CASE REPORT

Acute Kidney Injury, Myocardial Infarction and Death Following Brake Fluid Poisoning; A Case Report

RATHNAYAKA MUDIYANSELAGE MITHUN KAUSHIKA NAMAL RATHNAYAKA[1,2*], PANWILAHENE ELLAWATTE ANUSHA NISHANTHI RANATHUNGA[3]

[1]Intensive care unit, Provincial General Hospital, Ratnapura, Sri Lanka
[2]Department of Veterinary Pathobiology, Faculty of Veterinary Medicine &Animal Science, University of Peradeniya, Sri Lanka
[3]Medical Unit, Provincial General Hospital, Ratnapura, Sri Lanka

Abstract

Background: Ethylene glycol is a toxic alcohol which is used in brake fluid, antifreeze, coolants, preservatives and chemical solvents. Ethylene glycol poisoning usually results in depression of the central nervous system, renal insufficiency and cardiopulmonary compromise, while laboratory findings include metabolic acidosis, increased anion gap, increased osmolar gap and calcium oxalate crystalluria.
Case presentation: A 24-year-old previously healthy person died 13 days after self-ingestion of brake fluid (ethylene glycol). He developed multi-organ failure including acute kidney injury, metabolic acidosis, respiratory failure, myocardial infarction, low Glasgow coma scale, and elevation of liver enzymes. He also developed hypotension for which 3 inotropes were started. He had ST elevation myocardial infarction (STEMI) on day 4 of the poisoning associated with a reduction of ejection fraction of up to 25% with septal anterior wall hypokinesia. He needed intensive care treatment via ventilator and inotropic support. Five cycles of hemodialysis were carried out for acute kidney injury. His autopsy examination revealed sub-endocardial hemorrhages.
Discussion: Acute kidney injury and metabolic acidosis are frequently seen following ethylene glycol poisoning from brake fluid ingestion. The cardiotoxic effect of its poisoning could be due to multiple microcalcifications of the myocardium. This clinical report highlights the severity and the sequence of events following ethylene glycol poisoning.
Conclusion: STEMI may result following ethylene glycol poisoning in addition to other cardiac effects such as hypotension, tachycardia, myocarditis and ischemic changes in ECG.

Key words: Acute Kidney Injury; Brake Fluid; Ethylene glycol; Metabolic Acidosis; Myocardial Infarction

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INTRODUCTION

Brake oil is an automobile transmission fluid composed of mainly ethylene glycol $(CH_2OH)_2$, a toxic alcohol used also in antifreeze, coolants, preservatives and chemical solvents (1). Poisoning occurs as a result of intentional ingestion in suicide attempts or accidental ingestion (2). Ethylene glycol poisoning results in the depression of the central nervous system, renal insufficiency and cardiopulmonary compromise; laboratory features include increased anion gap metabolic acidosis, increased osmolar gap and calcium oxalate crystalluria (3). We report a fatal case of brake fluid poisoning complicated with metabolic acidosis, acute kidney injury and ST elevation myocardial infarction (STEMI).

CASE REPORT

A 25-year-old previously healthy married man was admitted to the medical ward at 2115 h following self-ingestion of 250ml brake oil (DOT 3) around 1730 h. He was a non-smoker and non-alcoholic. On admission, the patient had nausea, faintness and headache. On examination he was afebrile, not dyspneic; blood pressure (BP) was 120/80mmHg; pulse rate was 68 beats/min; bilateral lung fields were clear and findings of abdominal and nervous system were normal. As pure ethanol was not available at the hospital 33.5% arrack was given, 1.8ml/kg (90ml) as a loading dose and 0.2ml/kg/hr (10ml) as the maintenance dose orally. On admission his ECG was normal (Figure 1). Laboratory findings are mentioned in Table 1. Arterial blood gas analysis done on FiO2-40% and Hb-14.7g/dL showed severe metabolic acidosis (pH-6.854, pCO_2-9.8mmHg, pO_2-140.6mmHg, HCO_3-17mmol/L, base excess (-)32.1mmol/L, SO_2-96.1% and PO_2/FiO_2- 234.4 mmHg) and 8.4% $NaHCO_3$ 100ml intravenous (IV) bolus was administered. The following day serum K^+ and Ca^{2+} levels were 6.39mmol/L and 1.78mmol/L respectively, so a soluble insulin/dextrose and IV calcium gluconate were administered. Then around 1700 h (24 hours after the ingestion), the patient developed dyspnea associated with lowering of SpO_2 and few bilateral crepitations were heard in both lung fields. As he developed

