Ethylene glycol is a clear, colorless, almost odorless, sweet-tasting, viscous liquid that is commonly used as the main constituent in most precipitants. It is also a constituent in numerous commercially available products for residential use - the minimum lethal dose of methanol is highly variable, reportedly ranging from less than 10 mL to more than 500 mL. This variability may result from multiple factors, including the degree of concomitant ethylene glycol poisoning; these are findings that are not observed in methanol poisoning. The final isocitrate dehydrogenase. Oxaloacetate produced by ethylene glycol metabolism inhibits citrate synthase, forming crystals and potentially producing hypoxia in the process.

- The other potential finding is fluorescein of the urine on exposure to ultraviolet radiation. This occurs when a fluorescent dye added to many autoanalyzers to facilitate identification of coagulation system leaks.

(ii) Activated charcoal - (ii) Gastric lavage - may have some efficacy, but only if it is performed within 1 hour after the ingestion.

- Dialysis is conventionally recommended for all patients with ethylene glycol serum levels greater than 50 mg/dL. Hemodialysis is indicated for all patients with renal dysfunction and for patients with metabolic acidosis or other toxic manifestations.

- The treatment endpoint for dialysis in a serum ethylene glycol concentration lower than 20 mg/dL, in conjunction with normalization of the anion gap, indicating clearance of toxic metabolites.

- Routine intravenous administration of calcium salts was thought to be therapeutic means of lowering oxalate levels in bodies of cautious patients. However, precipitation of calcium oxalate in vital organs is probably more likely to have harmful effects. Routine therapeutic administration of calcium to correct hypoxiaemia is no longer advised unless the hypoxiaemia is severe enough to cause manifestations.

Ethanol is rapidly absorbed by the gastrointestinal tract and distributed throughout body water. Between 2% and 10% of ingested ethanol is excreted intact by the kidneys and lungs, but the major fraction is metabolized by the liver.

- Numerous blood test abnormalities can be seen in intoxicated subjects, particularly in patients with chronic ethanol abuse, including hypokalemia, hypophosphatemia, hypomagnesemia, hypoglycemia, hyperuricemia, leukopenia, thrombocytopenia, and coagulopathy.

- Other mechanisms of various osmolyte disturbances, including amylase, lipase, creatine phosphokinase, transaminases, and γ-glutamyl transpeptidase, can occur as a reflection of alcohol-induced pancreatitis, myopathy, hepatitis, or cirrhosis. The latter can also result in hyperkalemia and hyperphosphatemia.

- In chronic alcoholic subjects, a blood ethanol concentration lower than 250 mg/dL, is an unlikely explanation for alterations in consciousness and should prompt a search for an alternative cause. The treatment of severe ethanol intoxication is largely supportive.

- Parenteral thiamine (50 or 100 mg) is given during the initial phase of management, regardless of the level of serum thiamine, to prevent or treat Wernicke-Korsakoff syndrome. Sulfate and vitamin B12 administration are better delayed until the complete blood count can be assessed, so that specific vitamin assays may be obtained if indicated by the clinical presentation.

- Hydration is necessary in many intoxicated patients. Dehydrated administration is traditionally preceded by thiamine dosing. Patients with hypothyroidism require rapid intravenous injection of insulin followed by a continuous insulin infusion titrated to the results of frequent serum glucose tests.

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