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Current Management of Ethylene Glycol Poisoning

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Contents

Abstract

Ethylene glycol, a common antifreeze, coolant and industrial solvent, is responsible for many instances of accidental and intentional poisoning annually. Following ingestion, ethylene glycol is first hepatically metabolised to glycoaldehyde by alcohol dehydrogenase. Glycoaldehyde is then oxidised to glycolic

acid, glyoxylic acid and finally oxalic acid. While ethylene glycol itself causes intoxication, the accumulation of toxic metabolites is responsible for the potentially fatal acidosis and renal failure, which characterises ethylene glycol poisoning.

Treatment of ethylene glycol poisoning consists of emergent stabilisation, correction of metabolic acidosis, inhibition of further metabolism and enhancing elimination of both unmetabolised parent compound and its metabolites. The prevention of ethylene glycol metabolism is accomplished by the use of antidotes that inhibit alcohol dehydrogenase. Historically, this has been done with intoxicating doses of ethanol. At a sufficiently high concentration, ethanol saturates alcohol dehydrogenase, preventing it from acting on ethylene glycol, thus allowing the latter to be excreted unchanged by the kidneys. However, ethanol therapy is complicated by its own inherent toxicity, and the need to carefully monitor serum ethanol concentrations and adjust the rate of administration.

A recent alternative to ethanol therapy is fomepizole, or 4-methylpyrazole. Like ethanol, fomepizole inhibits alcohol dehydrogenase; however it does so without producing serious adverse effects. Unlike ethanol, fomepizole is metabolised in a predictable manner, allowing for the use of a standard, validated administration regimen. Fomepizole therapy eliminates the need for the haemodialysis that is required in selected patients who are non-acidotic and have adequate renal function.

Ethylene glycol poisoning is not a rare event. Nevertheless, it represents a clinical challenge to the physician who must properly diagnose and treat this poisoning. Successful treatment depends on astute and rapid diagnosis, aggressive supportive care, appropriate use of specific antidotes and, in selected patients, haemodialysis.

This review will focus on the current pharmacotherapy and management of ethylene glycol poisoning. For extensive monographs on this poisoning, the reader is referred to several excellent publications.[1,2]

1. Chemistry and Use of Ethylene Glycol

1.1 Sources

The most common sources of ethylene glycol are antifreeze, coolant and deicing solutions. Supplied in a relatively pure form, it is typically diluted to a 50% aqueous solution for this use. Other sources of ethylene glycol, as well as other toxic glycols, include brake and hydraulic fluids, solvent in inks, window and other household cleaners. The compound is also used in the manufacture of explosives, plastics and synthetic fibres.[3]

1.2 Properties

Ethylene glycol, 1,2-ethanediol, has molecular formula $C_2H_6O_2$ and a molecular weight of 62.07 g/mol. It is a sweet tasting, viscous, nonvolatile, colourless and very hygroscopic liquid. The estimated lethal dose of 100% ethylene glycol is approximately 1.4 ml/kg.[3]

2. Ethylene Glycol Toxicity

2.1 Epidemiology

The American Association of Poison Control Centers collected data on over 2 million poison exposures from 65 poison centres in the US in 1998. These participating centres served a population of 257 million people, or 95% of the US population.[4] Of these, 5376 were potentially toxic exposures to automotive ethylene glycol, resulting in 14 deaths and 81 life-threatening poisonings. Other forms of ethylene glycol were responsible for an additional 905 exposures resulting in 9 deaths and 67 life-

threatening poisonings.[4] It is likely that the poison centre data capture only a minority of the total number in the US. Many instances of ethylene glycol poisonings are the result of suicidal intent[4] and these generally involve ingestion of large volumes of ethylene glycol and late presentation for healthcare.

