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Review

Cardiac Involvement in Scorpion Envenomation: A review of Literature

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Abstract

Scorpion envenomation (SE) causes cardiac complications. Pubmed, Scielo, Embase, and google scholar were searched using the keywords scorpion: cardiac, heart, arrhythmia, electrocardiograph, and myocarditis in the abstract or text. 115 were selected. Cardiotoxicity can occur within 2 hours of SE and include hyper/hypotension, arrhythmias, myocarditis, and heart failure. The postulated mechanisms are autonomic storm, inflammation, direct venom toxicity, and metabolic derangement. Haematological and biochemical derangement suggests increased severity. Cardiac biomarkers, electrocardiography, and transthoracic echocardiography helps detect cardiotoxicity and guide management. Early use of antivenom and/or alpha-adrenergic blockade may prevent or reverse cardiotoxicity. Hypertension is best managed by alpha-adrenergic blockers. Arrhythmias are usually transient. Cardiovascular complications of SE are associated with morbidity and mortality. A clear consensus on the indication and utilization of antivenom administration in cardiac involvement SE is needed.

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Keywords: *Scorpion, cardiotoxic, venom, toxins, myocarditis, heart failure*.

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Introduction

Scorpion species are diverse, but only a few dozen lead to medically significant human envenomation, with most belonging to the Buthidae family.[1] Their geographic distribution leaves a third of the world's population at risk, more so in the tropics and subtropics. This results in developing nations being more at risk.[2] with the burden of human scorpion envenomation (SE) annually exceeding a million cases worldwide.[3,4]

SE manifests with local symptoms but systemic involvement may occur in 1/3. However cardiac manifestation has been documented quite commonly in studies with clinical presentations and abnormal investigational findings favoring cardiac insult being noted to be as high as 79.1%. The presence of cardiac involvement increases morbidity and inappropriate or delayed management worsens outcomes.[5]

SE can be a medical emergency and a better understanding of its health implications and management is required.[6] This review of the current literature on scorpion envenomation aims to describe the incidence of cardiac involvement after scorpion envenomation, its pathogenesis, cardiac manifestation, management, and outcomes.

Methods

Pubmed, Scielo, Embase, and Google Scholar were searched for papers containing the keyword scorpion combined with any of the following: cardiac, heart, arrhythmia, ECG, and myocarditis in either the abstract or text. The search was restricted to articles published over the past four decades. There were 1222 abstracts found during the combined search. Endnote X9 was used to filter the papers. Related references were also included. Two authors independently reviewed the abstracts and 115 papers were selected. The review aimed to answer the following questions

1) What is the pathophysiology of the cardiovascular toxicity of SE?
2) What are the cardiac manifestations of SE?
3) How can cardiac complications of SE be managed?

1. Pathogenesis of Scorpion venom induced cardiovascular toxicity

Venom composition

Scorpion venom is a mixture of water-soluble components including mucopolysaccharides, hyaluronidase, phospholipase, serotonin, histamine, enzyme inhibitors, and proteins namely neurotoxic peptides.[7] However, there is speculation whether it can also include toxins that are cardiac-specific in the form of cardio-toxins due to significant cardiovascular system involvement.[8,9] Some species have acetylcholine and noradrenaline, but this is unlikely to cause direct cardiovascular toxicity and mainly cause local and peripheral effects.[10]

Pathophysiology of cardiovascular dysfunction

Cardiac involvement after envenomation may be the result of multiple pathways. (Figure 1).

Autonomic storm theory

Effects of venom on sodium & potassium voltage-gated channels have diverse effects leading to catecholamine release in the adrenal glands and release of acetylcholine in the postganglionic parasympathetic neurons.[7] The rise in catecholamines can be 30-40 times the normal range and is proportionate to the severity of envenomation.[11] Catecholamine response is usually primary, with a rise in adrenaline, noradrenaline, sodium levels, activation of RAS and a fall in potassium and calcium levels. Severe envenomation causes an inverse pattern of a cholinergic response with a rise in bradykinin, NO, and kalikrein.[12,13]

