Review Article

A Review on Arrhythmias due to Snakebite

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Abstract

An estimated 1.8 to 2.5 million venomous snakebites occur worldwide each year and result in at least 100,000 to 125,000 deaths. It is a public health problem particularly in tropical and subtropical regions. It is known that 4 species of snakes (Viperidae) Elapidae, Colubridae, and Lamprophiidae are determined to cause harm to humans. Envenoming by these snake species may cause myocardial damage and electrocardiographic deterioration. Common manifestations of cardiovascular toxicity are acute myocardial infarction, electrocardiogram abnormalities and arrhythmias. In this narrative review, we aimed to conduct a comprehensive analysis of cardiovascular toxicity due to snake bite.

Keywords: Snakebite, envenomation, arrhytmia

Introduction

Snake envenoming is a common medical emergency particularly in tropical countries [(1)]. Every year, approximately 2.5 million cases of snake bite occur and 81,000-138,000 deaths occur due to snake bites [(2)]. Most of the snake bites result in local symptomsincluding bleeding, edema, and ecchymosis, However, they may also cause systemic reactions such as acute kidney injury, rhabdomyolysis, stroke, coagulopathy, thrombocytopenia, neurotoxicity and cardiotoxicity. Up to now, 4 species of snakes are determined to cause harm to humans. These are Viperidae, Elapidae, Colubridae, and Lamprophiidae. Each species has potential to cause cardiovascular toxicity [(3)].

Envenoming by certain snake species may cause myocardial damage and electrocardiographic deterioration [(4)]. Common manifestations of cardiovascular toxicity are acute myocardial infarction, electrocardiogram abnormalities and arrhythmias [(5)]. Atherothrombotic disease may occur as a result of coagulation disorders which may lead to acute coronary syndromes, left ventricular function impairment, and arrythmia. The most common rhythm disturbances are atrial fibrillation, ventricular fibrillation, ventricular tachycardia, atrioventricular block, and non-specific ECG abnormalities (ventricular extrasystoles, prolonged QTc intervals, left bundle branch block, T-wave inversion, T-wave flattening, tall T-waves, and presence of U waves). Bradyarrhythmia is a less common manifestation. However, atrioventricular block or sinus node dysfunction (sinus node arrest) may also be observed [(3)].

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In snake envenomation; besides acute manifestations, subacute and late-onset complications may be determined. Thus, observation period and ECG follow-up period should be longer than typical cardiac patients [3]. Treatment modality for snake envenomation should be selected on a case-specific basis. When cardiac arrest due to ventricular arrhythmias occur, IV continous amiodarone should be administered. Also, cardiopulmonary resuscitation and intubation may be required. In case myocardial infarction emerges, standart procedure for revascularization should be initiated. If thrombocytopenia and coagulopathy is determined, the risk for bleeding increases and thus, the use of thrombolytic agents may be limited. If myocarditis occurs, anti-inflammatory medications should be initiated [3].In a study, 52 patients with snake envenomation were investigated in terms of treatment modality. Better results were obtained in the group that received both hydrocortisone and chlorpheniramine when compared to single hydrocortisone group and placebo group. Additionally in group that received both hydrocortisone and chlorpheniramine, less adverse reactions were observed[6].

In this narrative review, we aimed to perform a comprehensive analysis of snake envenomation literature and create awareness on optential cardiovascular effects emerge in patients with snake bite.

Action Mechanism of Snake Venom

Snake venom has both presynaptic and postsynaptic neurotoxins [1]. In certain species, venom is produced in specific glands and secreted to their saliva. The venom is a mixture of protein toxins and various enzymes [7].

Potential mechanisms of venom toxicity are increased capillary permeability, endothelial damage, and disruption of the coagulation system leading to hemorrhagic activity and edema. These mechanisms are executed with proteins and enzymes in the venom. However, myotoxicity, myonecrosis, and cardiotoxicity due to venom are rare [8].

There are two classes of venom toxins that may cause myotoxicity; phospholipases and L-aminoacidoxidases [8]. The main components of venoms are phospholipase A2, three-finger peptides, serine proteases and metaaloproteinases. Phospholipase is a compound found in all snake venom. Phospholipase is cardiotoxic and can cause hypotension when combined with other components such asnatriuretic peptides, bradykinin potentiating peptides, vascular endothelial growth factors, snake venom metalloproteinases, and snake venom serine-protease [3].