2.2 Pharmacokinetics

Ethylene glycol is poorly absorbed by dermal and pulmonary routes, but is readily absorbed from the gastrointestinal tract. Peak serum ethylene glycol concentrations occur 1 to 4 hours after ingestion. Ethylene glycol is not protein bound and, because it is highly water-soluble, distributes evenly throughout body tissue with a volume of distribution ≈0.5 to 0.8 L/kg.[5] The step-wise hepatic metabolism of ethylene glycol proceeds in an nicotinamide adenine dinucleotide (NAD)-dependent fashion. The first step is oxidation to glycoaldehyde by alcohol dehydrogenase (the antidotal treatment of ethylene glycol poisoning is based on the inhibition of this enzyme) [fig. 1]. Subsequently, glycoaldehyde is oxidised to glycolic acid, to glyoxylic acid and finally to oxalic acid. Because the conversion of glycolic to glyoxylic acid is the rate-limiting step in this process, the accumulation of glycolic acid is largely responsi-

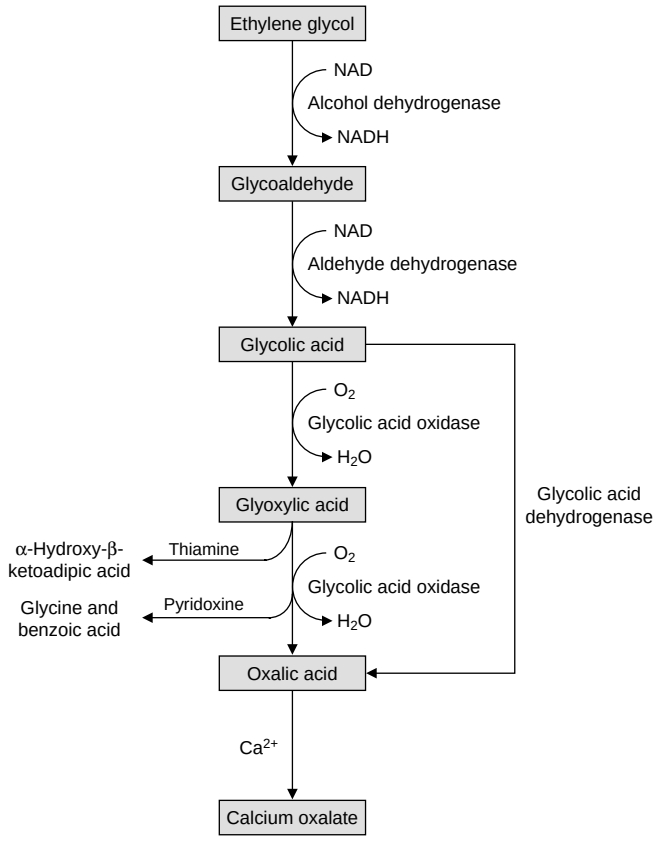

Fig. 1. The major pathway in the metabolism of ethylene glycol and the rationale for therapeutic intervention. **NAD** = nicotinamide adenine dinucleotide; **NADH** = dihydronicotinamide adenine dinucleotide.

ble for the metabolic acidosis seen in this poisoning.[6] Approximately 80% of an absorbed dose of ethylene glycol is hepatically metabolised, with the remainder renally excreted unchanged. In the rhesus monkey, the kidney excretes 0.5 to 10% of a dose of ethylene glycol as calcium oxalate.[7] The average elimination half-life of ethylene glycol is about 3 hours,[8] however inhibition of alcohol dehydrogenase results in a prolonged half-life of approximately 17 hours.[8]

2.3 Pathophysiology

2.3.1 CNS

Several mechanisms are responsible for the CNS effects seen in ethylene glycol poisoning. Early in the course of poisoning, CNS effects are the result of the direct action of ethylene glycol. Like ethanol, low doses of ethylene glycol cause euphoria and intoxication, whereas high doses cause CNS depression leading to coma. As the poisoning proceeds, the increase in metabolic acids, especially glycolic acid, contribute to CNS depression.[6,9] Persistent coma may be due to encephalopathy or cerebral oedema.[9,10] Seizures may be caused by a direct CNS-toxic effect. Hypocalcaemia may also contribute to the aetiology of seizures.[1,2,10]

