

# Reversible Myocarditis Following Black Widow Spider (*Latrodectus* spp.) Bite in Egypt

## A case report

Ahmed G. Emara,<sup>1</sup> AbdelRhman A. Aboshady,<sup>2</sup>\* Omar A. Aboshady,<sup>3</sup> Mohamed M. Shawqi<sup>4</sup>

**ABSTRACT:** Black widow spiders (BWSs) are poisonous spiders of the *Arthropoda* phylum that live in the Mediterranean region. The effects of BWS bites ranges from local damage to systemic manifestations including paresthesia, stiffness, abdominal cramps, nausea, vomiting, headache, anxiety, hypertension and tachycardia. However, cardiac involvement following a BWS bite is uncommon. We report a 35-year-old male patient who presented to a tertiary hospital in Menoufia, Egypt, in 2019 and developed acute pulmonary oedema with electrocardiogram (ECG) changes that showed ST elevation in leads I and aVL with reciprocal ST segment depression in infero-lateral leads with elevated cardiac biomarkers. Echocardiography showed regional wall motion abnormalities with an impaired ejection fraction of 42%. The condition was reversible after one week of supportive treatment and the patient was discharged from the hospital with normal electrocardiogram, ejection fraction and negative cardiac markers. A routine cardiac evaluation, serial ECG, serial cardiac markers and echocardiography should be considered for any patient exposed to a BWS bite for detection of any potentially fatal cardiac abnormalities.

**Keywords:** Black Widow Spider; Spider Bites; Myocarditis; Heart Failure; Kounis Syndrome; Acute Coronary Syndrome; Case Report; Egypt.

**B**LACK WIDOW SPIDERS (BWSs) ARE A RARE but very poisonous species of the *Arthropoda* phylum that generally live in moderate climatic conditions.<sup>1</sup> These spiders are shiny black with a ventral red hourglass mark on females, while males have various dorsal red marks. Their size averages 3-10 mm, with females up to 13 mm in length. The spider venom includes a main toxic protein ( $\alpha$ -latrotoxin) that primarily affects the motor nerve endings, leading to increased catecholamine release and acetylcholine consumption.<sup>2</sup>

Patients who have been bitten by a BWS typically complain of various clinical symptoms that range from local to systemic manifestations; a BWS bite can cause soft tissue damage at the site of the bite, with local to generalised pain and/or paresthesia.<sup>3-7</sup> In addition, priapism, stiffness, abdominal cramps, nausea, vomiting, headache, tremors and/or anxiety have been reported.<sup>3-5,8,9</sup> A few patients have hypertension, tachycardia and/or chest pain.<sup>3,4,7-11</sup> Only one study reported acute kidney failure and rhabdomyolysis.<sup>1</sup> Myocardial involvement after BWS bites is uncommon, and only a limited number of cases have been recorded with no cases from Egypt.<sup>3,5-12</sup> Here, we report on a 35-year-old previously healthy man who developed myocarditis complicated by acute heart failure and pulmonary oedema following a BWS bite, which is the first case reported from Egypt.

## Case Report

A 35-year-old previously healthy man presented to a tertiary hospital in Menoufia, Egypt, in 2019 12 hours after having been bitten by a BWS on the lateral aspect of his right leg, 15 cm below the knee joint. After being shown various photos of spiders, the patient identified a BWS as the culprit. Within a few minutes of the bite, he developed local severe burning pain that rapidly involved all of his thigh. Subsequently, 15 minutes later, he became nauseous with severe diffuse abdominal pain, back pain, dizziness, headache and severe muscle cramping in his lower limbs. On examination, he had priapism and generalised tremors.

On admission, he was noted to appear anxious and diaphoretic. His vital signs were as follows: blood pressure = 150/100 mmHg, pulse rate = 110/min, respiratory rate = 40 breaths/min, oxygen saturation = 98% and temperature = 37.3°C. Physical examination revealed a 3 × 2 mm area of erythema at the bite site, board-like abdominal rigidity and hyperactive stretch reflexes. Cardiac examination revealed rapid S1 and S2 with S3 and no murmur or rub. Other than a slight leukocytosis (total leucocyte count =  $15 \times 10^3$ , normal range:  $4-10 \times 10^3$ ) with mild elevation in the absolute eosinophilic count ( $0.9 \times 10^3/L$ , normal range:  $0.0-0.4 \times 10^3/L$ ), laboratory findings and arterial blood gases were normal.