*Correspondence to: Rathnayaka Mudiyanselage Mithun Kaushika Namal Rathnayaka; MBBS, MA, MSc, Dip.in Toxicology, Dip.in OH&S. Intensive care unit, Provincial General Hospital, Ratnapura, Sri Lanka
Tel: 09714337784, E-mail: namalrath10@yahoo.com
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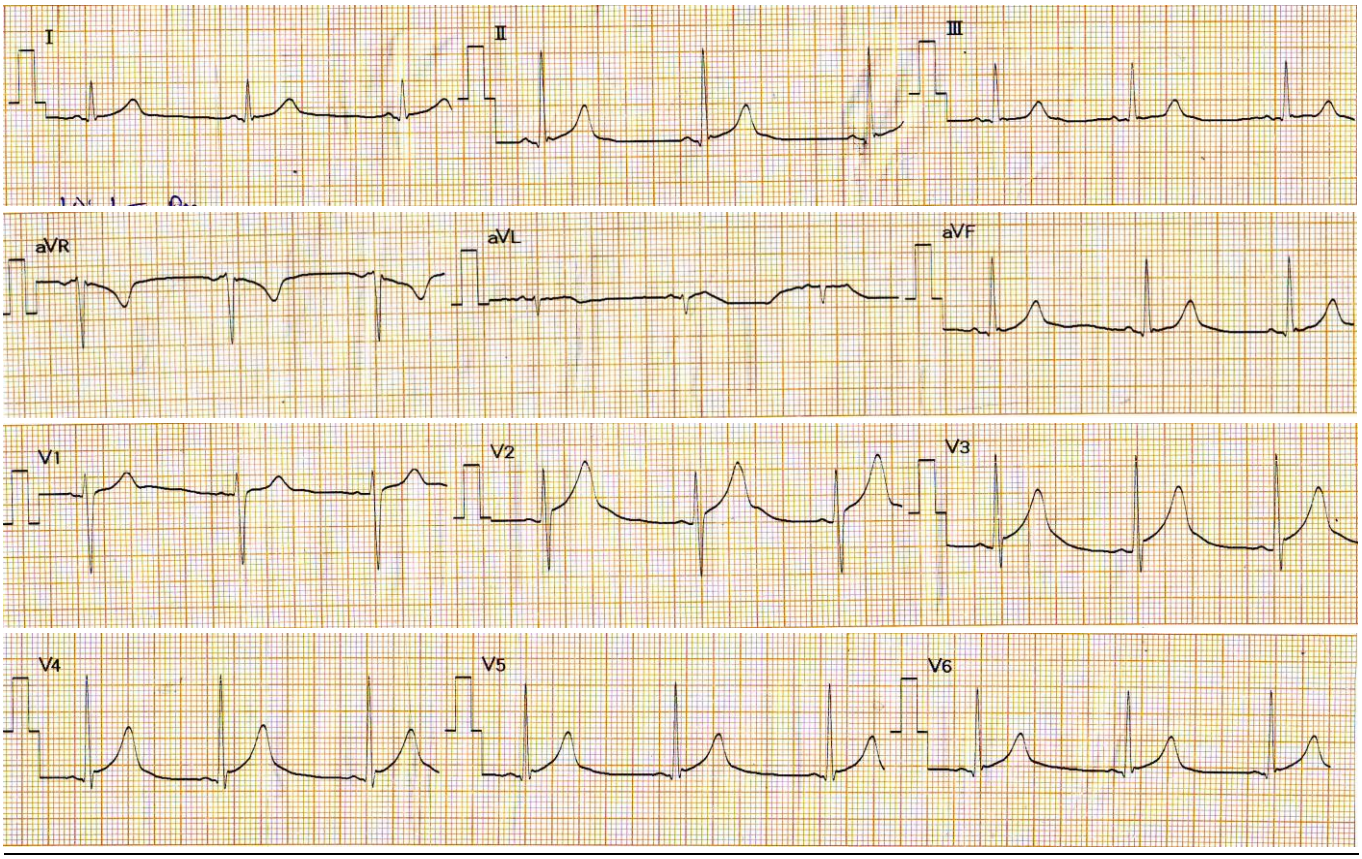

