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A RARE CASE OF REACTIVE ARTHRITIS SECONDARY TO RUSSEL'S VIPER BITE

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Abstract

In tropical areas, Russell's viper (Daboia russelii) bite envenomation poses a serious threat to public health since it can result in coagulopathy, severe renal failure, and neurotoxicity. In tropical areas, Russell's viper (Daboia russelii) bite envenomation poses a serious threat to public health since it can result in coagulopathy, severe renal failure, and neurotoxicity. Rarely, inflammatory joint conditions like reactive arthritis occur as sequelae. This report details a 61-year-old farmer presenting with reactive arthritis following a Russell's viper bite on his right heel. Initial management included anti-snake venom therapy and ICU care for coagulopathy. On day four, the patient developed high-grade fever, knee swelling, and pain, followed by ankle involvement. Laboratory findings showed elevated inflammatory markers, including leukocytosis, ESR, and CRP, while autoimmune and infectious markers were negative. Synovial fluid analysis excluded septic arthritis, supporting an inflammatory etiology. Treatment involved NSAIDs and intravenous dexamethasone, leading to marked clinical improvement and symptom resolution. This case emphasizes the need for recognizing rare complications like reactive arthritis in snakebite victims. A thorough clinical evaluation and multidisciplinary approach are essential for early diagnosis and management. Limited literature exists on reactive arthritis secondary to snakebite envenomation, emphasizing the importance of reporting such cases. Further research is warranted to understand the pathophysiological link between snake venom and post-inflammatory joint conditions to improve diagnostic and therapeutic strategies.

INTRODUCTION

In tropical areas, especially South and Southeast Asia, snakebite envenomation poses a serious threat to public health. Russell's viper (Daboia russelii) is one of the most deadly venomous snakes and is the cause of many snakebite deaths each year. The venom of Russell's viper is complex, containing hemotoxins, neurotoxins, and cytotoxins, leading to a wide spectrum of clinical manifestations. Common complications include coagulopathy, acute renal failure, and neurotoxicity. Rarely, envenomation can result in hypopituitarism, a condition characterized by decreased hormone production from the pituitary gland (1,2).

An infection or, in rare instances, exposure to certain chemicals can cause reactive arthritis, an inflammatory joint disease. Though not all of the symptoms are usually present, it is typified by the triad of conjunctivitis, urethritis, and arthritis. Joint inflammation is the result of an abnormal immune response, which is part of the pathophysiology. Reactive arthritis is extremely uncommon after snake envenomation, however it is frequently linked to gastrointestinal or genitourinary diseases. (3,4).

Russell's viper envenomation presents with a variety of clinical symptoms, primarily due to its hemotoxic effects, which can lead to bleeding disorders and renal complications. Neurological manifestations, though less common,

have also been documented. However, the development of reactive arthritis as a sequela of snakebite, particularly from Russell's viper, is scarcely reported in medical literature (2,4).

This case report details a rare instance of reactive arthritis following a Russell's viper bite. The objective is to highlight this uncommon complication, discuss its possible pathophysiological mechanisms, and emphasize the importance of considering such atypical presentations in the clinical management of snakebite victims (5).

CASE PRESENTATION

A 61-year-old farmer with no known comorbidities presented to the emergency room (ER) following a Russell's viper bite on his right heel. Initial assessment revealed a positive 20-minute whole blood clotting time test, indicating coagulopathy. The patient was promptly admitted to the intensive care unit (ICU) for observation and management. He was started on anti-snake venom (ASV) therapy with 10 vials, which he tolerated without any adverse reactions. Over the initial days, his condition improved symptomatically, and plans were made to transfer him out of the ICU.

However, the patient experienced acute pain and edema in his right knee along with a high-grade fever and chills on the fourth day of stay. Similar symptoms appeared in his left ankle two days later. With a blood pressure of 130/80 mmHg, a pulse rate of 89 beats per minute, a respiratory rate of 18 breaths per minute, and an oxygen saturation of 98% on room air, his vital signs were stable at that point.

Local Examination (L/E):

- Bite site (right heel): No swelling, blisters, or tenderness.
- Right knee: Swelling, warmth, and tenderness present.
- Left ankle: Warmth and tenderness present.

Laboratory investigations revealed significant changes between admission and day five of hospitalization. On admission, his total leukocyte count (TLC) was 8,670/µL, with 68% neutrophils and C-reactive protein (CRP) at 14 mg/L. By day five, these parameters had escalated, with a TLC of 15,120/µL, neutrophils at 80%, erythrocyte sedimentation rate (ESR) of 77 mm/hr, and CRP elevated to 162 mg/L. Tests for liver and kidney function (RFT, LFT) stayed within normal ranges. Rheumatoid factor (RA), anti-cyclic citrullinated peptide (Anti-CCP), and antinuclear antibodies (ANA) were among the autoimmune indicators that came back negative.

Ultrasound examination of the right knee revealed 50-60 mL of fluid collection. Synovial fluid analysis showed translucent yellow fluid with a total cell count of 13,000/µL, comprising 80% neutrophils and 20% lymphocytes. Acid-fast bacilli (AFB) staining, Gram staining, and culture yielded no evidence of infection, ruling out septic arthritis. Additionally, blood and urine cultures were negative.

The patient had initially been started on ASV, prophylactic intravenous (IV) antibiotics, and supportive care. When he developed fever on day four, further evaluation pointed towards an inflammatory etiology. A rheumatology consultation was sought, and the team recommended initiating IV dexamethasone (8 mg once daily) alongside non-steroidal anti-inflammatory drugs (NSAIDs).

