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PARAPARESIS AND PARAESTHESIA FOLLOWING A BLACK ANT STING

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ABSTRACT

Hymenoptera stings can lead to a different range of local and systemic reactions. However, rare and unexpected reactions can develop and may be life threatening. In this paper, we present a 29-year-old female who developed anaphylactic shock followed by paraparesis and paraesthesia after a black ant sting. Her symptoms completely resolved after treatment with plasma exchange. Several studies have linked these neurological complications to delayed immunological reactions that are triggered by insect antigen.

KEYWORDS

Black ant sting, anaphylaxis, paraparesis and paraesthesia

LEARNING POINTS

- Environmental antigen exposure can present with immediate and delayed reactions.
- Weakness and paraesthesia are an unusual presentation reported after an ant sting that might be triggered due to an immunological response to an antigen, or antigen that directly causes neurotoxicity.
- Plasmapheresis might be an option for management of unexplained acute paraparesis and paraesthesia.

INTRODUCTION

Hymenoptera is a very large order of insects that includes different types of ants, wasps, bees and sawflies[1,2]. Exposure to Hymenoptera stings may cause various toxic and immunological reactions. These reactions may vary from mild and local reactions to severe and life-threatening reactions[1-3]. Anaphylactic shock is the most dangerous and life-threatening reaction and may result in multiorgan failure[2]. However, Hymenoptera stings may lead to rare and unusual complications including cardiac, renal, hepatic, haematological and neurological complications. Neurological complications may manifest as neuritis, ischemic stroke, encephalomyelitis[1-5].

These complications may occur hours to days after exposure to Hymenoptera stings but may take up to weeks to manifest[3-5]. In this paper, we report the case of a young female who presented with paraparesis after she was stung by a black ant.

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CASE DESCRIPTION

A 29-year-old female, who was previously healthy, presented to emergency room (ER) after she was stung by a black ant on her right shoulder. She presented initially with sudden swelling in her face, breathing difficulty, nausea, vomiting, and a generalized body rash. Her symptoms started one hour after she was exposed to a black ant sting. When she arrived at the ER, her blood pressure (BP) was 89/49 mmHg, her heart rate (HR) was 140 beats per minute (bpm), her respiratory rate (RR) was 26 breaths per minute and her oxygen saturation was 96% on room air. She was given intramuscular epinephrine 0.5 mg, intravenous (IV) methylprednisolone 120 mg, 5 mg nebulized salbutamol and IV diphenhydramine 50 mg by ER team. Her condition improved after treatment, and her vital signs were stable. After initial treatment, her BP was 118/76, her HR 109 bpm, her RR 19 breaths per minute and her oxygen saturation 98% on room air. The patient was admitted for observation. Initial investigations upon admission including complete blood count, renal function profile and liver function profile which were within normal range. Four hours after admission, the patient started to complain of tingling on her lower limbs that progressed to heaviness in both legs, difficulty standing and moderate to severe lower back pain. On examination she was alert and oriented to time, place and person, fully cooperative, and cranial nerves were intact. Muscle power was 5/5 in both upper limbs with normal tone, preserved +2 reflexes, and normal pinprick sensation. Examination of her lower limbs revealed muscle weakness and heaviness with concomitant painful sensation upon movement. Muscle power of her hip flexors was 2/5 bilaterally, hip extensors 2/5 bilaterally, knee flexors and extensors 3/5 bilaterally, ankle dorsiflexors and planter flexors 3/5 bilaterally. Sensory examination showed decreased pinprick, proprioception and vibration sensation distally up to both mid-legs. Muscle tone examination showed hypotonia in both lower limbs. Deep tendon reflex examination showed +1 reflex (preserved but diminished) in bilateral patellae and Achilles tendons.

The patient was admitted to the intensive care unit (ICU) to prevent any further deterioration and for monitoring her vital signs and neurological status. A lumber puncture was done, and cerebrospinal fluid (CSF) was sent for cell count, meningitis multiplex, CSF culture, protein and glucose which were all normal. Whole spine and brain magnetic resonance imaging (MRI) with contrast was unremarkable. The day after, a nerve conduction study (NCS) and electromyography (EMG) were done with normal results. A multidisciplinary team including internal medicine, neurology, toxicology and ICU teams decided to start treatment for presumptive neurotoxicity as a result of black ant sting. The patient was started on plasma exchange for 5 sessions with significant improvement of her symptoms. After completion of therapy, lower limb muscle power improved to be 5/5 in both limbs, paraesthesia and lower back pain resolved and the patient was able to walk independently. The patient was discharged in good condition. When she was seen one week later in the neurology clinic, she was in good health with no more weakness or numbness. Her physical examination was completely unremarkable.

DISCUSSION

Different local and systemic reactions can be caused by hymenoptera stings. These reactions can be classified into local reactions such as skin erythema and swelling, systemic reactions such as toxic and anaphylactic reactions and unusual reactions like neurological complications. These neurological complications may include polyneuropathies, Guillain-Barré syndrome, cerebral infarction, autoimmune encephalomyotonia and encephalomyelitis. While most of acute and systemic allergic reactions caused by insect stings are well known as most of them are IgE mediated venom hypersensitivity, unusual reactions and neurological complications are rarely reported in the literature and of unknown pathophysiology[1-5]. Some studies have hypothesized that these complications are due to direct toxicity by hymenoptera venom that contains active amines, amino acids, polypeptides, phospholipase A2 which has direct neurotoxic activity as it blocks transmission on neural synapses and hyaluronidase which breaks hyaluronic acid in the extracellular matrix leading to spread of venom into the bloodstream, while other studies have indicated that it is related to delayed immunological reactions to insect antigen[6-8]. Peter Bánovčin et al. reported a case of a10-year-old boy who presented with quadriparesis 24 hours after a wasp sting. The patient's clinical status significantly improved after starting IV dexamethasone[6]. Moreover, Likattanasombut et al. reported a case of young man who presented with quadriplegia and coma after a wasp sting. His clinical condition significantly improved after initiation of IV methylprednisolone and plasma exchange[7]. Ridolo et al. have described the delayed autoimmune reaction using in vitro test in a 51-year-old male who developed acute polyradiculoneuropathy after he was stung two times by a paper wasp[8]. Additionally, Liu et al. have identified the major risk factors that may lead to development of severe manifestations after wasp stings. These risk factors include the number of was stings, serum lactate dehydrogenase level, serum total bilirubin and tea coloured urine. They developed a validated severity score called wasp sting severity score (WSS). A score of ≥3 points is considered ahigh score, and the patient should be considered a severe case. The authors recommended early treatment with plasma exchange for patients who are classified as severe cases[9]. Our patient presented initially with anaphylactic shock which responded immediately to standard therapy including epinephrine, IV steroids and diphenhydramine but later she developed these unusual neurological symptoms consisting of lower limb weakness and paraesthesia. Given that such a presentation is extremely rare, we initially focused on common causes that might explain her symptoms such as autoimmune diseases, demyelinating diseases, central nervous system infections and neuromuscular diseases. We did whole spine MRI

with contrast, brain MRI with contrast, lumbar puncture, NCS and EMG which were all normal. The unremarkable workup was reassuring for us but made it difficult to decide about management. After discussion and after reviewing the literature, a multidisciplinary team decided that the most likely explanation for the patient's clinical course was neurotoxicity or delayed immunological reaction to insect antigen and we decided to start treatment with plasma exchange in order to prevent further deterioration of the patient's condition. Fortunately, the patient responded very well to plasma exchange and significantly improved with complete resolution of her neurological symptoms.

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