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Cost-effective Detection Methods of Corrosive Poisons in Post-mortem Forensic Samples

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ABSTRACT: ***Background:*** Forensic Toxicology plays a key role in explaining the events surrounding suspicious deaths, yet financial constraints often impede access to advanced detection technologies. This review study explores the effectiveness of traditional chemical methods for detecting poisons in post-mortem samples, particularly corrosive poisons, as a cost-effective alternative to modern analytical tools and techniques. ***Method:*** A literature review is carried out to explore cheap and effective analytical methods for corrosive poisons. Our review encompassed forensic toxicology literature dating back to 1955 and more recent publications on the subject. We also examined online resources to compare our findings with the latest advancements in analytical technologies. ***Results:*** Various cost-effective methods are available and can be used for the detection of both organic and inorganic corrosive poisons including acetic acid, ammonia, carbolic acid, creosote, citric acid, hydrochloric acid, hydroxide and carbonates of potassium, hydroxide, and carbonates of sodium, nitric acid, oxalic acid, picric acid, potassium permanganate, salicylic acid, sulfuric acid, and tartaric acid. ***Conclusions:*** Classical chemical methods remain relevant and practical for daily toxicological analyses in resource-constrained settings due to their affordability, ease of use, and reliability. Despite the advantages of modern tools, the high costs and complexity make traditional methods a viable and necessary alternative for small-scale forensic laboratories.

Keywords: Forensic Toxicology, Corrosive Poisons, Toxicological Analysis, Chemical Method, Cost-Effective, Poison Detection.

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INTRODUCTION

The investigation of suspicious and sudden deaths within the area of forensic science (toxicology) hinges upon the precise identification of poison(s) present in the post-mortem samples. In such cases, toxicological analysis is a key tool for determining the circumstances behind the deceased's death. However, this is frequently hampered by the cost limits inherent in forensic investigations. Achieving a balance between the demand for accuracy and the limited budgetary resources remains a constant challenge for forensic professionals worldwide, particularly in developing nations.

Detecting corrosive poisons in post-mortem forensic samples is an important aspect of forensic toxicology, playing a key role in determining the cause of death in cases where foul play is suspected. Corrosive poisons, including strong acids and alkalis, are substances that cause significant chemical burns and tissue damage, leading to severe injuries or death. Their presence in the body can provide critical evidence in criminal investigations, therefore their detection is a key focus for forensic scientists.

Detection of corrosive poisons involves sophisticated and often expensive laboratory equipment, such as gas chromatography-mass spectrometry (GC-MS) and high-performance liquid chromatography (HPLC). A brand-new gas chromatograph (GC) costs between \$30,000 and \$50,000.[1] Liquid chromatography (LC) equipment can range from \$10,000 to \$30,000 for entry-level devices. The cost of an advanced LC fitted with high-pressure pumps, autosamplers, and detecting modules ranges from \$40,000 to \$100,000.[2] The cost range for High-Performance Liquid Chromatograph (HPLC) systems is \$15,000 to \$100,000.[3] Depending on the model, the cost of a mass spectrometer (MS) can vary from \$50,000 to \$1,000,000 for MALDI-Time of Flight (TOF); from \$100,000 to \$1,000,000 for Q-TOF; from \$75,000 to \$1,000,000 for Triple Quadrupole; from \$20,000 to \$500,000 for Ion Trap; and from \$150,000 to \$2,500,000 for Fourier Transform.[4]

The introduction of these affordable detection techniques has the potential to transform forensic toxicology. These advancements can assist in guaranteeing that forensic labs can offer precise and timely results, irrespective of their financial limitations. This is also crucial in legal settings, where rapid and accurate forensic evidence can greatly impact legal proceedings.

Analytical requirements are one of the many difficulties the forensic laboratory encounters due to the growing need for toxicology. However, the resources available to meet the endless demands of public justice are finite.[5] This article reviews cost-effective techniques that rival contemporary expensive approaches to detecting poisons, primarily focusing on chemical methods employed for identifying corrosive poisons, encompassing both organic and inorganic acids. These techniques offer a viable option for regions and organizations with limited financial resources, enabling them to contribute to justice despite lacking access to costly laboratory equipment.

Sample Preparation

In deceased individuals, the source of poison detection is stomach contents, visceral organs, body tissues, and bowels. Most commonly stomach contents are used for poison detection.[6] Water is added to the stomach content. If poison cannot be detected from symptoms and examination, divide the mixture into three parts for the detection of mineral poisons, vegetable poisons, and volatile poisons.[7]

Mineral poisons are extracted from the organic mixture by either the dry method or the wet method. In the dry method, organic content is exposed to heating, and nitric acid is added to the ashes. Heat the solution to remove free acid and let the nitrates dissolve. Add potassium hydroxide, only if the solution becomes more acidic. The wet method involves the addition of concentrated hydrochloric acid, about a third portion, to the organic mixture. Heat the solution and add potassium chlorate in small portions during heating. Filter the solution as it contains chloride of the metal. The solution thus obtained either by applying the wet or the dry method is used for confirmatory tests.[7, 8]

Corrosive Poisons

Corrosive poisons are chemicals, such as mineral and organic acids, which cause tissue destruction.[9] On contact with body organs such as the oral cavity, throat, esophagus, and stomach, corrosive acids show signs of partially deep thick second-degree burns. These poisons result in cell death due to ischemia (coagulative necrosis) and Esher formation.[10] This leads to sloughing and perforation of the stomach lining. These events along with systemic acidosis, hemolysis, mediastinitis, sepsis, shock, and decreased cardiac output lead to death.[11]

Acetic Acid

Acetic Acid (CH_3COOH) is a colorless organic acid with a pungent odor.[12] It occurs in combination with alcohol and acts as a corrosive poison in its concentrated form, glacial acetic acid. Its household preparation, *sirka* or vinegar, contains 4-5% acetic acid which can be poisonous if taken in large quantity. Acetic acid poisoning results in softening of areas of contact, discoloration to yellowish-white, pain, swallowing difficulty, convulsions, cough, suffocation, and hemoglobinuria.[13]

Acetic acid poisoning can be detected by the following methods.

A. Perform distillation on the poisonous sample. If the distillate has acetic acid, it has a distinctive odor. Heat the distillate with alcohol and sulfuric acid, if acetic acid is present, ethyl acetate is formed (Figure 1). Ethyl acetate is recognized by its unique aromatic smell.

B. Perform distillation on the poisonous sample. Neutralize the distillate with ammonia and add ferric chloride to it. If acetic acid is present, a deep red-colored solution is formed. Boil this solution, red color shifts to red-brown precipitates of ferric subacetate (Figure 2).

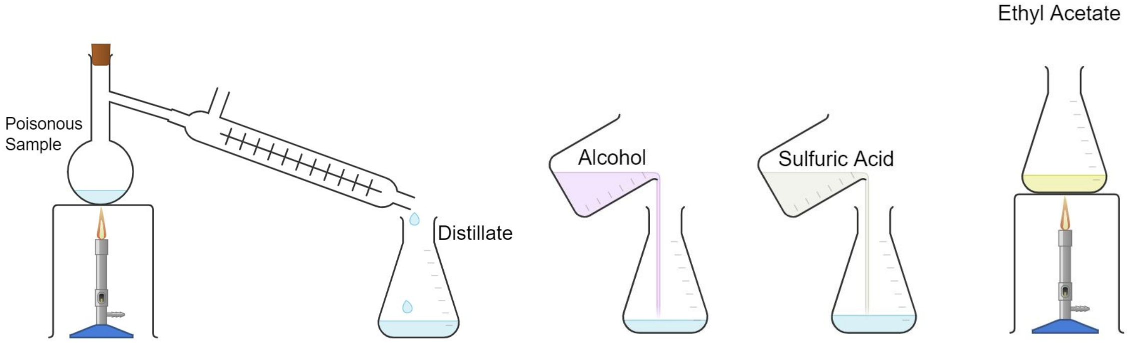

Figure 1: Illustration of Alcohol and Sulfuric Acid for Detection of Acetic Acid Poisoning