Alpha & beta-adrenoceptor stimulation leads to vasoconstriction, increased afterload along, and a positive chronotropic and inotropic effect. This manifests as hypertension, tachycardia, arrhythmia, and diaphoresis.[14] The increased workload results in biventricular dysfunction. The catecholamine surge also may lead to adrenergic myocarditis. The autonomic storm theory is strengthened by the failure of re-envenomation to re-elicit a catecholamine surge or hemodynamic alteration equivalent to the first envenomation.[15]

Immune response and inflammation

Activation of classical and alternate complement

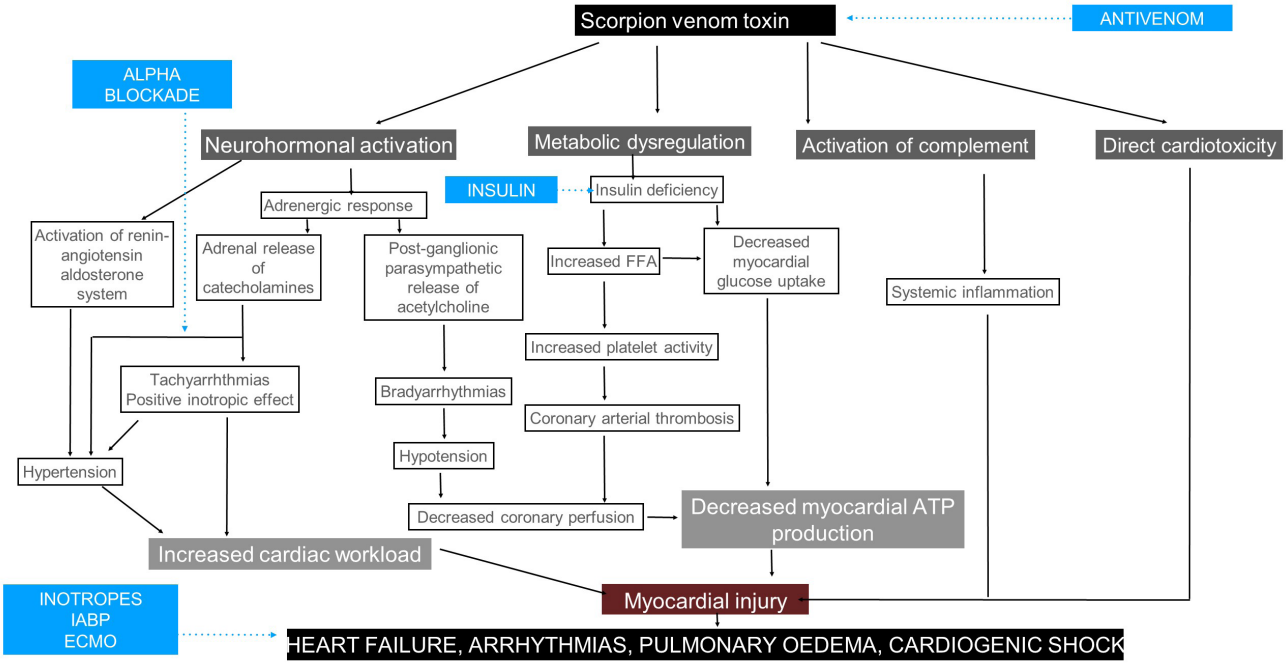

Figure 1. Demonstrates the different mechanisms by which scorpion envenomation causes myocardial injury and cardiac dysfunction. (FFA-Free fatty acid, ATP- Adenosine Triphosphate).

