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Electrocardiographic Profile of Cardiotoxic Plants and Animals

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ABSTRACT

There are numerous plant and animal toxins that can cause electrocardiogram changes, even in patients without history of cardiac pathology. Despite of extensive studies of poisons of plant and animal origin, only few articles are reported with integrated approach to their cardiac toxicity. This article presents the most common and important plant and animal poisoning which are typically involved in cardiac toxicity and with special reference to their electrocardiographic changes.

Keywords: plant poisoning, animal poisoning, cardiovascular toxicity, electrocardiographic Changes

INTRODUCTION

There are numerous plant and animal toxins that can cause cardiac manifestations. They contain alkaloids and toxic agents that rapidly and significantly affect the heart. Cardiac effects include changes in heart rate and rhythm that culminate in death. Although many commonly occurring plants are toxic, only some plants are potentially lethal when consumed in sufficient quantity. A number of plants contain cardiotoxic and neurotoxic alkaloids. Similarly some bites and envenomation exhibit similar cardiovascular toxicity.

The diagnosis of patients with an abnormal Electrocardiogram (ECG) encountered in a specific poisoning and their management can challenge experienced physicians. One must have serious knowledge of basic cardiac physiology of such poisons, in order to understand the ECG changes associated with various plant and animal poisoning. ECG is a valuable source of information in poisoned patients and has the potential to enhance and direct their care. The ECG must be an indispensable tool in the emergency department for the detection and diagnosis of cardiotoxicity. Further knowledge of the characteristic ECG changes may provide early clues to the presence of these plant and animal toxins, the prompt recognition of which can be lifesaving. It is therefore appropriate for every patient with cardiovascular disorders to discuss the hypothesis of poisoning by plants and animals. This article makes an attempt to appraise the importance of clinical toxicology by describing various cardiotoxic plants and animals and their electrocardiographic changes, and also focuses on prerequisite of special training programs and fellowship courses of the healthcare providers for handling these cases.

REVIEW

They are potentially poisonous plants and animals which contain alkaloids, toxic agents and enzymes which results in clinically significant cardiac toxicity.

1. Plants

1.1. Cardiac glycosides alkaloids: Cardenolide type

The active principles in the plants Digitalis, Nerium, Thevetia, Convallaria, Strophanthus, Cheiranthus and Asclepias act as Na+/K+ ATP ase Blockers. Digitalis species like *Digitalis lanata* and *Digitalis purpurea* contains active principles like digitoxin, digoxin, gitoxin and lanatoside C digitoxigenin. These cardenolides act by inhibiting

the Na + / K + ATPase membrane. Leaves are most poisonous. Ventricular paroxysmal tachycardia (bidirectional, ventricular fibrillation, deformation of the ST segment, lowering of the T wave, shortening of the Q-T duration, and prolongation of the P-R interval, narrow QRS interval, rapid atrial arrhythmias (paroxysmal tachycardia, flutter, and fibrillation, second degree or complete Atrioventicular block are effects of digitalis poisoning.[1, 2]

Cardiac adverse effects are typical after the ingestion of *Nerium oleander* with active principles oleandrin, nerioside, oleandroside and saponins. The clinical picture resembles digitalis toxicity, which includes life-threatening ventricular tachyarrhythmias, bradycardia, and heart block.[3, 4] In acute cardiac toxicity of *N.oleander/indicum* Poisoning electrocardiogram revealed decreased QRS-T interval, and T wave flattening or inversion, inverted P wave and prolonged PR interval, with varying degree AV blocks.[5] The cardiac toxic effects include cardiac arrhythmias (atrial and ventricular ectopic arrhythmias) and excito-conduction disturbances (sinus bradycardia, sinoatrial and atrioventricular node blocks) has also been reported in case of accidental oleandrin poisoning after the ingestion of snails that had eaten oleander leaves. [6] In yellow oleander, *Cascabela thevetia* or *Thevetia peruviana* poisoning, changes in the ST segment in ECG, and conduction blocks are noted. [7] The toxic principles are cardiac glycosides similar to digoxin. They are thevetin A and B, peruvoside, neriifolin, thevetoxin, ruvoside and theveridoside. All parts of the plant including nectar are poisonous. Inhalational poisoning has been reported when dried twigs are burnt as biomass fuel.

