# Ethylene Glycol Intoxication Following Brake Oil Ingestion: A Case Report

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Brake oil is a type of hydraulic fluid used in automobiles. It consists of toxic compounds like ethylene glycol, diethyleneglycol and additives. Brake oil poisoning is rare and it can either be due to intentional or accidental[1,2]. Toxic manifestations are mainly due to metabolites rather than the compounds in brake oil[3,4]. Immediate haemodialysis and other supportive carecan improve the outcome of intoxicated patients. Here we present a case report of successful management of a patient presented following brake oil poisoning.

**Key words:** Brake oil, Poisoning, Acute kidney injury

## Introduction

Brake oil is a type of hydraulic fluid used in hydraulic brake and clutch applications in automobiles. It is a mixture of toxic compounds such as glycol ether, ethylene, or diethylene glycol and additives. Ethylene glycol, diethylene glycol and its metabolites are responsible for the toxic effects. Intoxicated patient will have neurological, cardiorespiratory, renal and gastrointestinal complications during the course. Brake oil poisoning may present either as accidental or intentional. However, most deaths reported due to intentional poisoning and deaths from accidental poisoning are rare.

## Case history

A 51-year-old gentleman was admitted to the Intensive care unit(ICU)with a history of shortness of breath, agitation and reduced urine output following brake oil ingestion two days back. On admission to ICU patient's respiratory rate was 46/min and SpO2 was 100% with 60% of oxygen. He had a pulse rate of 140/min and blood pressure of 140/80 mmHg.

On admission, patient was severely acidotic with stage 3 AKI. His arterial blood gas values on admission were as follows; pH 6.946, pCO2 8.5mmHg, pO2 65.4mmHg, HCO3 1.9mmol/l, $sHCO_3$ 6.6mmol/l, BE – 25.4mmol/l, lactate 1.5mmol/l, anion gap 25mmol/l. His urine output had reduced and it was 155ml for 12 hours and his serum creatinine was 4.26 mg/dl. However, his serum electrolytes were within normal range. Serum calcium was 7.8 mg/dl. Full blood count showed haemoglobin of 143 g/l, white cell counts of 23600/ml and platelet of 117000/ml. His C- reactive protein was 67.4mg/l. His blood sugar was 144mg/dl. His urine analysis revealed oxalate crystals.

He was immediately treated with IV 100ml of 8.4% $NaHCO_3$and he was intubated and started on mechanical ventilation. Throughout ICU stay he was tachycardic and hypertensive. ECG showed sinus tachycardia and there was no QT prolongation.

Continuous renal replacement therapy(CRRT) using veno-venous haemodialysis was initiated within the first hour in intensive care unit. In spite of 12 hours of dialysis, he remained acidotic. pH was 7.0 with $HCO_3$ of 6.7mmol/l. Therefore, he was prescribed 100 ml of 8.4% $NaHCO_3$ three doses for next 24 hours. CRRT was continued for 04 days. The patient was extubated on day 04. However, he remained anuric and creatinine was on the rise. His blood gas values and renal functions during ICU stay were given in table below.

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***Table 1****: The patient was transferred to the nearest nephrology unit for haemodialysis and further management on day 06.*

| | On admission | Day 1 | | Day 2 | Day 3 | Day 4 | Day 5 |
|---|---|---|---|---|---|---|---|
| pH(7.35-7.45) | 6.946 | 7.0 | 7.1 | 7.32 | 7.33 | 7.34 | 7.36 |
| pCO2 (35-45mmHg) | 8.5 | 25 | 24 | 26 | 30 | 34 | 30 |
| pO2(90-110mmHg) | 65.4 | 450 | 179 | 172 | 138 | 158 | 140 |
| Lactate(<2mmol/l) | 1.5 | 0.5 | 1.4 | 0.9 | 1.6 | 0.7 | 0.5 |
| HCO3(24-28 mmol/l) | 1.9 | 6.7 | 9.4 | 14.1 | 16.2 | 17.2 | 18.2 |
| SHCO3(24-28 mmol/l) | 6.6 | 9 | 12.4 | 16.7 | 18.4 | 18.7 | 19.1 |
| BE(+/- 2mmol/l) | -25.4 | -24.4 | 19 | -12.1 | -9.9 | -8.8 | -8.5 |
| Aniongap(8-12mmol/l) | 25.0 | 24.3 | 23.0 | 17.1 | 17.0 | 15.2 | 15.9 |
| Creatinine (<1.2 mg/dl) | 4.26 | 3.28 | 3.28 | 2.92 | 3.26 | 2.64 | 2.23 |
| Na (135-145mmol/l) | 135 | 130 | 132 | 134 | 138 | 135 | 134 |
| K (3.5-4.5mmol/l) | 4.0 | 3.8 | 3.6 | 3.8 | 4.2 | 4.4 | 4.0 |