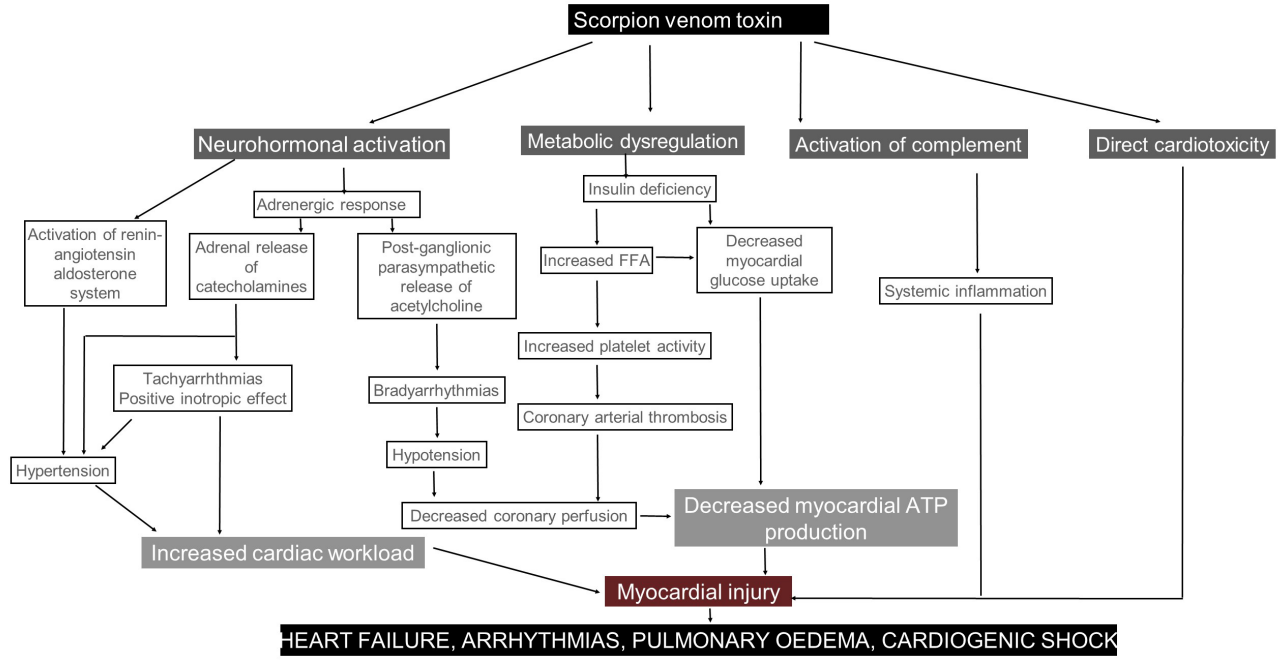

Figure 2. Shows how different therapeutic pathways target and counter the effects of scorpion envenomation. (FFA-Free fatty acid, ATP- Adenosine Triphosphate, IABP- Intra aortic balloon pump, ECMO-extracorporeal membrane oxygenation)

pathways following SE leads to the release of anaphylatoxins and pro- and anti-inflammatory cytokines. This results in a systemic inflammatory response-like syndrome with an excessive pro-inflammatory response leading to cardiovascular tissue injury and dysfunction.[7,8,16-18]

Metabolic response

A decrease in serum insulin levels following SE leads to increased free fatty acids (FFA) and hyperglycemia. The increased FFAs disrupt glucose uptake by the myocardium and may aggravate ischaemic myocardial injury in the setting of increased energy requirement in SE. FFAs also alter platelet functionality, increasing the risk of arterial thrombosis and injury.[19,20]

Direct myotoxicity

Some authors favor direct toxicity as a primary mechanism with other pathways being synergistic.[21] Immunofluorescence studies have demonstrated the direct binding of scorpion toxins to the myocardium with a direct correlation with venom concentration and dose.[22] This results in myocardial cell injury and interstitial necrosis within 3-6 hours of SE.[23] Myofibrillar degeneration and loss of sarcomere structure affect cardiac muscle integrity and function. A human necropsy finding has demonstrated coagulative myocytolysis in the myocardium occurring within a few hours of envenomation.[24] Toxins also directly affect myocardial sodium, potassium, and calcium channels affecting both conduction and contractility contributing to myocardial dysfunction, heart failure, and arrhythmias.[25,26]

Cardiac dysfunction secondary to hypoperfusion

Hypoperfusion can result in cardiac dysfunction. Hypotension may occur as a result of vasodilation due to anaphylaxis after SE. It can also be accentuated by venous pooling due to vasodilation mediated by nitric oxide (NO) release. In severe envenomation, bradykinin release worsens hypotension. Lastly, vasoactive and thrombogenic peptide components of the toxins have been suggested to cause coronary vasospasm or vessel occlusion secondary to platelet plug formation mediated by directly compromising myocardial perfusion.[27-30]

