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RESEARCH ARTICLE

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 tetranortriterpenoids, 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 organ 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 Poisons 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 km^2 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

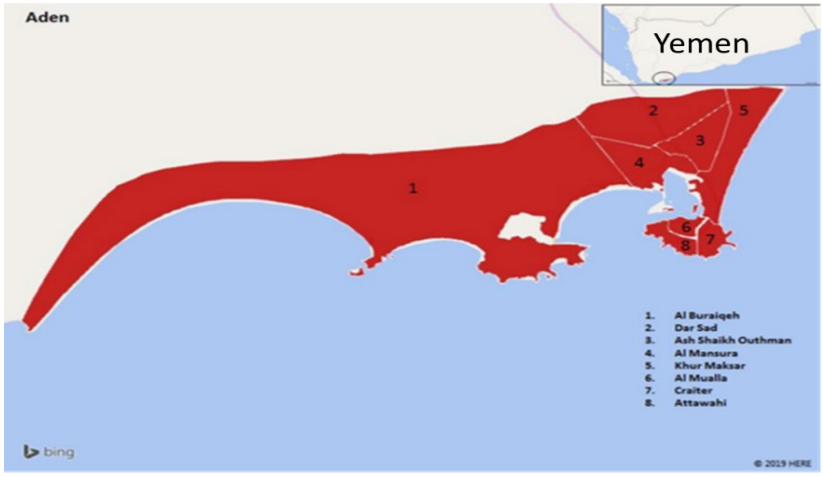

Fig 1. Map of the study areas in Aden governorate districts [13]

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. Aristolochiaceae, 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

Plant species/ Family			
Local/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 Outhman 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 × 1-1.5 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 bracteolata* Lam. (1783)/ Aristolochiaceae, Fig. 2			
Liyah, Ghaga, Loaeja, Loiya, Iqleet [14, 22-24].	Gawala, Madinat ash-Sha'b (Al Buraiqeh district)	• Coastal areas, Tihama foothills, Taiz, Adhala, Yafaa, Abyan, Lahej, Hadhramout, 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 0.5-4.5 cm long. Flowers solitary or 2-3 together, axillary. Perianth-tube yellowish-green, up to c. 2.5-4 cm long, with a bulbous globose swelling at the base, limb flat, narrowly oblong, up to 30 × 8 mm, dark reddish brown. Capsule cylindrical or obpyriform, c. 2 cm long, glabrous, 12-ribbed. Seeds triangular, rugose. [Modified after 14, 19 & 22].
3. *Azadirachta indica* A. Juss. (1830)/ Meliaceae, Fig. 2			
Neem, Muraymirah [20 & 22].	Al-Kamisri nursery, Al-Sheikh Outhman 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 Australia, and in southern Florida and California, United States of America [20 & 28].	Evergreen tree up to 15 m tall. Young shoots glabrous. Leaves petiolate, pinnate compound, up to 40 cm long, leaflets 8-18 pairs, usually opposite, lanceolate, oblique, up to c. 9 × 3cm, long-acuminate at the apex, with coarsely serrate margin, glabrous. Inflorescence an axillary panicle up to 35 cm long. Calyx white. Corolla white. Fruit ellipsoid, 1.5-1.8 cm long, yellow [Modified after 15 & 20].
4. *Caesalpinia pulcherrima* (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 Qashin (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 Outhman 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 Tihama, the escarpment foothills and particularly in the Mashriq where with *Tamarix* it is the most characteristic species of wadi margins and Socotra [22 & 24]. • 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 latexand rather weak spreading branches; bark fissured. Leaves sessile, weakly cordate, obovate or elliptic, 10-25 cm long. Flowers in dense, pedunculate, 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, ovoid to subglobose, smooth, 5-15 cm long [Modified after 22].
6. *Catharanthus roseus* (L.) G. Don (1837) / Apocynaceae, Fig.2			
Biftah, Winka, Finka, Ain al-bazoon, Fol afranki [16, 24 & 29]	Al-Kamisri nursery, Al-Sheikh Outhman 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 subshrub 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

