



A Case Report

Fatal Carbon Disulfide Exposure

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Abstract :

Carbon disulfide (CS₂) is a solvent most commonly used in manufacturing of rayon. In industrial setting, CS₂ is rarely released with isolation; it is almost always accompanied by hydrogen sulfide (H₂S). This may complicate the clinical and diagnostic picture. Consequently, while chronic toxicity is well documented, acute fatal poisoning from isolated or mixed with H₂S inhalation is seldom reported in recent literature. This is a rare case report which describes the rapid clinical deterioration and detailed gross autopsy findings following occupational CS₂ exposure. Autopsy revealed severe cerebral edema and visceral congestion, demonstrating the intense neurotoxic and hypoxic effect of the gas on the tissues.

Keywords : Carbon disulfide; hydrogen sulfide; occupational exposure; autopsy findings; neurotoxic substance; acute fatal poisoning

Introduction:

Carbon disulfide with chemical formula CS₂ is a colorless, highly flammable, extremely volatile with faint, sweet smelling odor when pure. CS₂ is widely used as solvent in manufacture of rayon, cellophane, dyes, and rubber.[1,2,3] Due to its rapid absorption through inhalation, even at lower concentrations may cause mucosal irritation, while higher concentrations can lead to respiratory distress, central nervous system (CNS) depression and coma. The clinical spectrum of acute exposures ranges from mild, self-limiting symptoms to severe toxicity requiring intensive critical care management. Although safety regulations have reduced its incidence in developed settings, accidental and occupational exposures have been reported.[4,5] Chronic exposure to CS₂ has been documented in the literature over the past six decades. In contrast, reports of fatal acute CS₂ poisoning are lacking, and overall documentation of CS₂ toxicity in recent literature remains limited. This is a rare case report of a factory worker's rapid clinical deterioration following alleged occupational exposure of CS₂, along with autopsy findings.

Case Details:

Brief history: A 47-year-old man working in Rayon industry was allegedly exposed to Carbon disulphide (CS₂) gas while performing maintenance work on a gas pipeline of the chemical production division. He developed sudden onset of discomfort followed by loss of consciousness shortly after the exposure. He was initially managed at a local hospital and was referred to a tertiary healthcare centre on the

second day following the incident. Despite appropriate medical management, he expired on the third day. A medico-legal autopsy was subsequently conducted.

Autopsy findings:

On external examination, post-mortem lividity was pinkish in colour, well-fixed, and distributed over the back, sparing the pressure areas, cyanosis was observed over the nail beds of both the fingers and toes. No other significant external findings. On opening of skull cavity, pungent (foul, unpleasant and strong) smell was appreciated. Brain weighed 1250 grams was found to be edematous and congested. Bilateral pleural effusion of approximately 500 ml of fluid each. Lungs appeared edematous and congested on cut section. Approximately 80 mL of fluid was present within the pericardial sac. The stomach contained approximately 100 grams of brownish, thick semi solid material with pungent smell and gastric mucosa appeared haemorrhagic.

Discussion:

CS₂ is most commonly used in the production of Viscose Rayon fibers. As environment consciousness arises among consumers regarding synthetic fibers, demand for sustainable materials such as rayon has surged. China followed by India are the largest market for rayon fibers in Asia-Pacific region.[6,7] For every kilogram of viscose used, about 20-30g of CS₂ and 4-6g of H₂S is emitted. Approximately 0.6-1.0kg of viscose is used per hour in textile yarn production. However, carbon disulfide exposure is considerably higher during staple fiber and cellophane manufacturing, where viscose consumption reaches about 70-100 kg/hour and 1800-2000 kg/hour, respectively.[1] It is primarily an occupational industrial toxin, with significant harmful exposure mainly occurring in the rayon industry, while the impact on nearby populations remains inadequately assessed due to limited data.

Acute exposure to CS₂ often occurs concomitantly with H₂S, making it challenging to delineate the independent toxic effects of CS₂. [1] In 1994, fatalities were reported in India following the accidental release of large quantities of CS₂ along with H₂S and sulphuric

acid from a viscose rayon plant.[9] Chronic exposure to carbon disulfide is far more commonly reported than acute exposure. A study conducted in Korea during 1980s-1990s investigated workers exposed to CS₂ in the viscose rayon industry and demonstrated long-term neurotoxicity, including peripheral neuropathy and neurobehavioral impairment.[5] Numerous other studies have documented CNS toxicity with CS₂ exposure.[10,11,12,13] In addition to its neurotoxic effects, CS₂ has also been linked to an increased risk of cardiovascular disorders.[5,10,14,15] In contrast to the predominantly reported chronic toxicity, the present case describes a fatal episode of acute occupational carbon disulfide inhalational exposure with detailed post-mortem findings, highlighting the severe consequences of high-level exposure in an industrial setting.