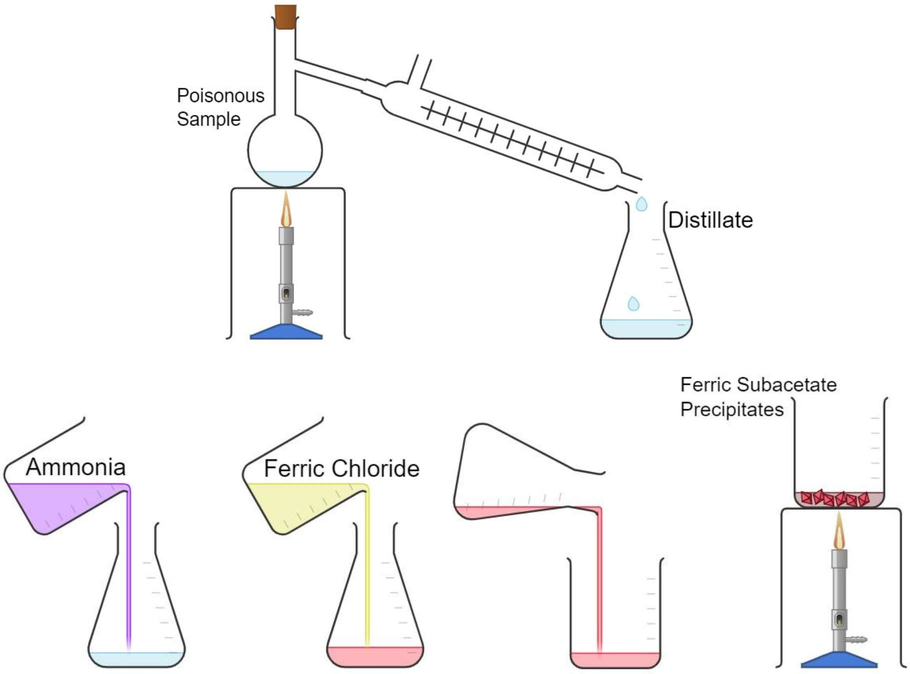

Figure 2: Illustration of Ammonia and Ferric Chloride for Detection of Acetic Acid Poisoning

Ammonia & derivatives

Ammonia (NH_3) is an alkali that exists as a colorless gas with a strong and unpleasant odor.[14] Inhalation may cause poisonous symptoms. Its liquid form, Hartshorn, involves dissolving gaseous ammonia in water forming a strong, colorless solution of ammonia with similar characteristic odor. Ammonium carbonate, $(NH_4)_2 CO_3$, exists as a luminous and hard crystalline mass. On exposure to air, it becomes porous and lumpy. Ammonia gas causes irritation and poisoning leading to death due to suffocation because of inflammation of the glottis, and pneumonia.

To detect ammonia poisoning, perform distillation to separate the organic mixture and ammonia and apply the following methods.

A. Mix the poisonous sample (distillate) with caustic potash and heat it. If it contains ammonia, ammonia gas is produced, identifiable by its odor (Figure 3). Bring red litmus paper near near the gas, it turns blue. Dip a glass rod in HCl and bring it near the NH_3 gas, white fumes of ammonium chloride will be produced.
B. Mix the poisonous sample (distillate) with Nessler's reagent (K2HgI4). If it contains ammonia, brown precipitates are formed (Figure 4).

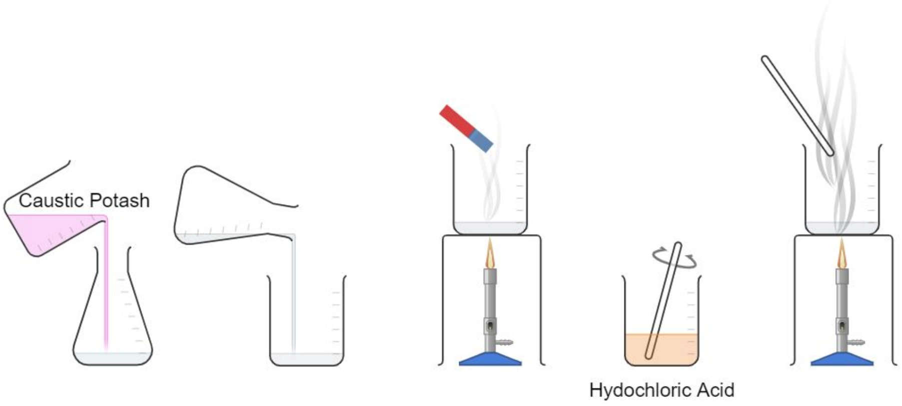

Figure 3: Illustration of Caustic Potash for Detection of Ammonia Poisoning

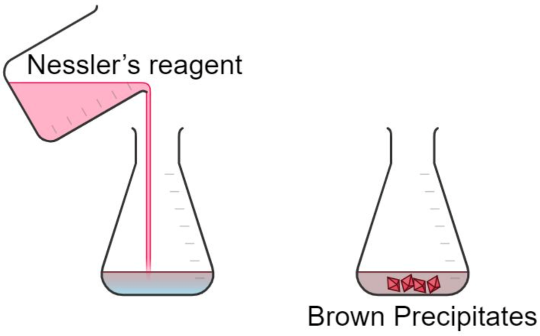

Figure 4: Illustration of Nessler's Reagent for Detection of Ammonia Poisoning

Carbolic Acid

Carbolic Acid (C_6H_5OH) is an organic corrosive aromatic hydrocarbon commonly used as a disinfectant.[15] Carbolic acid has many derivatives such as cresol, lysol, phenol, dettol, etc. In pure form, it exists as a colorless, prismatic, and needle-like structure. Pure carbolic acid needles turn pink and liquefy on exposure to light and moist air. It has a sweetish-burning taste and a unique phenolic or carbolic smell. Commercially available carbolic acid is a dark brown liquid with impurities such as cresol. Household preparations contain 5% phenol in water. Dettol is constituted of chlorinated phenol with terpineol. Carbolism, poisoning of carbolic acid, may lead to death due to paralysis of the cardiac and respiratory systems.[16]

Carbolism can be detected by the following methods. First, Separate the carbolic acid from organic matter. Wash the poisonous sample with ether. Pour the liquid into another container and wait for the ether to evaporate. Distillation (with dilute H_2SO_4) can also be performed for this purpose.

A. Mix one part of strong nitric acid with one part of mercury. Dilute the mixture twice with the water of its volume. Let the solution stand overnight and then pour down the clear liquid in another container and use it as a reagent. Heat the poisonous sample distillate with the reagent. If it contains carbolic acid, a red color is produced (Figure 5).
B. Add bromine water to the poisonous sample distillate. If it contains carbolic acid, whitish-yellow precipitates (tribromo-phenol) are formed (Figure 6). To confirm the presence of carbolic acid, the precipitates are treated with alcoholic preparation of sodium amalgam. It results in the formation of free carbolic acid.
C. Add some drops of dilute ferric chloride to the poisonous sample distillate. If it contains carbolic acid, the solution becomes violet-blue or purple (Figure 7). To confirm the presence of carbolic

acid and eliminate the possibility of salicylic acid, with the addition of acetic acid, the color of the solution remains the same.

D. Heat the poisonous sample distillate with ammonia and calcium hypochlorite. If it contains carbolic acid, the solution becomes blue (Figure 8). On adding acid, it changes to yellow or red.

E. Dissolve a minute quantity of poisonous sample distillate in Conc. H_2SO_4 and cool the mixture. Add a small crystal of sodium nitrite, if it contains carbolic acid, the solution becomes deep blue or green (Figure 9). Pour it into a water beaker, it turns red. On adding alkali, the red changes to blue or green.