		world as an ornamental garden plant [20 & 21].	a darker center; up to c. 3-4 cm across, tube up to c. 3 cm long, narrow cylindrical, lobes broadly obovate. Fruit green, of two cylindrical striate follicles c. 2-4 cm long, with many-seeded black [Modified after 16, 20, 21 & 30].
7. *Citrullus colocynthis* (L.) Schrad. (1838) / Cucurbitaceae, Fig.2			
Shari, Handhal, Dabak, Alkam, Hadag, Hagangal, Ketha'a an-neaam, Anb alhayah, Mur as-sahari [14, 23, 24 & 29].	Dar Sa'ad district	• Coastal areas, western mountain, Lahej, Abyan, Marib, Shabwa, Hadhramout, Socotra and Toor Al-Baha district, Yemen [24 & 26]. • Northern Africa (Algeria, Egypt; Libya, Morocco, Tunisia; Northeast Tropical Africa: Chad, Ethiopia, Somalia; East Tropical Africa: Kenya; West Tropical Africa: Mali; Asia: Kuwait, Saudi Arabia, Iraq, Jordan, Lebanon, Syria, Yemen, Afghanistan, Iran, Turkey, India, Pakistan, Sri Lanka; Europe: Greece, Italy, Spain; and Australia [31].	Monoecious trailing perennial herb. Stems shortly retrorse scabrid. Leaves scabrid, narrowly ovate in outline, up to 10 cm long, palmately lobed with the lobes pinnatisect. Tendrils simple or bifid. Male and female flowers solitary on short pedicels; petals united below, yellow-green c. 1 cm long. Fruit shortly pedunculate, globose, 5-10 cm diameter, glabrous, mottled green when young becoming yellow with maturity. Seeds ovate in outline, dark brown, smooth. [Modified after 19 & 22].
8. *Cryptostegia grandiflora* Roxb. ex R. Br. (1819)/Apocynaceae, Fig. 2			
NA	Al-Kamisri nursery, Al-Sheikh Outhman district	• Distribution throughout Yemen as ornamental [24] • Native to Madagascar, but it is widely distributed throughout tropical and subtropical regions of the world. The plant is introduced to Australia as an ornamental, has become an aggressive [32].	Many stemmed shrub, which can climb 30 m into tree canopies, or grow 1–3 m high when unsupported in open areas. Leaf blade elliptic to ovate, 6-9 × 3-5 cm, cuneate to tapering at base, usually acuminate at apex, glabrous; petiole 5-15 mm long, glabrous to rarely slightly hairy. Internodes of cymes 5-15 mm long; pedicels 3-7 mm long, usually hairy. Calyx lobes narrowly ovate to ovate, 14-20 × 4.2-8.8 mm, with reflexed margins. Corolla tube 18-30 mm long; lobes 32-56 × 15-30 mm. Corona lobes 8-11 mm high, bifid near apex only or cleft almost to the base. Staminal cone 3-4.5 mm high; anther 2.7-4.5 mm. style 1.3-3.4 mm long. Follicles 8-13.5 × 2-3.5 cm glabrous. Seeds 5-8 mm long; hairy 3-4 cm long [Modified after 33].
9. *Lantana camara* L. (1753) / Verbenaceae, Fig. 2			
Lantana, Mena, Hashaf, Hantakes [24]	Al-Kamisri nursery, Al-Sheikh Outhman district	• introduced in gardens, road sides, wadis & neglected areas of Yemen [24]. • Originally American. Now a cosmopolitan introduction in warm tropical and subtropical regions. Cultivated or undesirable weedy shrub [16].	Shrub, up to 2 m or more tall; armed with recurved prickles or unarmed. Leaves opposite, petiolate, blades ovate to ovate-oblong, crenate-serrate, rugose, scabrid, acute to acuminate. Flowers in axillary flat heads, red, purple, pink, yellow, orange or white. Drupes small, fleshy, purple or black, globose [Modified after 16, 21 & 34].
10. *Nerium oleander* L. (1753)/ Apocynaceae, Fig. 2			
Daflah, Dafl, Dafly, Taflah, Ward Al-Hameer, Sum Al-Hemar, Ghar Wardi, [29].	Al-Kamisri nursery, Al-Sheikh Outhman district	• cultivated in different cities of Yemen at gardens and streets [29]. • Trop Africa, SW Asia, introduced elsewhere [16].	Much branched shrub up to about 3-4 m tall. Leaves opposite or whorled, leathery thick, shortly petiolate, lanceolate, tapering at both ends, up to c. 15 × 2 cm, prominent-nerved, entire, acute. Flowers usually pink but variable from white to rose-red, (single in wild forms often double in cultivated forms). Calyx small, linear-lobed. Corolla tube c. 20 mm long with a corona of 5 scales at the throat; lobes obovate, each c. as long as the tube; fragrant. Stamens included; apical anther-appendages slender, twisted. Follicles up to c. 20 × 2 cm, narrow, long, ribbed, brown and open from one side. Seeds hairy, oblong-ellipsoid, c. 6 mm long with an apical tuft of hairs about as long as a seed itself [Modified after 16].
11. *Plumeria rubra* L. / Apocynaceae, Fig.2			
Indian Jasmine [24]	Khur Maksar district	• Introduced; gardens throughout Yemen as ornamental [22 & 24]. • Native to Mexico, Central America, the Caribbean and South America as far south as Brazil but now widely cultivated throughout the world's tropics in different color forms [21 & 35]	Shrub or small tree up to 8 m high; young branches pubescent. Leaves alternate, glossy dark green on long stout petioles up to 2.5-6 cm. Leaf-blades lanceolate-elliptic to obovate, 12-40 × 3-15 cm, acute at the apex, with flat margins, glabrous beneath. Flowers large and showy, sweetly fragrant, pentamerous, 5-7 cm diameter. Calyx-lobes c. 1 mm long. Corolla white to red, often with yellow throat; tube 13-18 mm long; lobes 25-40 mm long, obovate. Follicles 20-30 × 2-3 cm [Modified after 21].
12. *Ricinus communis* L. (1753) / Euphorbiaceae, Fig.2			
Kharwa'a, Tubshah, Tamra Gar, Rasba [22, 23 & 29].	Madinat ash-Sha'b -Al Buraiqeh district	• Widespread in Yemen e.g. in Tihama, Taiz, Al-Barh, Hagda, Shara'b, J. Sabar, Al-Hujeriyah, Ibb, Dhisufal, Assayani, Yarim, J. Summara, Al-Udeyn, Al-Qaeida, Annagd Al-Ahmer, Al-Qafr, Dhamar, Sana'a, Hajjah, Lahej, Adhala, Damt, Qataba, Al-Husha, Jihaf, Al-Azareq, Al-Hussein, Al-Mahara, Hadhramout, Socotra and Toor Al-Baha district [24 & 26].	Monoecious; perennial herb or shrub with herbaceous stems, up to 2 m; stems erect, branched, glabrous, older stems hollow. Leaves alternate, palmately 5-11 lobed, up to 20 cm long and wide, lobes acute, margin serrate, glabrous, petiole 20-30 cm. inflorescence terminal, paniculate; flowers monoeciuos with female flowers above the male flowers. Male flowers; sepals 2-5 mm, connate at base; petals absent; stamens numerous with branched filaments. Female flowers; sepals 2-5 mm, connate at base; petals absent; ovary 3-

