A REVIEW ON SOME CULTIVATED AND NATIVE POISONOUS PLANTS IN ADEN GOVERNORATE, YEMEN

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Abstract

Plant poisoning is a health concern in many countries where plants are used either accidently, especially among children, or intentionally for purposes such as assassination, suicide, hunting, fishing and treating various diseases. Presently, despite the implementation of toxicology surveillance systems in many countries, plant poisoning continues to be a preventable cause of morbidity and mortality.

In the Aden governorate of Yemen, there are no laws or regulations for the prevention of plant poisoning, despite the existence of several poisonous species in gardens, and as roadside trees planted by the local authority, or growing wildly in public areas. In addition, there is a lack of scientific studies on the risks of these poisonous plants. Therefore, we undertook this study, based on scientific review, to document and illustrate the botanical, geographical and toxicological characteristics of fourteen poisonous plants collected from different districts of Aden governorate.

The documented poisonous species (6 species) belong to Apocynaceae followed by Fabaceae (2 species), whereas Aristolochiaceae, Cucurbitaceae, Dracaenaceae, Euphorbiaceae, Meliaceae, and Verbenaceae are represented by one species each. The toxic parts of the majority of studied poisonous species are the whole plant, latex, seeds, and fruits. Cardiotoxicity, cytotoxicity, gastrointestinal toxicity, and inflammation of skin and mucous membrane are the main clinical manifestations. They are caused by varying amounts of plant toxins such as cardiac glycosides in *Calotropis procera*, *Cryptostegia grandiflora*, *Nerium oleander* and *Thevetia peruviana*, and cytotoxic toxins such as toxalbumins in *Abrus precatorius* and *Ricinus communis*, aristolochic acids in *Aristolochia bracteolate*, and vinca alkaloids in *Catharanthus roseus*, as well as gastrointestinal toxins such as cucurbitacins in *Citrullus colocynthis*, and tannins in *Caesalpinia pulcherrima*. Inflammation of skin and mucous membrane is caused by calcium oxalate crystals in *Calotropis procera* latex, and soluble protein in *Cryptostegia grandiflora* latex. Moreover, *Azadirachta indica* caused a number of toxicities attributed partially to tetrannortriterpenoids, while *Sansevieria trifasciata* toxicity was reported to be low.

The significance of this work is to promote the awareness among the local authority to take legal actions against plant poisoning. In addition, it provides the physicians with scientific information for the diagnosis and treatment of poisoning by some plants. It is hoped that this study motivates researchers to conduct further research on poisonous plants throughout Yemen.

Keywords: Aden, Cultivated, Native, Poisonous plants, Toxicity, Yemen.

1. Introduction

A poison is a substance, which, when administered locally, inhaled or ingested, is capable of acting deleteriously on the human body. Poisonous plants are widely distributed over the world and used for different purposes such as a method of murder, self-harm, execution, hunting, fishing and treating various diseases [1 & 2]. Plant poisoning in animals is usually accidental, and most frequently occurs during unfavorable conditions when pastures are poor, for example due to
drought or consumption of hay contaminated with poisonous plants. In humans it may be accidental or intentional. Accidental poisoning in humans may be due to confusing poisonous with edible plants, contamination of food or water with poisonous plants, use of the plants by children, or utilization of plants as remedies [3 & 4]. Poisonous plants can affect the entire spectrum of the organism systems. The dominant effect may depend on the plant species, growth stage of the plant, part of the plant used and the amount consumed, as well as susceptibility of the victim [3]. Poisonous effects are due to the production of substances such as alkaloids, glucosides, picrotoxins, resins, terpenoids, saponins, tannins, and toxalbumins in the toxic parts of the plants [1, 2 & 3]. Surveys of various poison centers in different countries showed the involvement of toxic plant exposures among the registered cases of poisoning. The American Association of Poison Control Centers' (AAPCC) National Poison Data System (NPDS) reported that poisonous plant exposures were corresponding to 1.67% of all exposures in 2018 [5]. The Poisons Information Centre Erfurt registered that poisonous plant exposures were responsible for 8.2% of all inquiries from the beginning of 2001 to the end of 2010 [6]. A 10-year retrospective cohort study of plant poisoning registered by the Ramathibodi Poison Center, Bangkok, Thailand from January 2001 to December 2010, indicated poisonous plant exposure cases comprising 3.1% of all cases recorded during the study period [7]. Of all inquiries related to acute human exposures received by the New Zealand National Poison Centre from 2003-2010, 6.4% involved plants [8]. Moreover, several studies have reported on the poisoning (intentionally or accidentally) of adults and children, in different countries, with a variety of toxic plants [9, 10, 11 & 12].

In Aden governorate, several poisonous species can be found as wild plants growing in public areas or planted in gardens, and along roadsides by the municipal Department. The lack of knowledge on the risks of these poisonous plants among the local authority, health care professionals and general public and the absence of laws and regulations and consequently national programs for prevention of or response to plant poisoning, as well as the absence of scientific works on poisonous plants in Aden governorate, encourage us to conduct this scientific literature review with the goals to document a number of poisonous plants in Aden governorate and provide scientific information on the botany, geography, and the toxins contained in the toxic plant parts and their toxicological mechanisms of action, main clinical manifestations and managements. We hope that this work will evoke the interest of the local authorities and health care professionals to take actions for preventing plant poisoning.