2. Manifestations of cardiovascular toxicity

Following SE, all patients should at least have 6 hours of observation regardless of symptomatology (possibly longer in pediatric patients). Admission is required for any symptoms other than local manifestation.[31,32]

Clinical features

Symptoms include dizziness, palpitations, sweating, chest pain, intractable cough and breathlessness.[28,33] Fever can occur, and a temperature above 39°C has a high positive predictive value for pulmonary edema.[5] Pallor and hypothermia can suggest global hypoperfusion. Clinical signs depend on whether the primary response was predominantly cholinergic or adrenergic.[34] Predominantly adrenergic response results in tachycardia or irregular pulse suggesting arrhythmia or hypertension. Cholinergic responses lead to bradycardias and hypotension occurs. Pulmonary oedema may be present and, while classically bilateral, can be unilateral.[33]

Classification of SE regarding cardiac involvement

Several systems have been developed to classify the severity of cardiac involvement. Abroug et al's classification is the most widely accepted and classifies cardiac involvement form class 1- 3. Class 1 is mild, excessive sweating and hypertension occur in class 2 and in class 3 cardiovascular decompensation is seen.[35]

Investigations to detect cardiotoxicity

Blood tests

Cardiac biomarkers

Biomarkers including Troponin-I, creatine kinase (CK), Creatine kinase- myocardial/brain isoenzyme (CK-MB), lactate dehydrogenase (LDH), and N-terminal Pro-Brain natriuretic peptide can be elevated after SE and suggest myocardial injury. Troponin-I titer has an inverse relationship with the left ventricular (LV) ejection fraction and more ECG abnormalities.[36-38]

Other hematological and biochemical investigations

Hyperglycaemia along with marked leucocytosis and thrombocytosis is seen following severe envenomation in the presence of cardiac involvement and shows an inverse correlation with left ventricular function.[5, 39-42]

ECG abnormalities

ECG abnormalities in scorpion envenomation are common and observed more with severe SE. As changes can occur early or hours after the first presentation serial ECGs every 6 hours may be useful.[2] The spectrum of abnormalities are vast and ranges from QRS variation and bundle branch blocks, ST/T wave changes, conduction abnormalities (1st-degree, 2nd-degree heart blocks), atrial arrhythmias, and ectopics to sinister rhythms eg., ventricular tachycardia, torsade de pointes, ventricular fibrillation or pulseless ventricular tachycardia. ECG changes may have a dose-response relationship. Most changes resolve early within 24 hrs but can persist for up to 1 week and rarely a month.[2,30,40,43-47]

Cardiac Imaging

Echocardiographic changes

Transthoracic 2-dimensional echocardiography (TTE) is useful following SE. LV dilation and functional mitral and tricuspid regurgitation can be seen. It can also demonstrate diastolic dysfunction, regional wall motion abnormalities, and impaired LV and RV function. Pulmonary hypertension has also been documented following SE. Echocardiographic abnormalities usually resolve 24-48 hours post-envenomation but can persist for up to 1 month when systolic function is severely impaired. TTE is useful in in moderate to severe envenomed patients to predict outcomes and help facilitate a safe discharge.[3,44,47-51]

Cardiac MRI (CMRI)

Magnetic resonance imaging is an investigation modality limited by access, but when available CMRI can help identify myocarditis following SE by demonstrating myocardial inflammation and oedema.[52,53]

Myocardial Perfusion Imaging (MPI)

MPI with thallium-201 scintigraphy can be a supplementary investigation and also demonstrate transient myocardial ischaemia due to myocardial hypoperfusion caused by microvasculature spasm precipitated by the released catecholamines.[54,55] The role of this investigation is limited by access and cost.

