Severe quadriparesis caused by wasp sting

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Besides the typical symptoms of allergic reaction after wasp sting, unusual and unexpected reactions may also develop. In this report, a case of severe peripheral quadriparesis and sphincteric disorder (urinary incontinence) in a 10–year-old boy occurring within 24 hours after wasp sting is presented. Corticosteroids had very good therapeutic effect, and improvement in clinical status was observed within 72 hours. The exact pathogenic mechanism of peripheral nervous system damage is not very well known. Several studies have suggested that besides the neurotoxic effect of wasp venom, delayed immunological response to wasp antigens followed by an allergy-triggered autoimmune reaction is possible. Wasp venom may activate an allergic reaction or effects by toxic impacts; however, typical clinical symptoms of allergic reaction are not necessarily present.

Key words: children, quadriparesis, sphincteric disorder, wasp.

Wasps together with hornets and bees are members of the order Hymenoptera. Their sting can induce a variety of responses - from slight local reactions to severe life-threatening events. Hymenoptera venom can activate allergic reaction or effects by toxic impacts; however, typical symptoms of allergic reaction are not necessarily present. It is well known that wasp and bee venoms belong to the strongest animal allergens. Wasp venoms show variability in their composition among species. In general, wasp venom consists of three bases – active amines (serotonin, histamine, tyramine, epinephrine and dopamine) and amino acids (tryptophan, histidine), enzymes and polypeptides (vespakinin, mastoparan and chemotactic peptides). The major allergens in wasp venoms are phospholipase A2, hyaluronidase and antigen 5. Phospholipase A2 acts as a cytotoxin and an indirect cytolysin and also has neurotoxic activity by blocking neurotransmission on neural synapse.

We report a case of severe peripheral quadriparesis in a 10-year-old boy occurring after wasp sting.

Case Report

A 10-year-old boy with weakness in his upper extremities and difficulties with standing and walking was presented for evaluation.

He was born at term as a fifth child after physiological pregnancy. His morbidity was appropriate and he did not suffer from any chronic disease or take any drugs. There was no previous clinical history of allergic reaction. He had been bitten by a tick nine months before without any local or subsequent systemic reaction. In the previous month, before the development of clinical signs, the patient had no health problems or accidents and he had not been vaccinated.

His older sister was treated for epilepsy; his father had diabetes and suffered three strokes at the age of 53 years.

The patient had been stung by a wasp, and local reaction on his right hand developed within 24 hours. He felt well and had no other complaints. The local reaction had regressed slightly after local application of antihistamine (dimetindeni maleas 0.1%). After 28 hours, weakness in his upper extremities developed and he soon had difficulties with standing and walking. He complained of mild persistent headache. After 32 hours, he was admitted to a regional Department of Pediatrics. He was afebrile and conscious, and vital signs were normal. Physical examination revealed allergic swelling on his right hand. Neurological

evaluation showed peripheral paraparesis of the lower extremities, accented on the right side, and minimal weakness of the upper extremities. Upper and lower meningeal signs were negative, and there were no sensitive deficit, paresthesias or leg pain. In laboratory tests on admission, inflammatory markers and complete blood cells count were within normal range, whereas creatinine phosphokinase (11.91 μkat/L; normal value: 0-4.12 μkat/L) and aspartate aminotransferase (0.85 μkat/L; normal value: 0.1-0.58 μkat/L) were elevated in blood chemistry. Intravenous anti-allergic (antihistamine, systemic corticosteroids) and polyvitamin therapy were started immediately. However, the disease had progressed despite therapy. After 40 hours, quadriparesis and sphincteric disorder (urinary incontinence) had developed.

The patient was referred to our department on the second day after the wasp sting. The general physical examination was normal at admission. Neurologically, he was alert and awake, without objective sensory abnormalities and paresthesias, and cranial nerves innervation was intact. He had diffuse muscle weakness, evident on lower extremities and less marked on upper extremities. Deep tendon reflexes were not present on lower extremities, and were markedly decreased on upper extremities. Sphincteric disorder - urinary incontinence developed. Guillain-Barré syndrome, central nervous system inflammatory disease, toxo-allergic polyradiculoneuropathy, and neuroborreliosis were taken into consideration in the context of differential diagnosis. In laboratory tests, persisted inflammatory markers and complete blood cells count were within the normal range, while elevated levels of creatinine phosphokinase and aspartate aminotransferase were determined. Urinalysis and evaluation of renal function revealed no abnormality. Kidney and urinary tract ultrasound revealed no pathology. The immunological examination showed combined immunodeficiency of both specific and non-specific cellular immunity (decreased absolute T-cell count and phagocytic activity), and immunoglobulins G, A and M levels were within physiological ranges. Polymerase chain reaction (PCR) analysis for neurotropic viruses (influenza A, B; herpes simplex virus 1, 2; varicella-zoster virus; Epstein-Barr virus; cytomegalovirus) and serology for Borrelia (IgG, IgM) were negative. Total IgE levels were not elevated and venom-specific IgE levels to wasp and bee were low (class I positivity, which usually does not cause reactions), and also again two months after wasp sting. A lumbar puncture was done within 48 hours after wasp sting. The cerebrospinal fluid was clear and liquor pressure was normal. Basal chemistry tests - cell counts (erythrocytes 2; normal value (N): 0-5/3; polymorphonuclear cells 0, N: 0-3/3; lymphocytes 2, N: 0-5/3), glucose (2.6 mmol/L, N: 1.8-4.6 mmol/L), chloride (121 mmol/L, N: 113-131 mmol/L) and total protein (0.27 g/L, N: 0.10-0.45 g/L) were within the normal range, without albumin-cytologic dissociation. Electrophoresis of cerebrospinal fluid proteins detected elevated albumin (0.608 U; N: 0.371-0.459 U) and alpha 2 globulin (0.077 U; N: 0.035-0.063 U) levels. The cerebrospinal fluid culture was sterile. Imaging studies (magnetic resonance imaging [MRI] scan of the brain and spinal column, MRI-angiography of brain blood vessels) did not reveal any pathology. Electromyogram performed on the fifth day after the wasp sting showed normal findings without any signs of peripheral nervous system neuropathy, with the exception of the F wave absence (the absence of this graphoelement is considered to be a non-specific sign of polyneuropathy). We had proceeded with corticotherapy (dexamethasone 1 mg/kg/day intravenously in 4 divided doses for the first 4 days and then successive detraction of the dose for the next 10 days), neuroprotective therapy and rehabilitation. After 72 hours, we observed slow improvement in his clinical state –motility of first the upper and then of the lower extremities improved. Mild headache completely disappeared within two weeks. He was discharged after three weeks with a diagnosis of toxo-allergic polyradiculoneuropathy caused by wasp sting. Neurological findings included mild peripheral paraparesis of lower extremities accented on the right side and urinary incontinence; laboratory tests were normal.