2. Materials And Methods

2.1. Study Area

The study was conducted in Aden governorate of Yemen (Fig. 1). It is located on the coast of the Gulf of Aden and consists of eight districts. It has an area of 741 km² and population of 925,000 people [13]. Field tours were performed in different districts of Aden governorate (Al-Kamisri nursery in Ash Shaikh Outhman district, alt. 14 m a.s.l., 12° 52' N, 44° 58' E; Dar Sad district, alt. 25 m a.s.l., 12° 57' N, 45° 02' E; Khur Maksar district, alt. 7 m a.s.l., 12° 48' N, 45° 15' E, and Gawala, Madinat ash-Sha'b in Al Buraiqeh district, alt. 19 m a.s.l., 12° 54' N, 44° 57' E) to collect samples of poisonous plants. The collected plants were photographed and voucher specimens of them were deposited in the Department of Pharmacognosy- Faculty of Pharmacy- Aden University

Fig 1. Map of the study areas in Aden governorate districts [13]

2.2. Literature Review

Data on botanical name, family name, local and Arabic names, brief botanical description, and distribution of the collected plants were obtained from several references [14-22]. In addition, electronic databases such as Google, Google scholar, PubMed, Science Direct and published e-books were searched using a combination of different terms such as the name of the plant with the toxic plant parts, the toxic chemical constituents, the toxicological activities and associated mechanism of actions.
3. Results And Discussion

Fourteen plant species belonging to 14 genera and 8 families collected from different districts of Aden governorate were found to possess poisonous effects. The family Apocynaceae (6 species) was the most represented family followed by Fabaceae with 2 species. Euphorbiaceae, Cucurbitaceae, Dracaenaceae, Euphorbiaceae, Meliaceae, and Verbenaceae were represented by only one species each. Table 1 presents data on local/Arabic name, collection location, distribution in Yemen and globally and brief botanical description of the collected poisonous plant species.

Table 1. Botanical characteristics and biogeography of the collected poisonous plant species from Aden governorate districts

| Loc./Arabic names | Collection location | Distribution in Yemen & globally | Brief botanical description |
|-------------------|---------------------|----------------------------------|-----------------------------|
| 1. Abrus precatorius L. (1767) subsp. precatorius / Fabaceae, Fig. 2 |
| Shaklam, Sous, Ain al-Afreet, Byllia [15 & 23] | Al-Kamisri nursery, Al-Sheikh Othman district | • Al-Kamisri nursery (Aden) [24]  
• Native to India, from the Himalayas down to southern India and Sri Lanka, but now grows in tropical Asia and Australia [19 & 25]. | It is a climbing shrub 1-4.5 m tall, with greenish yellow branches. Leaves compound with 10-20 leaflet-pairs, leaflets deciduous, subsessile, oblong, obovate-oblong or ovate, 0.6-2.7 × 0.3-1 cm, glabrous above, sparsely appressed pubescent beneath. Inflorescences robust, usually curved, 2-7 cm long with subsessile flowers in dense clusters, peduncles 1.5-6 cm long. Calyx 3 mm long. Corolla yellow, white, pink or mauve, 9-15 mm long. Pod oblong, 2-4 × 1.5 cm, with a hooked beak, with dense short reddish-brown appressed pubescence. Seeds red or scarlet with a black spot round the hilum, almost globose, 5.7 × 4.5 mm, shining. [Modified after 18]. |
| 2. Aristolochia baucelata Lam. (1783)/ Aristolochiaceae, Fig. 2 |
| Liyah, Ghaga, Loaeya, Loiya, Isleet [14, 22, 24]. | Gaivala, Madinat ash-Shab (Al Burniqh district) | • Coastal areas, Tihamah foothills, Taiz, Adhala, Yafya, Abyan, Lahj, Hadhramaut, Toor Al-Baha, Yemen [24 & 26].  
• Somalia, Djibouti, Ethiopia and westwards to Nigeria, East Africa, Saudi Arabia, Oman, UAE, Pakistan, India, Ceylon [14, 18 & 27]. | Prostrate glabrous, glaucous perennial herb. Leaves alternate, ovate, c. 4.5 cm long, base hastate to subcordate, margin irregularly crenate, glabrous; petiole 0.4-5.4 cm long. Flowers solitary or 2-3 together, axillary. Perianth-tube yellowish-green, up to 2.5-4.4 cm long, with a bulbous globose swelling at the base, limb flat, narrowly oblong, up to 30 × 8 mm, reddish brown. Capsule cylindrical or obpyriform, c. 2 cm long, glabrous, 12-ribbed. Seeds triangular, rugose. [Modified after 14, 19 & 22]. |
| 3. Azadricha indica A. Juss. (1830)/ Meliaceae, Fig. 2 |
| Neem, Muraaymeh [20 & 22]. | Al-Kamisri nursery, Al-Sheikh Othman district | • distributed throughout Yemen as ornamental [22 & 24].  
• Indigenous to India and Burma and widely distributed in South and South-East Asia. Cultivated in drier parts of Africa, Arabia, the South Pacific Islands, South and Central America and America, and in southern Florida and California, United States of America [20 & 28]. | Evergreen tree up to 15 m tall. Young shoots glabrous. Leaves petiole, pinnate compound, up to 40 cm long. Leaflets 8-18 pairs, usually opposite, lanceolate, oblique, up to c. 9 × 3 cm, long-acuminate at the apex, with coarsely serrate margin, glabrous. Inflorescence an axillary panicule up to 35 cm long. Calyx white. Corolla white. Fruit ellipsoid, 1.5-1.8 cm long, yellow [Modified after 15 & 20]. |
| 4. Caesalpinia pulcherima (L.) Sw. (1791)/ Fabaceae, Fig. 2 |
| Barbados Pride [22] | Dar Sa'ad district | • introduced and cultivated in gardens and streets in Taiz, Aden and West of Qshin (Al-Mahara, Yemen) [22 & 24].  
• Probably native to tropical America. It is now cultivated elsewhere [18]. | Glabrous ornamental shrub, unarmed or with small prickles; pinnae 3-10 pairs; leaflets 5-11-(13) pairs per pinna, oblong-elliptic. Flowers in long racemes, with scarlet, red and yellow, orange-red or yellow petals 15-25 mm long [Modified after 19]. |
| 5. Calotropis procera (Ait.) Ait. f. (1811)/ Apocynaceae, Fig.2 |
| Ushar [24] | Al-Kamisri nursery, Al-Sheikh Othman district | • The most conspicuous plants in Yemen, which is widespread up to 2300 m though at higher altitudes mostly occurring as scattered individuals on roadsides and in waste ground. It is abundant along sandy wadis in the Tihamah, the escarpment foothills and particularly in the Marshiq where with Tamarix it is the most characteristic species of wadi margins and Socotra [22 & 26].  
• Somalia, Djibouti, Eritrea, Ethiopia, drier parts of tropical Africa, Arabia and India; naturalized elsewhere in the tropics [21]. | Soft-wooded shrub, up to 4 m high, with plentiful white latex and rather weak spreading branches; bark fissured. Leaves sessile, weakly cordate, obovate or elliptic, 10-25 cm long. Flowers in dense, pendunculate, lateral cymes; corolla deeply 5-lobed, the lobes triangular, c. 10 mm long, white on outside, purple inside; corona prominent, white in the center. Follicles usually solitary, inflated, ovate to subglobose, smooth, 5-15 cm long [Modified after 22]. |
| 6. Catharanthus roseus (L.) G. Don (1837)/ Apocynaceae, Fig.2 |
| Bifah, Winka, Finka, Ain al-bazoon, Fol afranki [16, 24 & 29] | Al-Kamisri nursery, Al-Sheikh Othman district | • distributed throughout Yemen as ornamental [22 & 24].  
• Native from Madagascar to India, the plant has now spread throughout the tropics and is cultivated naturalized in many areas of the | Perennial herb or shrub up to 75 cm tall, stems glabrous or pubescent. Leaves opposite, petiolate, oblong to obovate, tapering to base, up to c. 7 cm × 2 cm, obtuse at the apex, pubescent or glabrous. Flowers relatively large, solitary axillary, short pedicelled; corolla white or pink, usually with