**Discussion**

Brake oil consists of glycol ether, ethylene, or diethylene glycol and additives. Ethylene glycol, diethylene glycol and its metabolites are responsible for the toxic effects[4]. Ethylene glycol is metabolised in the liver to glycoaldehyde, glycolic acid, glyoxylate and oxalate. Diethylene glycol is metabolized to 2-hydroxy-ethoxyacetaldehyde and 2-hydroxyethoxyacetic acid. All these are responsible for metabolic acidosis. Furthermore, these metabolites are nephrotoxic and neurotoxic.

The end product of metabolism of ethylene glycol is oxalate, which chelates with calcium to form insoluble calcium oxalate which may result in hypocalcaemia. Calcium oxalate crystals deposit in proximal renal tubules causing renal injury. Moreover, calcium oxalate crystals deposit in brain and lung tissues[4].

Three distinct clinical stages are clearly identified in brake oil poisoning. Severe poisoning may go through all three clinical stages[5].They are nervous system involvement, cardiopulmonary involvement and renal involvement from 0.5 hrs to 12 hrs, 12 to 36 hrs and 24 to 72 hrs respectively[6-7]. We observed all three clinical stages in this patient.

Neurological manifestation varies from mild symptoms like visual disturbances to severe symptoms like seizures. At very high doses it may cause coma and brain death[2].The only nervous system finding in this patient was agitation.

Cardiopulmonary manifestations are tachycardia, hypertension, tachypnoea, cardiac failure and QT prolongation. Morbidity and mortality are high in the patients who has cardiac failure[8]. In this case patient remained tachycardic and hypertensive throughout ICU stay, but he didn't show any features of cardiac failure.

Renal involvement of ethylene glycol poisoning is characterized by acute kidney injury[2].In this case, on admission to ICU he was in acute renal failure. His pH was 6.946 with a bicarbonate of 1.9 mmol/l. In addition, other positive findings identified were hypocalcaemia and oxalate crystals in his urine.

Management of ethylene glycol intoxication includes stabilization, correction of acidosis, management of acute kidney injury, use of antidotes and other supportive care[2]. Haemodialysis is the mainstay of management of ethylene glycol intoxication. Haemodialysis removes ethylene glycol and its metabolites from the blood. Traditional recommendation is

to offer haemodialysis, when the ethylene glycol level is more than 500mg/l[9]. Moreover, it is recommended to initiate haemodialysis when there is severe metabolic derangement irrespective of the ethylene glycol level. In the absence of facilities to measure ethylene glycol levels, initiation and duration of haemodialysis was mainly guided by clinical condition, renal functions with electrolytes and blood gas values. In spite of continuation of haemodialysis, he remained acidotic for about 12 hours, which required three doses of intravenous sodium bicarbonate infusion 8hourly over next 24 hours to achieve pH of 7.1.

Antidotes used in ethylene glycol poisoning are ethanol and fomepizole[9]. These two antidotes act differently. Ethanol competitively inhibits metabolism of ethylene glycol by alcohol dehydrogenase, whereas fomepizole is a potent inhibitor of alcohol dehydrogenase[9]. As a results production and effects of metabolites are minimal. In addition, vitamins like thiamine and pyridoxine prevent the formation of oxalic acid by facilitating the conversion of glyoxylic acid(toxic metabolite of ethylene glycol)to non-toxic metabolites[2].

**Conclusion**

Brake oil poisoning can lead to fatal outcome. Although it is rare, understanding of pathophysiology is paramount important in the management of such cases. Immediate stabilization, correction of acidosis, management of AKI, use of antidotes and other supportive care improve the outcome of these patients.

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