2.3.2 Metabolic

The metabolism of ethylene glycol results in the production of several organic acids and glycolic acid is the predominant metabolite found in plasma (fig. 1). As the conversion of glycolic to glyoxylic acid is the rate-limiting step, accumulated glycolic acid is primarily responsible for the characteristic ethylene glycol-induced metabolic acidosis.[11,12] The first 2 steps of ethylene glycol metabolism cause the reduction of NAD to dihydronicotinamide adenine dinucleotide (NADH). The resulting high NADH to NAD ratio facilitates the conversion of pyruvate to lactate, thus causing a lactic acidosis.[11] The formation of aldehyde and acid metabolites also causes inhibition of other metabolic pathways, such as oxidative phosphorylation.

2.3.3 Cardiovascular

Ethylene glycol-induced acidosis and hypocalcaemia are believed to be responsible for the dysrhythmias and myocardial depression seen in patients poisoned with ethylene glycol. While calcium oxalate crystals in myocardial tissue and focal haemorrhaging have been noted on autopsy of fatally poisoned victims, the clinical significance of these findings remains unknown.[13]

2.3.4 Renal

The well known renal toxicity of ethylene glycol has traditionally been thought of as renal tubular injury caused by the accumulation of calcium oxalate crystals. However, only a small fraction of an ingested amount of ethylene glycol results in calcium oxalate formation. In addition, the degree of necrosis correlates poorly with the amount of oxalate crystals deposited, suggesting there may be other mechanisms of renal toxicity, such as direct cytotoxic effects of the glycolate metabolite. The net effect is a reversible oliguric or anuric renal failure.[14]

2.3.5 Histopathology

Patients fatally poisoned with ethylene glycol exhibit calcium oxalate crystals in various tissues including the brain, heart, lungs and the kidneys, in which oxalate crystals are seen in the proximal renal tubules.[13-18] Other postmortem changes include oedema of the brain and lungs, and petechial haemorrhaging in the lungs, pleura, pericardium and heart.[13,15]

2.4 Clinical Effects

In the past, ethylene glycol poisoning has often been described as occurring in 3 classical stages.[19] However, poisoned individuals will not always develop each stage or follow a specific time frame.[1] Also, the presence of ethanol, or other alcohols or glycols, as a co-ingestant can greatly delay the development of toxic signs and create a confusing clinical picture.

2.4.1 Stage 1: Neurological (0.5 to 12 hours)

Ethylene glycol produces an intoxication resembling ethanol but, because of its low volatility,

Table I. Suggested criteria for the presumptive diagnosis of ethylene glycol poisoning

(a) A history or suspicion of ingesting ethylene glycol plus any 2 of the following:
Arterial pH < 7.3
Serum bicarbonate of < 20 mEq/L
Osmolal gap > 10 mOsm/L
Presence of urinary oxalate crystals
OR
(b) A history or suspicion of ethylene glycol ingesting within the last *1 hour* and osmolal gap > 10 mOsm/L

the characteristic odour of ethanol is not detected. Early effects may include slurred speech, ataxia and somnolence. Gastric irritation may cause nausea and vomiting. As the metabolism of ethylene glycol proceeds and metabolic acidosis develops, the CNS depression deepens. Other commonly reported effects include ataxia, nystagmus, areflexia, myoclonic movements, cerebral oedema and seizures.[10]

2.4.2 Stage 2: Cardiopulmonary (12 to 36 hours)

The second phase is caused by the accumulation of organic acids formed by the metabolism of ethylene glycol. Commonly reported signs include tachycardia, hypertension and metabolic acidosis with compensatory tachypnoea, Kussmaul's respiration and congestive heart failure. Serious hypocalcaemia may cause hyper-reflexia, muscle spasms, and QT interval prolongation. In untreated patients, death most commonly occurs during this period.[10,18]

2.4.3 Stage 3: Renal (24 to 72 hours)

The final phase of ethylene glycol poisoning is the result of acute renal injury and is characterised by renal tubular necrosis, haematuria, proteinuria, flank pain, oliguria, anuria and renal failure.[20] Renal failure is generally reversible although weeks, or rarely, months of haemodialysis may be required. Occasional bone marrow suppression with pancytopenia and leucocytosis has been reported.[20] Neuropathy involving cranial nerves has been reported as a late finding,[21] occurring up to 2 weeks after the ingestion of ethylene glycol.[22]