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Aristolochiaceae, Cucurbitaceae, Dracaenaceae, Euphorbiaceae, Meliaceae, and Verbenaaceae were represented by only one species each. Table 1 presents data on local/Arabic name, collection location, distribution in Yemen and globally and brief botanical description of the collected poisonous plant species.
world as an ornamental garden plant [20 & 21].

7. Citrullus colocynthis (L.) Schrad. (1838) / Cucurbitaceae, Fig.2

- Shari, Handhal, Dabak, Alkm, Hadag, Hawgargal, Ketha'a an-neaam, Aab alhayah, Mus-sahari [14, 23, 24 & 29].

- Dafly, nursery, Al-Kamisri district

- Al-Kamisri nursery, Al-Sheikh Othman district

- Al-Kamisri nursery, Al-Sheikh Othman district

- Al-Kamisri nursery, Al-Sheikh Othman district

- Al-Kamisri nursery, Al-Sheikh Othman district

- Khur Maksar

- Madinat ash-Shab - Al Burquis district

- Madinat ash-Shab - Al Burquis district

- Lantana, Men, Hashaf, Hantakes [24]

- Lantana, Men, Hashaf, Hantakes [24]

- Dafly, Daf, Dafly, Taflah, Ward Al-Hameer, Sum Al-Hemar, Ghar Wardi, [29].

- Indian Jasmine [24]

- Kharwa'a, Tubshah, Tamra Gar, Rasba [22, 23 & 29].

- Kharwa'a, Tubshah, Tamra Gar, Rasba [22, 23 & 29].
Searching electronic databases have revealed several studies reporting on the toxicological properties of the collected poisonous plants. In Table 2, we grouped 12 of the collected poisonous plants according to their main toxic properties and presented the major clinical manifestations of actions. On the other hand, the mechanistic study of the actions of the toxins contained in *Azadirachta indica* and *Sansevieria trifasciata* have not yet been reported in the literature reviewed. Toxic plant parts and the main clinical manifestations following the exposure to the poisonous plants are also presented in Table 2.

### Table 2. Mechanisms of toxicity and main clinical manifestations of plant toxins of the studied poisonous plants from Aden governorate

| Scientific name | Toxic part | Toxins | Main clinical manifestations |
|-----------------|------------|--------|------------------------------|
| **Cardiototoxic plants** | | | |
| **Mechanism of toxicity:** Inhibition of the cellular Na+/K+-ATPase, which indirectly increases the intracellular Ca2+ concentration in myocardial cells. Therapeutically, this both enhances cardiac inotropy (contractility) and slows the heart rate. However, excessive elevation of the intracellular Ca2+ concentration also increases myocardial excitability, predisposing to the development of ventricular dysrhythmias. In addition, enhanced vagal tone, mediated by the neurotransmitter acetylcholine, is common with poisoning by these plants and produces bradycardia and heart block. | | |
| Calotropis procera (Ait.) Ait. f. | Leaves, stems roots, and latex [39 & 40]: | Cardiac glycosides e.g., usharadin, calotocin, calotropin, in all parts of the plant [41 & 42]: | - Gastrointestinal effects: burning in throat, stomatitis, abdominal pain, nausea, vomiting, diarrhea and hepatitis. - Cardiovascular effects: tachycardia, hyperkalemia. - Dilated pupils, tremors, vertigo and convulsions [39, 40 & 43]. |
| Cryptostegia grandiflora Roxb. ex R. Br. | All parts [39 & 44]: | Cardiac glycosides e.g., cryptostigmin I, II, III, and IV [44 & 45]: | - Gastrointestinal disturbances: abdominal pain, nausea, vomiting, and anorexia. - Cardiovascular effects: hypotension, bradycardia, hyperkalemia, cardiac arrhythmias (Mobitz type 1, Mobitz type 2, junctional rhythm, AV dissociation and atrial fibrillation), second degree AV block and complete heart block, followed by a residual first-degree heart block. - Neurological symptoms: hypertonia, hyperreflexia, subtle higher mental function derangement, weakness, lethargy, drowsiness, disorientation and delirium. - Hematologic manifestations: bleeding manifestations and epistaxis due to thrombocytopenia [44 & 46]. |
| Nerium oleander L. | All parts and especially the roots [39, 47-49]: | Cardiac glycosides e.g., oleandrin and neroine [39, 47 & 48]: | - Gastrointestinal effects: nausea, vomiting, increased salivation, abdominal pain and diarrhea. Additional symptoms are irritation of the mucous membranes, resulting in buccal erythema, numbness, dysesthesias and a burning sensation in mouth. - Cardiac symptoms: dysrhythmias include sinus bradycardia and other arrhythmias, atrioventricular (AV) block, atrial fibrillation and/or ventricular fibrillation. In severely poisoned patients, fatal cardioversion-resistant ventricular fibrillation or refractory cardiogenic shock may follow. Sever toxicity was also demonstrated with prominent hypotension and hyperkalemia. Typical features of digoxin poisoning such as atrial or ventricular tachyarrhythmias or ventricular ectopic beats were observed in relatively few patients affected by *T. peruviana* poisoning. - Neurological symptoms: tremor, drowsiness, ataxia, confusion, dizziness, visual disturbances, mydriasis and weakness [39, 47, 48, 50 & 51]. |

