Hemodialysis Management In A Patient With Methanol Intoxication: A Case Report

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ABSTRACT

Methanol, also known as methyl alcohol or H_3COH, is a toxic alcohol with the characteristics of a clear liquid that is colorless at room temperature and has a sweet smell (Allata et al., 2023; Barceloux et al., 2002). Methanol is obtained through the distillation process of wood and used as an ingredient in mummy processing (Nekoukar et al., 2021). As time went by, methanol is now used as a mixture of raw materials for various commercial industries and household purposes such as formaldehyde, dyes, vehicle fuels, cleaning fluids, and handrubs (Allata et al., 2023; Mousavi-Roknabadi et al., 2022).

Keywords: process, materials, commercial, fluids

Methanol, also known as methyl alcohol or H_3COH, is a toxic alcohol with the characteristics of a clear liquid that is colorless at room temperature and has a sweet smell (Allata et al., 2023; Barceloux et al., 2002). Methanol is obtained through the distillation process of wood and used as an ingredient in mummy processing (Nekoukar et al., 2021). As time went by, methanol is now used as a mixture of raw materials for various commercial industries and household purposes such as formaldehyde, dyes, vehicle fuels, cleaning fluids, and handrubs (Allata et al., 2023; Mousavi-Roknabadi et al., 2022).

Methanol began to be widely used as an alcohol mixture solution since the method of producing methanol cheaply was discovered around 1890 (Suit & Estes, 1990). Each year no less than 800 cases of methanol intoxication are reported in various countries (Kraut & Kurtz, 2008; World Health Organization, 2014, 2024). Methanol mixtures are commonly found in illicitly traded liquor. Intentional mixing of methanol into liquor based on social and economic factors increases

the incidence of methanol intoxication due to the consumption of "mixed" liquor (Alnefaie et al., 2024; de Wit & Sayette, 2018).

In human, methanol is easily absorbed through inhalation, skin absorption, and oral routes. Methanol is then metabolized through a series of enzymatic processes that produce toxic metabolites in the form of formic acid. The accumulation of formic acid is what then causes symptoms of intoxication such as nausea, vomiting, acidosis, acute kidney injury, visual impairment, and death (Bruckner et al., 2021; Clary, 2013; Howard & Ferrier, 2024).

The management of methanol intoxication is based on accelerating the elimination of methanol from the body and inhibiting the formation of formic acid through the inhibition of related enzymes (Barceloux et al., 2002; Kusumobroto, 2015). Renal replacement therapy is one of the modalities in the management of methanol intoxication. In addition to accelerating the excretion of methanol and its toxic metabolites, renal replacement therapy is also able to improve the condition of acidosis and the clinical symptoms that arise (Kute et al., 2012; Roberts et al., 2015; Zakharov et al., 2017).

CASE

A 20-year-old man came to the emergency room with complaints of nausea and vomiting. Nausea and vomiting had been felt by the patient for the last 24 hours before being taken to the emergency room. Based on the information, the patient claimed to have vomited more than 10 times. The vomit said to be containing water and food scraps with the volume of each vomit ranging from 100 to 200 ml. This condition caused the patient to experience a decrease in appetite. The patient did not have time to take any medication to reduce symptoms before being taken to the emergency room. In addition, the patient also complained of blurred vision since 1 day ago and was still worsening. The patient also felt shortness of breath without coughing that appeared since 1 day ago.

From further anamnesis, it was discovered that 3 days prior to the presence, the patient had consumed mixed liquor with 3 other friends. The patient did not remember how much liquor he had consumed. The day after the patient and his friends consumed the mixed liquor, the patient went home and rested. At that time the patient only felt sleepy and dizzy. According to the patient, other symptoms such as vomiting, shortness of breath, and blurred vision were only felt 2 days after consuming the mixed liquor.