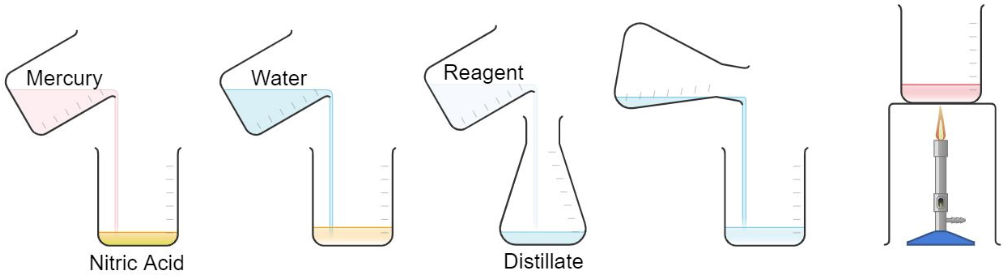

Figure 5: Illustration of Mercury and Nitric Acid for Detection of Carbolic Acid Poisoning

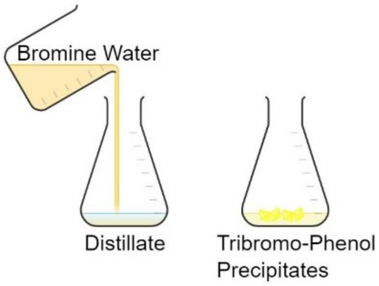

Figure 6: Illustration of Bromine Water for Detection of Carbolic Acid Poisoning

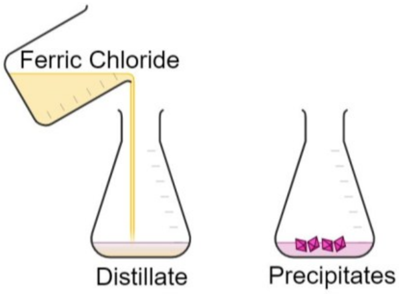

Figure 7: Illustration Of Ferric Chloride for Detection of Carbolic Acid Poisoning

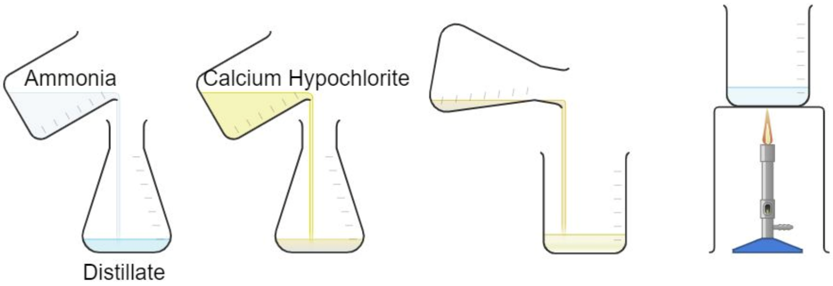

Figure. 8 Illustration of Ammonia and Calcium Hypochlorite for Detection of Carbolic Acid Poisoning

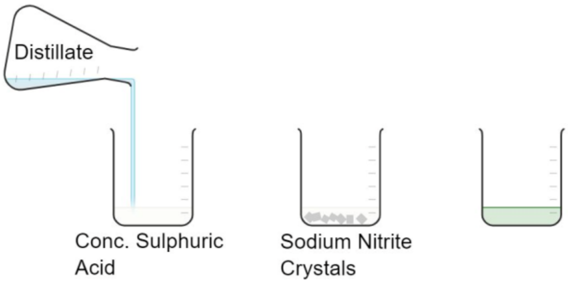

Figure 9: Illustration of Concentrated Sulfuric Acid and Crystals of Sodium Nitrite for Detection Of Carbolic Acid Poisoning

Citric Acid

Citric Acid ($C_6H_8O_7$) is an organic acid present in sour-taste fruits and about 0.05-1% in cow and human milk.[17] It is manufactured by heating lemon extract followed by neutralizing it with calcium carbonate. Physically it exists as white powder or colorless or white crystalline solid. It is not commonly used to induce harm to life but there are some instances where it is used as poison and to cause abortion.[34]

Citric acid poisoning can be detected by the following methods.

A. Boil a poisonous sample with calcium chloride. If it contains citric acid, white precipitates will be formed (Figure 10).
B. Add potassium permanganate to a poisonous sample solution. If it contains citric acid, the color changes to green (Figure 11).

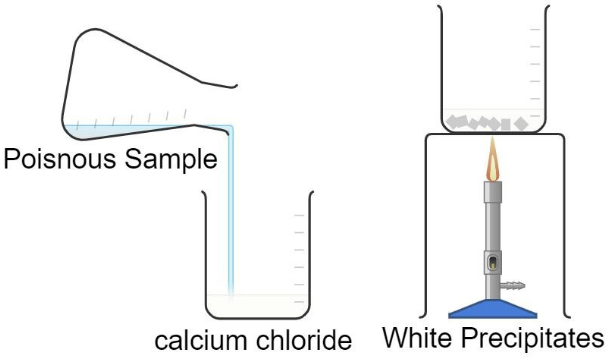

Figure 10: Illustration of Calcium Chloride for Detection of Citric Acid Poisoning

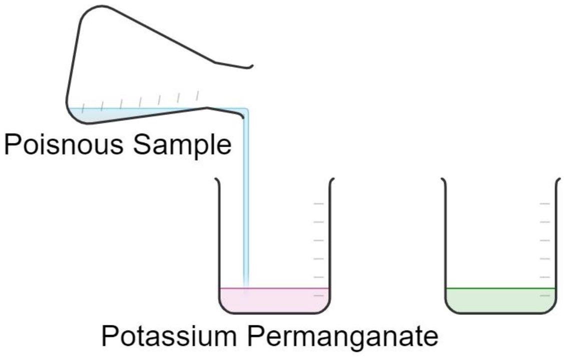

Figure 11: Illustration of Potassium Permanganate for Detection of Citric Acid Poisoning

Creosote

Creosote is an organic acid consisting of creosol, guaiacol, and phenols. It is obtained from tar distillation from wood or coal. It is oily, and colorless when fresh but turns brown on exposure to light. It is used for toothache, tuberculosis, wood preservation, and pesticides.[18] The poisoning effects caused by creosote resemble carbolism.

Creosote acid poisoning can be detected by the following method.

A. Mix a poisonous sample solution with ferric chloride. If it contains creosote, green or brown color formation occurs (Figure. 12). This color can be cleared by adding water.

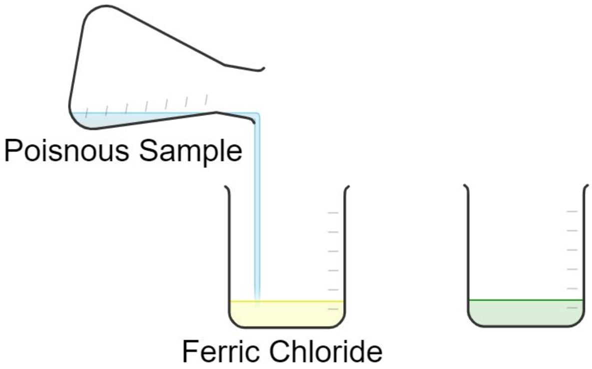

Figure 12: Illustration of Ferric Chloride for Detection of Creosote Poisoning

Hydrochloric Acid

Hydrochloric Acid (HCl) is an inorganic acid that exists as a colorless gas. Its watery solution exhibits a yellow color and is known as a spirit of salts/muriatic acid.[19] HCl is also present in the stomach (0.2 percent) as a digestive juice, its property as corrosive is relatively less than other inorganic acids but inhalation of its fumes causes irritation and damage to mucous membrane, throat, and lungs.[20]

HCl can be detected by applying tests on the distillate of vomit and stomach content.

A. When a solution of silver nitrate reacts with a poisonous solution. If it contains HCl, it forms thick lumpy, and white precipitates of silver chloride are formed (Figure 13). These white precipitates turn grey on exposure to sunlight.
B. A poisonous solution is heated with manganese dioxide. If it contains HCl, it produces chlorine gas (Figure 14). Such gas has a greenish-yellow color with an irritating smell.
C. When a poisonous solution is brought near ammonia gas. If it contains HCl, white-colored fumes of ammonium chloride are produced (Figure 15).