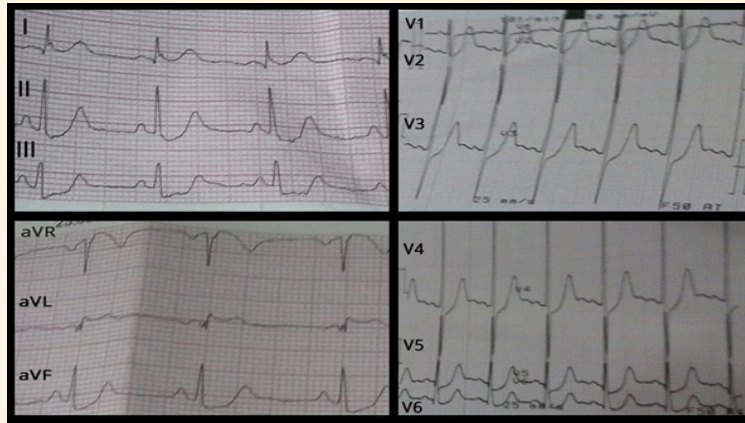
Departments of <sup>1</sup>Cardiology, <sup>2</sup>Critical Care Medicine Unit and <sup>3</sup>Clinical Pharmacology, Faculty of Medicine, Menoufia University, Shebin ElKoum, Egypt; <sup>4</sup>Faculty of Medicine, Benha University, Benha, Egypt

\*Corresponding Author's email: [omr.ali@med.menofia.edu.eg](mailto:omr.ali@med.menofia.edu.eg)

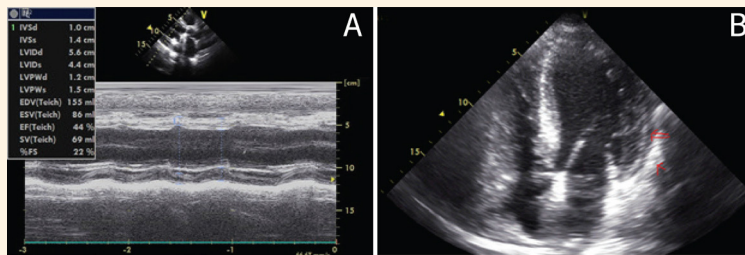
The patient was given tetanus prophylaxis with intravenous analgesics, hydrocortisone, anti-histamine (pheniramine maleate at 22.75 mg/day) and fluids (Ringer's lactate at 1.5 L/day). Anti-venom was not given because it is unavailable in Egypt.

Four hours later, the patient developed progressive dyspnoea, orthopnoea and retrosternal chest pain. An electrocardiogram (ECG) was obtained

that showed an ST-segment elevation of 0.5 mm in leads I and aVL with ST-segment depression in leads II, III, aVF and V2–V6 [Figure 1]. Cardiac biomarkers were CK-MB 89.9 IU/L (0–25 IU/L) and cTnI 5.1 ng/ml (0–0.6 ng/mL). A chest radiograph showed exaggerated pulmonary vascular markings consistent with pulmonary oedema. Echocardiography, done 17 hours after his presentation, revealed impaired left



**Figure 1:** Initial electrocardiogram showing ST-segment elevation in leads I, aVL and ST-segment depression in leads II, III, aVF and V2–V6.



**Figure 2:** A: Echocardiography showing normal left ventricular end-diastolic diameter and impaired left ventricular systolic function with an ejection fraction of 42%. B: Echocardiography showing thickening of the pericardium with rim of pericardial effusion on lateral wall and right atrium.

**Table 1:** Electrocardiogram, cardiac enzymes and ejection fraction findings of a 35-year-old male patient who was bitten by a black widow spider over the admission period and one week after discharge

Timeframe	ECG	Cardiac enzymes		Ejection fraction in %
		CK-MB in IU/L (normal range: 0–25)	cTnI in ng/mL (normal range: 0–0.6)	
Four hours after admission	ST-segment elevation (0.2 mv) in leads I, aVL with reciprocal depression (0.3 mv) in II, III, aVF and V2-V6	89.9	5.1	42
10 hours after admission	ST-segment elevation (0.1 mv) in leads I, aVL with reciprocal depression (0.2 mv) in II, III, aVF and V2-V6	79.08	Not done	Not done
One day after admission	ST-segment elevation (0.1mv) in leads I, aVL with reciprocal depression (0.2 mv) in II, III, aVF and V2-V6	24.07	3.2	43
Two days after admission	Normal	6.5	0.5	51
One week after discharge	Normal	6.1	0.5	56

ECG = electrocardiogram.