An electrocardiogram of *Convallaria majalis* which acts by inhibiting Na+ ,K+ - ATPase revealed third-degree AV block. [8] Active principles are Convallarin, convallamarin and Convallatoxin. *Strophanthus gratus* exhibited occurrence of toxic arrhythmias with induction of ST-T wave changes and ventricular fibrillation. Active principles are ouabain g/k/e-strophanthin. [9] Marijuana, the most commonly abused form of *Cannabis sativa* develops sinus tachycardia, QT interval prolongation, ST elevation and T wave changes like inverted T wave. [10, 11] The cannabinoid delta 9-tetrahidrocannabinol (THC) is the principal psychoactive constituent of *C.sativa* (marijuana consists of the leaves and flowering parts of the plant). In *Cheiranthus cheiri*, the active principles are cheirotoxin, cheirosid A and other cardenolides, glucosinolates (glucocheirolin) causes inverted T wave and bradycardia. [12] In milkweed (*Asclepias syriaca*) poisoning, sinus bradycardia was reported.[13]

Antiaris toxicaria with cardioactive steroids antiarin results in sinus tachycardia, T wave inversion and ST segment elevation. [14] *Calotropis procera* causes bradycardia and significant increase in the force of ventricular contraction, and increase in T-wave amplitude.[15] Milky wax exuding from the stem contains cardiac glycosides (calotropin, uscharin) and fatty acids. In *Cerbera manghas* poisoning, where the active principle is cerberin, the ECG findings showed non-specific ST-T changes, and 1st, 2nd, 3rd degree heart block and sinus bradycardia. Atrial fibrillation was the tachyarrythmias.[16, 17] *Cerbera odollum* poisoning results in atrioventricular block, ST segment depression, shorten DT interval, peaked T waves.[18] *Cryptostegia grandiflora* poisoning results in bradycardia. Cardiac glycosides inhibit the cellular Na+/K+-ATPase which enhances cardiac inotropy (contractility) and slows the heart rate.[19, 20]

1.2. Cardiac glycosides alkaloids: Bufadienolide type

Urginea maritima or *Drimia maritima* plants producing cardiac glycosides bufadienolide Glucoscillarene A, proscillaridine A, scillarene A, scilliglaucoside and scilliphaeoside act as Na+/K+ ATP ase Blockers. It's toxicity causes bradycardia and complete atrio-ventricular heart block.[21] Depressed ST segment, increased amplitude of QRS wave and T wave is observed in *Moraea polystachya* poisoning.[22] Cardiac glycoside poisoning caused by bufadienolides hellibrigenin 3-acetate in *Kalanchoe lanceolata* demonstrates arrhythmias, widening of QRS wave, ST elevation, increased PR interval and AV blocks. [23] Tachycardia, ST elevation and complete AV dissociation was noted in *Thesium lineatum* poisoning.[24] *Tylecodon wallichii* poisoning detected slight increases in the amplitudes of the QRS and T-waves, QT segment depression and a depression of the ST-segment.[25, 26]

1.3. Other alkaloids

In Aconitum species like *Aconitum napellus*, all organs of the plant, but especially the roots and seeds contain diterpene alkaloids, aconitine and mesaconitine. It has Na+ channel binding properties. In poisoning ECG revealed decreased PR interval, cardiac dysarhythmias, frequent multifocal ventricular ectopics, which later turned to short runs of ventricular tachycardia i.e. polymorphic ventricular tachycardia like ventricular fibrilation, premature ventricular contraction, accelerated idioventricular rhythm and non-sustained ventricular tachycardia, including episodes of prolonged bidirectional ventricular tachycardia.[27-31]