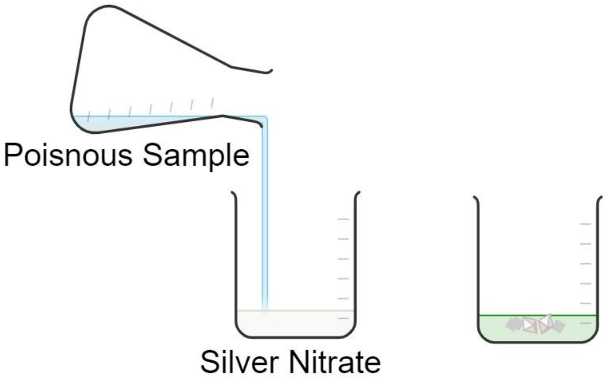

Figure: 13 Illustration of Silver Nitrate for Detection of Hydrochloric Acid Poisoning

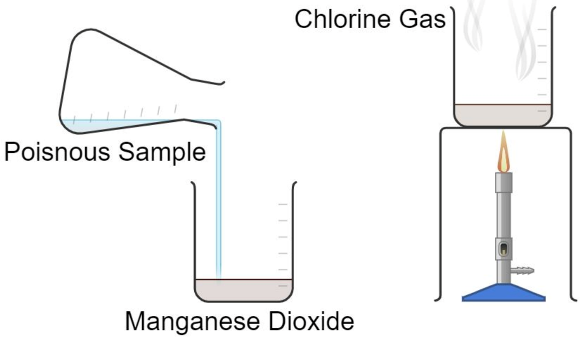

Figure 14: Illustration of Manganese Dioxide for Detection of Hydrochloric Acid Poisoning

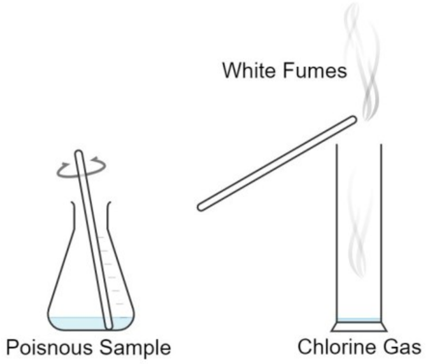

Figure 15: Illustration of Ammonia Gas for Detection of Hydrochloric Acid Poisoning

Hydroxides and Carbonates of Potassium

Potassium Hydroxide (KOH) is an alkali and inorganic compound that exists as an odorless lumpy solid, also known as caustic potash.[21] Potassium Carbonate (K_2CO_3), pearl ash, is an inorganic alkali compound that exists as a white crystalline powder. KOH and K_2CO_3 dissolve in water and form a strongly alkaline solution. These are mostly used in soap manufacturing. Poisoning causes more severe injuries than acids, dissolves proteins, saponification of fats, liquefaction necrosis, deep burns, edema, etc.[22]

Take a porcelain capsule containing organic matter (from the poisoned individual) and burn it to remove organic content in the form of ash. Acidulated water is added to this residual ash and the solution is tested for the presence of potassium by following methods.

A. Mix the poisonous sample with a solution of strong tartaric acid and alcohol. If it contains potassium, white precipitates are formed (Figure 16).
B. Mix platinic chloride with the poisonous sample. If it contains potassium, yellow crystalline precipitates are formed (Figure 17).
C. Take a clean nichrome wire and dip it in the poisonous sample. Introduce the wire to the flame. If it contains potassium, the flame color changes to violet (Figure 18).

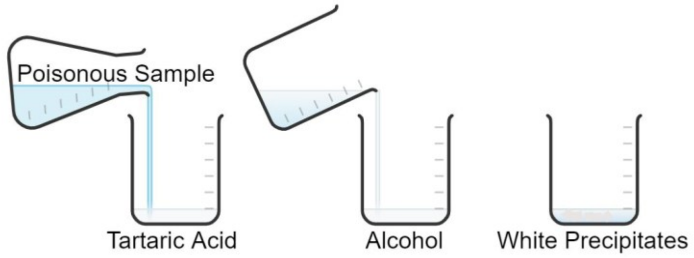

Figure 16: Illustration of Tartaric Acid and Alcohol for Detection of Potassium Poisoning

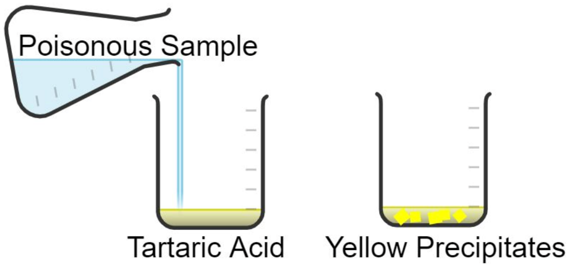

Figure. 17 Illustration of Platinic Chloride for Detection of Potassium Poisoning

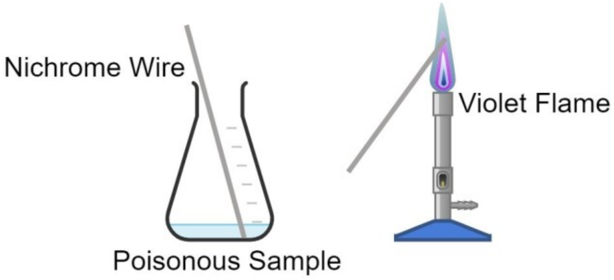

Figure 18: Illustration of Flame Test for Detection of Potassium Poisoning

Hydroxides and Carbonates of Sodium

Sodium Hydroxide (NaOH) is an alkali compound that exists as a white crystalline solid. It is commonly known as lye or caustic soda and is used in the manufacture of soap and cleaning products.[23] Sodium Carbonate (Na_2CO_3) exists as a white, odorless salt that forms an alkaline solution in water. It is commonly known as washing soda, soda ash, or soda crystal. A combination of caustic soda (NaOH) and washing soda (Na_2CO_3) is known as soap lye. Soap lye was used as a poisoning agent in the past.[23]

Take a porcelain capsule containing organic matter (from the poisoned individual) and burn it to remove organic content in the form of ash. Acidulated water is added to this residual ash and the solution is tested for the presence of sodium by following method.

A. Take a clean nichrome wire and dip it in the poisonous sample. Introduce the wire to the flame. If it contains sodium, the flame color changes to yellow (Figure 19).

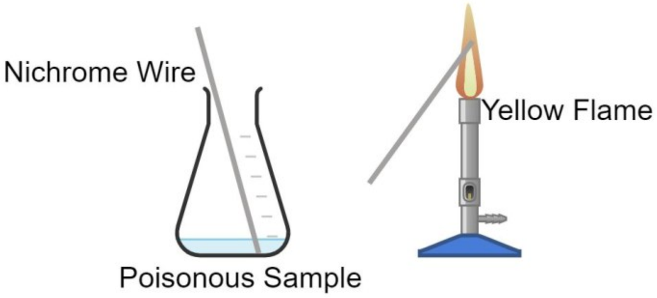

Figure 19: Illustration of Flame Test for Detection of Sodium Poisoning

Nitric Acid

Nitric Acid (HNO_3), also known as the red spirit of nitre or aqua fortis, is a colorless inorganic acid with a pungent odor.[25] The formulas available in the commercial sector vary in color from yellow to dark red. One of the commonly available solutions (of HNO_3) saturated with red oxides of nitrogen is known as fuming nitric acid. Inhalation of such fumes may cause cough, dyspnoea, eye irritation, and suffocation (leading to death). On contact with body organs, it undergoes a xanthoproteic reaction producing picric acid, and the area of contact turns yellow. Nitric acid poisoning can even turn the color of blood to yellowish-brown.

HNO_3 poisoning samples can be detected by tests such as:

A. Add a strong ferrous sulfate solution and sulfuric acid to a solution containing a poisoning sample. If it is nitric acid poisoning, a brown ring is formed at the junction of ferrous and sulfuric acid solution (Figure 20).

B. Add a poisoning sample to the brucine solution. If it is nitric acid poisoning, the solution turns blood-red color (Figure 21).

C. Heat a poisoning sample with strong sulfuric acid and copper. If it is nitric acid poisoning, reddish-brown fumes of nitric oxide are given off and the solution turns green (Figure 22).

D. Add a poisoning sample to the morphine solution. If it is nitric acid poisoning, the solution turns orange color (Figure 23).