2.5 Diagnosis

The direct measurement of serum ethylene glycol concentration is the most definitive means of establishing the diagnosis. However, many hospital laboratories are unable to perform this useful assay. In the absence of a quantitative ethylene glycol level the presumptive diagnosis of ethylene glycol poisoning must be made based on the clinical presentation of the poisoning. Table I provides a set of criteria useful for making this diagnosis presumptively.[2,12] Although a presumptive diagnosis can be used to initiate treatment, a definitive diagnosis should ultimately be established with a quantitative serum ethylene glycol determination. A potentially useful test is the measurement of an osmolal gap. Using this technique, the serum osmolality is measured, ideally by freezing point depression,[1] and compared with the predicted osmolality based on the patients' measured sodium, glucose, blood urea nitrogen and any ethanol that may be present [table II(a)]. Although the presence of a large osmolal gap supports a suspicion of ethylene glycol poisoning, the absence of a gap does not rule it out because of wide individual variability.[1,23] The osmolal gap is caused by the ethylene glycol itself, not its acidic metabolites. Thus, as

Table II. Calculation of osmolal and anion gaps

(a) Osmolal gap:
Osmolal gap = measured osmolality – calculated (or predicted) osmolality
Normal osmolal gap < 10 mOsm/L
Calculation of Predicted Osmolality
If using SI units:
Predicted osmolality = 2 (Na^+ + glucose + BUN + ethanol)*(all in mmol/L)
If using mass units:
Predicted osmolality = 1.86 Na^+ + glucose/18 + BUN/2.8 + ethanol*/0.46 (in mg/L)
* If present
(b) Anion gap:
Anion gap = $Na^+ – (Cl^- + HCO_3^-)$
The normal anion gap is typically between 3 and 16, depending on the methodology used. Calculated anion gaps should therefore be compared with the stated laboratory normal values.

BUN = blood urea nitrogen; **SI** = Systèm International

metabolism decreases the serum ethylene glycol concentration, the serum osmolal gap will decrease despite worsening toxicity.[1,24] Finally, it should be remembered that the presence of other alcohols (e.g. ethanol, isopropanol or methanol) and pathological conditions such as alcoholic or diabetic ketoacidosis, lactic acidosis, renal failure and shock may also produce an elevated osmolal gap.

Serum calcium levels may diminish as the concentration of oxalate increases. The calcium oxalate thus formed is often detectable in urine in the form of mono- (needle-shaped) and dihydrate (envelope-shaped) crystals. As the monohydrate form resembles hippurate crystals, the dihydrate form is a better indicator of ethylene glycol poisoning. The presence of oxalate crystals in the urine can only be used as supporting evidence as only 50% of patients poisoned with ethylene glycol have this finding on admission.[25] Another abnormal laboratory finding which may suggest ethylene glycol poisoning is the presence of a metabolic acidosis, particularly if it is characterised by a large anion gap [table II(b)]. The anion gap is largely due to a decrease in serum bicarbonate levels and usually follows the development of acidosis. This observation is manifested initially during Stage 1 (section 2.4.1). The presence of a high anion gap metabolic acidosis may also be the result of other types of poisoning, such as methanol, salicylates, iron, isoniazid, paracetamol (acetaminophen), theophylline, and disease states such as uraemia, or diabetic and alcoholic ketoacidosis.[2] However, the diagnosis of ethylene glycol or methanol poisoning must always be seriously considered in any patient with a severe unexplained metabolic acidosis.