		• Indigenous to tropical regions of Africa, probably native to Ethiopia. In the Arabian Peninsula found in Kuwait, Qatar, Saudi Arabia, UAE and Yemen including Socotra [17].	locular, covered in green soft spines. Capsule 10-18 × 10-15 mm, with soft spines, dehiscing explosively. Seeds oblong-ellipsoid, pale brown with dark brown blotches, with a white appendage at the apex [Modified after 17].
13. *Sansevieria trifasciata* Prain (1903) / Dracaenaceae, Fig.2			
Danaq Haraq [29]	Al-Kamisri nursery, Al-Sheikh Outhman district	• Singly cultivated in Yemen as a houseplant [29]. • Native to Africa (Nigeria and Zaire), naturalized elsewhere (e.g. India). Now widely cultivated throughout warmer regions of the world [36 & 37].	Perennial stemless herb with erect leaves arising from an underground rhizome. Leaves thick and fibrous, up to 1 m long, with pointed apices, the blade splotched with bands of whitish and darker green. Flowers 6-parted, with green and white perianth parts, fragrant, borne on terminal racemes. Fruit a reddish berry with 1-3 seeds (Modified after 37].
14. *Thevetia peruviana* (Pers.) K. Schum. (1895) / Apocynaceae, Fig.2			
Thevetia, Daflah Safra [21].	Al-Kamisri nursery, Al-Sheikh Outhman district	• Introduced in gardens, Yemen [22 & 24]. • It is native and common in America, from Mexico to Argentina, France and Africa, but now cultivated all over the world in the tropics and even sometimes in the subtropics [38].	Shrub or small tree 1-4 m tall, with white latex in all parts. Leaves alternate, shortly petiolate; blade very narrowly elliptic to linear, 5-15 × 0.5-1 cm, acute to acuminate at the apex. Inflorescence axillary, few-flowered. Sepals narrowly triangular, Calyx-lobes c. 8 mm long. Corolla bright yellow; tube 10-35 mm long; lobes obovate, 20-40 mm long. Stamens inserted at the apex of the narrow part of the corolla tube. Drupe obtriangular or nearly so c. 2 × 3.5 cm. Seeds 2(-4), c. 1 cm long [Modified after 21 & 38].