Even though mechanism of cardiotoxicity is in snake envenomation is not clear, there are some hypotheses that were proposed. Some of these are as follows: [1] direct cytotoxic effect, [2] toxin-induced arrhythmia, [3] acute coronary syndrome secondary to coagulopathy, [4] toxin-mediated coronaryspasm, [5] hyperkalemia secondary to acute renal failure and [6] inflammatory processes secondary to venom-induced hypersensitivity [8]. It was also reported that fatal cardiac rhythm changes ranging from bradycardia to ventricular tachycardia/fibrillation may develop in a short period following snakebite [9].

Venom components directly may cause hypotension. Also, hypotension may occur as a result of implications of envenomation (coronary ischemia or arrhythmias with hemodynamic instability) [3].

Experimental studies have shown that venom induces alteration of genes expressed in the heart, which is responsible for the mediators of immune response, apoptosis, ion transport, signal transduction, hypotension, energy metabolism and electron transport [10].

Primary action site of snake venom is known to be neuromuscular junction. Hence, it causes flaccid paralysis. Multiple toxin types in a single venom presents various actions [2]

Materials and Methods

We conducted a narrative review entering keywords “snakebite” and “arrhythmia” into the scientific database, Pubmed©. If the full text of the

article was not available and the researchers failed to retrieve it, then the article has been excluded if the abstracts were nonexplanatory. During the database search, we excluded studies, which did not relate with the objective by reading the title and abstract. Studies that were not published in English, studies without explanatory abstracts and studies that do not focus on cardiovascular effects of snake envenomation were excluded. Also, animal studies relevant to snake envenomation were excluded. It was observed that the majority of the studies in the literature consist of case reports on snake bites. Two reviewers conducted independent screening and data extraction. First, the reviewers independently screened titles and abstracts of the returned articles to decide if they met the inclusion criteria.

Results

A total of 64 articles were determined relevant to the topic. It was observed that most of the articles on snake envenomation in literature consisted of case reports. Twelve of the publications were excluded from the study because they were about dog exposure to snakebite. Two of them were horse exposure, 1 of them was swine exposure and 1 was rat exposure. Thus, when these articles were excluded, a total of 48 articles were included into the study.

Discussion

Snakebite is a common problem in rural areas of Asia[11]. Snake bites rarely cause cardiac manifestations and when present, main cause is vipers [1].

Cardiotoxicity is a rare manifestation of Russell's Viper's (Daboia russelii) bite.Russell's Viper toxicities are mainly bleeding disorder, nephrotoxicity, neuromuscular paralysis and respiratory failure[1,11]. Cardiac rhythm abnormalities include sinus bradycardia, sinus tachycardia, an atrioventricular conduction abnormality, and ventricular arrhythmia [12].

In a report, a healthy man presenting with a Russell's viper bite developed cardiac arrest from ventricular tachycardia. He was successfully treated by cardiovertion and amiodarone. Subsequently, antivenom for Russell's Viper was administered for correction of a bleeding disorder [11].In another report, a 35-year-old male patient presented with snakebite on his left ankle in 2 hours. The dead snake was detected and identified as a viper. The ECG of the patient revealed sinus node dysfunction characterized by sinus arrest with junctional escape rhythm and retrograde P waves with a rate of 40 beats per minute. It is probable that the venom alters electrophysiological features of cardiac cell membrane by effecting impulse generation and conduction [7].

A 14-year-old female patient was bitten by Russell's viper. The patient presented with mucosal bleeding, ptosis, and muscle weakness. A coagulopathy was determined in the laboratory analysis. The initial ECG of the patient revealed sinus tachycardia. Then, she developed bradycardia and her ECG revealed a delayed onset sinus node dysfunction[12].