An occasionally useful means for determining the presence of ethylene glycol is the use of fluorescence, typically using a Wood's lamp. Some commercial radiator antifreeze products have fluorescein added, enabling radiator leaks to be detected in the presence of ultraviolet light.[26] Therefore, a Wood's lamp may reveal fluorescence of a patient's perioral area, clothing, vomitus or urine. The Wood's lamp test is most likely to be positive within a few hours of ingestion. As this technique is insensitive, a negative finding should not be used to rule out poisoning.[26,27]

3. Treatment of Ethylene Glycol Poisoning

3.1 Stabilisation

The patient with serious ethylene glycol poisoning may present in a critical condition. As with all poisoned patients, initial stabilisation must be instituted before other possible treatments can be employed. Airway management is important in the obtunded patient and endotracheal intubation, ventilation and oxygenation should be a priority. Although not empirically validated, it is probably sound practice to administer sufficient sodium bicarbonate to correct a severe metabolic acidosis. Mild to moderate acidosis should be treated primarily with antidotal therapy (see below). Overaggressive alkalinisation may aggravate hypocalcaemia and cause hypernatraemia.[1] Calcium chloride or calcium gluconate should only be used to correct serious deficits in serum calcium concentration as the administration of calcium may promote undesirable calcium oxalate formation.[1]

Seizures should initially be treated with benzodiazepines. Barbiturates are an appropriate second-line therapy. Other anticonvulsants may be added as needed. Ethylene glycol-induced seizures may be caused by profound hypocalcaemia; therefore levels of ionised calcium should be determined immediately. If there is the suspicion of hypoglycaemia in a patient experiencing seizures, for example the presence of QT prolongation, calcium should be empirically administered. Other life-threatening events, such as hypotension and dysrhythmias should be treated according to advanced cardiac life support standards.

3.2 Gastric Decontamination

Because ethylene glycol is rapidly absorbed from the gastrointestinal tract and may induce rapid loss of consciousness, ipecac-induced emesis should not be employed. Similarly, although gastric lavage or aspiration of gastric contents may be

theoretically useful in the removal of unabsorbed ethylene glycol from the stomach, recent consensus documents have questioned the utility of this procedure in the treatment of poisonings.[28] Aspiration or lavage of gastric contents using a small bore nasogastric tube may remove some ethylene glycol, but only if done shortly after ingestion. The ability of orally-administered activated charcoal to adsorb ethylene glycol appears questionable and charcoal is likely to be of clinical value only if administered very soon after ingestion. However, it should be administered if the presence of a toxic dose of a co-ingestant is suspected.[1] The usual dose of activated charcoal is 50g in adults and 1 to 2 g/kg in children, administered as an aqueous slurry.

3.3 Inhibition of Metabolism

Following the diagnosis of ethylene glycol poisoning and the institution of supportive measures, the next, and crucial, step in the treatment of this poisoning is inhibition of further ethylene glycol metabolism. Traditionally, inhibition of alcohol dehydrogenase is considered if serum ethylene glycol concentrations are greater than 2.0 mg/L (3.2 mmol/L). However, this value has not been empirically validated. In the absence of a quantitative level, treatment should be considered for patients meeting the criteria in table I.[2,12] Currently there are 2 antidotes used for this purpose, both of which act by inhibiting alcohol dehydrogenase.

3.3.1 Fomepizole

Fomepizole (4-methylpyrazole) is a potent inhibitor of alcohol dehydrogenase and is commercially available as a solution for intravenous administration.[29] Fomepizole has recently been shown to be highly effective as an antidote for ethylene glycol poisoning.[12,30] Fomepizole is currently the only treatment for ethylene glycol poisoning approved by the US Food and Drug Administration (FDA). Based on recent Practice Guidelines by the American Academy of Clinical Toxicology, fomepizole administration is currently considered the standard of care for the treatment of this poisoning.[2] Therapeutic doses have been shown to be well tolerated in human subjects.[12,30-33]

The intravenous loading dose of fomepizole is 15 mg/kg, diluted to at least 100ml of normal saline or 5% dextrose and infused over 30 minutes. The loading dose is followed by an intravenous maintenance dose of 10 mg/kg every 12 hours for 4 doses, and thereafter by 15 mg/kg every 12 hours until the serum ethylene glycol concentration is <2.0 mg/L (3.2 mmol/L).[2,29] Because fomepizole is dialysable,[34,35] the administration interval should be reduced to 4 hours during haemodialysis.[29]

3.3.2 Ethanol

Ethanol is a competitive substrate for alcohol dehydrogenase. This enzyme has a much greater affinity for ethanol than for ethylene glycol and ethanol has therefore been used to inhibit ethylene glycol metabolism. Traditionally, the goal of ethanol therapy is to achieve a serum ethanol concentration of 10.0 to 12.5 mg/L (21.7 to 27.1 mmol/L). This concentration is believed to saturate the enzyme, thus inhibiting further ethylene glycol metabolism.