From the evaluation when the patient arrived at the ER, the patient was found to be conscious (GCS 456) with vital signs showing BP 132/100 mmHg, pulse rate 98 x/minute, respiratory rate 38 x/minute, body temperature 36.8°C, and peripheral saturation of 99% using 8 L/minute of simple mask oxygen. On head and neck examination, the pupils appeared mydriatic. There was not any icteric sclera or anemic conjunctiva. Chest wall movement appeared symmetrical. Single one and two heart sounds were heard without any murmurs or gallops. Vesicular breathing sounds were found throughout the lung field and there were no wheezing or rhonchi. From the physical examination, the abdominal area appeared flat. Auscultation heard bowel sounds eight times per minute. There was tenderness in the epigastric region without any enlargement of the liver or spleen. The patient's extremities felt cold, with a capillary refill time of more than two seconds.

The ER laboratory examination results showed: Hb 17.7 g/dL; HCT 53%; MCV 87.6 fL; MCH 29.3 pg; WBC 23,840 /µL; NEUT 87%; LYMPH 4.9%; PLT 347,000 µL; BUN 13.8 mg/dL; SCr 1.5 mg/dL; Na 130 mmol/L; K 5.3 mmol/L; Cl 98 mmol/L; AST 61 U/L; ALT 58 U/L; Alb 5.17 g/dL; Random Blood Glucose 116; non-reactive HBsAg; non-reactive anti-HCV; non reactive anti-HIV 3 methods; Blood Gas Analysis: Simple Mask 8 lpm pH 7.10, pCO2 17, pO2 36, HCO3 5.3, BE -24.4, SO2 45%; Urinalysis: Clear Yellow, BJ 1.017, pH 6, Negative Leukocytes, Negative Nitrite, Protein +2, Negative Glucose, Ketone +1, Normal Urobilin, Negative Bilirubin, Erythrocytes +3, ACR >=300, PCR >=0.5. Anion gap calculation

obtained 26.7 mEq/L or 23.8 mEq/L with albumin correction indicating a high anion gap . Serum osmolality was calculated to be 271 mOsm/kg. The results of the chest radiology examination showed no abnormalities in the heart and lungs.

From the results of anamnesis, physical examination, and other supporting examinations that have been carried out, the patient was diagnosed with methanol intoxication + severe metabolic acidosis + acute kidney injury + hyperkalemia + toxic optic neuropathy due to methanol. The patient was then treated with initial supportive therapy in the form of rehydration with crystalloid fluid, correction of hyperkalemia using intravenous insulin followed by intravenous dextrose fluid, bicarbonate infusion through the central vein, and antidote therapy using intravenous ethanol solution drip. The results of the blood gas analysis reevaluation still showed severe metabolic acidosis: pH 7.08, pCO2 10, pO2 164, HCO3 3, BE -27, SO2 99%. The patient was then proceeded for hemodialysis therapy to accelerate the excretion of remaining toxic substances and improve the severe metabolic acidosis that occurred.

The patient was consulted to the Department of Ophthalmology regarding the toxic optic neuropathy condition that occurred. From the examination, visus oculi dextra (VOD) was obtained: 1-meter counting finger (1/60) and visus oculi sinistra (VOS): 3-meter counting finger (3/60). Examination of the eyeball pressure with Schiotz obtained oculi dextra (OD) 11.2 mmHg and oculi sinistra (OS) 12.2 mmHg. Examination of the eyeball movement, both OD and OS, was normal without any pain in eyeball movement. Examination of the visual field of OD and OS found counting fingers in all quadrants. Examination of the anterior segment OD and OS found palpebra OD: negative edema, negative spasm; palpebra OS: negative edema, negative hematoma; conjunctiva OD and OS found negative hyperemia; cornea OD and OS were clear; anterior chamber OD and OS were deep; iris appeared radial in OD and OS; pupil was round with a diameter of 3 mm and positive corneal reflex and negative of relative afferent pupillary defect (RAPD) in OD and OS; lens was clear in OD and OS. Funduscopic examination in both OD and OS showed positive fundus reflex, N.II papilla with blurred superior area, hyperemic color, cup disk ratio difficult to evaluate, negative Pattons line, negative peripapillary hemorrhage, negative AV tortuosity, AV ratio 2:3, positive retinal hemorrhage, negative exudate, and positive macular reflex. The patient was given therapy in the form of intravenous steroid injection and neurotrophic to improve the toxic optic neuropathy that occurred.