Discussion

Reactions to insect stings are a frequent medical problem. The purpose of the diagnostic procedure is to classify a sting reaction by history, identify the underlying pathogenic mechanism, and identify the insect. Prevalence of Hymenoptera sting reactions varies. Reactions

to Hymenoptera stings are classified into normal local reactions, large local reactions, systemic toxic reactions, systemic anaphylactic reactions, and unusual reactions. Most of the studies reported that large local reactions occur in 10.6-26.4%[1, 2] and systemic reactions in 0.66-3.3%[1-3] of the general population. Venom hypersensitivity may be mediated by immunologic mechanisms (IgE-mediated or non-IgE-mediated venom allergy) but also by non-immunologic mechanisms, which are responsible for a minority of insect sting reactions. They are mediated by short-term sensitizing IgG antibodies or complement activation by IgG-venom immune complexes[4]. Negative venom-specific IgE could also be due to insufficient sensitivity of tests used or to a long interval from the sting-induced reaction to the testing with spontaneous decline in venom-specific IgE.

Local and acute systemic allergic reactions caused by wasp stings are well known. Unusual and unexpected reactions, which can develop from within a few hours to weeks, have been rarely reported in relation to wasp sting. Most often, renal diseases have been described following single or multiple wasp stings. Acute renal failure after wasp sting, which is the most frequent event, is caused by acute tubular necrosis in consequence of hemolysis or rhabdomyolysis[5], direct nephrotoxic effect[5] or acute interstitial nephritis due to hypersensitivity reaction to the wasp venom[6,7]. Wasp venom can seldom induce acute myocardial infarction. Cardiovascular symptoms are provoked by vasoactive, inflammatory and thrombogenic components of wasp venom; allergic reaction is not necessarily present[8,9]. Other reactions, such as rhabdomyolysis[5] or lupus erythematosus were also rarely mentioned in the literature. Neurological complications after wasp sting are rarely reviewed in the literature. They generally develop from within a few hours to weeks after the sting. Reactions such as Guillain-Barré, myasthenia gravis, multiple sclerosis, cerebral infarction, encephalomyelitis, neuropathies (polyneuropathy or optic neuropathy), and demyelinating diseases have been observed[10-15]. A few cases of polyneuropathy after wasp sting are known. Agarwal et al.[16] presented a case of progressive generalized weakness and quadriparesis of three hours duration in a 30-year-old man after wasp sting. Anti-allergic therapy was given nine hours after the accident. However, symptoms appeared after five hours and were probably caused by acute inflammatory demyelinating polyneuropathy following hypersensitivity to the wasp sting. Likattanasombut et al.[13] described development of encephalomyeloradiculopathy with impaired consciousness and quadriplegia in an 18-year-old man 16 days after wasp sting. His clinical status improved after methylprednisolone treatment and plasma exchange. Ridolo et al.[15] reported a case of a 51-year-old man with acute polyradiculoneuropathy occurring 40 hours after a second wasp sting. After the first wasp sting, the man underwent venom immunotherapy. He had muscle weakness, paresthesias, and difficulties in standing up and walking. Skin tests and specific IgE measurement confirmed IgE-mediated allergy to wasp.

The exact pathogenic mechanism of peripheral nervous system damage after wasp sting is not very clear. It was hypothesized that besides the neurotoxic effect of wasp venom, delayed immunological response to wasp antigens followed by an allergy-triggered autoimmune reaction could be responsible for the clinical reactions[13,15]. Corticosteroids showed very good therapeutic effect in previous cases.

In this case report, we want to stress that besides the typical symptoms of allergic reaction after wasp sting, unusual and unexpected reactions may also develop. To our knowledge, this is the first report of sphincteric disorder following wasp sting as a part of a complex clinical presentation in the Central European region. A possible explanation for the incontinence could be direct toxicity of wasp venom, or this clinical sign could be the consequence of a slowly developing immune reaction, although sufficient explanatory data in the literature does not exist.

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