In an attempt to achieve this goal, the loading dose of ethanol is generally 0.6 to 0.7 g/kg. To maintain the desired serum ethanol concentration, a maintenance infusion must also be administered. Because the rate of ethanol metabolism varies widely, depending on individual variability and history of chronic alcohol consumption, the rate of ethanol administration depends on the patient's history of alcohol use. Thus, the rate of infusion must be carefully titrated based on serial serum ethanol determinations. The maintenance dose of ethanol will range from 66 to 154 mg/kg/hour.[2] Because it is readily dialysable, ethanol administration must be increased by 2- to 3-fold in patients undergoing simultaneous haemodialysis.

Pharmaceutical grade ethanol can be administered intravenously as a 10% solution in 5% dextrose. Because of the low ethanol concentrations used, the volumes of administered intravenous solutions are large. This can be a major disadvantage in small children or patients prone to fluid over-

load. Unless known to be pyrogen-free, all intravenous solutions of ethanol must be filtered prior to administration.

As an alternative to intravenous infusion, ethanol, either pharmaceutical grade or alcoholic spirit beverage, to may be diluted to 20% in any palatable liquid and administered orally. Ethanol that has been denatured in any way must never be used by either route.

Ethanol therapy is usually continued until the serum ethylene glycol concentration is <2.0 mg/L (3.2 mmol/L). Currently, the accepted indications for ethanol therapy for ethylene glycol poisoning are unavailability of, or hypersensitivity to, fomepizole.[2]

3.3.3 Ethanol vs Fomepizole

Ethanol has a few distinct advantages over fomepizole: it is relatively inexpensive, readily available, and can be administered intravenously as well as orally. Although it has never been approved by the US FDA, it has been the traditional antidote for ethylene glycol poisoning for many years. However, ethanol therapy has many disadvantages. Frequent ethanol dosage adjustments are required to maintain a serum ethanol concentration of 10.0 to 12.5 mg/L (21.7 to 27.1 mmol/L), which mandates frequent (every 1 to 2 hours) monitoring of serum ethanol concentration. Because ethanol is removed during haemodialysis, ethanol infusions must be increased in patients undergoing dialysis. Finally, ethanol solutions often require manual filtration by pharmacy staff prior to intravenous administration. The significant adverse effects of ethanol therapy include CNS depression,[1] hypoglycaemia (especially in paediatric and malnourished patients), and possible hepatotoxicity. Thus, ethanol therapy may further complicate the already complex clinical course of ethylene glycol poisoning. Patients treated with ethanol must be kept in an intensive care unit.

In contrast, fomepizole therapy has several advantages over ethanol for the treatment of ethylene glycol poisoning. The standardised administration regimen reliably maintains therapeutic concentrations,[12] eliminating the need for constant serum monitoring. The adverse effects of therapy are minimal: most commonly dizziness, headache and nausea. Tolerability is good even at plasma fomepizole concentrations that were over 10 times higher than those considered therapeutic.[36] Fomepizole is the only antidote approved by the US FDA for the treatment of ethylene glycol poisoning. Like ethanol, the dose of fomepizole must be increased during haemodialysis. However, the use of fomepizole eliminates the need for haemodialysis in non-acidotic patients[1,12,30,37] and may also prove useful in the treatment of other types of glycol poisonings.[38]

The only disadvantage of fomepizole is the acquisition cost. However, there are many potential cost savings with fomepizole. Because it is a more reliable antidote than ethanol, morbidity from ethylene glycol poisoning may be reduced. Fewer fomepizole-treated patients may need haemodialysis, intubation or admission to intensive care units, and here is no need for frequent checking of serum fomepizole concentrations.