Table 1. The clinical development of a methanol intoxication patients receiving hemodialysis therapy

	7/1 (Emergency Installation)	**8/1 (Room)**	**9/1 (Room)**	**10/1 (Room)**	**11/1 (Room)**	**12/2 (Outpatient)**
Hb (g/dL)	17.7	15.2				
WBC (/µL)	23,840	10,710				
NEUT (%)	87	71.1				
LYMPH (%)	4.9	15.5				
PLT (/µL)	347,000	259,000				

ALT (U/L)	58			
AST (U/L)	61			
Albumin (g/dL)	5.17	3.9		
GDA (mg/dL)	116			
BUN (mg/dL)	13.8	17		9.6
Cr (mg/dL)	1.5	1.1		0.9
Na (mmol/L)	130	129		
K (mmol/L)	5.3	3.3		
Cl (mmol/L)	98	91		
Blood Gas Analysis	SM 8 lpm	Udara bebas		
pH	7.08	7.38		
pCO2	35	35		
pO2	85	85		
HCO3	3	20.7		
BE	-27	-4.4		
SO2	99	96		
HBsAg	Non Reactive			
Anti-HCV	Non Reactive			
Anti-HIV	Non Reactive			
Urine production (cc/Kg/hour)	1.7			

Visual Acuity	VOD 1/60; VOS 3/60	VOD 1/60; VOS 3/60	VOD 6/36; VOS 6/36	VOD 6/18; VOS 6/24	VOD 6/18; VOS 6/9	VOD 6/12; VOS 6/6

Note: Hb = hemoglobin; WBC = white blood cells count; NEUT = neutrophil count; LYMPH = lymphocyte count; PLT = platelet count; ALT = alanine transaminase; AST = aspartate transaminase; GDA = Random Blood Glucose; BUN = blood urea nitrogen; Cr = creatinine; Na = sodium; K = potassium; Cl = chloride; HBsAg = hepatitis b surface antigen; Anti-HCV = anti-hepatitis C; VOD = Visus occuli dextra; VOS = Visus occuli sinistra

On evaluation 1 day later, the patient's condition improved. The patient was in stable condition with compos mentis consciousness (GCS 456), vital signs showed BP 116/70 mmHg, pulse rate 84 x/minute, respiratory rate 20 x/minute, body temperature 36.8°C, and peripheral oxygen saturation of 96% with free air. Laboratory evaluation showed improvement in acute kidney injury (AKI) and the acidosis experienced previously, with blood gas analysis reevaluation results: pH 7.38, pCO2 35, pO2 85, HCO3 20.7, BE -4.4, and SO2 96%; and serum creatinine 1.1.

From the results of the evaluation of visual acuity development, improvements were also obtained. The patient was admitted with a visual acuity of VOD 1/60 and VOS 3/60. On the 3rd day of treatment, improvements were obtained to VODS 6/36, then gradually improved to VOD 6/18 VOS 6/24 on the 4th day, and VOD 6/18 VOS 6/9 on the 5th day. On the 5th day, the patient's steroid therapy was tappered down to oral dose and the patient was discharged for planned outpatient care.

During the follow-up period at the outpatient clinic, the patient was in stable and good condition without any complaints. Laboratory results also showed improvement. Evaluation of kidney function showed serum creatinin of 0.9. The patient also underwent a visual acuity examination to monitor the progress of visual acuity improvement, and obtained VOD 6/12 and VOS 6/6.