NA= not available

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 the main toxins present in these plants, and their toxicological mechanism of actions. On the other hand, the mechanism 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
Cardiotoxic plants			
Toxins: Cardiac glycosides **Mechanism of toxicity:** Inhibition of the cellular Na^+/K^+- ATPase, which indirectly increases the intracellular Ca^{2+} concentration in myocardial cells. Therapeutically, this both enhances cardiac inotropy (contractility) and slows the heart rate. However, excessive elevation of the intracellular Ca^{2+} 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 [39].			
Calotropis procera (Ait.) Ait. f.	Leaves, stems roots, and latex [39 & 40]	Cardiac glycosides e.g., uscharidin, calotoxin, 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. - Neurologic 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 nerine [39, 47 & 48].	- Gastrointestinal effects: nausea, vomiting, increased salivation, abdominal pain and diarrhea. Additional symptoms are irritation of the mucus 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].
Thevetia peruviana (Pers.) K. Schum.	All parts particularly the kernel of seeds [39 & 47]	Cardiac glycosides e.g., thevetin A, B, thevetoxin and peruvoside [39, 47 & 51]	
Cytotoxic plants			

<table>
<tr><td colspan="4">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 even 0.1 µg and less per kg body weight can be lethal [39, 52 & 53].</td></tr>
<tr><td>Abrus precatorius L.</td><td rowspan="2">Seed coat (Ingestion of well-chewed, broken or pulverized seeds to release the toxin from hard water impermeable seed coat) [39].</td><td>Abrin [39, 52 & 53]</td><td rowspan="2">- 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].</td></tr>
<tr><td>Ricinus communis L.</td><td>Ricin [39, 52 & 53]</td></tr>
<tr><td colspan="4">Alkylating and intercalating DNA toxins: Aristolochic acids</td></tr>
<tr><td colspan="4">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].</td></tr>
<tr><td>Aristolochia bracteolata Lam.</td><td>Herb [56-58).</td><td>Aristolochic acids [52, 56-58]</td><td>- 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 urothelial carcinomas [57 & 58].</td></tr>
<tr><td colspan="4">Mitotic inhibitors: Vinca alkaloids</td></tr>
<tr><td colspan="4">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].</td></tr>
<tr><td>Catharanthus roseus (L.) G. Don</td><td>All parts [39]</td><td>Vinblastine and vincristine [12, 39 & 59]</td><td>- Ingestion may cause initial oropharyngeal 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].</td></tr>
<tr><td colspan="4">Gastrointestinal toxic plants</td></tr>
<tr><td colspan="4">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 tannic acid-containing diets [60].
Cucurbitacins isolated and purified from plants, as well as cucurbitacin containing plant material are violent irritants of the intestinal mucosa. Colonoscopy of C. colocynthis intoxicated patients indicated mucosal erosion [61].</td></tr>
<tr><td>Caesalpinia pulcherrima (L.) Sw.</td><td>Mature seeds [39 & 62]</td><td>Tannins (protein precipitants) [39].</td><td>- Gastrointestinal effects after a latent period of 30 minutes to 6 hours: nausea, vomiting, abdominal cramping, diarrhea, and dehydration [39 & 62].</td></tr>
<tr><td>Citrullus colocynthis (L.) Schrad.</td><td>Pulp of the peeled fruit [61, 63-65]</td><td>- Cucurbitacin A, B, C, D, E, F, I, J, K, L and glycosides [64 & 66]
- Colocynthin (Cucurbitacin E 2 O-β-D-glucopyranosie) [67]</td><td>- Three cases with toxic acute colitis were presented with dysenteric diarrhea, diffuse abdominal pain. Colonoscopy indicated lesions mostly in the sigmoid and descending colonic sections, consisting of thickened edematous and inflammatory folds with exudates but without ulceration or pseudopolyp 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 colonoscopic observation was mucosal erosion [61].</td></tr>
<tr><td>Lantana camara L.</td><td>-Immature berries [39, 69 & 70].
- Unripe and ripe berries, flower, leaf, stem and seeds [71]
-Leaves by grazing livestock [39 & 69].</td><td>- Unknown for human toxicity [39].
- Animal toxicity primarily by pentacyclic triterpenoids (lantadenes), of which lantadene A is the most significant toxic principle [69 & 72].</td><td>- 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].</td></tr>
<tr><td colspan="4">Plants with irritant sap or latex</td></tr>
</table>