### Cytotoxic plants
Protein toxins: Toxalbumins

Mechanism of toxicity: The toxalbumins (abrin and ricin), belong to the group of type 2-ribosome-inactivating protein, work specifically by inhibiting the function of ribosomes, the subcellular organelle responsible for protein synthesis. The toxins typically have two linked polypeptide chains (A-chain linked by a disulfide bond to a B-chain). One of the chains (B-chain) binds to cell-surface glycoproteins to allow endocytosis into the cell. The other chain (A-chain) upon cell entry binds the 60S ribosomal subunit and impairs its ability to synthesize protein. These lectins are extremely poisonous and an oral dose of 1 mg/kg body weight is enough to kill a human and an injection of 0.1 μg and less per kg body weight can be lethal [52 & 53].

| Plant Name | Description | Toxicity |
|------------|-------------|----------|
| Abrus precatorius L. | Seed coat (Ingestion of well-shewed, broken or pulverized seeds to release the toxin from hard water impermeable seed coat) [39]. | Abrin [39, 52 & 53] - Mild to severe gastrointestinal toxicity depending upon the amount of toxin exposure and include nausea, vomiting, abdominal pain, diarrhea. Gastrointestinal bleeding may ensue with bloody diarrhea and/or hematemesis. Patients may exhibit tachycardia, headaches, dilated pupils, irrationality, hallucinations, drowsiness, weakness, tetany, tremors, seizures, fever, flushing of the skin and dysrhythmias (unspecified) [39, 54 & 55]. - Parenteral administration or inhalation, or perhaps large ingestion, may produce life-threatening systemic findings, including multisystem organ failure, even with small exposures [39 & 55]. |
| Ricinus communis L. | Ricin [39, 52 & 53] - One of the chains (B-chain) binds to cell-surface glycoproteins to allow endocytosis into the cell. The other chain (A-chain) upon cell entry binds the 60S ribosomal subunit and impairs its ability to synthesize protein. These lectins are extremely poisonous and an oral dose of 1 mg/kg body weight is enough to kill a human and an injection of 0.1 μg and less per kg body weight can be lethal [52 & 53]. |

Alkylating and intercalating DNA toxins: aristolochic acids

Mechanism of toxicity: Aristolochic acids I (AAI) and II (AAII), two structurally related nitrophenanthrene carboxylic acids, are the major components of the AA mixture contained in the plant extract of the Aristolochia species. Several enzymes have been demonstrated to metabolize AAI and AAII to a cyclic N-acylnitrenium ion with a delocalized positive charge able to covalently bind to the exocyclic amino groups of purine bases and to form DNA adducts. If alkylated DNA bases are not repaired, they can cause mutations and even cancer [52 & 56].

| Plant Name | Description | Toxicity |
|------------|-------------|----------|
| Aristolochia bracteolata Lam. | Herb [56-58]. | Aristolochic acids [52, 56-58] - Causing a syndrome of kidney injury, termed aristolochic acid nephropathy (AAN), which is marked by elevated serum creatinine, significant anemia, and histopathologic changes demonstrating a hypocellular interstitial infiltrate with severe fibrosis. Progression towards end-stage renal disease is rapid, with most patients having chronic kidney disease for less than 2 years. In addition, AAN is associated with a 40-45% prevalence of ureterohelial carcinomas [57 & 58]. |

Mitotic inhibitors: vinca alkaloids

Mechanism of toxicity: The plant vinca alkaloids interfere with the polymerization of microtubules, which must polymerize for mitosis to occur, leading to metaphase arrest. Rapidly dividing cells (e.g., gastrointestinal or bone marrow cells) typically are affected earlier and to a greater extent than those cells that divide slowly. In addition, microtubules are important in the maintenance of proper neuronal function [39].

| Plant Name | Description | Toxicity |
|------------|-------------|----------|
| Catharanthus roseus (L.) G. Don | All parts [39] | Vinblastine and vincristine [12, 39 & 59] - Ingestion may cause initial ophthalmic pain followed in several hours by intense gastrointestinal symptoms. (abdominal pain, vomiting and severe, profuse, persistent diarrhea). Vinca alkaloids may subsequently produce peripheral neuropathy, bone marrow suppression, and cardiovascular collapse [39 & 59]. |

Gastrointestinal toxic plants

Mechanism of toxicity: Plants containing tannins are gastrointestinal irritant due to the strong astringency of tannins and their protein-binding ability that results in destruction of the mucosal lining of the digestive tract. It has been reported that the excretion of mucoprotein, sialic acid, and glucosamine was increased in feces of rats fed on tannin acid-containing diets [60]. Cucurbitacins isolated and purified from plants, as well as cucurbitacin containing plant material are violent irritants of the intestinal mucosa. Coloscopy of C. colocynthis intoxicated patients indicated mucosal erosion [61].