3. Treatment of SE

Prompt treatment of SE is required to control the extent of systemic involvement and symptom severity as it will impact the overall outcome.[56,57] This is achieved by early consultation, anticipating cardiac dysfunction, and implementing treatment.[58] A delay of 4-6 hours post-SE increases cardiovascular morbidity and mortality.[59-61] When treatment is delayed, venom neutralization becomes ineffective and the focus shifts towards stabilizing organ dysfunction.[62] Despite years of experience, a unified treatment protocol to guide management is lacking. The lack of consensus on the preferential neutralizing agent and treatment could be due to the failure to understand their specific role in the scorpion envenomation's complex process and is mostly based on experience.[63,64] Treatment of SE from a cardiac aspect focuses on neutralizing the toxin and its effects while providing supportive therapy (Figure 2).

Specific therapy

Antivenom

Anti-venom(AV) are purified immunoglobulins[65] Equine-derived antivenom F(ab')2 is approved for human use and is commercially available. Systemic involvement benefits from scorpion-specific F(ab')2 AV. Intravenous administration is recommended as a bolus rather than an infusion and is superior to the intramuscular route, with better outcomes and lower systemic toxicity.[66]

Scorpion toxins can be present in circulation within 2 hours after SE,[67] meriting early utilization of AV. Proactive treatment within 2-4 hours after SE with AV can prevent and even reverse cardiac changes.[68,69]

When response is inadequate repeated doses of AV have been shown to reverse systemic abnormalities,[70] and improve echocardiographic abnormalities. Combining AV with prazosin showed better outcomes compared

to using AV alone as an antidote.[64] AV has a place in delayed presentation due to the long half-life of scorpion venom (26-33 hours) and continued venom absorption from the sting site.[71] However clinical experience has been variable.[72] A randomized control trial by Abroug et al., compared AV against a placebo and did not show an effect in preventing complications or reversing changes, and did not support routine administration.[73] Frassone et al. suggested moderate SE should be managed conservatively without AV.[74]

Correction of the metabolic derangement

Insulin confers metabolic support reduces the harmful effects of FFA and helps control the adverse effects of catecholamines. It stabilises the ischaemic myocardium resulting in improved cardiac contractility and output. Insulin is recommended when blood sugar levels exceed 10mmol/L. A suggested protocol is a continuous infusion of soluble insulin at the rate of 0.3 Units/gm glucose with concurrent administration of intravenous glucose at the rate of 0.1 g/kg/hr with supplemental potassium in addition to AV for a period of 48-72 hrs.[19,41,75-77]

Countering the vasoactive effects

Prazosin is an alpha-adrenergic blocker (AAB). It increases insulin secretion and neutralizes the catecholaminergic effects helping to reverse hemodynamic, hormonal, and metabolic derangement due to SE. It improves LV function, reduces PE, and reduces morbidity and mortality. Comparative studies demonstrate the benefit of prazosin over AV use. However, combining both is a more effective approach. Dosing for adults and paediatric patients is oral 30ug/kg per dose QDS for two days or until the resolution of SE. Its ease of administration, effectiveness, affordability, and availability should encourage its early use even in the presence of hypotension. Doxazocin is an alternate that can also be used.[40, 60, 64, 65, 78-82]

Supportive therapy

The principles of supportive management include an adequate period of observation, stabilizing blood pressure, managing PE, cautious volume resuscitation, and countering potential arrhythmias.

Blood pressure control

Catecholamine-induced hypertension is largely due to vasoconstriction. Prazosin counters this effectively. The mechanism of action is discussed above. Alternatively, a calcium channel blocker (CCB) such as Nifedipine can be used. Combining both AAB and CCB yielded better control versus each being used individually. Hydralazine is another alternative. Side effects include sympathetic tachycardia and possibly hypotension.

Angiotensin-converting enzyme (ACE) inhibitors role in SE is controversial. It is hypothesized to counter the catecholamine effect, cause vasodilation, and aid in PE. However the effect on the accumulation of bradykinin is similar to that following SE which may worsen PE or precipitate hypotension. Sifi et al. demonstrated that the use of an ACE inhibitor or angiotensin II receptor blocker, conferred protection as cardiac and aortic tissue samples showed less inflammatory and infiltrative changes following envenomation.[17, 46, 83]

The role of beta-blockers (BB) in SE is controversial. Trejo et al., compares the SE mechanism of action to that of adrenergic crisis in phaechromoctytoma.[84] implying BB should not be used in isolation but can be considered with simultaneous utilization of AAB if needed.[63, 85]