3.4 Vitamins

3.4.1 Thiamine

Thiamine is often administered to the patients with ethylene glycol poisoning in an attempt to prevent the formation of oxalic acid by facilitating the conversion of glyoxylic acid to α-hydroxy-β-ketoadipic acid, a nontoxic metabolite (fig. 1). The standard administration regime is 100mg thiamine given intravenously every 6 hours until ethylene glycol is no longer measurable in the serum.[2]

3.4.2 Pyridoxine

The administration of pyridoxine may prevent the formation of oxalic acid by converting glyoxylic acid to harmless glycine and hippuric acid metabolites (magnesium is a necessary cofactor) [fig. 1]. These metabolites are then renally excreted. However, it is not clear if pyridoxine has this effect in ethylene glycol poisoning. The typical dose of pyridoxine is 50mg administered intravenously every 6 hours. Excessive cumulative doses of pyridoxine over a short time may induce a toxic sensory

peripheral neuropathy. Thus, pyridoxine therapy should not be continued for longer than 24 hours.

3.5 Enhanced Elimination

3.5.1 Haemodialysis

A key step in the treatment of ethylene glycol poisoning is enhancing the elimination of unmetabolised ethylene glycol, as well as glycolic acid, in selected patients. Haemodialysis has been shown to be highly effective in the removal of both toxins from the blood.[11,39] There have also been anecdotal reports of the use of continuous venous-venous haemofiltration to detoxify patients poisoned with ethylene glycol. In addition, haemodialysis has the advantage of correcting the other metabolic derangements caused by ethylene glycol ingestion.

Haemodialysis has traditionally been indicated for ethanol-treated patients with a serum ethylene glycol concentration greater than 500 mg/L (8 mmol/L). Other indications are any serum ethylene glycol concentration in the presence of severe metabolic acidosis, renal failure, severe electrolyte imbalance, or generally deteriorating condition despite supportive measures.[2]

Patients are typically haemodialysed until the serum ethylene glycol concentration is less than 500 mg/L (8 mmol/L). Haemodialysis of patients who have been treated with an alcohol dehydrogenase-inhibitor will reduce the serum half-life of ethylene glycol from ≈17 hours to 2.5 to 2.7 hours.[8,40]

The disadvantages of haemodialysis include the additional cost of the procedure, the cost of increased doses of antidotes as these are removed by dialysis, and the risk to the patient associated with the invasive nature of haemodialysis. Recent evidence suggests that in patients who are treated with fomepizole prior to the development of acidosis, haemodialysis is unnecessary because this agent reliably inhibits ethylene glycol metabolism, allowing for urinary excretion of the parent compound.[1,12,30,37] Thus patients receiving fomepizole who are non-antidotic and have normal renal function do not routinely require haemodialysis. Unlike the situation with ethanol, it is not necessary to consider the serum ethylene glycol concentration when deciding upon haemodialysis in patients receiving fomepizole.

4. Conclusion

Ethylene glycol poisoning is a medical emergency that requires immediate and aggressive treatment. In the absence of a good history, proper treatment depends on correct interpretation of clinical clues, such as metabolic acidosis (particularly with a high anion gap), a high osmolal gap, signs of alcoholic inebriation without evidence of alcohol intoxication, or oxaluria.

Appropriate treatment includes aggressive supportive care, proper use of the antidotes fomepizole or ethanol to inhibit the synthesis of toxic metabolites, haemodialysis when necessary to enhance the elimination of unmetabolised ethylene glycol and its toxic metabolites, and adjunctive therapy aimed at conversion of ethylene glycol to nontoxic metabolites. As each case of ethylene glycol poisoning presents with unique circumstances, the foregoing discussion can only be used as a general guideline.

In the event of a confirmed or suspected case of ethylene glycol poisoning, the clinician unfamiliar with this medical emergency is urged to contact a Regional Poison Center or consult a medical toxicologist for assistance.

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