Figure 1. Normal ECG on admission (4 hours after brake fluid ingestion)

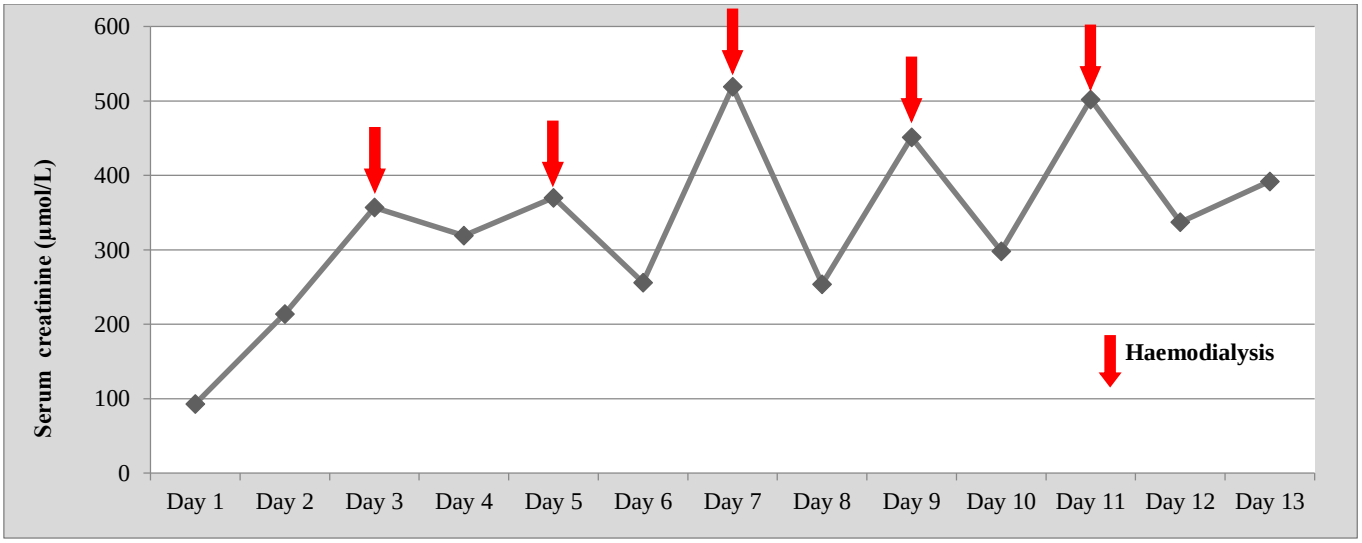

Figure 2. Daily changes of serum creatinine level and points of haemodialysis

impending respiratory failure, he was intubated and transferred to intensive care unit (ICU) for the ventilator support. The chest X-ray at this stage showed few bilateral inflammatory shadows. As BP was low (88/46mm Hg) IV dopamine infusion (15µg/kg/min) and then IV noradrenalin infusion was started. On day 2 of ICU hemodialysis was started and subsequently 5 cycles were done (Figure 2). During ICU stay the patient did not produce urine, in spite of