The cholinergic response to SE can also result in hypotension. Other contributing factors to low blood pressure include impaired cardiac function, vasodilation, catecholamine depletion, and hypovolaemia. Hypotension may be addressed by appropriate fluid resuscitation and inotropic or vasopressor support e.g., dobutamine, dopamine, noradrenaline, and adrenaline. There is good experience with dobutamine which improves both cardiac output and blood pressure. The recommended dose is 10ug/Kg/min infusion, tapered as per response. Using dobutamine and prazosin concomitantly has been shown to reduce mortality. Dobutamine should be considered in PE as it reduces mortality. When medical therapy fails intra-aortic balloon pump has been used to bridge and achieve stability. In non-responsive cardiogenic shock and PE due to severe LV dysfunction, a temporary heart-lung bypass in the form of extracorporeal membrane oxygenation has been used successfully.[11, 65, 79, 86-94]

Managing arrhythmia and ECG abnormalities

ECG changes and arrhythmias tend to be benign and transient.[95] Rarely, sustained arrhythmias may need intervention e.g., cardioversion.[14] In the presence of severe bradycardia or complete atrioventricular block, atropine can be useful.[65, 96] Magnesium treatment may be useful in non-sustained and sustained VT including torsade de pointes episodes.[89]

Amiodarone can deplete norepinephrine in the sympathetic neurons. This neuromodulatory sympatholytic action explains its antiarrhythmic property. It has been used as a rescue therapy in severe LV dysfunction secondary to SE. It can also be used to terminate sinister arrhythmias successfully e.g., VT.[14, 97]

There is limited evidence for using non-dihydropyridine CCB in arrhythmia related to SE.

Correction of electrolyte abnormalities

Electrolyte imbalances maybe observed following scorpionism. Though hypokalaemia occurs after SE it is due to an intracellular shift and not reduction in whole-body potassium levels. It tends to normalize after the autonomic storm and following AV treatment.[14, 98]

Role of corticosteroids

The data regarding the role of corticosteroids in the treatment of SE is limited. In a study done critically ill paediatric population following SE the use of corticosteroids had no impact on morbidity and mortality. It's use routinely following SE is not recommended.[99] However, there may be a role of prophylactic use of corticosteroids to prevent hypersensitive reactions arising from anti-venom.

Managing pulmonary oedema and heart failure

PE after SE may result from heart failure or adult respiratory distress syndrome(ARDS). Histopathological evidence suggestive of ARDS has been seen after SE.[100] However, the preferred hypothesis of PE is LV failure as evidenced by echocardiographic parameters and observed response to the treatment.[49, 101, 102] Acute PE and cardiac failure are the most frequent causes of death following scorpionism.[103] PE can occur within 30 min but may occur even 3-8 hours post envenomation.

Specific therapy with AV and prazosin prevents and improves established PE and prevents further deterioration of LV function. Inotropes will improve LV failure and improve PE, thus should be instituted early. Inotropes also improve right ventricular function. The experience with dobutamine is good in PE. It's recommended to be used early in PE as an infusion of 7-20ug/kg/min as required.[91,102] Alternate inotropes e.g., dopamine, and vasopressors such as norepinephrine have been utilized successfully.[72,104,105] If hypoxia, oxygen will be beneficial with an appropriate modality for delivery. A low threshold for oxygen use is suggested in the presence of PE.[102] The use of non-invasive pressure support ventilation and if required invasive mechanical ventilation is effective and beneficial.[106] Both inotrope use and mechanical ventilation help resolve PE and can even prevent further formation.