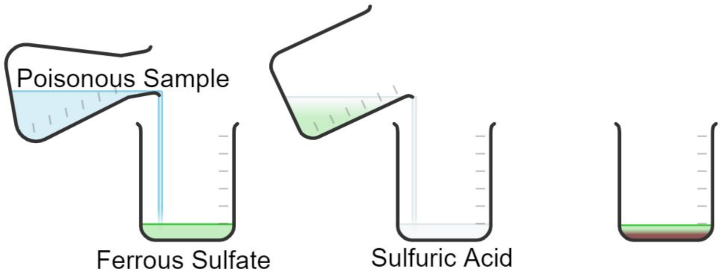

Figure 20: Illustration of Ferrous Sulfate and Sulfuric Acid for Detection of Nitric Acid Poisoning

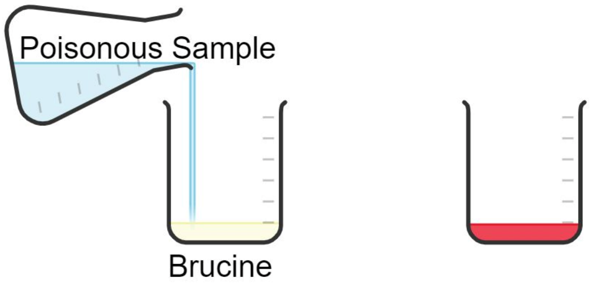

Figure 21: Illustration of Brucine for Detection of Nitric Acid Poisoning

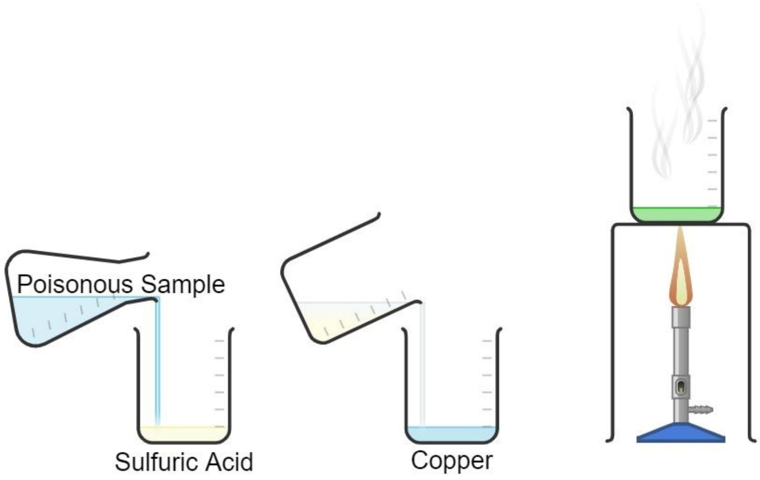

Figure 22: Illustration of Sulfuric Acid and Copper for Detection of Nitric Acid Poisoning

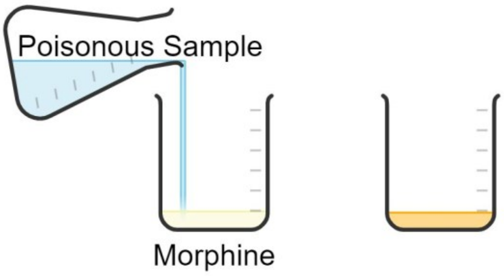

Figure 23: Illustration of Morphine for Detection of Nitric Acid Poisoning

Oxalic Acid

Oxalic Acid ($C_2H_2O_4$) is an organic acid, also known as salt of sorrel or acid of sugars, produced by oxidizing sucrose with nitric acid. It exists as colorless, transparent, and prismatic crystals in solid form.[26] In plants, it is present as oxalate and it is excreted (20mg) in urine daily. Concentrated doses of oxalic acid cause corrosive action while acting as an irritant in weaker solutions. It acts as poison if applied directly to the wounds. Oxalic acid poisoning causes hypocalcemia and renal failure leading to death.[27]

Oxalic acid poisoning can be detected by the following method.

A. Mix the poisonous sample solution with barium nitrate. If it contains oxalic acid, white precipitates of barium oxalate are formed (Figure 24). To confirm the presence of barium oxalate, add HCl or HNO_3 to dissolve precipitates without effervescence.
B. Mix the poisonous sample solution with silver nitrate. If it contains oxalic acid, white precipitates are produced (Figure 25). These precipitates are soluble in NH_3 and HNO_3.
C. Mix the poisonous sample solution with calcium chloride. If it contains oxalic acid, white precipitates are produced (Figure 26). These precipitates are soluble in HCl while insoluble in CH_3COOH.
D. Mix the poisonous sample solution with calcium sulfate. If it contains oxalic acid, white precipitates are produced (Figure 27). These precipitates are soluble in HCl while insoluble in CH_3COOH.
E. Mix the poisonous sample solution with lead acetate. If it contains oxalic acid, white precipitates are produced (Figure 28). These precipitates are insoluble in CH_3COOH while soluble in HNO_3.

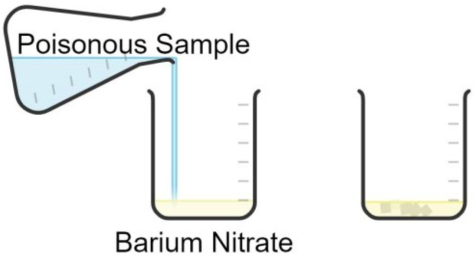

Figure 24: Illustration of Barium Nitrate for Detection of Oxalic Acid Poisoning

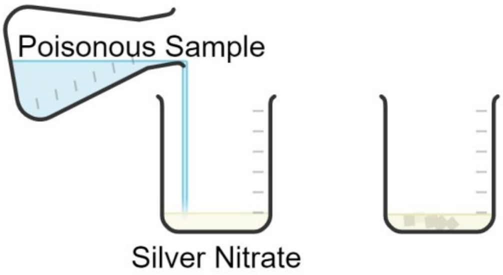

Figure 25: Illustration of Silver Nitrate for Detection of Oxalic Acid Poisoning

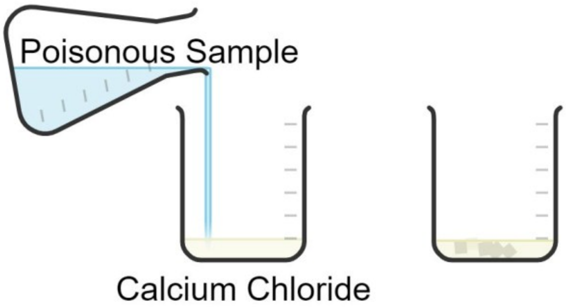

Figure 26: Illustration of Calcium Chloride for Detection of Oxalic Acid Poisoning

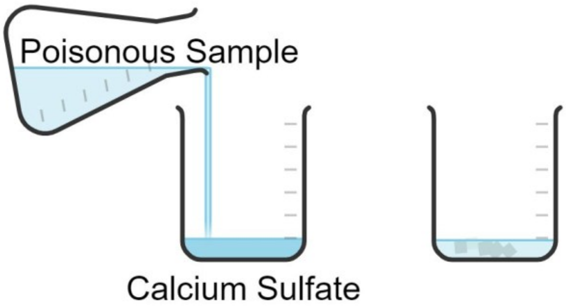

Figure 27: Illustration of Calcium Sulfate for Detection of Oxalic Acid Poisoning

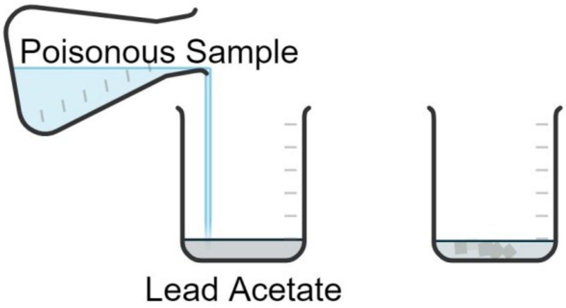

Figure 28: Illustration of Lead Acetate for Detection of Oxalic Acid Poisoning

Picric Acid

Picric Acid ($C_6H_3N_3O_7$) is an organic acid produced by the reaction of phenol with concentrated sulfuric acid and concentrated nitric acid. This results in odorless pale-yellow crystals.[28] It has toxic effects (may lead to death) in solution form and by inhalation of volatilized picric acid. Picric acid poisoning causes local necrosis, precipitation of albumin, decomposition of red blood corpuscles, production of methemoglobin, and irritation of the central nervous system leading to convulsions.

Picric acid poisoning can be detected by the following methods.

A. Add copper ammonia sulfate to a solution of a poisonous sample. If it contains picric acid, the formation of green precipitates will take place (Figure 29).
B. Heat the aqueous solution of the poisonous sample with potassium cyanide. If it contains picric acid, the solution changes its color to blood-red (Figure 30).