DISCUSSION

A 20-year-old man came to the emergency room with complaints of nausea and vomiting. Nausea and vomiting had been felt by the patient for the last 24 hours before being taken to the emergency room. Based on the information, the patient claimed to have vomited more than 10 times. The vomit said to be containing water and food scraps with the volume of each vomit ranging from 100 to 200 ml. This condition caused the patient to experience a decrease in appetite. The patient did not have time to take any medication to reduce symptoms before being taken to the emergency room. In addition, the patient also complained of blurred vision since 1 day ago and was still worsening. The patient also felt shortness of breath without coughing that appeared since 1 day ago.

Symptoms of methanol intoxication can be divided into 3 phases along with the methanol metabolism process: 1) the early phase; 2) the latent phase; and 3) the advanced phase. The early phase of methanol intoxication symptoms begin within 30 minutes up to 4 hours after methanol consumption. This early phase is dominated by symptoms of the central nervous system and gastrointestinal tract (Howard & Ferrier, 2024; Kraut & Kurtz, 2008). Patients may complain of central nervous system symptoms such as headache and fatigue. In some rarer conditions, patients may experience extrapyramidal symptoms associated with putamen necrosis and decreased consciousness to seizures due to brain edema (Kraut & Kurtz, 2008; Randall Bond et al., 2002). Gastrointestinal symptoms are dominated by epigastric pain, nausea, and vomiting. In more severe conditions, pancreatitis may occur (Kusumobroto, 2015; Nekoukar et al., 2021).

After going through the initial phase, the patient then enter the latent phase. Basically, intoxication is not directly caused by methanol but rather the metabolite resulting from its oxidation, namely formic acid, which is catalyzed by the enzyme alcohol dehydrogenase (ADH) (Suit & Estes, 1990). The timespan required in

the oxidation process of methanol to formaldehyde and further into formic acid causes the latent phase to occur. The average latent phase ranges from 24 to 72 hours and may be longer if there is a mixture of ethanol in the liquor consumed (Barceloux et al., 2002; Kusumobroto, 2015).

Within 24 hours of the methanol consumption, enough formic acid will form and accumulate in body tissues, causing the advanced-phase symptoms. In this advanced phase, symptoms of visual disturbances, metabolic acidosis, multiorgan failure, and death begin to appear (Bruckner et al., 2021; Howard & Ferrier, 2024).

From further anamnesis, it was discovered that 3 days prior to the presence, the patient had consumed mixed liquor with 3 other friends. The patient did not remember how much liquor he had consumed. The day after the patient and his friends consumed the mixed liquor, the patient went home and rested. At that time the patient only felt sleepy and dizzy. According to the patient, other symptoms such as vomiting, shortness of breath, and blurred vision were only felt 2 days after consuming the mixed liquor.

The term mixed liquor refers to illegal drinks mixed with various mixtures such as ethanol to methanol with the aim of obtaining high alcohol content at a cheap price (Dedy Heryawan & Arie Mangesti, 2021; Presidential Regulation of the Republic of Indonesia Number 74 of 2013 concerning Control and Supervision of Alcoholic Beverages, 2013; Rusdi et al., 2023; Wirasuta et al., 2019). Family and peer culture are the strongest influencing factors in drinking habits from adolescence to young adulthood. Although the family has a strong influence in shaping the character of adolescents, the habit of drinking due to family influence begins to decrease after the age of 15. Many studies have shown a shift in social influence from family to peers in the context of alcohol consumption, especially after the age of 15 (Sudhinaraset et al., 2016). De Wit and Sayette explained how psychological factors such as the desire to socialize and better acceptance by social groups also influence a person's decision to consume alcohol (de Wit & Sayette, 2018).

