MANAGEMENT OF EMERGENCY CASES IN CLINICAL TOXICOLOGY

THESIS

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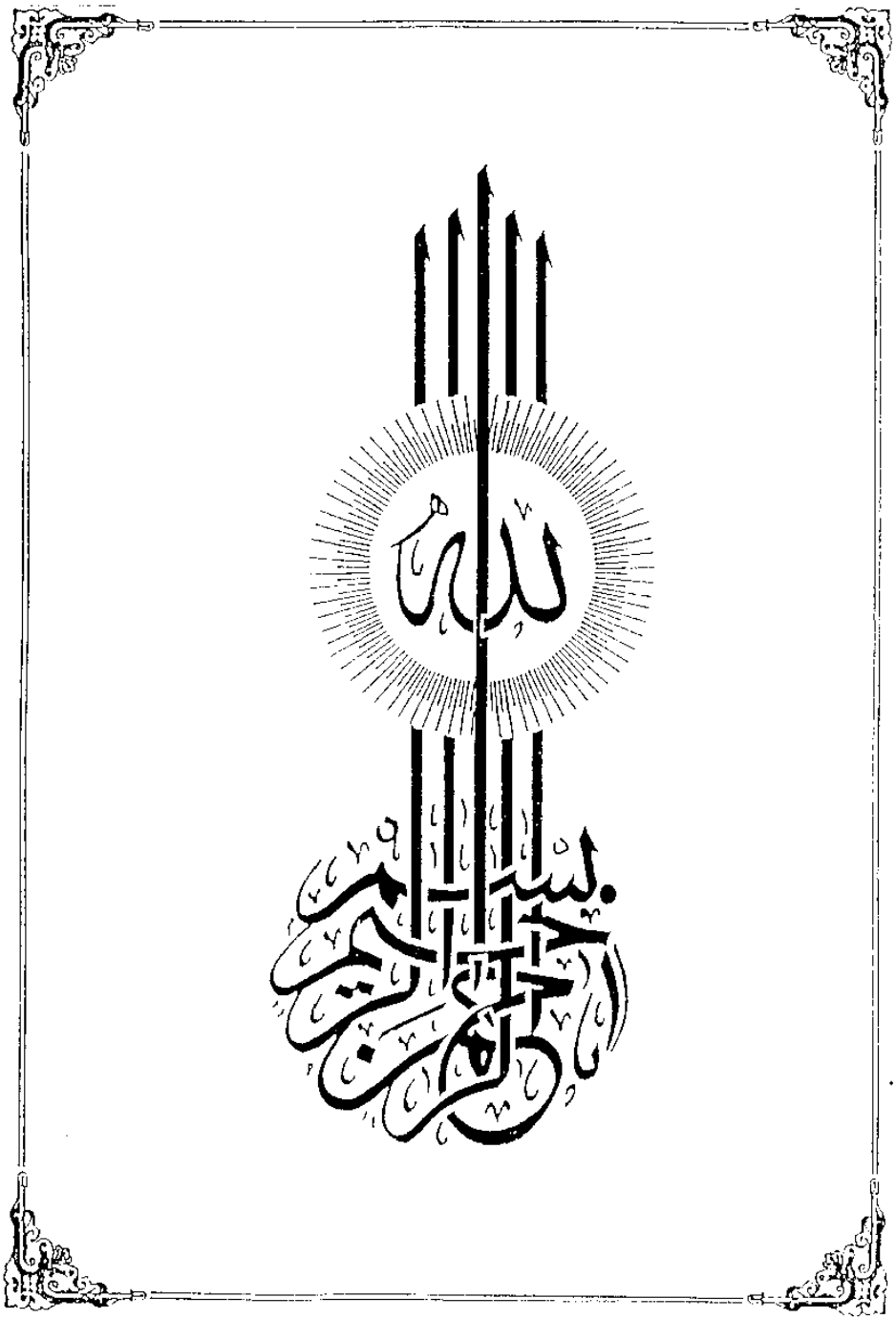

C O N T E N T S

ABREVIATIONS

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- Poison Control Center: P.C.C.
- Intensive Care Unit : I.C.U.
- Central Nervous System: C.N.S.
- Intravenous : I.V.
- Intramuscular : I.M.
- Body weight : b.wt.

INTRODUCTION

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Poisoning may occur following either accidental or intentional exposures, including both suicidal attempts and drug misuse or abuse. Accidental poisoning comprises the vast majority of poisoning expoures reported to major poison control centers (Temple and Mancini, 1980). Intentional poisonings occur almost exclusively in the adolescent and adult age group, and are the most serious poisonings. Among children below 5 years, essentially all poisonings are accidental and are not generally serious (Hanson, 1984).