**Table 2:** Reported cases with cardiac involvement after black widow spider bites<sup>3,5-12</sup>

Author and year of publication	Age/sex	Cardiac presentation	ECG	Echocardiography	Cardiac markers	Diagnosis
Piscopo <i>et al.</i> <sup>6</sup> (2020)	50/M	Not mentioned	- Diphasic T wave in the lateral leads at admission. - At day 3, ECG showed sinus rhythm and negative T wave in the lateral and inferior leads.	- Abnormalities in left ventricular wall motions and moderate systolic dysfunction (hypokinesia of LV middle/basal segment of inferior, lateral and inferior-lateral wall. - LVEF = 48%	Positive	Acute myocarditis
Yaman <i>et al.</i> <sup>7</sup> (2015)	15/M	Pulmonary oedema/heart failure	- ST depression in II, III, aVF, aVL and V3-V6	- EF = 22% - Global hypokinesia- Rim of pericardial effusion	Positive	Reversible myopericarditis
Dendane <i>et al.</i> <sup>10</sup> (2012)	35/M	Pulmonary oedema/ heart failure	- Sinus tachycardia - Hyperacute T in V3-V6	- EF = 48% - Septal and lateral wall hypokinesia	Positive	Reversible myopericarditis
Levine <i>et al.</i> <sup>5</sup> (2010)	22/M	Pulmonary oedema	- Incomplete right bundle branch block - ST uptake in V1-V6	- EF = 35% - Mild to moderate tricuspid regurgitation	Positive	Reversible myopericarditis
Sari <i>et al.</i> <sup>11</sup> (2008)	65/M	Chest pain	- ST elevation in II and aVF- Hyperacute T in V3-V6	- Normal	Positive	Kounis syndrome
Erdur <i>et al.</i> <sup>8</sup> (2007)	22/M	Chest pain, severe hypertension	- Inverted P in leads II, III, aVF, aVL and V1	- EF = 40% - Anteroseptal wall hypokinesia	Positive	Reversible toxic myocarditis
Pneumatikos <i>et al.</i> <sup>9</sup> (2003)	19/F	Cardiogenic shock	- Atrial fibrillation - Incomplete right bundle branch block	- EF = 20 % - Global hypokinesia	Positive	Acute fatal toxic myocarditis
Bucur <i>et al.</i> <sup>3</sup> (1999)	Seven cases (13–57 years)	Ranging from chest pain to pulmonary oedema	Not mentioned	Not mentioned	Not mentioned	All cardiac events were reversible
Pulignano <i>et al.</i> <sup>12</sup> (1998)	16/M	Typical chest pain	- ST-T changes in precordial leads	- Akinesia of interventricular septum - Depressed left ventricular function	Positive	Reversible toxic myocarditis

ECG = electrocardiogram; LVEF = left ventricular ejection fraction; EF = ejection fraction.

ventricular systolic function with an ejection fraction of 42%. There were regional wall motion abnormalities including hypokinesia of the mid-basal anterior, mid-basal posteroseptal, mid-lateral and basal inferior walls with preserved thickness. In addition, the pericardium was noted to be thickened with a rim of pericardial effusion on the lateral wall [Figure 2].

The patient was admitted to the intensive care unit and was treated with intravenous furosemide at 20 mg/8 h, nitroglycerine infusion, intravenous morphine, captopril at 12.5 mg/8 h, and prophylactic enoxaparin at 80 IU/24 h. Later, beta-blocker (bisoprolol at 2.5 mg/24 h for one month) was added to maintain a heart rate of 60–70 bpm and good coronary perfusion.

The dyspnoea improved rapidly after this supportive therapy. The pain, headache, dizziness, tremors and muscle cramps disappeared after 48

hours. However, hyperreflexia and priapism continued to the fourth day. He was discharged on the sixth day with resolution of his symptoms. At that point, his ECG had normalised and the ejection fraction was estimated to be 51% on repeated echocardiography [Table 1].

