusion

Break oil poisoning has several clinical evolutions through deposition of harmful metabolites. Precise dose of ethylene glycol was not defined. After intoxication patient can present in any phases of toxicity irrespective of time. Some studies defined Renal failure as critical end point. Cardiopulmonary involvement and delayed treatment can be fatal as seen in this case. Early treatment of metabolic acidosis should be done to prevent vital organs from toxicity. Low albumin binding capacity, water solubility and decreased volume of distribution are factor which made hemodialysis ideal in cases with late presentation, electrolytes disturbance and severe metabolic acidosis.

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