From the evaluation when the patient arrived at the ER, the patient was found to be conscious (GCS 456) with vital signs showing BP 132/100 mmHg, pulse rate 98 x/minute, respiratory rate 38 x/minute, body temperature 36.8°C, and peripheral saturation of 99% using 8 L/minute of simple mask oxygen. On head and neck examination, the pupils appeared mydriatic. There was not any icteric sclera or anemic conjunctiva. Chest wall movement appeared symmetrical. Single one and two heart sounds were heard without any murmurs or gallops. Vesicular breathing sounds were found throughout the lung field and there were no wheezing or rhonchi. From the physical examination, the abdominal area appeared flat. Auscultation heard bowel sounds eight times per minute. There was tenderness in the epigastric region without any enlargement of the liver or spleen. The patient's extremities felt cold, with a capillary refill time of more than two seconds.

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disturbances that can cause mydriasis (Clary, 2013; Nekoukar et al., 2021; Suit & Estes, 1990).

Metabolic acidosis that occurs in methanol intoxication is caused by the accumulation of formic acid metabolites resulting from the oxidation of methanol. In the initial conditions, the amount of methanol oxidized into formic acid does not significantly increase the anion gap when serum osmolality is at its highest point. The anion gap will gradually increase as formic acid accumulates in the body followed by a significant decrease in HCO3 and serum osmolality. The arrival of patients in the inbetween period may show an increase in both the anion gap and the osmolar gap (Double gap) (Kraut & Kurtz, 2008; Nekoukar et al., 2021).

AKI in the context of methanol intoxication, according to a study by Verhelst, can be caused by its direct effects on the kidneys, such as high blood methanol levels and formed formate concentrations, or indirect effects such as hemolysis and myoglobinuria. About 66% of patients with methanol intoxication can experience AKI which is predictive of mortality in hospital care up to 19 times. Therefore, patients with AKI should receive faster and more aggressive management (Chang et al., 2019).

From the results of anamnesis, physical examination, and other supporting examinations that have been carried out, the patient was diagnosed with methanol intoxication + severe metabolic acidosis + acute kidney injury + hyperkalemia + toxic optic neuropathy due to methanol. The patient was then treated with initial supportive therapy in the form of rehydration with crystalloid fluid, correction of hyperkalemia using intravenous insulin followed by intravenous dextrose fluid, bicarbonate infusion through the central vein, and antidote therapy using intravenous ethanol solution drip. The results of the blood gas analysis reevaluation still showed severe metabolic acidosis: pH 7.08, pCO2 10, pO2 164, HCO3 3, BE -27, SO2 99%. The patient was then proceeded for hemodialysis therapy to accelerate the excretion of remaining toxic substances and improve the severe metabolic acidosis that occurred.

The management of suspected methanol intoxication is based on the algorithm recommended by the American Academy of Clinical Toxicology (AACT). AACT recommends that 1 of 3 criterias to be met before proceeding with further management: 1) documented plasma methanol concentration >20 mg/dL, OR 2) documented history of previous ingestion of toxic amounts of methanol with an osmolar gap of >10 mOsm/Kg H2O, OR 3) history or strong suspicion of methanol intoxication AND at least 2 of the following: A) arterial pH <7.3, B) serum bicarbonate <20 mEq/L, C) osmolar gap >10 mOsm/kg H2O. When the criteria is met, the patient can be treated for methanol intoxication (Barceloux et al., 2002).

The principles of managing methanol intoxication in addition to supportive management and stabilization of general conditions refer to 2 things: 1) preventing further metabolism of methanol; and 2) accelerating the elimination of methanol and its metabolites from the body (Gallagher & Edwards, 2019). There have been no studies that describe the benefits of gastrointestinal decontamination in treating methanol intoxication. Administration of emetics is highly discouraged due to the risk of aspiration, especially in patients with decreased consciousness. The use of activated charcoal as a chelating agent, although it has been widely tested on ethanol, has not been widely used on methanol so its use is not recommended (Barceloux et al., 2002).