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], 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].			
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; leucocytosis 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 Prain	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 plants 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 calotropis 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 male 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 LD_{50} of ricin for human was estimated as 1–20 mg/kg, bw, and the LD_{50} 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 layers's mash containing castor seed husks in meal), ducks (ingestion of castor seeds), horses and cows (ingestion of flaxseed 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 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 c1, 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^6-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 toxicosis within 24 h of ingestion of the plant with all animals manifesting salivation, incoordination, staggering, recumbency, dyspnea, anorexia, 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 sever 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 dessert 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, catarrhal 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 lantadenes (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. procera* 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 nimbolide 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*, *Abrus 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.

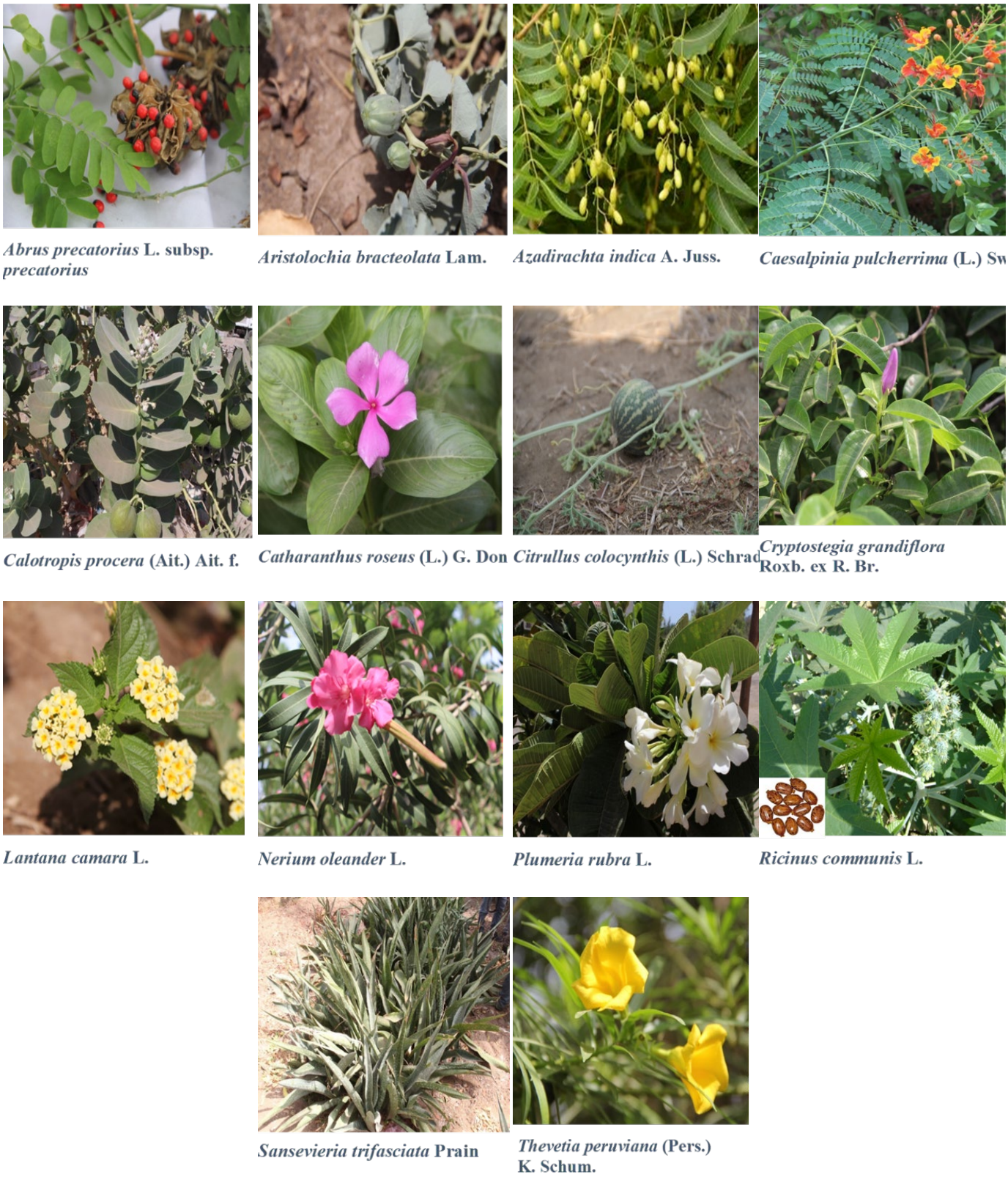

Fig 2. Poisonous plants collected from Aden Governorate

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مقالة بحثية

مراجعة لبعض النباتات السامة المنزرعة والبرية في محافظة عدن ـ اليمن

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المُلخّص

يعتبر التسمم بالنبات مشكلة صحية في كثير من البلدان، نظراً لتناول النبات عن جهل بسميته وخاصة بين الأطفال، أو عن عمد عند البالغين بغرض الاغتيال أوالانتحار أوالصيد وحتى في بعض الأحيان لعلاج أمراض مختلفة، وبالرغم من تنفيذ أنظمة مراقبة السموم في العديد من البلدانً، إلَّا أنَّ التسمم بتناول النباتات السامة برغم إمكانية الحماية والوقاية منه يظل سببًا للمرض والوفاة.