Informed written consent for publication of this case report and figures was obtained from the patient.

## Discussion

Cardiac involvement following a BWS bite is uncommon. Only a few cases have been reported in the literature, with effects ranging from reversible myocarditis to acute severe fulminant heart failure and cardiogenic shock.<sup>1,5,8-12</sup> Most cases have been reported in males and most had myocarditis after a

BWS bite [Table 2].<sup>3,5-12</sup> The majority of cases presented with chest pain or other manifestations suggesting pulmonary oedema or heart failure.<sup>3,5-12</sup> Eight cases showed elevated levels of cardiac biomarkers.<sup>5-12</sup> Only a few cases showed ST segment changes that were similar to the current findings.<sup>5,7,9,11,12</sup> Cardiac dysrhythmia, such as atrial fibrillation and incomplete bundle branch block, have also been reported.<sup>5,9</sup>

Although the underlying mechanism of cardiac affection after a BWS bite is still not fully understood, there are many possible explanations, such as the direct toxic effect of  $\alpha$ -latrotoxin on cardiomyocytes producing a form of toxic myopericarditis.<sup>5,8,9,11,12</sup> Recently, the hyperadrenergic state was claimed to primarily be involved (broken heart syndrome).<sup>10</sup> In addition,  $\alpha$ -latrotoxin, which is a foreign protein, might induce an allergic reaction producing a form of hypersensitivity myopericarditis.<sup>1</sup>  $\alpha$ -latrotoxin also induces inflammatory mediator release, which could induce coronary artery spasm (Kounis syndrome).<sup>1</sup>

From these proposed mechanisms of cardiac affection, the heart can be affected by two main pathologies: myopericarditis and/or coronary artery spasm. However, the clinical presentation depends on which of the two pathologies is predominant. When coronary artery spasm is the dominant pathology, the main presentation is typically chest pain or even acute coronary syndrome. However, when myopericarditis is predominant, the main presentation is heart failure and pulmonary oedema. In echocardiography, hypersensitivity myopericarditis usually shows heterogeneous segmental wall motion abnormalities. In contrast, coronary artery spasm shows segmental wall motion abnormalities in certain territories. Late gadolinium enhancement in cardiac magnetic resonance shows patchy sub-epicardial distribution which is not consistent with any coronary territory. Distribution in coronary artery spasm, however, is usually in the sub-endocardium and consistent with the infarct-related artery. In the current case, the authors suspect the pathology was mostly combined, with greater spasm, which was reflected in the ECG.

The current patient developed the commonly reported symptoms of latrodectism such as nausea, pain, muscle rigidity, headache, tremors, and muscle cramping from a BWS bite.<sup>3-9</sup> In addition, a moderate degree of priapism was reported, which is also recorded in the literature.<sup>3</sup> The findings of hypertension and tachypnoea that the patient developed was similar to previous studies.<sup>7-10</sup>

Treatment of the BWS bites depends mainly on the severity of presentation.<sup>13</sup> Most cases are mild and only require oral pain medication and tetanus prophylaxis. In severe cases, however, parental

opioids or/and benzodiazepines might be required.<sup>13</sup> Antivenom administration is reported to reduce pain duration to less than 24 hours in approximately 80% of cases; it is reported to reduce severity, with home discharge in 90% of patients.<sup>13-15</sup> However, allergic reactions, serum sickness and rare reports of fatalities have been reported from antivenom administration.<sup>13,15,16</sup> Unfortunately, given that BWS bites are rare in Egypt, the antivenom was not available at the presenting centre.

## Conclusion

To the best of the authors' knowledge, this is the first case reported from Egypt of a BWS bite to present with ECG changes typical of acute myocardial infarction in the literature. Clinicians should be aware that reversible myocarditis can occur after a BWS bite. Moreover, it is recommended that a complete cardiac evaluation be performed for every case of BWS bite to screen for myopericarditis and coronary artery spasm.

## AUTHORS' CONTRIBUTION

AGE and AAA managed the case clinically. All authors contributed equally to the literature review, drafting and critically revising the final version of the paper. All authors approved the final version of the manuscript.