| Plant Name | Description | Toxicity |
|------------|-------------|----------|
| Caesalpinia pulcherrima (L.) Sw. | Mature seeds [39 & 62] | Tannins (protein precipitants) [39] - Gastrointestinal effects after a latent period of 30 minutes to 6 hours: nausea, vomiting, abdominal cramping, diarrhea, and dehydration [39 & 62]. |
| Citrullus colocynthis (L.) Schrad. | Pulp of the peeled fruit [61, 63-65] | - Cucurbitacin A, B, C, D, E, F, I, J, K, L and glycocides [64 & 66]; - Colocynthin (Cucurbitacin E 2 O-D-glucopyranoside) [67] - Three cases with toxic acute colitis were presented with dysenteric diarrhea, diffuse abdominal pain. Coloscopy indicated lesions mostly in the sigmoid and descending colonic sections, consisting of thickened edematous and inflammatory folds with exudates but without ulceration or pseudopolyph formation. [63]. - Acute severe bloody diarrhea [68]. - One case was presented with dizziness, mild abdominal pain, watery diarrhea, moderate to severe hypotension, moderate hypoglycemic, and hepatic injury with increasing of hepatic enzymes [65]. - Four cases were presented with acute rectorrhagia preceded by mucosal diarrhea with tenesmus, which gradually progressed to bloody diarrhea and overt rectorrhagia within 3 to 4 hours. The only colonscopic observation was mucosal erosion [61]. |
| Lantana camara L. | - Immature berries [39, 69 & 70]. - Unripe and ripe berries, flower, leaf, stem and seeds [71]. - Leaves by grazing livestock [39 & 69]. | - Unknown for human toxicity [39]. - Animal toxicity primarily by pentacyclic triterpenoids (lantadene), of which lantadene A is the most significant toxic principle [69 & 72]. - Effects after ingestion of unripe berries are most commonly gastrointestinal, including nausea, vomiting, abdominal cramping, and diarrhea. Severe toxicity may cause weakness, lethargy, large pupils, and respiratory depression [39]. - Of the 17 children who ingested unknown quantities of unripe berries, 4 children developed symptoms including nausea, vomiting, dilated pupils, depressed mental status, deep respirations and depressed deep tendon reflexes and one patient (a two-year-old girl) died 90 minutes after presentation [71]. - Effects after pediatric (ages range from 1 to 16 years) ingestion of berries (ripe and unripe), flowers, leaves, stem, and seeds of the plant were vomiting, abdominal pain, diarrhea, throat/mouth irritation, nausea, agitation, tachycardia, drowsiness and mydriasis [71]. |

Plants with irritant sap or latex
**Mechanism of toxicity:** Poisoning by plants with irritant sap or latex could be attributed to 1): the content of mechanical irritant such as calcium oxalate needles in *C. procera* that are released by chewing in a projectile fashion and penetrate mucous membrane and induce the release of histamine and other inflammatory mediators [39, 40]. 2): Chemical irritants that induce inflammatory activity such as histamine in *C. procera* latex that is released upon local contact with skin [73 & 74] and 3): stimulation of inflammation by the release of mast cell histamine and the induction of prostaglandin synthesis through the activation of cyclooxygenase-2 by *C. procera* latex [73 & 74] and stimulation of neutrophil migration, enlarging vascular permeability and increasing myeloperoxidase activity by soluble protein fraction in *C. grandiflora* latex [75].

| Scientific name | Toxic part | Toxins | Main clinical manifestations |
|-----------------|------------|--------|-----------------------------|
| Calotropis procera (Ait.) Ait. f. | All parts of the plant and the latex [39 & 40] | - An unidentified vesicant allergen in the latex [39] - Calcium oxalate crystals [39]. | - Local administration induces intense inflammatory response. The acute inflammation induced by latex involves edema formation and cellular infiltration [74]. - Latex caused immediate mild to moderate or severe corneal damage with painless blurring of vision. Low endothelial cell count was also reported by some studies suggesting that the cause of corneal oedema is endothelial toxicity [76-79]. In addition, cases of iridocyclitis [76 & 77] and associated secondary glaucoma [76] have been reported. - Applying latex to right upper posterior carious molar by a patient to soothe toothache resulted in the burning of the mucosa, inflammation and formation of oro-antral communication [80]. - Ingestion of calcium oxalates cause a painful burning sensation of the lips and mouth. There is an inflammatory reaction, often with edema and blistering. Dysphonia, and dysphagia may also result [39]. |
| Cryptostegia grandiflora R. Br | Latex [75] | Soluble protein fraction of the latex [75] | - A potent inflammatory [75]. - Skin irritant and burning of exposed skin [81]. |
| Plumeria rubra L. | Latex [39] | NS | - Skin irritation, pink rash and dermatitis [82 & 83]. |

**Other Poisonous Plants**

| Scientific name | Toxic part | Toxins | Main clinical manifestations |
|-----------------|------------|--------|-----------------------------|
| Azadirachta indica A. Juss. | Seed fixed oil [84-87] | - NS - Nimbolide and 6-deacetylnimbin isolated from methanolic extract of the seeds showed hepatotoxicity [88]. | - Toxic encephalopathy particularly in infants and young children (after oral administration of ‘Droplets’ and 5ml oil). Usual symptoms included vomiting, drowsiness, tachypnoea and recurrent generalized seizures; leukocytosis and metabolic acidosis were also observed [89]. - 12 children from South India, who were given single dose of *A. indica* oil (neem oil) (25–60 mL) for cough, 10 of them died. In this group, 10 children presented with seizures and altered sensorium. Metabolic acidosis was seen in four with very low bicarbonate values. Liver biopsy done in one of the fatal cases showed fatty infiltration with patchy necrosis [85]. - A case of accidental neem oil poisoning in a 5-year-old child was manifested with refractory seizures and metabolic acidosis. Late neurological sequelae in the form of auditory and visual disturbances and ataxia were present [84]. - A suicidal consumption of neem oil for 5 days by a 35-year-old female patient was found to cause ophthalmopathy and loss of bilateral vision [87]. - Vomiting, seizures, metabolic acidosis, and toxic encephalopathy were evident after neem oil poisoning of a 73-year-old male [86]. |
| | Leaves | NS | A case of ventricular fibrillation and cardiac arrest [90]. |
| | Pesticide, named NeemAzal-T/S containing Azadirachtin 1% [85]. | Azadirachtin [85] | A case of 35-year-old lady who had consumed a 250 mL of the pesticide, in an attempt of deliberate self-harm was manifested with neurotoxicity [85]. |
| Sansevieria trifasciata | All parts [91] | Saponins and organic acids [91] | Minor toxic affects if eaten such as excessive salivation, or minor skin irritation [91]. |

NS= not specified

Poisonous plants are widely distributed throughout the world. More than 700 plant species are recognized as being potentially dangerous in the world and a large number (around 500 species) of these are frequently used as ornamentals. Dangerous plants are classified in two large groups according to the type of interaction with the organism: those that cause injuries by external contact and those that do so after ingestion and/or aspiration [92]. The expected toxicity of ingested toxic plants depends on various factors. Some depend on the plant (type, growth and maturation, part ingested (intact or crushed before or during ingestion), and amount, as well as toxin concentration in the plant part ingested), while others depend on the patient (weight, age, health at the time of ingestion, vomiting after ingestion) or the time elapsed since ingestion. When it comes to age, the most frequent exposure to toxic plants in the early years of life is unintentional. The exploratory behaviors and the limited risk perception characteristic of children are the reason for the higher incidence (up to 85% especially younger than 5 years) in this population. Moreover, poisoning can also occur in the context of child abuse or the use of plant materials for suicidal purposes or misuse (medicinal, hallucinogenic or food) [9, 47 & 92]. The correct diagnosis of plant poisoning in children is particularly difficult, so they are probably under diagnosed. Part of the reason for this is that often neither the patient nor the family associates the symptoms with exposure to a plant, and sometimes the exposure was not even witnessed by the adults. The correlation between taxonomy and toxicology is also poor, as members of the same plant family can cause different clinical manifestations. This is
compounded by the fact that the doctors frequently lack the botanical knowledge required to guide the differential diagnosis [9 & 92].