Three young farmers died 15, 52 and 36 h after being bitten by Russell's vipers were presented byThan-Than et al. Clinical features included local swelling, spontaneous systemic bleeding, defibrination, shock, cardiac arrhythmia, hypoglycemia, coma and oliguria [13]

In another report a 60-year-old male presented to the ED following Russell's viper bite on the left ankle. His heart was irregularly irregular with a rate of 146 beats/minute. His ECG revealed atrial fibrillation with fast ventricular rate of 126 beats/minute. An echocardiography was performed and it was normal without wall motion abnormality. Russell's viper venom contains factor V and factor X activators within. These factors induce coagulopathy with their procoagulant potential. Rarely, Russell's viper venom causes myocardial infarction and atrial fibrillation [12].

In a study on patients with European adder (Vipera berus) bites were retrospectively analyzed. Of 26 patients, 14 patients presented systemic symptoms that require antivenom. The cardiac dysrhythmias

observed in 2 patients (ventricular arrhythmias) on admission and did not require treatment [14].

Vipera berus envenomation is well-known with its morbidity while its mortality rate is low. A 69-year-old male patient was bitten by V. Berus on the right thumb and presented to the Emergency Department 30 minutes after being bitten. In his medical history, it was determined that he had heart attack 10 years previously. His initial complaints were abdominal pain and vomiting. As he presented to the Emergency Department, he developed Diarrhoea. An ECG was performed and it revealed showed intermittent 2: 1 second degree heart block [15]. In another study with V. berus, 76 patients were determined in 7 years. In concordance, electrocardiographic finding of cardiac effect (T-wave inversion) was seen in 9% of the 54 patients in whom an electrocardiography had been performed [16].

A male patient at the age of 56 presented with severe coagulopathy following Vipera ammodytes ammodytes envenomation. The patient was hypotensive and a tachycardia was determined in the ECG [17].

In a study with 30 patients with snake bite, it was determined that majority of the patinets were bitten by Viperine snake and the rest by elapide snake. In 25% of cases with Viper bite, cardiotoxicity was determined. The most common cardiac manifestations were disturbance in heart rate, rhythm disturbance, tachycardia and bradycardia, respectively. Also, evidence for myocardial ischemia was detected in 10% of the patients. Another significant finding was that 10% of the patients died and all of these patients had abnormal electrocardiogram [18].

A 42-year-old healthy male presented to the Emergency Department due to hump-nosed pit viper (Hypnale hypnale) bite. The patient was admitted to the Emergency Department with cardiac arrest and following CPR the patient developed atrial fibrillation on ECG. Atrial fibrillation reverted to normal rhythm following synchronized electrical cardioversion was applied. However, the patient died due to multi organ failure in 16th day of follow-up [19].

A 49-year-old male patient with hump-nosed pit viper (Hypnale hypnale) experienced cardiac arrest 30 minutes after bite. Hump-nosed pit viper, up to then, was known only with its local effects. An ECG was performed and it revealed ST elevation in leads II, III and aVF with reciprocal changes in leads I and aVL, suggestive of an inferior wall infarction. Also, atrial fibrilllation with a heart rate of 132 per minute was determined [20].

In a report, a 14-year-old boy presented to a hospital mild envenomation symptoms after a spitting cobra (Naja sumatrana) bite. Premature multiple ventricular complexes were determined on his ECG. On the physical exam, ptozis as a sign of paralytic envenomation was determined. After antivenom administration, ptozis and ECG abnormalities have improved [21].

A 23 year-old male patient was bitten by a cobra. He had complaints of nausea and vomiting. On his examination, hypotension and tachycardia were determined. On ECG, infrequent ventricular ectopics that progressed to ventricular bigeminy was detected. Sinus rhythm was obtained following monovalent antivenom against Naja kaouthia venom was administered [22].

Agarwal et al. presented a case of a 26 -year -old male farmer was admitted to the hospital with complaints of breathlessness six hours after being bitten by a snake while working in the fields. The dead snake was brought in and identified as Bungarus caeruleus. On admission, the patient had repeated bradycardia and tachycardia episodes. Patient was also sweating despite sedation and maintenance of normoxemia. Patient developed pulmonary edema due to snakebite. This clinical finding was found to be associated with myocarditis due to venom. It was emphasized that neurotoxic snake venoms may cause cardiotoxicity. In order to prevent and complications like pulmonary edema, recognition of cardiac involvementis essential in these patients [1].

