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Letter to the Editor
EDİTÖRE MEKTUP

Etilenglikol Zehirlenmesine Bağlı Gelişen Akut Böbrek Yetmezliği

Acute Renal Failure Due To Ethyleneglycol Poisoning

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ÖZET

Etilenglikol, dihidrik alkoller grubundan, renksiz, kokusuz, tatlı ve suda çözünen bir maddedir. Etilenglikol içeren maddelerin kaza ya da intihar amaçlı alınması sonucu ağır metabolik asidoz, santral sinir sistemi depresyonu ve oligürik akut böbrek yetmezliği meydana gelir. Erken tanı ve tedavi ile mortalite anlamlı bir düzeyde azalabilmektedir. Biz kaza ile antifriz içtikten sonra etilenglikol zehirlenmesine bağlı olarak ağır metabolik asidoz, koma ve akut böbrek yetmezliği tablosu gelişmiş bir vakayı sunduk.

Anahtar kelimeler: Akut böbrek yetmezliği, Etilenglikol zehirlenmesi, Metabolik asidoz

ABSTRACT

Ethylene glycol is a colorless, odorless, sweet, water-soluble substance from the group of dihydric alcohols. As a result of taking ethylene-containing material for suicide purpose or as an accident, serious metabolic acidosis, central nervous system depression and oliguric acute renal failure occur. Early diagnosis and treatment can reduce mortality at a significant level. We present a case with severe metabolic acidosis, coma and acute renal failure due to ethylene glycol intoxication after drinking antifreeze accidentally.

Key words: ethylene glycol poisoning, metabolic acidosis, acute renal failure

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INTRODUCTION

Ethylene glycol (EG) poisoning can result in serious morbidity and mortality [1]. It is used in construction of glass cleaners and antifreeze solution. EG rapidly absorbes after oral administration and peaks in the blood within 1-4 hours, it's half-life is 3-5 hours.

When EG is drunk accidentally or with a suicide attempt, it is transformed into its active metabolites, and then metabolic acidosis, renal failure, hypocalcemia, oksalüri, central nervous system disorders and cardiovascular failure ocur [2]. Kidney failure is formed due to double crushing oxalate crystals, one of EG metabolites, accumulation in the distal tubule and collecting duct [3]. We present a case with acute renal failure, metabolic acidosis, coma after drinking antifreeze accidentally.

CASE REPORT

Eighty-years-old male patient was brought to emergency room at 8:30 in the morning with unconsciousness and vomiting sypmtoms. According to information received from relatives, the patient drank 100 cc antifreeze solution as thinking grape juice at 23:30 the day before because of his visual impairment. He awakened with vomiting in the morning and followed by confusion. On physical examination of the patient during the application process; fever was 36.3ºC, pulse was 105 / min, BP was 155/90 mm Hg, respiratory rate was 28 / min, Glasgow Coma Score was 4 points. On laboratory tests WBC was 12.79 10x3 / mL, Neu was 11.5, Hgb was 10.4 g / dL, Plt was 71000 10x3 / µ, Urea was 87 mg / dl, creatinine was 2.7 mg / dL, NA was 138 mmol / L, CL was 87 mmol / L, K was 5.8 mmol / L, CA was 6.2 mg / dL, pH was 6.93, HCO 3 was 7 mmol / L, calcium oxalate crystals were found in the urine sediment. Because of confusion cranial computed tomography was done and CVA or brain edema were not observed. Renal ultrasonography revealed normal. There was a prolonged QT interval on the electrocardiogram. With the story of antifreeze drinking and patient's current clinical and laboratory findings, EG poisoning was considered. 150 cc / hr 0.9% NaCl and bicarbonate infusion were administered to the patient, after then hemodialysis with bicarbonate was done. After dialysis, Ph was 7.26, HCO 3 was 15.5 mmol / L, respectively. After 24 hours from the incident, the patient remained oliguric and creatinine levels gradually increased to 5.6 mg / dL. Patient was monitored, 200 cc/h 0.9% NaCl isotonic solution and 8 mEq/h bicarbonate infusion were started after the lack of evidence of heart failure on echocardiography. While asidosis improved after 48 hours of hospitalization, creatinine levels gradually decreased until the 7th day of admission, and fixed at 2.3. At follow-up renal biopsy was performed because of to the lack of decline in creatinine levels.

On renal biopsy patchy interstitial mononuclear inflammatory cells containing a small number of eosinophils and in the tubule lumen, epithelial cast and calcium oxalate crystals that crushing polarized light double revealed. With these findings nephrotoxic acute tubular damage was considered. The patient's creatinine levels decreased to 1.4 at follow-up.

DISCUSSION

EG is used in antifreeze content, cooling and many industrial solvents. It causes numerous accidents or intentional poisoning every year. Following oral administration, it is primarily metabolized to glikoaldehide by alcohol dehydrogenase enzyme in the liver. Glikoaldehid then transforms to glycolic acid, glioxilik acid and finally oxalic acid [4]. These metabolites are responsible for the toxic effects. The dose for toxic effects is usually 0.1 mL / kg [5]. Clinic symptoms of EG poisoning generally develop in three stages. However, three stages might not emerge in all intoxications. In the first 12 hours, neurological and gastrointestinal tract symptoms like dizziness, incoordination of muscle movements, nystagmus, headache, speech disorders, coma, nausea and vomiting are seen. In the second stage (12-36 hours slic, tachycardia, hypertension, hyperventilation, metabolic acidosis, hypocalcemia, congestive heart failure may develop. Death is the most commonly seen in this process if it is left untreated [4]. In the third

stage (36 to 72 hours period) acute renal failure occurs. At this stage, hematuria, proteinuria, oliguria, hyperkalemia are seen [6]. Our case was presented with nausea, coma, hypocalcemia, hyperkalemia, severe metabolic acidosis about ten hours after the incident. At follow-up, acute renal failure developed.

In the diagnosis of a patient presenting with these symptoms EG levels should be checked directly. If this is not avaliable in your center, it can be diagnosed according to the criterias in table 1 [7].

Clinical entities such as diabetic and alcoholic ketoacidosis, uremia, lactic acidosis, aspirin and methanol poisoning should be considered in the differential diagnosis. We diagnosed our case with existing criterias in table 1 after exclusion of possible causes of metabolic acidosis. In treatment, gastric lavage, alcohol dehydrogenase inhibition, fluid-electrolyte replacement and hemodialysis can be done after hemodynamic stability is supplied [4]. In our case, after hemodynamic stability is supplied, fluid-electrolyte replacement and hemodialysis were applied.

Table 1. The criterias for the pre-diagnosis of Ethyleneglycol poisoning.

a) with ethyleneglycol poisoning or suspicion, 2 of the following criterias inclusion Arterial pH <7.3 Serum bicarbonate <20 mEq / L Osmolal gap> 10 mOsm / L Urinary oxalate crystals
OR
b) with ethyleneglycol poisoning or suspicion in the last one hour, being osmolal gap> 10 mOsm / L

RESULT

Because EG is colorless, odorless, sweet and water-soluble substance, it can be drunk for purposes other than suicide accidentally. Because it is a toxic and lethal substance, patient should be referred immediately to the emergency room.

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