Our work documented and described the botanical and geographical characteristics as well as presented the toxicological effects and clinical managements of 14 poisonous plants collected from different districts of Aden governorate (Table 1 and 2). Among the collected poisonous plants, those containing cardiac glycosides (C. procera, C. grandiflora, N. oleander and T. peruviana), toxalbumins (A. precatorius and R. communis), alkylating and intercalating DNA toxins (A. bracteolate) and mitotic inhibitors (C. roseus), are the most toxic species and can be potentially fatal. The cardiovascular effects induced by C. procera, C. grandiflora, N. oleander and T. peruviana were reported to be usually associated with gastrointestinal disturbances as the early symptoms of poisoning and neurological effects (Table 2). In addition, in a woman who died by calotrops poisoning, the small intestines, liver, spleen and kidney were congested [40]. Furthermore, C. procera leaves and latex were found to cause severe pathological changes in the liver, kidneys, heart, lungs, brain and intestines of sheep and goats [93-95]. C. grandiflora was also found to cause death in cattle, sheep, goats, horses and donkeys and hence is unpalatable and rarely eaten by the animals [44, 81 & 96]. Poisoning with N. oleander has been reported not only from the ingestion of any part of the plant but also from the ingestion of sap or honey produced from this plant or the ingestion of meat or marshmallows roasted on stems or the drinking the water in which the flowers have been placed as well as inhaling smoke from burning the plant [4 & 39]. Several nonfatal cases have been reported with different levels of toxicity after ingesting varying amounts of N. oleander leaves, leaf extract, flowers and root extract. On the other hand, a number of fatalities after ingestion of known or unknown amount of N. oleander have also been reported [47]. A suicidal use of N. oleander was reported by a case of a 55-year-old man who prepared a cocktail comprising of 25 N. oleander leaves and five flowers which he blended with a soft drink and consumed. He survived this suicide attempt due to specialized medical treatment and the fact that he vomited severely after ingestion of the blend [4]. On the other hand, there was no toxicity or deaths reported from topical administration or contact with N. oleander [49]. Accidental and/or experimental N. Oleander toxicities have been reported in cattle, horses, sheep, goats, donkeys, camels, cats, dogs, monkeys, budgerigars, geese, ducks, turkeys, toed sloths and bears. However, rodents and birds were observed to be relatively insensitive to oleander cardiac glycosides [49, 97 & 98]. Deliberate self-harm with seeds of yellow oleander (Thevetia peruviana) was found to cause significant morbidity and mortality each year in South Asia including Sri Lanka. T. peruviana was reported to be the commonest plant poison in adults with a case fatality rate of up to 10% in a number of studies in Eastern, Northern, Southern and North-Central of Sri Lanka [11, 47, 50, 51, 99 & 100]. Poisoning through accidental ingestion of T. peruviana seeds was common in young children [47]. Deliberately as well as accidental ingestion of T. peruviana seeds has been reported among Sri Lankan children in urban area [101]. Among 325 children involved in a multicenter study of plants poisoning in rural Sri Lanka, 68 children (20.9%) were reported to be deliberately poisoned with T. peruviana seeds with 4 lethal cases [11].

Variations in the severity of toxicity of A. precatorius and R. communis seeds may be related to the degree to which the seeds are ground or chewed before ingestion and the amount of toxins (abrin and ricin, respectively) released (from the hard water impermeable seed coat) and absorbed by intestinal cells. The oral LD₅₀ of ricin for human was estimated as 1–20 mg/kg, bw, and the LD₅₀ of abrin for humans has been reported to be from 10 to 1000 μg/kg via oral ingestion and 3.3 μg/kg if injected [55 & 102]. The cause of death is related to toxin-induced damage to the endothelial cells, resulting in a vascular leak syndrome characterized by hypoalbuminemia and edema. This leads to vascular collapse and shock, with death occurring after 3 or more days [53]. Worbs et al. have presented a summary of human and veterinary intoxications with R. communis seeds. The number of human cases (the majority were children aged 1-8, and <19 years) reported were presented either as accidental intoxications (875 oral cases with 13 fatal cases among them and 1 injectional case, fatality rate 1.5%) and intended intoxications (5 oral cases without fatality and 6 injectional cases with 5 fatal cases among them, fatality rate 45.5%). Veterinary intoxications with fatal cases were reported in dogs (ingestion of castor seeds (R. communis), fertilizer based on castor seed cakes, motor oil based on castor oil, and soil conditioner with 10% oil cake, with fatality rate of 35.3% in Germany and 23.5% worldwide) as well as in other animals such as pigs, heifers and cattle (ingestion of layer’s mash containing castor seed husks in meal), ducks (ingestion of castor seeds), horses and cows (ingestion of fluxseed flour contaminated with castor seeds), sheep and goats (ingestion of garden waste containing castor beans). Animals showed similar symptoms as humans after intoxication with ricin, which were weakness, profuse watery diarrhea, dehydration with sunken eyes, dilation of pupils, depression, tachycardia, dyspnea and colic. These signs and symptoms developed most frequently within 6–24 h [55]. In addition, there have been several reports of allergy to castor seeds. Cases of asthma have been reported not only in employees in the oil industry, but also in seamen and laboratory workers exposed to the seeds. Three important allergens from the crude drug have been identified: a 2S storage albumin with a molecular weight of 11 kDa, a 11S crystalloid protein with bands at 50 kDa and a protein doublet of 47 and 51 kDa. These allergens have been named Ric c 1, Ric c 2 and allergen 3. Castor oil extractors, fertilizer workers and farmers may acquire occupational dermatitis from handling plants or seeds [103]. A retrospective study (over a period of 7 years (January 2009-December 2015) documented 112 patients (age >13 years) that were poisoned by A. precatorius seeds and admitted to medicine wards [104].
In addition, several studies demonstrated some cases of human poisoning with A. precatorius seeds either accidentally or intentionally [105-109]. Young children are at great risk by ingestion of A. precatorius seeds due to their bright colors; their poisoning by accidental ingestion of A. precatorius seeds has been reported in some studies [110-112]. In addition, a prospective hospital study (from June 1984 to December 2001), carried out on children with a history of having ingested a part of a poisonous plant and admitted to the Lady Ridgeway hospital in Colombo, Sri Lanka, has reported that 46 (19%) and 8 children (0.6%) out of 243 children were poisoned by R. communis and A. precatorius seeds, respectively [101]. Moreover, a multicenter study of plants poisoning involving 325 children in rural Sri Lanka indicated that 60 (18.5%) and 17 children (5.2%) were poisoned by A. precatorius and R. communis seeds, respectively [11]. The ubiquitous nature of A. precatorius is responsible for poisoning in livestock (mainly cattle) owing to their open grazing habit. Toxicity may occur following topical injection of A. precatorius seeds preparation, called Sui or Sitari, in the muscles of the limbs of cattle grazing in neighbor’s field as a means of grievance in rural India [113].