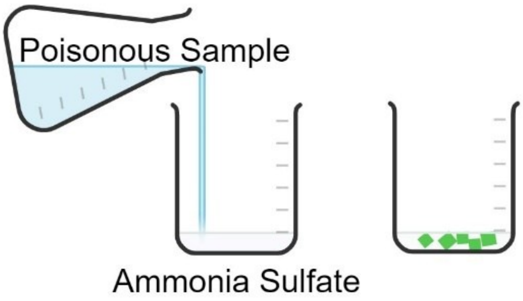

Figure 29: Illustration of Ammonia Sulfate for Detection of Picric Acid Poisoning

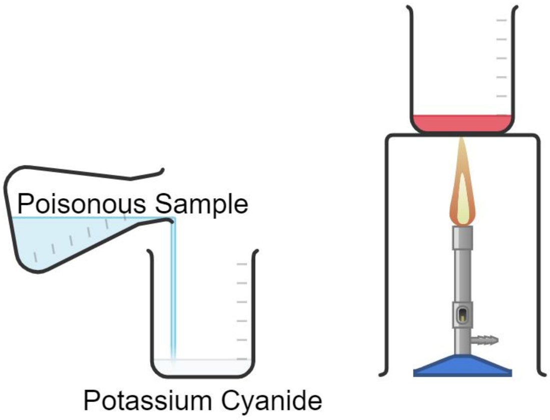

Figure 30: Illustration of Potassium Cyanide for Detection of Picric Acid Poisoning

Potassium Permanganate

Potassium Permanganate ($KMnO_4$) is a corrosive base that is a dark, purplish-red crystalline solid.[29] It is rarely used as a poison but was used in some fatal and suicide events.[30] KMnO4 induces burning pain in the oral cavity, and digestive tract leading to the stomach. A poisoned individual experiences difficulty in breathing, swallowing, and continuous vomiting. The tongue and pharynx become black or dark brown due to corrosion by the base.[35] Paralysis of cardiac tissue leads to death.

Potassium Permanganate poisoning can be detected by the following methods.

A. Add oxalic acid to a solution of a poisonous sample. The solution starts to decolorize if it contains potassium permanganate (Figure 31).
B. Mix the poisonous sample solution with KOH. If it contains potassium permanganate, green precipitates are produced (Figure 32).
C. Add cocaine hydrochlorate to a solution of a poisonous sample. If it contains potassium permanganate, precipitates of distinctive appearance will be formed which can be viewed under a microscope.

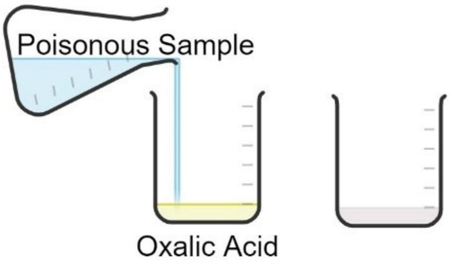

Figure 31: Illustration of Oxalic Acid for Detecting Potassium Permanganate Poisoning

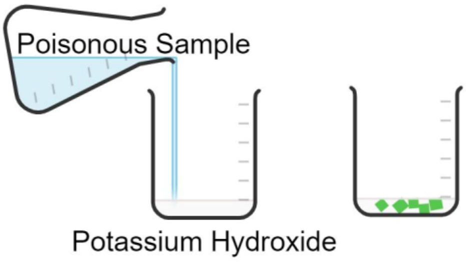

Figure 32: Illustration of Potassium Hydroxide for Detection of Potassium Permanganate Poisoning

Salicylic Acid

Salicylic Acid ($C_7H_6O_3$) is an organic acid formed by the reaction between sodium carbonate and carbon dioxide, but it exists in nature also. It is an odorless crystalline solid in appearance.[31] Unlike other acids it has a sweet taste. Derivatives of salicylic acid like methyl salicylate (oil of wintergreen), sodium salicylate, acetyl salicylic acid (aspirin), etc. are also fatal but in different doses. Salicylic acid poisoning may lead to abortion and death due to cessation of the cardiovascular and respiratory systems.

Salicylic acid poisoning can be detected by the following method.

A. Mix a solution containing a poisonous sample with ferric chloride. If it contains salicylic acid, a violet color appears in the solution (Figure 33), which disappears with the addition of acetic acid, but carbolic acid doesn't affect the violet color.

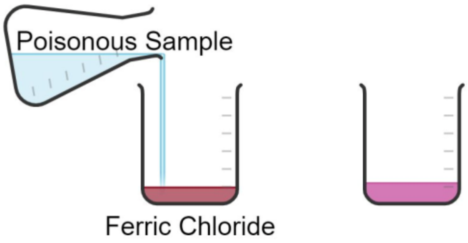

Figure 33: Illustration of Ferric Chloride for Detection of Salicylic Acid Poisoning

Sulfuric Acid

Sulfuric Acid (H_2SO_4) is a commonly known inorganic acid. When it comes in contact with body parts it starts to burn them, and they appear black. Its action is so fast that it starts burning, the upper layers of skin, in 1 second, and within 30 seconds a deep skin burn occurs.[32] Pure H_2SO_4 is colorless while the one commercially available is of dark or brown color. Nordhausen Acid (pyrosulphuric acid $H_2S_2O_7$), a stronger form of H_2SO_4, and brown in color, is used in the production of indigo (1 part of indigo dissolves in 9 parts of sulfuric acid) and has the same symptoms such as swollen tongue, spillage of acid from chin as well as neck, discoloration of teeth etc. except that the vomit, mouth and urine are of blue color.

To detect H_2SO_4 in poisoning samples, these tests can be used:

A. Mix barium nitrate with a poisoning sample. If it is sulfuric acid poisoning, this combination produces a white precipitate (Figure 34). This white precipitate remains insoluble even with the addition of HCl.
B. Heat the poisoning sample with mercury (copper filings or wood chips can also be used). If it is sulfuric acid poisoning, sulfurous acid gas with a sulfur-like odor is released. Test the presence of sulfurous acid by using starch paper dipped in a solution of potassium iodide or iodic acid. The gas turns the starch paper blue and then bleaches it (Figure 35).
C. Bring blotting paper to the poisoning sample and heat them. If it is sulfuric acid poisoning, the paper turns black even when dilute acid is used for poisoning (Figure 36).

D. Heat and evaporate the poisoning sample with a veratrine on a porcelain dish. If it is sulfuric acid poisoning, the formation of a crimson deposit occurs (Figure 37).

E. Bring wood to the poisoning sample. If it is sulfuric acid poisoning, the wood turns black.

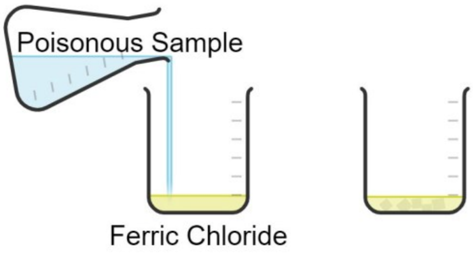

Figure 12: Illustration of Ferric Chloride for Detection of Creosote Poisoning

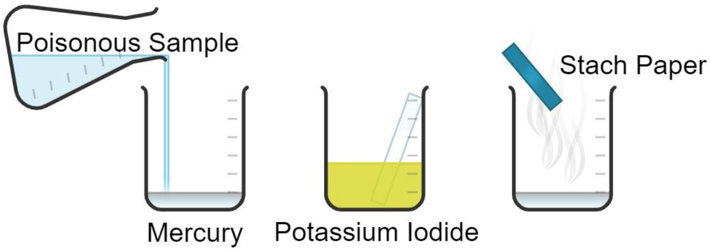

Figure 35: Illustration of Mercury for Detection of Sulfuric Acid Poisoning

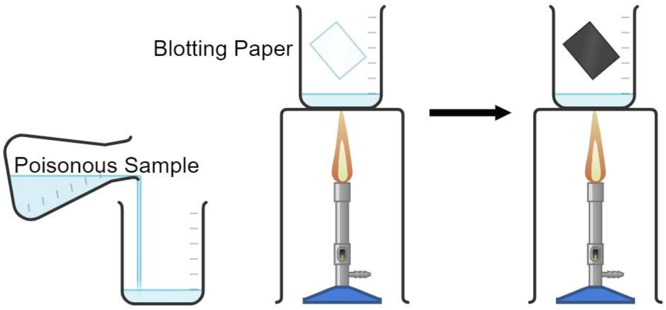

Figure 36: Illustration of The Utilization of Blotting Paper for Detection of Sulfuric Acid Poisoning

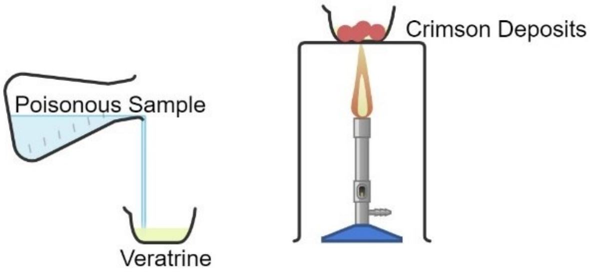

Figure 37: Illustration of Veratrine for Detection of Sulfuric Acid Poisoning

Tartaric Acid

Tartaric Acid ($C_6H_6O_6$) is an odorless organic acid that is a colorless crystalline solid or white powder. It occurs naturally in fruits and can also be synthesized from potassium acid tartrate.[33] It is not commonly a substance that someone used as a poison but there were some incidents where poisoning was caused by tartaric acid. It produces burning sensations in the regions of the throat and stomach, vomiting, and diarrhea.

