

Awareness, among primary health care physicians, of acute cyanide poisoning being a possible consequence of fire accidents

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Abstract

Objectives: To discern the degree of awareness amongst the primary health care physicians about the fact that cyanide poisoning is a possible consequence of fire accidents.

Methods: Using an anonymous questionnaire, thirty allopath primary health care physicians were tested for awareness of acute cyanide poisoning being a possible consequence of fire accidents.

Results: Only one (3%) of the physicians was aware of the possibility of acute cyanide poisoning due to fire accidents.

Conclusions: We report dismal awareness among the primary health care physicians. Medical education curriculum must cater for emerging healthcare issues.

Keywords: Acute cyanide poisoning, primary health care physician, fire accident, awareness

1. Introduction

During a disaster preparedness workshop for medical doctors, it was noticed that some of the primary health care physicians (physicians) seemed to lack awareness of acute cyanide poisoning being a possible consequence of residential or industrial fire accidents. Fire smoke inhalation is associated with acute cyanide poisoning. There is no test available for rapid confirmation of acute cyanide poisoning and treatment is based on a presumptive diagnosis. Quick recognition & immediate administration of antidote can prevent an otherwise devastating outcome. The antidote is to be administered at the scene immediately after removal of the patient from the source of cyanide exposure [1]. It cannot be, thus, overemphasized that successful outcome necessitates awareness and ability to recognize early signs of cyanide poisoning on part of the physicians. This study was designed to test the awareness of acute cyanide poisoning being a possible consequence of fire accidents.

2. Material & Methods

Thirty allopath primary health care physicians (physicians) from urban region of Western Maharashtra volunteered for the study. Informed consent was obtained. Participants were tested for awareness of acute cyanide

poisoning being a possible consequence of fire accidents. A questionnaire with a single question asking 'which all among those listed, you believe to be the possible health outcomes of a fire accident?' was administered. Response 'Acute cyanide poisoning' was tucked among others, namely, burns, injuries, asphyxia, carbon monoxide poisoning & post-traumatic stress disorder. The participants were asked to mark their responses by checking in the list supplied. Any participant checking the response 'Acute cyanide poisoning' was interviewed to check whether the response was based on content knowledge or guesswork. The questionnaire responses were kept anonymous.

2.1 Statistical Methods

Maintaining anonymity, the responses were allotted serial numbers & were tabulated in an excel sheet. The frequency of checking the response 'Acute cyanide poisoning' was derived and calculated as a percentage of all the responses.

3. Results

Only one (~ 3%) participant checked the response 'Acute cyanide poisoning'. However, detailed interview of the physician who did check the response, revealed sound

knowledge of the cause of association of acute cyanide poisoning with inhalation of fire smoke. He happened to be preparing for higher studies in medicine.

The study shows that awareness, among primary health care physicians, of acute cyanide poisoning being a possible consequence of fire accidents is dismal. Those preparing for higher studies can be expected to be better informed.

Table 1: Knowledge of the participants with respect to the question

Category of participants	No. (%)
Checked the response 'Acute cyanide poisoning'	01 (03)
Did not check the response 'Acute cyanide poisoning'	29 (97)
Total	30 (100)

4. Discussion

Certain cyanogenic plants' yields contain cyanide precursors. These include apricot seeds [2-10], cassava - the tapioca root consumed commonly as sago [11], peaches [10,12], flax seeds [13], bitter almonds [14] apple seeds [10], cherry [15, 16], papayas, lima beans, clover and sorghum [17]. Human body has built in metabolic mechanisms capable of detoxifying small quantities of cyanide ingested. Endogenous enzymes, rhodanese & 3-MPST normally metabolize small quantities of cyanide present in the dietary sources to relatively harmless thiocyanate [18]. However prolonged and severe exposure can overwhelm these mechanisms, thereby resulting in poisoning.

The most frequent sources of cyanide poisoning include smoke inhalation, suicidal ingestion and industrial exposure. Prolonged use of certain cyanide-containing compounds (cyanogens) like vasodialator sodium nitroprusside that are metabolized to cyanide, leads to iatrogenic exposure. Cyanide has also been used as a chemical warfare agent [19].

Otherwise rare, cyanide poisoning is more common in fire accidents. Although morbidity data for India is not available, it is estimated that cyanide may be a major contributor to approximately 5000-10,000 deaths from smoke inhalation occurring each year in the United States [17].

Acrylonitrile, a derivative of hydrogen cyanide is used as a solvent in the production of acrylic fibers (pillows, quilts), synthetic plastic (melamine in dishware, acrylonitrile in plastic cups), rubber & polyurethane foam (furniture cushions) [20]. Burning of these materials during fire accidents leads to discharge of hydrogen cyanide in the smoke. Many natural products, like wool & silk, also produce hydrogen cyanide as a combustion product. Cyanide laden smoke inhalation results in acute cyanide poisoning [21]. Cyanide (CN⁻) is a cellular poison that inhibits mitochondrial cytochrome oxidase a3 by reversibly binding to the iron within. This leads to disruption of the electron transport chain, decreased tissue utilization of oxygen and ultimately halting

of aerobic production of ATP [22]. CNS and heart are particularly affected due to heavy dependence on aerobic respiration.

Individuals with smoke inhalation from enclosed space fires who have soot in the mouth or nose, altered mental status or hypotension may have significant cyanide poisoning [17].

Early symptoms attributable to involvement of the CNS include anxiety, headache, giddiness, inability to focus the eyes and mydriasis. Early respiratory signs include increased rate & depth of breathing & normal arterial oxygen saturation. Progressively lower levels of consciousness, seizures and coma follow. CVS involvement is manifested as hemodynamic instability [23]. Early administration of one of the antidotes holds the key to treatment [24].

Antidotes include Hydroxocobalamin, Nitrites, Thiosulfate & Sulfanegentriethanolamine among others. Hydroxocobalamin acts by binding with cyanide to form non-toxic cyanocobalamin that is excreted by kidneys [23, 25, 26]. Nitrites oxidize iron present in hemoglobin to the ferric state, leading to formation of methemoglobin which has a higher affinity for cyanide. This releases cyanide from cytochrome oxidase a3 which binds to & converts methemoglobin to cyanmethemoglobin. Thiosulfate acts as a sulfur donor for the endogenous cyanide metabolizing enzyme, rhodanese. Sulfanegentriethanolamine too acts by improving the efficiency of endogenous cyanide detoxifying enzyme pathways [27]. 3-Mercaptopyruvate acts as a substrate for the enzyme endogenous cyanide detoxifying enzyme 3-MPST [28].

Industrialization brings in its own hazards along with the economic benefits. Excessive dependence on acrylonitrile, a cyanide derivative, for production of household items of routine use like pillows, quilts, plastic, rubber & furniture cushions, has increased the likelihood of cyanide poisoning in fire accidents. Urbanization has as such increased the incidence of fire accidents. It cannot be overemphasized that our medical education curriculum must cater for emergence of such newer health challenges.

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