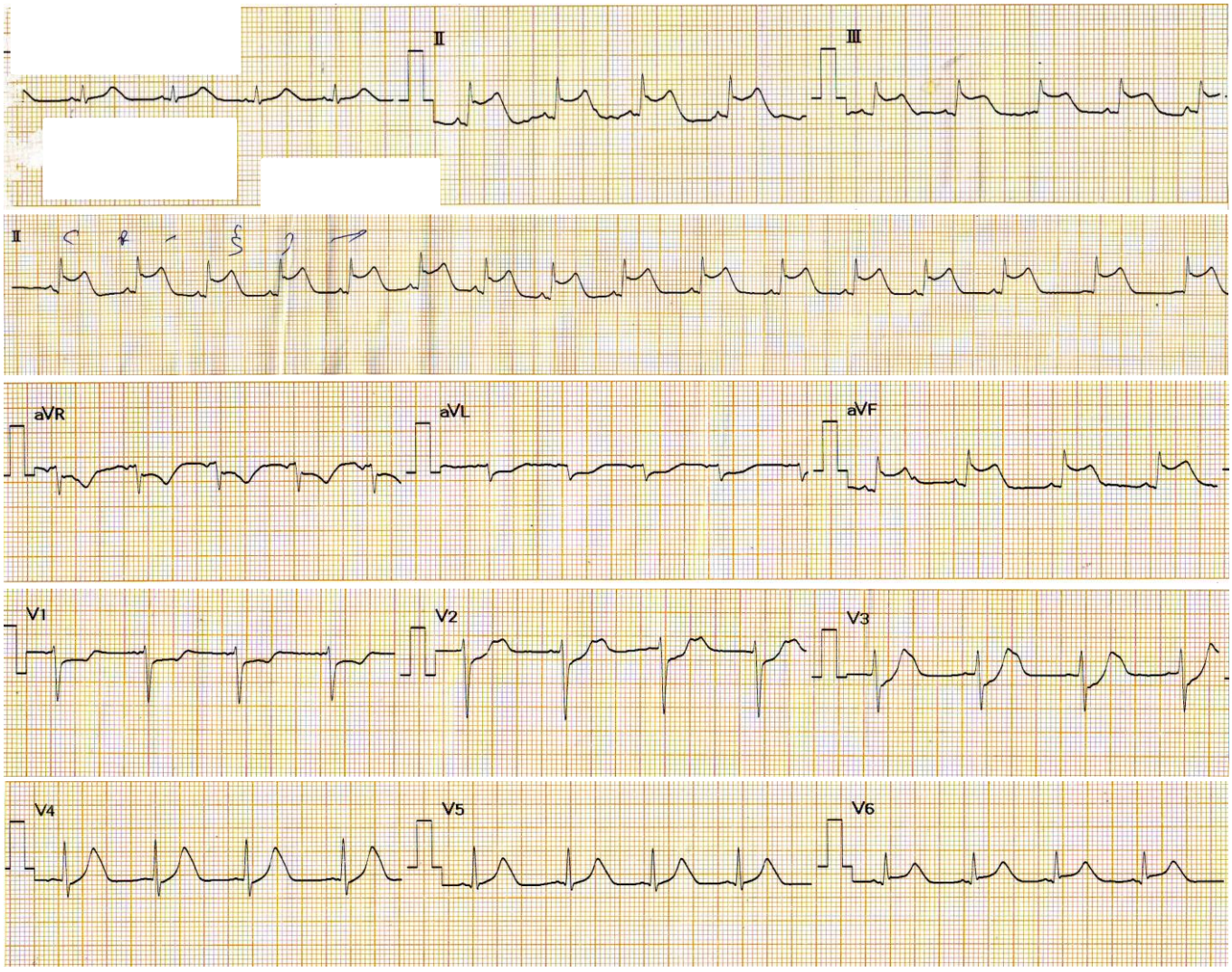

Figure 3. Inferior STEMI (ST elevation in lead II, III, aVF)

having IV frusemide infusion 10mg/hour in the first day. On day 3 of ICU (day 4 of the ingestion) the patient developed ST elevation myocardial infarction (STEMI-Figure 3) for which a daily dose of 1.25 mg of subcutaneus fondaparinux was administered. Aspirin, clopidogrel or atrovastatin was not started at this time due to a low platelet count and elevated liver enzymes (SGPT/SGOT). The Troponin I that we did on ICU day 5 was positive (> 180ng/mL, normal < 1ng/mL). At this stage a 2D echocardiogram showed 25% of ejection fraction with septal and anterior wall hypokinesia. Blood picture done at ICU day 8 was suggestive of sepsis and disseminated intravascular coagulation (DIC). As his Glasgow coma scale (GCS) was low throughout (< 8), non-contrast computed tomography (NCCT) of brain was done on ICU day 9 and it showed petechial hemorrhages in parietal and occipital lobes. An IV 8.4% $NaHCO_3$ 100ml slow bolus was administered daily for metabolic acidosis and IV calcium glucanate 10ml (one vial) was given daily for hypocalcaemia. However, the patient had cardiorespiratory