Aggressive therapy may be necessary for life-threatening PE. IV sodium nitroprusside is useful.[33,107] IV nitroglycerine for refractory PE has been shown to reduce the number of patients ultimately requiring mechanical ventilation. Aggressively managing PE will help reduce mortality.[61]

Generally, diuretics are useful in PE. Though furosemide has been used as an infusion in documented case reports with good outcomes,[79,104] its use in scorpionism is controversial. When blood pressure is satisfactory and in the absence of hypovolemia, diuretic bolus doses can be safely used in PE.[97]

Refractory heart failure following scorpion envenomation can be challenging. Though only a short-term solution, Levosimendan, a calcium sensitizer, can increase cardiac contractility in severe LV dysfunction when conventional therapy fails.[108,109]

Role of Anticogulants

There is no role for routine anticoagulation in SE. Even in the presence of acute ECG changes which is more likely due to myocarditis rather than acute ACS as shown by negative angiographic studies.[110] Anticoagulation may potentially be harmful as certain species of SE can cause prolongation of Activated partial thromboplastin time (PTT), and prothrombin time (PT).[111]

Outcomes and prognosis

Mortality and outcomes following SE have improved due to AV, supportive therapy, and intensive care facilities.[5, 64] However, age above 50 years with a background of cardiovascular disease as well as the paediatric age group are more vulnerable to cardiac complications including death.[35,112,113]

A delay in treatment directly correlates with more complications and increased mortality.[5,35,59-61] It can be compared to a "golden hour" principle in instituting necessary antidote treatment.[114]

The need for mechanical ventilation also correlates with a poor prognosis.[5] In the absence of LV dysfunction, ward-based management, and discharge is possible in 24-48 hours from a cardiac perspective. Mild to moderate LV dysfunction necessitates high dependency unit monitoring, inotropic support, and extended hospital stay. Severe LV dysfunction and decompensated cardiovascular states necessitate ICU stay.[104,115]

Observed ECG changes and LV dysfunction on echo are transient. Rarely in delayed presentations and severe LV dysfunction, pathogenic ECG changes or ECHO abnormalities may persist for up to a month.[43,44,86] but usually there is no long-lasting sequela for the patient.

Conclusions

Cardiovascular involvement usually occurs in the setting of intermediate or severe levels of SE. There are multiple aetiopathogenesis pathways leading cumulatively to cardiac insult.

Pre-existing cardiovascular conditions pose a higher risk for SE. Early detection of alterations in haemodynamic parameters and blood tests may aid patient triage. Leucocytosis, thrombocytosis and high glycaemic levels may predict myocarditis and left ventricular failure. Troponin I has prognostic value.

An ECG if abnormal indicates cardiac involvement. Transthoracic echo objectively defines cardiac dysfunction and will guide treatment and discharge.

Early use of AV or AAB as antidote therapy can potentially prevent and even reverse cardiac manifestations and compromise including PE and LV dysfunction. Evidence favours using a combination of AV and prazosin.

Hypertension is well managed using Prazosin. Alternate options include nifedipine & intravenous hydralazine.

PE, cardiogenic shock, or moderate to severe LV impairment may benefit from use of inotropic support. Conventional heart failure treatment with ACE inhibitors and beta-blockers is controversial. Insulin infusion in the presence of hyperglycemia has shown to improve cardiovascular instability.

Arrhythmias are usually transient. Regular prophylactic use of antiarrhythmics agents is not recommended.

SE causes cardiovascular morbidity and mortality. Treatment addressing the pathogenetic mechanisms have been used successfully. It may be useful to develop standardized guidelines for antivenom and pharmacological agents in the management of these patients.

Declarations

Authors' contributions

MRN conceptualized the review. Both MRN & DRW contributed in searching for and filtering the articles. Both authors helped with composition of the manuscript. Both authors read and approved the final manuscript.

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List of Abbreviations

ARDS	Adult Respiratory Distress Syndrome
AV	Anti-Venom
ACE	Angiotensin-Converting Enzyme
AAB	Alpha-Adrenergic Blocker
BB	Beta-Blockers
CCB	Calcium Channel Blocker
CK	Creatine Kinase
CK-MB	Creatine Kinase- Myocardial/Brain Isoenzyme
FFA	Free Fatty Acids
LDH	Lactate Dehydrogenase
LV	Left Ventricular
MPI	Myocardial Perfusion Imaging
NO	Nitric Oxide
PTT	Partial Thromboplastin Time
PT	Prothrombin Time
LV	Right Ventricular
SE	Scorpion Envenomation
TTE	Transthoracic 2-Dimensional Echocardiography

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