## References

1. Al Bshabshe A, Alfaifi M, Alsayed AF. Black widow spider bites experience from tertiary care center in Saudi Arabia. *Avicenna J Med* 2017; 7:51-3. <https://doi.org/10.4103/2231-0770.203606>.
2. Ushkaryov YA, Volynski KE, Ashton AC. The multiple actions of black widow spider toxins and their selective use in neurosecretion studies. *Toxicon* 2004; 43:527-42. <https://doi.org/10.1016/J.TOXICON.2004.02.008>.
3. Bucur IJ, Obasi OE. Spider bite envenomation in Al Baha Region, Saudi Arabia. *Ann Saudi Med* 1999; 19:15-19. <https://doi.org/10.5144/0256-4947.1999.15>.
4. Kara H, Ak A, Bayir A, Avci A. Reversible myocarditis after spider bite. *BMJ Case Rep* 2013; 2013:bcr2013008957. <https://doi.org/10.1136/BCR-2013-008957>.
5. Levine M, Canning J, Chase R, Ruha AM. Cardiomyopathy following latrodectus envenomation. *West J Emerg Med* 2010; 11:521-3.
6. Piscopo A, Massari F, Scicchitano P, Sanasi M, De Palo M, Caldarola P, et al. Acute myocarditis after black widow spider bite: A case report. *Cardiol Ther* 2020; 9:569-75. <https://doi.org/10.1007/s40119-020-00178-3>.
7. Yaman M, Mete T, Ozer I, Yaman E, Beton O. Reversible myocarditis and pericarditis after black widow spider bite or kounis syndrome? *Case Rep Cardiol* 2015; 2015:768089. <https://doi.org/10.1155/2015/768089>.
8. Erdur B, Turkcuier I, Bukiran A, Kuru O, Varol I. Uncommon cardiovascular manifestations after a Latrodectus bite. *Am J Emerg Med* 2007; 25:232-5. <https://doi.org/10.1016/J.AJEM.2006.11.005>.

9. Pneumatikos IA, Galiatsou E, Goe D, Kitsakos A, Nakos G, Vougiouklakis TG. Acute fatal toxic myocarditis after black widow spider envenomation. *Ann Emerg Med* 2003; 41:158. <https://doi.org/10.1067/MEM.2003.32>.
10. Dendane T, Abidi K, Madani N, Benthani A, Gueddari FZ, Abouqal R, et al. Reversible myocarditis after black widow spider envenomation. *Case Rep Med* 2012; 2012:794540. <https://doi.org/10.1155/2012/794540>.
11. Sari I, Zengin S, Davutoglu V, Yildirim C, Gunay N. Myocarditis after black widow spider envenomation. *Am J Emerg Med* 2008; 26:630.e1–3. <https://doi.org/10.1016/j.ajem.2007.09.012>.
12. Pulignano G, Del Sindaco D, Giovannini M, Zeisa P, Faia M, Soccorsi M, et al. Myocardial damage after spider bite (*Latrodectus tredecimguttatus*) in a 16-year-old patient. *G Ital Cardiol* 1998; 28:1149–53.
13. Clark RF, Wethern-Kestner S, Vance MV, Gerkin R. Clinical presentation and treatment of black widow spider envenomation: A review of 163 cases. *Ann Emerg Med* 1992; 21:782–7. [https://doi.org/10.1016/S0196-0644\(05\)81021-2](https://doi.org/10.1016/S0196-0644(05)81021-2).
14. Ellis RM, Sprivulis PC, Jelinek GA, Banham ND, Wood SV, Wilkes GJ, et al. A double-blind, randomized trial of intravenous versus intramuscular antivenom for red-back spider envenoming. *Emerg Med Australas* 2005; 17:152–6. <https://doi.org/10.1111/J.1742-6723.2005.00720.X>.
15. Isbister GK, Brown SG, Miller M, Tankel A, Macdonald E, Stokes B, et al. A randomised controlled trial of intramuscular vs. intravenous antivenom for latrodectism--the RAVE study. *QJM* 2008; 101:557–65. <https://doi.org/10.1093/QJMED/HCN048>.
16. Murphy CM, Hong JJ, Beuhler MC. Anaphylaxis with *Latrodectus* antivenin resulting in cardiac arrest. *J Med Toxicol* 2011; 7:317–21. <https://doi.org/10.1007/S13181-011-0183-1>.