In a case, a healthy 39-year-old patient was bitten by Heloderma suspectum. The patient developed

tongue, lip swelling and stridor. When he was tranferred to Intensive Care Unit, he was hypotensive with hypokalemia. He developed a state of shock with atrial fibrillation and a electrical cardioversion was performed. Following cardioversion, patient's ECG revealed sinus tachycardia at 130 bpm, associated with diffuse ST depression in the majority of leads, particularly in aVR. In the follow-up, ECG returned to normal aside from an anteroseptal 1–2mm J point elevation. The elevation of troponin suggested non-ST elevation myocardial infarction. Even though it was not clear if myocardial infarction was strongly related to snake venom, the authors emphasized three possibilities following snake bite: angioedema, fluid loss associated with hypokalemia and metabolic acidosis,and cardiac disorders simulatingischemia[23].

In another study, 33 patients with snake bite were investigated. Sixteen of them were bitten by kratis and 14 of them manifested local swelling at the bite site and haemorrhagic manifestations. Three of the patients were bitten by cobras. However, in this study, authors do not focus on cardiovascular effects of the envenomation[24].

Khaldi et al. reported a 20-year-old male who developed atrial fibrillation following D.palaestinae snakebite. The patient was administered polyvalent anti-venom in the emergency department and relief in his pain was obtained. Then, he was transferred to the Intensive Care Unit for close monitorization. On the second day, the patient developed palpitations and shortness of breath. The patient did not mention a chest pain. His heart rate was 155 bpm, blood pressure130/65 mmHg, O2 saturation 95%, temperature 36.6. After appropriate consultations and medications, the patient was discharged with full recovery in three days[8].

In a study, 65 patients with snake bite were investigated in a 3-year period in terms of cardiovascular effects. Cardiovascular effect was defined as occurence of at least one of the followings: myocardial injury, shock, ventricular dysrhythmia, or cardiac arrest. In 9 patients, cardiovascular effects of envenomation were determined. Underlying cardiac problems were more common in these patients. None of the patients developed ventricular dysrhythmia or cardiac arrest[25].

A prospective study investigated electro-cardiographic changes in patients with snake bite inPapua New Guinea. Sixty-nine patients were involved into the study and 36 of these patients were bitten by taipan (Oxyuranus scutellatus), 2 were bitten by death adders (Acanthophis sp.) and 1 was bitten by the brown snake (Pseudonaja textilis). The most common cardiac abnormalities determined were septal T wave inversion and bradycardias, including atrioventricular block. Even though there were no patients with hemodynamic deterioration, 2 patients presented troponin elevation revealing myocardial damage[4].

23-year-old man bitten from finger was admitted to a hospital due to brown snake bite. On ECG; Intraventricular conduction delay, asymptomatic QRS and QT prolongation were determined. Following antivenom and supportive treatment, his symptoms resolved in 11 days (26).

In a report, a 73-year-old patient with co-morbidities bitten to his lower limb by Crotalinae was presented. Following hospitalized in the Intensive Care Unit, his electrocardiogram revealed new-onset atrial fibrillation. Amiodorone treatment was initiated and a sinus rhythm was obtained. A transthoracic echocardiogram revealed mild concentric left ventricular hypertrophy and an ejection fraction of 72%. The patient was discharged with baseline first-degree atrioventricular block as a sequela [5].

Of 10 patients with puff-adder (Bitis arietans) bite, 6 showed local signs and 4 showed systemic manifestations. Cardiovascular effects were hypotension, and bradycardia. Two patients died due to circulatory collapse and renal failure. Antivenom and supportive treatment helped with blood pressure in hypotensive patients[27]. The venomous snakes, their cardiac effects and treatment methods are summarized in Table 1.