The accessibility and high toxicity of ricin and abrin toxins lead to concerns that they could pose a severe threat to public health. Hence, ricin and abrin are classified as a Category B agent by the US Centers for Disease Control and Prevention (CDC). Agents in this category are considered moderately easy to disseminate, able to cause morbidity and low mortality [53]. Based on its history of military, criminal and terroristic use, ricin is a prohibited substance both under the Chemical Weapons Convention (CWC, schedule 1 compound) and the Biological Weapons Convention (BWC) and its possession or purification is strictly regulated and controlled by the Organization for the Prohibition of Chemical Weapons (OPCW) [55]. Abrin is also placed in the category of “Biological Select Agents or Toxins” by the US Department of Health and Human Services (HHS) [53].

Numerous reports from many countries (USA, Europe, Australia, Japan, Korea, Taiwan, China, and Hong Kong) have confirmed that plants from the Aristolochia species are the cause of the nephropathy. Aristolochic acid nephropathy has also been evidenced by various studies in humans and experimental animals and aristolochic acids (I and II) were found mutagenic in several test systems. [56-58 & 114]. A recent study has demonstrated that aristolochic acid is metabolized by nitroreduction to aristolactam, which can then be further metabolized by P450s. The resulting metabolites form DNA adducts, the main of which are 7-(deoxyadenosin-N^6-yl) aristolactam I and 7-(deoxyguanosin-N^2-yl) aristolactam I. Using UPLC−MS/MS, 7-(deoxyadenosin-N^2-yl) aristolactam I adducts were measured in renal cortex and upper tract urothelial carcinoma samples from two Taiwanese individuals [115].

Extreme toxicity was observed when humans consumed extracts of C. roseus orally and therefore, its cultivation, possession or sale was outlawed in the state of Louisiana, USA. The use of the plant was strictly for aesthetic, landscaping or decorating purposes but never for medicinal purposes [116]. Despite the benefits of the two main commercially important C. roseus alkaloids (vincristine and vinblastine) as invaluable antitumor agents, they possess many side effects (neurotoxicity, myelosuppression, alopecia, abdominal cramps, constipation, nausea/vomiting, paralytic ileus, ulcerations of the mouth, hepatocellular damage, kidney impairment, pulmonary fibrosis, urinary retention, amenorrhea, azoospermia, orthostatic hypotension, and hypertension) [117]. A report documented a 67-year-old woman with hepatitis C-related liver cirrhosis and hepatoma who had developed severe bone marrow suppression after taking C. roseus as an alternative anticancer treatment. The patient developed severe pancytopenia with initial presentations of vomiting, diarrhea, oral ulcer, and fever about 1 week after taking 5-days’ course of C. roseus. The patient also had severe gastrointestinal disturbances, bacteremia, urinary tract infection, and impaired renal and liver function [59]. Accidentally poisoning of a flock of 40 sheep (31 female and 9 male) with the leaves and flowers of C. roseus caused an acute toxicity within 24 h of ingestion of the plant with all animals manifesting salivation, incoordination, staggering, recumency, dyspnea, bloody diarrhea and dehydration. All the sheep died within two days after the start of the signs [118]. Toxicity studies with the use of C. roseus have been reported in a number of experimental animals (rabbits, mice, rats) [118-120].