In Taxus species, except the aril, all parts of the plant are poisonous and contain a mixture of alkaloids and complex structure pseudoalcaloïdes. The main cardiotoxic substances are taxol, taxines A and especially B taxines, diterpene taxane core substances that are antagonists of the calcium and sodium channels and whose properties are similar to those of the class of antiarrhythmic 1. *Taxus Baccata* poisoning causes ventricular fibrillation.[32] *Taxus cuspidata*

causes ST-segment elevation, QRS prolongation and ventricular tachycardia.[33] *Colchicum autumnale* poisoning, the electrocardiogram showed only diffuse nonspecific ST changes.[34] The main compound is crocus colchicines. Poisonous parts are leaves and seeds. Cardiovascular effects of *Adonis aestivalis* plant poisoning containing adonitoxin inhibit Na+ ,K+ - ATPase. It showed various ECG abnormalities like sinus arrhythmia, shortened and depressed S-T interval, and absence of P wave and flattened or inverted T wave. In addition, ventricular arrhythmias, bradyarrhythmias, atrioventricular block, ventricular premature beats, ventricular tachycardia and ventricular fibrillation. [35, 36] Acute hellebore (*Veratrum album*) contains many alkaloids protovératrines A and B, germerine and cyclopamine activate Na+ - channels. The main pathophysiological mechanism of poisoning is related to the increase in the permeability of sodium channels. Poisoning revealed sinus bradycardia, shortening of the PQ interval and QTc interval, and depression of ST-segment, low and (or) pointed T waves. [37, 38] The plant *Veratrum viride* poisoning causes T-wave inversions and second degree Atrioventricular block.[39] Myocardiopathy with nonspecific ECG changes nonspecific ST segment, and T-wave changes or atrial or ventricular extrasystoles were reported in *Argemone mexicana* Poisoning. [40]

The cardiotoxicity of coffee senna (*Senna occidentalis*) which contains sympathomimetic toxins showed reduction in RR interval.[41, 42] Electrocardiogram showed sinus bradycardia caused by the *Rhododendron japonicum* poisoning.[43] *Cleistanthus collinus* poisoning containing diphyllin glycosides manifests ECG abnormalities as nonspecific ST-T changes like ST segment depression, Flat P wave, QTc prolongation, prominent U waves.[44, 45] The toxic principles are cleistanthin A, cleistanthin B, diphyllin, saponin, tannin and oduvin. All parts of the plant are toxic.

Plants containing belladonna alkaloids exhibiting anticholinergic toxicity like *Datura stramonium*, *Atropa belladonna* and Hyoscamus niger caused sinus tachycardia and prolonged QT interval.[46] All parts of the plant contain tropane alkaloids such as atropine, hyoscyamine and scopolamine. Oral ingestion of crude opium from the plant, *Papaver somniferum* results in tachycardia, cardiac conduction defects. ST-T changes and presence of Q-wave are also reported in opium abusers.[47] In Nicotiana tabacum containing cholinomimetic toxins like nicotine and nornicotine, smoking cause shortening of duration of QRS complex and T-P interval, abnormal Q/QS wave patterns, T wave abnormalities and development of ST segment depression were noted.[48-50]

In ipecac poisoning from the dried rhizome and roots of *Carapichea ipecacuanha*, ECG abnormalities have been reported. The ECG showed diffuse T-wave inversion and QT-intend prolongation, premature atrial complexes, supraventricular and ventricular tachycardias, ventricular fibrillation, and PR-interval prolongation. [51, 52] In a cardiotoxicity study of the aqueous extract of corn silk (maize) in rats, the results showed significant changes in the heart rate, P-wave duration, QT interval and QTc. [53]

2. Fungus

Amanita phalloides contain toxic principles like cyclopeptides, amatoxins, amantine, phalloidin and phallotoxins. They exhibit anticholinergic toxicity. The most common ECG abnormalities in the patients with mushroom poisoning are P were sinus tachycardia, sinus arrhythmia, ST/T inversion, 1st degree AV block and QT prolongation.[54] In *Russula subnigricans* mushroom poisoning presents with ventricular tachycardia, sinus tachycardia with QT prolongation.[55]