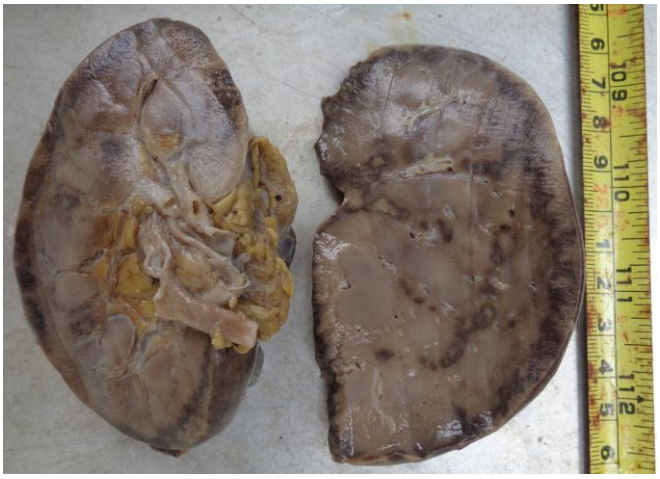

Figure 4. Reduced cortico-medullary demarcation in right kidney (autopsy findings)

arrest on day 13 of the poisoning (day 12 of ICU) and died while on resuscitation.

The postmortem examination revealed features of multi-organ involvement: reduced cortico-medullary demarcation in kidneys (Figure 4), petechial hemorrhages in cerebrum, sub-endocardial hemorrhages and features of pulmonary edema in both lungs. The investigation findings were mentioned in Table 1.

DISCUSSION

This previously healthy young person had intentionally consumed large amounts of brake fluid and died due to multi-organ failure involving toxic effects on the kidneys, heart, brain, liver and lungs. Ethylene glycol is at first metabolized into glycolaldehyde by the action of alcohol dehydrogenase which causes neurological features (depression of central nervous system) at the initial stage (stage I) of the poisoning (3). Glycolaldehyde is further metabolized to glycolic acid then to oxalic acid which results in profound metabolic acidosis characterized biochemically by increased anion gap. Metabolic acidosis is reported 55.5% of the poisoned patients following ethylene glycol ingestion (4). Cardiopulmonary phase (stage II) develops in 12-24 hours of post ingestion characterized by dyspnea, hyperventilation, tachycardia and blood pressure changes. Renal failure develops in stage III of the poisoning after 24-72 hours of the ingestion. Acute kidney injury and metabolic acidosis are frequently seen following ethylene glycol poisoning with brake fluid ingestion (4, 5). Initially the patient developed metabolic acidosis and then acute kidney injury for which 5 cycles of hemodialysis was performed. The renal failure could be due to a combination of hypotension and direct toxicity of oxalic acid as the deposition of calcium oxalate in renal tubules (6). The patient was found to have completely anuric during the ICU stay. There was persistent metabolic acidosis up to day 7 of the poisoning.

The antidote of ethylene glycol is ethanol or fomepizole. Ethanol acts by competing with ethylene glycol for alcohol dehydrogenase which has about a 100-times greater affinity than ethylene glycol, then blocks the breakdown of ethylene glycol into glycolaldehyde and prevents its toxic effects. Alternatively, Fomepizole is a potent inhibitor of alcohol dehydrogenase which is not available in Sri Lanka. As IV ethanol preparations were not available in the hospital, the patient was given 33.5% arrack as a loading dose (1.8ml/kg) and then the maintenance dose (0.2ml/kg/hr) for 48 hours via nasogastric tube (7). The patient had persistently low GCS (< 8) and petechial hemorrhages were observed in the cerebrum in both NCCT brain and autopsy examination. This may be due to deposition of calcium oxalate crystals in cerebral vessels in ethylene glycol poisoning (8). In histopathological slides Riccardo Zoja et al. (2013) observed multiple microcalcifications of different sizes in various areas of the myocardium, glomeruli and renal tubules associated with local necrosis of the renal parenchyma (9). The minimum fatal dose of ethylene glycol in adults is 1.4-1.6ml/kg or 100ml in total (10). We were unable to measure serum ethylene glycol level, anion gap or serum osmolality of this