Among the gastrointestinal toxic plants, C. colocynthis pulp poisoning causes severe cases of gastrointestinal disturbances. In addition, moderate to severe hypotension, moderate hypoglycemic, and hepatic injury with increasing of hepatic enzymes were recorded in a case of acute C. colocynthis toxicity (Table 2). Moreover, the extract of fruit pulp of C. colocynthis was reported to cause teratogenic effects if given during the early stage of pregnancy in rats [121]. Studying the effects of 100 or 200 mg/kg/day of either pulp or seed extracts of C. colocynthis on male rabbits, for one month, indicated that 200 mg/kg/day of pulp extract was fatal for all animals and 50% of animals treated with pulp extract at 100 mg/kg/day did not survive to the end of the study. Survival animals treated with 100 mg/kg/day of pulp extract displayed severe lesions in the small intestine, kidney, and liver. Interestingly, animals treated with either 100 or 200 mg/kg/day of seed extract displayed only minor intestinal symptoms. It has been suggested that the main pathophysiological mechanism underlying these toxic effects is likely to be the membranolytic activity of saponin present in the C. colocynthis pulp extract [122]. Very high toxicity of C. colocynthis was also shown in studies on Nubian goats, sebu calves, and desert sheep. The common feature of all toxic effects reported in experimental and domestic animals exposed to cucurbitacins isolated and purified from plants, as well as cucurbitacin containing plant material is a violent irritation of the intestinal mucosa, and after prolonged
exposure appearance of hepatic fatty changes, catarhral enteritis, pulmonary emphysema and necrosis of the cells of the renal tubuli [66]. Contrary to a study in 1964, which reported that ingestion of Lantana camara unripe berries resulted in serious toxicity and even death, Carstairs et al. have demonstrated that ingestion of L. camara (ripe and unripe berries, flowers, leaves, stem, and seeds) was not associated with significant toxicity, and patients who ingested unripe berries did not exhibit more-frequent or more-severe symptoms than patients who ingested ripe berries or other plant parts. Most patients also displayed no or minimal symptoms, and children with asymptomatic ingestions and those with mild symptoms could be treated at home [71]. Animal toxicity with L. camara was reported in some studies. Typical signs were inappetence, constipation, cholestasis, hepatotoxicity and photosensitization [39, 69 & 72]. Hepatotoxicity, caused primarily by lantadene (with lantadene A as the most significant toxic principle), was an important cause of livestock morbidity and mortality in lantana-infested regions. Lantana poisoning has been demonstrated in cattle, buffaloes, sheep, goats and horses. However, neonatal lambs and calves were found to be resistant to poisoning by lantadene A [69 & 72]. In addition to ruminants, nonruminant animals such as guinea pigs, rabbits, and female rats were found susceptible to the hepatotoxic action of lantana toxins [69]. However, the hepatotoxicity that was well described for animals has not been known to occur in humans [71]. Poisoning with C. prodera latex is attributed to its ability to induce inflammation (Table 2). On the other hand, the dry latex-induced inflammation, tested in different models, was considered useful to evaluate the anti-inflammatory drugs [73 & 74]. It has been reported to avoid using P. rubra not only because of its irritant latex (Table 2) but also to its content of significant amounts of immunoreactive cardiac glycoside [123]. The toxicity of A. indica was reported in several acute, subacute and semi-chronic toxicity studies on animals. It has been demonstrated that oral administration of leaves, seeds, seed oil, aqueous and non-aqueous extracts of leaves and seeds as well as pure bioactive compounds such as azadirachtin, 6-deacetylnimbin and nimbidole and commercially available neem-based pesticides such as praneem (a purified seed extract) produced a variety of toxicological effects such as nervous symptoms, neuro-psychopharmacological effects, genotoxicity, teratogenic effect, hepatotoxicity, hepatorenal toxicity, reproduction disturbances and antifertility, reduction in the organ weight and varying degrees of damage of different organs (e.g. liver, kidney, lung) of the treated animals. Some of these toxic effects ends with the death of the experimental animals [88-90 & 124-127]. Moreover, reproduction and antifertility effects were reported after vaginal administration of seed oil to experimental animals (rats, rhesus monkey) [90 & 126]. It has been suggested that other compounds than azadirachtin are responsible for the toxic effects of A. indica [89]. The risk of toxicity with Sansevieria species, was reported to be low. There have been information suggesting that the foliage of this species could be toxic to some domestic animals with the potential to cause vomiting, salivation and diarrhea [128].

The treatment of the studied plants poisoning is generally supportive and symptomatic with prolonged observation of symptomatic patients. When oral poisoning is suspected, the following measurement should be performed as appropriate: applying activated charcoal to prevent further systemic absorption of the toxins, gastric lavage to remove the remaining of the toxins in the stomach (if the toxins have necrotizing action, such as ricin and abrin, gastric lavage is not advisable for fear of inducing even greater damage to the stomach mucosa tissues), and if the emesis and/or diarrhea become excessive, replacement of fluids and electrolytes should be performed. Failure to do so may lead to development of shock, myoglobinuria, and renal failure [39, 53, 62, 71, 84, 106 & 128]. The management of organ specific poisonings such as the cardiac toxicity is performed not only by applying appropriate supportive and symptomatic management but also by using digoxin specific antibody (Fab) fragments as the treatment of choice in the case of serious cardioactive steroid toxicity [39, 40, 44, 47, 51 & 65]. Aristolochic acid nephropathy is treated by glucocorticoids, which delay the progression of the disease. As most patients progress to end-stage renal disease, dialysis or kidney transplant are usually performed. Due to the high malignant potential of this disease, care must be taken to minimize future development of upper urinary tract cancers by performing prophylactic bilateral nephroureterectomies and aggressive cancer surveillance [58].

The current literature study highlights the importance of the issue of poisoning by plants grown anywhere in the cities of Aden governorate and can pose serious risks to the public health. The scientific information on the botany, geography, toxicity, clinical manifestations and management of 14 poisonous plants could serve as a quick reference for the physicians in Aden to manage any poisoning by these plants. In addition, the study could raise the awareness of the local authority and health care professionals on the subject of plant poisoning prevention. Thus, we stress the need for an effective multidisciplinary teamwork including the local authority, health care professionals, botanists, toxicologists and pharmacists to address the issue of plant poisoning exposures and prevention by creating legislation and regulation for response to and prevention of plant poisoning. It is also hoped that our work will promote further research on more poisonous plants throughout Yemen.

4. Conclusion

To conclude, poisonous plants can be present anywhere in the cities and may cause poisonings, which in some cases are severe but preventable. In order to prevent plant poisonings, the general population as well as health care providers need to be better informed on the toxicity of plants. Our work achieved its main objectives by documenting and providing scientific information on 14 poisonous plants collected from different districts of
Aden governorate. Some of these plants such as *Calotropis procera*, *Cryptostegia grandiflora*, *Nerium oleander*, *Thevetia peruviana*, *Abras precatorius*, *Ricinus communis*, and *Aristolochia bracteolate* can cause severe toxicities. Unfortunately, in Yemen there is no laws that regulate plants poisoning prevention and protect the public, especially the children from unintentionally use of the dangerous plants grown on roads, gardens, and public areas. It is hoped that this scientific review could evoke the interest of the authorities and health professionals in Yemen to take the issue of poisonous plants more seriously and create laws, regulations, and national programs for the prevention of plants poisonings.

![Poisonous plants collected from Aden Governorate](https://ejua.net)
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مقالة بحثية

مراجعة لبعض النباتات السامة المنزوعة والبرية في محافظة عدن - اليمن

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