3. Arthropod

In scorpion poisoning, RST segment and T waves are most frequently affected. Arrowhead tented T wave look indicates acute injury, while tent shaped indicates recovery. Toxins act by binding to the Na+ channels. Early myocardial infarction like pattern, atrial arrhythmias, non-sustained ventricular tachycardia and conduction defect due to injury to the conducting system. PQRST or T waves alternans suggest serious myocardial injury. Prolonged QTc and LV dysfunction, conduction defect, T wave inversion, Low voltage, wide QRS complex, tachycardia, hemiblock and mark ST segment depression carries bad prognosis.[56, 57] Other cardiac effects are sinus tachycardia, atrial ectopic beat, bradycardia, and ventricular ectopic beat are also seen.[58] Chilopoda (centipede) biting developed bradycardia and ST-T wave changes.[59]

Black widow spider envenomation showed ST-segment elevation and accompanying augmentation in T-wave amplitude and prolonged QT interval. [60, 61] Black widow spider bites involves an excess of catecholamines, and cholinomimetic toxins. Electrocardiographic changes following bee-sting anaphylaxis produced widespread T wave inversion and ST segment changes.[62] In a study of Effects of bee (*Apis mellifera*) venom on the electrocardiogram three types of changes were noted like marked shifts in the ST-T segments, various degrees of atrioventricular block and severe ventricular arrhythmias.[63] Hymenoptera stings, histamine plays a role in pathogenesis. ECG demonstrated demonstrated ventricular tachycardia, concave ST-segment elevation, T-wave inversion, QTc prolongation and abnormal Q-wave in honey bee sting.[64, 65] Wasp sting showed inverted T waves.[66]

4. Reptiles
In sea snake poisoning, ECG changes showed hyperkalaemic arise in T waves, Dominant R waves and widening of the angle between S and ST segment.[67] In viperine snake bite, common electrocardiographic changes were sinus tachycardia, sinus arrhythmia, sinus bradycardia, tall T-wave, non-specific ST-T changes like ST elevation, and T-wave inversion and atrioventricular block.[68, 69] Second-degree heart block was associated with envenomation by *Vipera berus*.[70] ECG changes (usually in the ST segment or T-wave) prominent in severe cobra bites (Naja sp.) and some viper bites- *V. berus* is also reported.[71] Septal T wave inversion and bradycardias, including atrioventricular block, were the commonest abnormalities in taipan snake bite of elapid family.[72]

5. Amphibian
Bufo toad producing cardiac glycosides, bufadienolide has Na+/K+ ATP ase Blockers. Toad evenomation from Rhinella species or *Bufo melanosticus* or *Bufo* marinus causes wide QRS duration, prolonged PR interval and low QRS amplitude.[73] Bufalin, marinobufagenin, Telocinobufagin are cardioactive steroids.

6. Fish
Jellyfish envenoming reported ECG changes included atrial and ventricular ectopy, atrioventricular conduction defects, ST-segment elevation, T-wave abnormalities, and nonsustained ventricular tachycardia. [74] Ciguatera fish and Puffer fish causes Bradycardia and arrhythmias.[75] The ECG showed sinus tachycardia , widespread ST segment displacement (depression or elevation) with T wave changes in scombroid poisoning.[76]

7. Anemone
Sea anemone (*Anemonia sulcata*) toxicity casues an increase of the QR-voltage, delayed QTintervals and enlarged and broadened T-waves. [77]

CONCLUSION

ECG as a valuable source of information in poisoned patients has the potential to enhance and direct their care and to prevent further morbidity and mortality. Further more research is required to determine the detailed mechanism of their electrocardiographic changes. All the healthcare providers should have knowledge about the commonly occurring and potentially lethal poisonous plants and animals of the region. An ECG should be examined extremely early in the initial evaluation of most poisoned patients. Emergency physicians should have the proficiency to deal with exposure to lethal poison, bites and envenomation and their management. As most of them do not have antidotes and treatment is mainly supportive, early recognition and management plays an important role in reducing morbidity and mortality. A blood sample to assess measurement of specific alkaloids is necessary in order to confirm the diagnosis and to advance in the study of a clinicopathologic correlation for cases of poisoning. Further clinical toxicology knowledge is also lacking among most healthcare providers. Short training courses of undergraduates and postgraduates as well as fellowship in clinical toxicology with special reference to cardiotoxic plants and animals will be helpful in improving the knowledge and the patient care significantly.

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The authors declare that there is no conflict of interest.

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