Acute poisoning is likely to remain one of the most common clinical emergencies which represent a critical problem. Management of such cases should, therefore, receive effective symptomatic and specific treatment as well as laboratory support in suspected cases of poisoning. The present study is aimed mainly towards the establishment of the pattern of acute poisoning in the Poison Control Center (P.C.C.) Ain-Shams University during the year 1986. Cases intoxicated by the most common poisons are to be statistically analysed. This statistical analysis comprises the number of the received cases, representation of the age,

sex, social class, mode of intoxication, grading of severity of cases, time delay between intoxication and consultation of P.C.C., clinical picture, laboratory investigations, management, indication of admission to the Intensive Care Unit (I.C.U.) and prognosis of patients in correlation with the type of the poison.

REVIEW OF LITERATURE

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KEROSENE

Kerosene is a mixture of hydrocarbons distilled from petroleum. It is a colourless or pale yellow oily liquid with a characteristic odour. It is used as a degreaser and cleaner and as illuminating and fuel oil in kerosene lamps and stoves. It is also used as a vehicle for various insecticides, fungicides, cleaning agents, furniture polishes and paint thinner (Blacow and Wade, 1973, Swinyard, 1975).

Kerosene poisoning usually results either from inhalation of the vapours or from ingestion of the liquid. Ingestion of kerosene is more hazardous than inhalation since vomiting and eructation, as well as the low surface tension of kerosene, favour its aspiration into the respiratory tract in toxic amounts (Swinyard, 1975).

Clinical picture:

Ingestion of kerosene leads to a characteristic smell, burning sensation in mouth, oesophagus and stomach, vomiting, eructation and diarrhea with blood tinged faeces (Gosselin et al., 1984). Ingestion of

kerosene also leads to central nervous system manifestations characterized by weakness, dizziness, coma and may be convulsions (Blacow and Wade, 1973). The same previous manifestations with addition of a transient euphoria, headache, tinnitus, muscular incoordination might occur with kerosene inhalation (Swinyard, 1973).

The prognosis in most cases of kerosene ingestion depends on whether or not aspiration occurred when the material was ingested or later during eructation or vomiting (Olstad and Lord, 1952, Foley et al., 1954, McNally, 1956, Baldachin and Melmed, 1964).

Early and widespread pulmonary changes may be found even in absence of clinical signs (Brunner et al., 1964) and conversely pulmonary symptoms may precede lung radiological changes (Vaziri et al., 1980). Respiratory manifestations include cough, dyspnoea, tachypnea, acute bronchospasm, pneumonitis, pulmonary oedema, pleural effusion and pneumothorax (Hanson, 1984). Acute fulminating haemorrhagic and often fatal bronchopneumonia, may appear within minutes or be delayed for several hours (Soule and Foley, 1957). Children who survive the pneumonitis usually recover clinically within 3-5 days but in adults, chronic respiratory diseases as bronchiectasis may result

(Gosselin et al., 1984). Occasionally pseudocysts or pneumatoceles develop and resolve spontaneously over several weeks or months (Bagdassarian and Weiner, 1965, Dragsted and Rodbro, 1965, Campbell, 1970).

Sensitization of the myocardium to epinephrine, resulting in cardiac dysrhythmias, has been reported with volatile hydrocarbons. In addition the myocardium is sensitized to their hypoxic effects (Goldfrank et al., 1982).

CORROSIVES

The strong corrosive alkalies include caustic soda, caustic potash, sodium and potassium hydroxides and carbonates. They are commonly used as cleaning agents and are present in many washing powders. Most of the reported cases of poisoning have resulted from the careless practice of leaving the solutions in beverage containers within the reach of a child (Gosselin et al., 1984).

Corrosive mineral acids include sulfuric, nitric, hydrochloric and phosphoric acids. Automobile battery acid is usually about 28% sulfuric acid. Corrosive organic acids include acetic, trichloroacetic acids, carbolic acid and oxalic acid (Gosselin et al., 1984).

Clinical picture:

Ingestion of corrosives causes severe burning pain in the mouth, pharynx and abdomen followed by vomiting and collapse. The vomitus may contain dark precipitated blood (Dreisbach, 1983).

Inhalation of the fumes causes cough, asphyxia from glottic oedema (Gosselin et al., 1984), pulmonary oedema with tightness of the chest, frothy sputum and cyanosis (Dreisbach, 1983).

Oesophageal perforation leads to mediastinitis, pericarditis, pleuritis, tracheobronchoesophageal fistulae and oesophagoaortic perforation which is a grave complication. Gastric perforation leads to peritonitis. Pyloric obstruction is a late complication (Goldfrank et al., 1982).

Skin contact causes severe pain with burn which may penetrate the full thickness of the skin and heal with scar formation. Eye contact causes conjunctival oedema and corneal destruction (Dreisbach, 1983).

Oxalic acid causes gastrointestinal manifestations, followed by convulsions, weak pulse, collapse and acute renal failure because of calcium oxalate blocking the renal tubules (Dreisbach, 1983).

Phenol ingestion causes little pain, because it destroys the peripheral afferent nerve endings. Rapid absorption from the gastrointestinal tract or the skin leads to convulsions, coma with respiratory depression, haemolysis, methaemoglobinaemia, discolouration of the urine with haemoglobinuria and renal damage (Goulding, 1981).