Table 1. Commonly Seen Venomous Snakes Involved in the Review

Snake Species	Cardiac Involvement (Reference Number)	Treatment (Reference Number)
Russell's Viper's (Daboia russelii)	Sinus bradycardia, sinus tachycardia, an atrioventricular conduction abnormality, and ventricular arrhythmia (12), cardiac arrest from ventricular tachycardia (11), sinus node dysfunction characterized by sinus arrest with junctional escape rhythm and retrograde P waves with bradycardia (7), bradycardia and her ECG revealed a delayed onset sinus node dysfunction following tachycardia (12), cardiac arrhythmia and coma (13), atrial fibrillation with fast ventricular rate, myocardial infarction and atrial fibrillation (18).	Cardiovertion and amiodarone(11), anti-venom (7,11,12)
European adder (Vipera berus)	Ventricular arrhythmias (14), intermittent 2: 1 second degree heart block (15), T-wave inversion (16)	Did not require treatment (14), anti-venom, antibiotics when cellulitis occurs (15)antivenoms, corticosteroids (controversial) (16)
Vipera ammodytes ammodytes	Tachycardia and hypotension (17)	Intravenous fluids, antihistamines, antibiotics, glucocorticosteroids, antivenom (17)
Hump-nosed pit viper (Hypnale hypnale)	Atrial fibrillation, cardiac arrest (19),inferior wall infarction, atrial fibrillation (20)	Synchronized electrical cardioversion (19)
Spitting cobra (Naja sumatrana)	Premature multiple ventricular complexes (21), infrequent ventricular ectopics that progressed to ventricular bigeminy (22), no cardiac involvement (24)	Anti-venom (21,22)
Bungarus caeruleus	Bradycardia and tachycardia episodes, myocarditis (1)	Antivenom, mechanical ventilation when needed (1)
Heloderma suspectum	Atrial fibrillation, hypotension, non-ST elevation myocardial infarction, shock (23)	Electrical cardioversion (23)
Daboiapalestinae	Atrial fibrillation (8)	Anti-venom (8)
Taipan (Oxyuranus scutellatus)	Septal T wave inversion and bradycardias, including atrioventricular block (4)	Anti-venom (4)
Death adders (Acanthophis sp.)	Septal T wave inversion and bradycardias, including atrioventricular block (4)	Anti-venom and supportive treatment (26)
Brown snake (Pseudonaja textilis)	Septal T wave inversion and bradycardias, including atrioventricular block (4), intraventricular conduction delay, asymptomatic QRS and QT prolongation (26)	Anti-venom and supportive treatment (26)
Crotalinae	Atrial fibrillation, concentric left ventricular hypertrophy, first-degree atrioventricular block (5)	Amiodorone treatment (5)
Puff-adder (Bitis arietans)	Hypotension, bradycardia,circulatory collapse (27).	Anti-venom and supportive treatment (27)

Limitations

There are also some limitations of our article. Firstly, the fact that the majority of the articles in the literature consist of case reports makes it difficult to establish a certain standard for the treatment of snake bites.Another reason why such a standard cannot be established is that snake species vary greatly depending on geographical regions and the spread of snake speciesaround the world for commercial purposes is increasing.Additionally, another limitation of our study, as in all other review articles on snake bites, is the possibility that there are still unidentified potentially venomous snakes and their effects on the cardiovascular system are unknown.

Conclusion

In summary, cardiovascular toxicity secondary to snake bite should be treated with antivenom therapy combined with hemodynamic support and specific cardiovascular interventions. Cardiac involvement in snake bite may complicate clinical status and alter the treatment course. If abnormalities are detected in ECG, the patients should be monitored serial ECGs and cardiac enzymes. As people interact with wildlife more, their chances of encountering poisonous snakes will increase.Clinicians' awareness of snake envenomation and timely intervention will contribute significantly to the reduction of morbidity and mortality.As a future recommendation, every snakebite should be assumed to be dangerous and poisonous on cardiovascular system as well as other systems until proven otherwise, and ECG should be considered a priority modality in these patients. It should be known that the initial ECG is not sufficient and serial ECGs should be taken to monitor potential ECG changes.Long-term follow-up of patients should be provided for long-term complications.The cases collected from all over the World can be compiled in an international database and the correlation between snake species and symptoms can be made easily accessible.Artificial intelligence (AI) can also be used to create these databases. If a photograph of the snake is available, it can be uploaded to an AI application, and clinicians can be informed about po ssible symptoms depending on the snake species.Additionally, ensuring collaborative working conditions between international laboratories, a method proven successful during the COVID-19 pandemic, will be beneficial in preventing deaths due to snake bites.

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