Tartaric acid poisoning can be detected by the following method:

A. Mix the poisonous solution with water or alcohol. If it contains tartaric acid, transparent crystals (of tartaric acid) are formed (Figure 38).[7]

B. Mix the poisonous solution with calcium chloride. If it contains tartaric acid, white precipitates are formed (Figure 39). To distinguish tartaric acid from oxalic acid, add acetic acid which results in the solubility of white precipitates.

C. Boil the poisonous solution. If it contains tartaric acid, it starts to darken. The addition of potassium permanganate results in decolorization of tartaric acid (Figure 40).

D. Mix the poisonous solution with silver nitrate. If it contains tartaric acid, white precipitates of silver tartrate are formed. Take precipitates in a cleaned (with caustic soda and distilled water) test tube. Add ammonium hydroxide until it starts to dissolve a bit. Put a crystal of silver nitrate at the bottom of a test tube. Take a beaker of water and start boiling it. Put the test tube in a beaker and wait for a few minutes. In case of a positive result, a metallic mirror of silver will form on the sides of the test tubes (Figure 41).

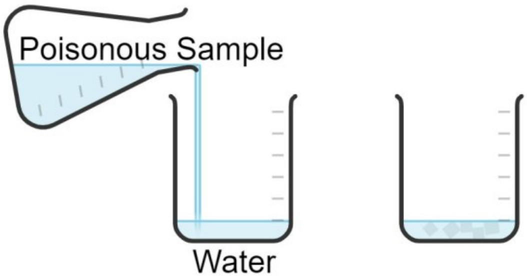

Figure 38: Illustration of Water for Detection of Tartaric Acid Poisoning

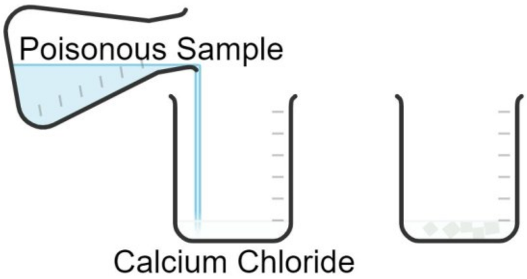

Figure 39: Illustration of Calcium Chloride for Detection of Tartaric Acid Poisoning

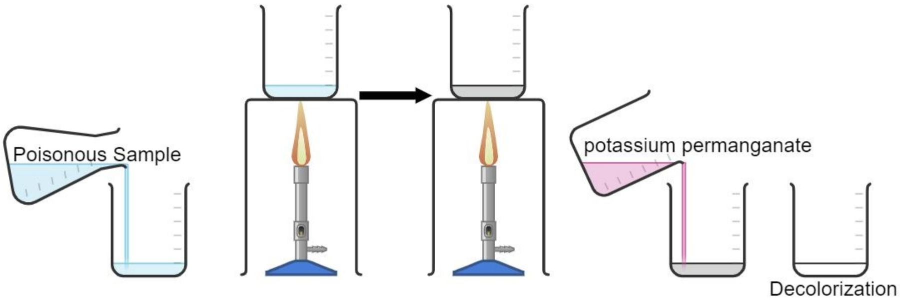

Figure 40: Illustration of Potassium Permanganate for Detection of Tartaric Acid Poisoning

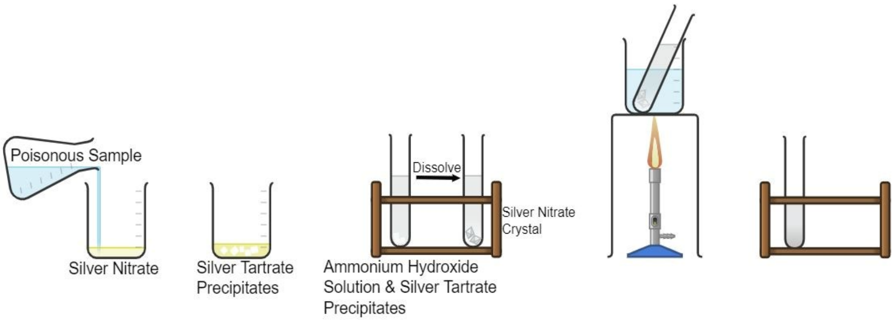

Figure 41: Illustration of Applying Silver Mirror Test for Detection of Tartaric Acid Poisoning

Comparison of Classical Methods and Modern Analytical Technology

Precise and accurate results, provided by instruments like MS and HPLC, are crucial in forensic investigations where even slight inaccuracies can lead to wrongful conclusions. These equipments can detect trace amounts of toxins. Their sensitivity is essential for identifying substances in complex mixtures such as biological samples. Modern equipments are capable of analyzing multiple compounds simultaneously. They also include automation functions, which reduce the demand for manual handling and the possibility of human error.

Modern equipments are expensive to purchase and maintain, making it beyond budget for forensic facilities with limited budgets. Advanced instruments require specialized training to work efficiently. Maintaining and calibrating sophisticated devices is both time-demanding and expensive. Operational disruptions can result from technological failures or downtime caused by maintenance or repairs.

Classical procedures mostly involve use of simple reagents and equipment making them inexpensive. These technologies often require less specialized training, allowing forensic scientists to readily adapt and utilize them in work processes. These procedures have been used for decades to provide uniformity and reliability in forensic toxicology analyses. These are more durable and less prone to slight fluctuations in sample preparation, making them appropriate for routine analysis.

Classical approaches may lack sensitivity for detecting and quantifying trace amounts of toxin, limiting their application when minute quantities are critical. These approaches may not be capable of analyzing and identifying many chemicals at once. Some of these methods rely on subjective interpretation of results, which can introduce bias and inconsistencies in forensic analysis. These involve manual handling of samples and reagents, increasing the risk of human error and variability between analyses.

Table 1: Postmortem Symptoms, Fatal Dose, Fatal Period, and Detection Tests of Corrosive Poisons

Corrosive Poison	Postmortem Symptoms	Fatal Dose	Fatal Period	Detection Test	Positive Result	References
Acetic Acid	Ecchymosed patches appear on the mucous membranes of the stomach, intestine, esophagus, and oral cavity due to corrosion.	Conc. Acetic Acid: 3.55 ml	2 – 36 hours or more	Reaction with alcohol and sulfuric acid Reaction with ammonia and ferric chloride	Ethyl acetate forms with its aromatic smell Dark red Color appears	Gardet *et al.*[7]
Ammonia	Signs of corrosion. Inflammation (with brown/black patches), softening, and exfoliation of the mucous membranes	5 – 10 ml 30g	24 hours or more	React with caustic potash React with Nessler reagent	Ammonia gas produces Brown precipitates	Gardet *et al.*[7], Ramadan,[10] Abou and Khaled[11]

	of the mouth, throat, gullet, stomach, and duodenum. Stomach contents are turbid, blood-stained, or coffee colored. Stomach perforation is possible to some extent. Stomach contents smell of ammonia with a soapy feel. Deeper tissues exhibit inflammation and congestion. Viscera is highly congested.					
Carbolic Acid	Brain is congested. Blood appears black and partially coagulated, or semifluid. Lungs are congested and edematous. Hemorrhagic nephritis (in case of delayed death). Brown and leathery with hemorrhagic spots, soft and greyish white in color. Stomach content emits an odor of carbolic acid and has reddish fluid mixed with mucus and epithelial shreds. The esophagus' mucous membrane is corrugated, stiff, white or grey, and arranged in longitudinal folds.	10 – 15 g	3 – 4 hours or more	React with mercury and nitric acid React with bromine water React with dilute ferric chloride Heat with ammonia and calcium hypochlorite React with conc. Sulphuric acid and sodium nitrite	Red Color appears Whitish-yellow precipitates formed Violet-blue or purple color appears Blue color appears A deep blue or green color appears	Gardet *et al.*[7], Ramadan[10], Abou and Khaled[11], Shamsi *et al.*[35]