توجد بمحافظة "عدن – في الجمهورية اليمنية" العديد من أنواع النباتات السامة المزروعة أو النامية في الاماكن العامة مثل الحدائق العامة، أو كأشجار زينة على جوانب الطرقات زرعتها السلطة المحلية، ولكن لا توجد قوانين أو لوائح للوقاية من التسمم بالنبات، بالإضافة إلى غياب الدراسات العلمية حول مخاطر النباتات السامة، وعليه فمن الأهمية معرفة النباتات السامة ومخاطرها، وهو هدف هذه الدراسة لتوثيق وتوضيح الخصائص النباتية والجغرافية والسمية لأربعة عشر نوعاً نباتياً سامًا تم جمعها من مختلف مديريات محافظة عدن.

وظهر من النتائج المتحصل عليها أن العائلة الدفلية Apocynaceae تمثلت بـ 6 أنواع سامة تليها العائلة البقولية Fabaceae بنوعين من الأنواع السامة، في حين تمثلت العائلة الاريستولوخية Aristolochiaceae والقرعية Cucurbitaceae والدراسينية Dracaenaceae واللبينية Euphorbiaceae و Meliaceae والفربينية Verbenaceae بنوع واحد لكل منها، وكانت الأجزاء السامة لغالبية الأنواع النباتية التي شملتها الدراسة هي النبات الكامل، والعصارة اللبنية، والبذور والثمار.

المظاهر السريرية الرئيسية للتسمم بالنباتات هي السمية القلبية، السمية الخلوية، السمية المعدية المعوية، والتهاب الجلد والأغشية المخاطية. حيث تنتج السمية القلبية عن وجود كميات متفاوتة من السموم النباتية مثل جليكوسيدات القلب في نبات العشر *Calotropis procera* و *Cryptostegia grandiflora* والدفلة *Nerium oleander* والتيفيتيا *Thevetia peruviana*، كما تنتج السمية الخلوية عن وجود مركبات مثل توكسالبومين في *Abrus precatorius* والخروع *Ricinus communis*، وأحماض أرستولوخيك في نبات اللاعية *Aristolochia bracteolata* وقلويد الفنكا في نبات الونكا *Catharanthus roseus*، أما السمية المعدية المعوية فتنتج عن وجود مركبات مثل cucurbitacins في نبات الحدج *Citrullus colocynthis*، والتانينات في نبات *Caesalpinia pulcherrima*، ويحدث التهاب الجلد والأغشية المخاطية بسبب وجود بلورات أكسالات الكالسيوم في عصارة نبات العشر *Calotropis procera*، والبروتين القابل للذوبان في عصارة *Cryptostegia grandiflora*، علاوة على ذلك، تسببت المريمرة/النيم *Azadirachta indica* في عدد من السمية التي تُعزى جزئيًا إلى tetranortriterpenoids، بينما تنخفض السمية في نبات جلد النمر *Sansevieria trifasciata*.

يكتسب هذا العمل أهميته في تعزيز الوعي، وفي تنبيه السلطة المحلية لاتخاذ الإجراءات القانونية لمكافحة التسمم النباتي، بالإضافة إلى تزويد الأطباء بالمعلومات العلمية لتشخيص وعلاج حالات التسمم الناتجة عن بعض النباتات، ونأمل أن تحفز هذه الدراسة الباحثين لإجراء المزيد من الأبحاث حول النباتات السامة في جميع أنحاء اليمن.

الكلمات المفتاحية: عدن، منزرع، بري، النباتات السامة، السمية، اليمن.

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