Table 1. Loboratory findings of the patient

Investigation	Day1	Day2	Day3	Day4	Day5	Day6	Day7	Day8	Day9	Day10	Day11	Day12	Day13
WBC ($x10^3/\mu L$)	12.7	30.2	25.8	10.2	9.6	12.8	8.8	7.2	10	16.2	17.6	15.1	9.3
Neutrophils%	87	89	89.2	87.7	87.7	92	87.1	86.4	86.9	87.4	87	88.4	92.3
Lymphocytes%	11.5	10	6.5	6.3	9.5	5.9	7	11.9	5.8	5.2	10	4.3	4.4
Platelets($x10^3/\mu L$)	376	434	262	97	71	59	46	42	27	45	93	91	84
Hb (g/dL)	14.7	16.7	16.5	11.8	10.4	10.4	9.5	9.6	9.9	9	10.3	8.5	7.1
Packed cell volume (PCV) %	43.9	50.6	50.9	35.2	31.5	31.1	29.1	38.3	30.4	28.3	30.9	26	22
Serum creatinine (µmol/L)	93	214	357	319	256	519	589.9	254	298	427	502	337	392
Blood urea (mmol/L)	3.7	9.9	12.8	12.4	13.6	17.8	33.6	16.6	17	27.4	45.3	25	32.3
Na^+ (mmol/L)	139	137	145	134	145	143.6	148	146	142	140	147	144	145.7
K^+(mmol/L)	4.1	6.8	4.1	4.2	3.6	4.5	3.9	3.4	4	4.1	5.3	4	3.8
PT (sec.)		16/12	16/12	20/12	18/12	13/12	14/12		15/12		15/12	15/12	15/12
INR		1.41	1.41	1.85	1.63	1.10	1.21		1.31		1.31	1.31	1.31
APTT (sec.)		46/25	33/25	40/25	31/25		20/25		49/25		24/25	34/25	26/25
SGOT (AST)[U/I]*		67.7	121.9	746		585			365	310			210
SGPT (ALT)[U/I]**		35.7	89.5	338		315			312.9	275			187
Total Bilirubin (µmol/L)			7.7						11.4	14.3			13.4
Calcium (mmol/L)	1.08	1.11	1.03			2.15			1.95		2.10		
pH	6.854	6.670	6.873	7.382	7.333	7.378	7.374	7.403	7.423	7.448	7.473	7.397	7.411

*SGOT (AST) - serum glutamic oxaloacetic transaminase
**SGPT (ALT) - serum glutamic-pyruvic transaminase

patient due to the poor resource setting. Cardiotoxic effects such as hypotension, tachycardia, myocarditis and ischemic changes in ECG following ethylene glycol poisoning have previously been reported (10). But extensive literature search did not find any case documented with STEMI. Therefore, this was the first case of having STEMI following brake fluid (ethylene glycol) poisoning. Since day 2 of the poisoning, this patient had low BP for which initially dopamine infusion and then noradrenalin infusion was started. But as the BP was further low, another inotrope (dobutamin) had to be started. By day 4 of the poisoning he developed inferior wall MI and ejection fraction reduced up to 25% with septal anterior wall hypokinesia. Further sub-endocardial hemorrages were found during the autopsy examination. This sequence of events will explain the cardiotoxic effects of ethylene glycol. On the other hand, the liver enzymes (SGOT/SGPT) were gradually elevated and this conveys the hepatotoxic effect of the poison. But if ethanol therapy is prolonged, patients are also at risk of developing hepatotoxicity and hypoglycemia. Hyperglycemia is relatively uncommon finding following ethylene glycol poisoning (11, 12). But in our patient random blood sugar levels were normal throughout. Accidental ethylene glycol poisoning is common in pediatric age groups but have a good recovery (13). In this patient respiratory failure may be due to the aspiration pneumonia and the later development of features of acute respiratory distress syndrome (ARDS) in the chest X-ray. It was reported that 38.8% of patients following ethylene glycol poisoning go in to respiratory failure and require ventilator support (4). The patient was admitted to the hospital around 4 hours after the ingestion. Therefore, gastrointestinal decontamination (gastric lavage and activated charcoal) of this patient has limited efficacy as ethylene glycol is rapidly absorbed in the stomach (3). It is rapidly distributed in all tissues of the body with a volume of distribution of 0.7-0.8L/kg and cleared by the kidneys (4). Supplemental thiamine and pyridoxine are recommended in poisoning management because they act as cofactors in the conversion of glycolic acid into nontoxic metabolites (3). Poisoning of ethylene glycol is fatal (4, 11) and this case report conveys the multi-organ failure following brake fluid (ethylene glycol) ingestion.

CONCLUSION

Ethylene glycol poisoning may be fatal and it may result in STEMI in addition to other cardiotoxic effects such as hypotension, tachycardia, myocarditis and ischemic changes in ECG.

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