Supportive management includes the correction of acidosis. The undissociated formic acid is a more potent inhibitor of the cytochrome oxidase enzyme. A high ratio of formic acid to formate increases the inhibition of the enzyme in the mitochondria, shifting cellular respiration to the anaerobic pathway, leading to lactate accumulation and worsening acidosis. There have also been reports of increased formate elimination with bicarbonate alone. Thus, bicarbonate administration not only corrects acidosis but also reduces the ratio of formic acid to formate and increases its elimination from the body. The AACT recommends intravenous bicarbonate administration in acidosis with a pH

below 7.3 (Barceloux et al., 2002; Mirrakhimov et al., 2017).

Both ethanol and fomepizole can be given with the aim of inhibiting further methanol metabolism into formic acid. Ethanol has a 10 times stronger affinity for the ADH enzyme so ethanol acts as a competitive inhibitor. Ethanol has its advantages and disadvantages in its use as an ADH inhibitor. Because it is cheap and easier to obtain, in some places ethanol may be the only choice of antidote. The disadvantages of using ethanol are its side effects such as hypoglycemia and central nervous system depression, therefore it requires monitoring of methanol and ethanol levels to remain at the therapeutic range (100-150 mg/dL). Fomepizole, which also acts as a competitive inhibitor of the ADH enzyme, has an affinity to ADH for up to 8000 times stronger than ethanol. Because of its strong affinity, the use of fomepizole remains effective even in low doses. In addition, the minimal side effects and no need for monitoring of blood concentration levels mean that patients can be given without the need for intensive care. Its expensive price and limited availability in some places are some of the obstacles to its use (Barceloux et al., 2002; Gallagher & Edwards, 2019; Kraut & Kurtz, 2008; Nekoukar et al., 2021). Both ethanol and fomepizole can be dialyzed and reduce their therapeutic levels in the body, so the administration of ethanol or fomepizole requires dose regulation in patients undergoing dialysis. With loading dose of 600 mg/kg, the maintenance dose of ethanol should be increased to 169 mg/kg/hours during dialysis or a lower 66 mg/kh/hours without dialysis. If Fomepizole is used, the loading dosage (15 mg/kg) and maintenance dosage (10 mg/kg every 12 hours for up to 48 hours, and then increased to 15 mg/kg) is the same bertween patient receiving dialysis and those without. But the maintenance dose in patient receiving dialysis should be given as early as 6 hours after the loading dose, and then continued every 4 hours (Barceloux et al., 2002; Nelson et al., 2019; Roberts et al., 2015).

Formic acid formed in the body will basically be converted into carbon dioxide by a series of enzymatic processes and then excreted. The enzymatic process in the excretion of formic acid in the form of carbon dioxide can be accelerated by administering folic acid which works as a substrate and catalyst (Barceloux et al., 2002). In addition, hemodialysis has been widely approved by the latest guidelines as a treatment option that can be used routinely to accelerate the elimination of methanol as well as its toxic metabolites and to improve the acidosis that occur (Barceloux et al., 2002; Roberts et al., 2015). Hemodialysis has also been reported to be able to improve the clinical outcomes of methanol intoxication patients including improved visual acuity (Karimi et al., 2021). The half-life of formate in patients undergoing hemodialysis therapy is much shorter (1.1 hours) compared to patients who do not undergo hemodialysis (3.7 hours) (Barceloux et al., 2002).

Hemodialysis management is recommended if 1 of the following conditions is found: 1) acidosis with pH <7.3, 2) the emergence of new visual disturbances, 3) worsening of the condition despite intensive supportive management, 4) renal failure, 5) significant electrolyte disturbances that do not respond to conventional therapy, 6) serum methanol concentration ≥50 mg/dL (Barceloux et al., 2002). Existing studies support the use of intermittent hemodialysis over continuous renal replacement therapy (CRRT), except in patients with unstable hemodynamic conditions (Kute et al., 2012; Kuusela et al., 2022; Roberts et al., 2015; Zakharov et al., 2017). The recommended duration of hemodialysis is 8 hours in the context of unavailable serum methanol analysis. However, recent studies have used shorter hemodialysis durations (4 to 6 hours), related to the high demand for hemodialysis machines and limited capacity. Therefore, the determination of the duration of hemodialysis should be determined according to each healthcare facility (Kute et al., 2012). The methanol clearance ratio ranges from 125 to 215 mL/min depending on the blood flow rate. At a blood flow rate of 100-400 mL/min, the average methanol clearance ratio reached 200 mL/min and formic acid reached 223 mL/min (Barceloux et al., 2002; Kute et al., 2012). It should be noted that ADH