	The mucous membranes of the lips, mouth, and throat are partially detached, corrugated, white, or ash-grey, and characterized by a large number of tiny submucous hemorrhages. Discolorations on the chin and around the mouth's angles that are white or brownish.					
Citric Acid	The mucosal surface of the esophagus shows black discoloration. Degeneration of the epithelium. Neutrophilic infiltration in the muscle layer of the esophagus. Degeneration of esophageal mucosal and submucosal Presence of vessel thrombosis in the esophagus and cellular infiltration in the esophageal tissues.	25 g	No specific time was recorded	Boil with calcium chloride React with potassium permanganate	White precipitates formed Green color appears	Gardet *et al.*[7]
Creosote	The mucous membranes of the mouth, esophagus, stomach, tongue, and lips are eroded and swollen in areas, and they have a grey or red tint. The brain and lungs are congested. Kidneys are usually congested and inflamed. Red and injected stomach.	Depends upon the age group. Infant: 1g Adult: 7 ml	Infant: 16 – 60 hours or more Adult: 36 hours – 5 days	React with ferric chloride	The green or brown color appears	Gardet *et al.*[7]

	Presence of creosote odor in both brain and stomach contents.					
Hydrochloric Acid	Mucous membranes are ash-grey or black and interspersed with erosions. The stomach wall is red (acute gastritis). The stomach contains brownish fluid. The whole stomach mucosa folds are brownish. Respiratory passages and lung tissue show acute inflammation and edema.	15 – 20 ml	18 – 36 hours or more	React with silver nitrate Heat with manganese oxide React with ammonia gas	White precipitates forms Chlorine gas forms White fumes form	Gardet *et al.*[7], Ramadan[10], Abou and Khaled[11], Shamsi *et al.*[35]
Nitric Acid	Mucous membranes and skin appear yellow. If bile is present, the mucous membrane of the stomach may have a greenish color. The stomach wall is soft, friable, and ulcerated. Congestion in the larynx, trachea, and bronchial tubes. Lungs may be edematous or show blood effusion. Inflammatory changes in the lining membrane of the right auricle of the heart. Lips and fingers stained yellow and corroded. Alimentary canal from lips to	10 – 15 ml	12 – 24 hours or more	React with ferrous sulfate and sulfuric acid React with brucine solution Heat with sulfuric acid and copper React with morphine solution	Brown ring forms between two solutions Blood-red color appears Brown fumes are formed Orange color appears	Gardet *et al.*[7], Ramadan[10], Abou and Khaled[11], Shamsi *et al.*[35]

	duodenum stained yellow. Necrosis of the stomach coats with perforation (rare).					
Oxalic Acid	Mucous membranes are white and shriveled, easily detached from underlying tissues, and sometimes black due to altered blood. The esophagus' inner surface is corrugated with longitudinal erosions. Blood vessels appear as dark brown or black streaks on the stomach's internal surface. The stomach contains dark-brown, grumous (thick and clot-like) liquid with an acidic reaction. Stomach may appear pale and uncorroded if death is immediate after ingestion. Perforation of the stomach is rare, but walls are often softened and easily torn. Kidneys are congested and loaded with oxalates.	15 – 20 g	1 – 2 hours or more	React with barium nitrate React with silver nitrate React with calcium chloride React with calcium sulfate React with lead acetate	White precipitates are formed White precipitates are formed White precipitates are formed White precipitates are formed White precipitates are formed	Gardet *et al.*[7], Ramadan[10], Abou and Khaled[11], Shamsi *et al.*[35]
Picric Acid	All internal organs (viscera) exhibit yellow staining.	Uncertain dose	No specific time was recorded	React with ammonia sulfate Heat with potassium cyanide	Green precipitates are formed Blood-red color appears	Gardet *et al.*[7],
Potassium	Lips, mouth, and throat exhibit corrosion. Tissues of the esophagus and stomach show	KOH: 5g K_2CO_3: 15 – 30g	24 hours or more	React with tartaric acid and alcohol React with platinic chloride Flame test	White precipitates are formed Yellow precipitates are formed Violet color appears	Gardet *et al.*[7], Ramadan[10], Abou and Khaled[11]

	corrosion and sliminess. Mucosa may turn brownish due to the formation of alkali hematin. Esophageal stricture					
Potassium Permanganate	Blackening, congestion, edema, and inflammation of mouth, pharynx, trachea, and esophagus.	10 – 42.5g	5 hours – 4 days	React with oxalic acid Mix with potassium hydroxide React with cocaine hydrochlorate	Decolorization of solution Green precipitates forms Distinctively appearing precipitates forms	Gardet *et al.*[7],
Salicylic Acid	Signs of inflammation in the stomach (gastritis), small intestine (enteritis), and kidneys (nephritis) may be present. Organs are typically found hyperaemic (indicating increased blood flow). Congestion of internal organs.	Salicylic acid: 70 – 80 gm Sodium salicylate: 15 – 20 gm Acetyl salicylic acid: 15 – 20 gm Methyl salicylate: 10 – 20 ml	Salicylic acid: 4 – 7 days or more Sodium salicylate: 1 – 3 days or more Methyl salicylate: 12 – 24 hours or more	React with ferric chloride	Violet color appears	Gardet *et al.*[7], Shamsi *et al.*[35]
Sodium	Lips, mouth, and throat exhibit corrosion. Tissues of the esophagus and stomach show corrosion and sliminess. Mucosa may turn brownish due to the formation of alkali hematin. Esophageal stricture	NaOH: 5g Na_2CO_3: 15 – 30g	24 hours or more	Flame test	Yellow color appears	Gardet *et al.*[7], Ramadan[10], Abou and Khaled[11]
Sulfuric Acid	Brown or brownish-black corroded spots on the mouth, and lips region. Dark-brown or black mucous membrane.	5 – 10 ml	12 – 24 hours or more	React with barium nitrate Heat with mercury/copper filings/wood chips	White precipitates are formed Sulfurous acid gas forms	Gardet *et al.*[7], Ramadan[10], Abou and Khaled[11], Shamsi *et al.*[35]

	Perforation of the stomach. In non-perforation cases, the stomach is collapsed and contracted. Stomach contents are a dark-brown and grumous liquid, mainly mucus and altered blood. The mucous membrane may be dark-brown or black, often corrugated and detached in shreds or patches. Folds are large and deep from swelling, sometimes softened to tear easily. Removal of the mucous membrane reveals red and intensely inflamed underlying coats of the stomach. The duodenum shows corrosion and inflammation if death occurs after eighteen or twenty hours of ingestion. Coagulated blood in the blood vessels.			Heat with blotting paper Heat with veratrine React with wood	Paper turns black Crimson deposits are formed Wood turns black	
Tartaric Acid	Erosions of the mucous membrane of the esophagus. The alimentary canal shows inflammation. Blood remains persistently fluid, red currant juice color.	9 – 184g	12 hours - 7 days	React with water/alcohol React with calcium chloride Boiling React with silver nitrate	Transparent crystals are formed White precipitates are formed Solution turns dark Metallic mirror of silver forms	Gardet *et al.*[7]

CONCLUSION

The development and use of cost-effective methods for detecting corrosive poisons in post-mortem forensic samples is important as these techniques provide reliable and accurate results while also reducing costs and complex procedures. They enable resource-limited laboratories to contribute effectively to criminal investigations and judicial processes. The adoption of these approaches not only enhances the efficiency and speed of toxicological analyses but also cements the integrity of forensic evidence, eventually supporting the pursuit of justice. Further research and development in cost-effective detection methods will be essential to meet the increasing demands and challenges of forensic toxicology.

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