Following the introduction of low-dose steroids and higher antibiotics, the patient showed marked symptomatic improvement. Fever spikes and joint pain resolved, and IV steroids were gradually tapered to an oral dose before being discontinued.

This case highlights the importance of thorough evaluation and multidisciplinary management in addressing unusual complications of snakebite envenomation, such as reactive arthritis.

DISCUSSION

An extremely uncommon case of reactive arthritis that occurred as a result of envenomation by a Russell's viper (Daboia russelii) patient is presented in this case report. Although snakebites constitute a major public health concern in tropical locations, particularly in South and Southeast Asia, the development of reactive arthritis as a sequela is extremely rare (6,7). Snakebites represent a substantial public health concern in these regions. On a yearly basis, the Russell's viper is responsible for a significant number of fatalities, making it one of the most lethal snakes in Asia. Its venom is a complicated mixture of enzymes and proteins, which includes hemotoxins, neurotoxins, and cytotoxins. As a result, it can cause a wide range of clinical symptoms. Coagulopathy, acute renal failure, and neurotoxicity are examples of common problems that might occur (8 9, 10). The venom-induced

consumption coagulopathy (VICC) that occurs after Russell's viper bite can lead to a syndrome that is consistent with thrombotic microangiopathy (TMA). This syndrome is characterized by the presence of thrombocytopenia, microangiopathic hemolytic anemia (MAHA), and acute renal injury (8). Inflammatory joint disease, also known as reactive arthritis, is a condition that manifests itself as a reaction to an infection that occurs in another part of the body. The trinity of arthritis, urethritis, and conjunctivitis is what distinguishes it from other conditions, although not all of its components are observed in every instance. The pathophysiology is characterized by an abnormal immune response, which ultimately results in inflammation of the joints. In spite of the fact that reactive arthritis is frequently linked to diseases of the genitourinary or gastrointestinal tract, the incidence of this condition following snake envenomation is quite uncommon (9,10).

In this particular patient, the temporal link between the snakebite and the beginning of arthritis, in conjunction with the exclusion of septic arthritis through synovial fluid analysis and negative cultures, lends support to the diagnosis of reactive arthritis as a result of snake envenomation. The patient's clinical course was significant for the onset of a high-grade fever with chills, along with swelling and intense pain in the right knee, which was then followed by similar symptoms in the left ankle. Each of these side effects occurred simultaneously. A strong inflammatory response was observed in the laboratory, as evidenced by an enhanced total leukocyte count, neutrophilia, an increased erythrocyte sedimentation rate (ESR), and significantly higher levels of C-reactive protein (CRP). Autoimmune markers, such as rheumatoid factor (RA), anti-cyclic citrullinated peptide (Anti-CCP), and antinuclear antibodies (ANA), were all negative, which provides additional evidence in favor of the diagnosis of reactive arthritis (3,4). The treatment of reactive arthritis often entails the use of non-steroidal anti-inflammatory medicines (NSAIDs) in order to reduce the amount of pain and inflammation that is experienced. When there are considerable symptoms, it may be necessary to use corticosteroids in order to effectively reduce inflammation. In this particular instance, the patient responded favorably to intravenous dexamethasone and nonsteroidal anti-inflammatory drugs (NSAIDs). Subsequently, oral steroids were gradually reduced, and eventually, the patient was stopped using them altogether as their symptoms improved (11). There are just a few examples of reactive arthritis occurring as a result of snake envenomation, according to a study of the relevant literature. One example was a man who was 44 years old and developed signs and symptoms of complex regional pain syndrome (CRPS) in his left upper extremity after being bitten by a snake that was only mildly venomous. This case demonstrates that even a little amount of envenomation can result in post-inflammatory problems (12). There was another instance that documented a woman who was 22 years old and experienced acute multiple cerebral infarctions as a result of Russell's bite from a viper. This case demonstrates that viper envenomation can cause neurological problems that are uncommon but severe (13). Because of the different and unpredictable nature of the difficulties that can occur as a result of snake envenomation, these cases illustrate the situation.
When attempting to diagnose patients who come with joint symptoms after being envenomated by a snake, it is essential to take into account the possibility of reactive arthritis as part of the differential diagnosis. Due to the rarity of this complication, it presents difficulties in diagnosis and management, which is why doctors need to have a high index of suspicion. Early detection and adequate management are absolutely necessary in order to forestall the occurrence of any problems and guarantee positive outcomes for patients. It is necessary to conduct additional research in order to shed light on the pathophysiological mechanisms that are responsible for this connection and to provide management guidelines that are supported by strong evidence.

CONCLUSION

This case highlights the rare occurrence of reactive arthritis as a complication of Russell's viper envenomation. While snakebites commonly lead to coagulopathy, neurotoxicity, and acute renal failure, the development of inflammatory joint conditions such as reactive arthritis is an uncommon and under-recognized phenomenon. The temporal association of symptoms, coupled with the exclusion of infectious and autoimmune causes, strongly suggests a post-inflammatory reaction to the venom. The successful resolution of symptoms with corticosteroids and NSAIDs bring out the importance of timely recognition and management of inflammatory complications in snakebite victims. This case emphasizes the need for a multidisciplinary approach involving toxicologists, rheumatologists, and infectious disease specialists to optimize outcomes in such atypical presentations. Future research should aim to elucidate the pathophysiological mechanisms linking snake venom to autoimmune and inflammatory responses, which could provide insights into better diagnostic and therapeutic strategies. This report also serves as a reminder to clinicians to maintain a high index of suspicion for uncommon sequelae of snakebites, especially in endemic regions where timely intervention can significantly alter patient outcomes.

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