inhibitor therapy should be continued even though the patient is undergoing hemodialysis therapy. This is because both ethanol and fomepizole can be dialyzed during the hemodialysis process causing their serum levels to be subtherapeutic (Roberts et al., 2015). Treatment with hemodialysis or ADH inhibitors can be stopped when serum methanol levels are known to have decreased to less than 20 mg/dL or the acidosis condition has improved (Barceloux et al., 2002; Kraut, 2016; Roberts et al., 2015).

The patient was consulted to the Department of Ophthalmology regarding the toxic optic neuropathy condition that occurred. From the examination, visus oculi dextra (VOD) was obtained: 1-meter counting finger (1/60) and visus oculi sinistra (VOS): 3-meter counting finger (3/60). Examination of the eyeball pressure with Schiotz obtained oculi dextra (OD) 11.2 mmHg and oculi sinistra (OS) 12.2 mmHg. Examination of the eyeball movement, both OD and OS, was normal without any pain in eyeball movement. Examination of the visual field of OD and OS found counting fingers in all quadrants. Examination of the anterior segment OD and OS found palpebra OD: negative edema, negative spasm; palpebra OS: negative edema, negative hematoma; conjunctiva OD and OS found negative hyperemia; cornea OD and OS were clear; anterior chamber OD and OS were deep; iris appeared radial in OD and OS; pupil was round with a diameter of 3 mm and positive corneal reflex and negative of relative afferent pupillary defect (RAPD) in OD and OS; lens was clear in OD and OS. Funduscopic examination in both OD and OS showed positive fundus reflex, N.II papilla with blurred superior area, hyperemic color, cup disk ratio difficult to evaluate, negative Pattons line, negative peripapillary hemorrhage, negative AV tortuosity, AV ratio 2:3, positive retinal hemorrhage, negative exudate, and positive macular reflex. The patient was given therapy in the form of intravenous steroid injection and neurotrophic to improve the toxic optic neuropathy that occurred.

Eye damage due to methanol intoxication is caused through 2 pathways: retinal damage (due to injury to Müller cells and photoreceptors) and optic neuropathy. An examination can show nystagmus, slow pupil movement, and disc edema to hyperemia. Formic acid formed as the result of methanol metabolism causes inhibition of the cytochrome oxidase pathway in the mitochondria, resulting in tissue hypoxia to optic nerve demyelination (Althwanay et al., 2020; Barceloux et al., 2002; Karimi et al., 2021; Kraut, 2016). Many studies have reported the benefits of steroid administration related to clinical outcomes of visual acuity in patients with methanol intoxication (Liberski et al., 2022). Recently, erythropoietin has begun to be widely studied and used in the therapy of optic neuropathy due to methanol intoxication. The use of erythropoietin is based on its neuroprotective effects on glial cells, anti-inflammatory, anti-apoptotic, antioxidant, and the ability to repair blood flow in damaged tissue (Alrobaian et al., 2024; Liberski et al., 2022).

SUMMARY

A case of a 20-year-old man with methanol intoxication treated with hemodialysis has been reported. The diagnosis was made based on clinical assessment referring to the methanol intoxication management algorithm. The patient was then given supportive treatment to stabilize the general condition and correct the acidosis that occurred. The patient then given an antidote with intravenous ethanol and hemodialysis with the aim of inhibiting the formation of further formic acid toxic substances and accelerating its elimination from the body. The patient was also given intravenous steroid therapy for the optic neuropathy that occurred.

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