Carbon disulfide exposure occurs predominantly via inhalation due to its high volatility and rapid pulmonary absorption. At low concentrations, it may produce irritative symptoms, whereas high concentrations can result in serious manifestations such as respiratory compromise, central nervous system depression and potentially coma. The severity of toxic effects is influenced by both the intensity and duration of exposure.[8] Occupational exposure occurs mainly through inhalation and skin contact, particularly in industries using carbon disulfide, with high concentrations found in the viscose rayon industry. It is rapidly absorbed and distributed in the body, primarily affecting the brain and liver, where it is metabolized into thiocarbamates, contributing to its neurotoxic effects. Excretion occurs mainly through the kidneys, with some unmetabolized carbon disulfide expelled via breath.[16]

After absorption, CS₂ is metabolized into reactive dithiocarbamate compounds, which are implicated in several toxic pathways, including inhibition of dopamine beta hydroxylase, thereby disrupting catecholamine synthesis and contributing to neurological manifestations. [17,18] Exposure has also been associated with pyridoxine (vitamin B6) deficiency and impaired GABA synthesis, potentially leading to seizures that may be resistant to benzodiazepines. Additional mechanisms include oxidative stress

and inflammatory injury. Hepatic effects include altered lipid metabolism with resultant hypercholesterolemia and accelerated atherosclerosis in chronic exposure. Inhibition of aldehyde dehydrogenase may predispose to disulfiram-like reactions.[19,20,21] Endocrine and reproductive dysfunction has also been described, possibly due to reduced nitric oxide synthesis and hormonal imbalance, leading to impaired spermatogenesis and infertility. [22,23,24] CS₂ is a highly volatile, lipophilic compound that is readily absorbed via inhalation, dermal contact, and, less commonly, ingestion, with inhalation accounting for the majority of systemic uptake (approximately 80–90%).[1] Once absorbed, it distributes widely due to its high lipid solubility. It undergoes hepatic metabolism primarily through cytochrome P450 enzymes, producing metabolites such as thiourea, thiocarbamates, carbonyl sulfide, and 2-thiothiazolidine-4-carboxylic acid (TTCA). [19,24] TTCA serves as a useful urinary biomarker of exposure and has been associated with impaired pulmonary function, including reduced FEV₁/FVC ratios at higher exposure levels.[25] Elimination occurs mainly via hepatic metabolism and renal excretion, with a half-life of approximately 1-4 hours and a volume of distribution of 0.7-1.0 L/kg.

Detailed autopsy descriptions in fatal carbon disulfide poisoning are scarce in the available literature. A fatal oral ingestion of approximately 15 ml of CS₂ was documented that lead to rapid clinical deterioration characterized by convulsions, dyspnea, cyanosis and coma, culminating in death within few hours; however, specific post-mortem finding was not elaborately described in that report. [8] In the present case, the autopsy findings were largely consistent with acute toxic inhalational injury and systemic hypoxic effects following CS₂ exposure, as evidenced by cyanosis over the nail beds of both the fingers and toes. The presence of pinkish, well-fixed post-mortem lividity may reflect impaired tissue oxygen utilization and circulatory compromise secondary to toxic exposure; however, this finding may also be attributable to concomitant hydrogen sulfide (H₂S) exposure, which is known to interfere with cellular respiration and can produce similar discoloration

due to altered oxygen dynamics at the tissue level. Marked cerebral edema and congestion are consistent with the well-recognized neurotoxic effects, reflecting rapid absorption via inhalation and preferential distribution to brain due to its high lipophilic nature, additionally brain is a highly perfused organ. Acute CNS depression, hypoxia and vascular endothelial injury may explain the observed brain swelling. CS₂'s high volatility facilitates rapid respiratory absorption, and acute exposure to high concentrations can result in capillary leakage, pulmonary edema, and respiratory compromise. The presence of pericardial effusion further suggests systemic vascular permeability and circulatory disturbance. The pungent odor appreciated on opening the cranial cavity and stomach may correlate with the volatile nature of un-metabolized CS₂, a fraction of which is excreted unchanged via the lungs, while the remainder undergoes hepatic metabolism through cytochrome P450 pathways to form reactive metabolites such as dithiocarbamates. As CS₂ is primarily metabolized in the liver and its metabolites are excreted via the kidneys with minor pulmonary elimination of unchanged compound the generalized visceral congestion and serous effusions observed at autopsy can be interpreted as secondary to widespread toxic endothelial injury, hypoxia, and oxidative stress. Overall, the constellation of findings correlates with acute high-level inhalational exposure, potentially compounded by concomitant H₂S toxicity.

CS₂ is highly lipophilic, initial pulmonary absorption is high (upto 80% in the first 15 minutes of high exposure), but falls significantly over time. Notably, red blood cells absorb approximately twice as much CS₂ as plasma. While the liver initially shows highest concentrations, CS₂ is absorbed slowly absorbed by the brain and retained for extended duration, which explains the persistent neurotoxic effects in chronic cases. [26] Iodine-azide reaction has been utilized as a qualitative, rapid, non-specific screening tool. This test detects sulfur-containing metabolites in the urine. However, its diagnostic value is limited in modern forensic practice due to lack of sensitivity at low exposure levels and poor specificity compared to chromatographic

methods. In the human body, CS₂ is metabolized & excreted primarily as TTCA (2 thiothiazolidine -4-carboxylic acid) in urine. Automated HPLC is preferred analytical technique due to its high sensitivity and reproducibility.[1,27] The coupling of Gas Chromatography (GC) with Mass Spectrometry (MS) allows for the selective identification of CS₂ even at low atmospheric levels. This method utilizes both retention times and mass spectra to reduce false positives, making it a "gold standard" for tracking volatile sulfur compounds.[28]. Advanced sensors, such as those based on Quartz-Enhanced Photoacoustic Spectroscopy (QEPAS) using quantum cascade lasers, have been developed for high-precision detection of CS₂ concentrations in industrial environments.

Conclusion:

Acute CS₂ inhalation remains a rare yet potentially fatal industrial hazard. This case report describes the multisystem manifestation of CS₂ inhalation and highlights gross autopsy findings. Recognition of the findings as well clinical manifestation while considering the concurrent H₂S exposure is important for accurate diagnosis and management. Industrial safety practices must be strengthened, appropriate use of protective gears and application of regular investigations for biomarker analysis such as urinary TTCA for exposure assessment and to prevent occupational exposure deaths whether it is chronic or acute.

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