



Letter

A case of vibratory anaphylaxis

Vibratory angioedema and urticaria are rare conditions that have been well documented in the literature.¹ Hereditary, acquired, and occupational forms of vibratory angioedema exist.² Identified stimuli include massage, toweling, motorcycling, lawn mowing, horseback riding, or jackhammer use.¹ Although vibration is a well-known cause of physical urticaria and angioedema, there is no clear published report of vibration as a primary cause of anaphylaxis.

There have been several previous case reports documenting vibratory angioedema with associated systemic symptoms. A 1972 report described an episode of vibratory angioedema associated with the systemic symptoms of headache and generalized erythema.² Another report documented a case of angioedema precipitated by exercise or vibration that was accompanied by dizziness, tachycardia, and hypotension.³ Most recently, a single case of venom-dependent, vibration-induced anaphylaxis was described.⁴ This was a case of severe anaphylaxis 18 hours after a wasp sting with a large local reaction. In this letter, we report a case of vibration as the primary cause of anaphylaxis.

A 53-year-old woman with hypertension, diabetes mellitus type 2, and hyperlipidemia used a vibratory massage bed for 10 minutes. The massage bed used water pressure to create vibration without any physical water contact. The massage bed was heated and had a latex-free plastic covering. Within minutes of stopping the massage bed, she developed diffuse pruritus, followed by diffuse flushing, nausea, vomiting, and presyncope. The patient was immediately brought to the emergency department (ED).

The patient had no history of anaphylaxis, food, or latex allergies. She also denied any new foods, medications, or substance ingestions. There was no exposure to Hymenoptera. This episode occurred in January in Minnesota, not in a season with stinging insects or in an area endemic for fire ants. She had been taking lisinopril for the past 3 years. She was taking aspirin, 81 mg/d, but no other nonsteroidal anti-inflammatory drugs or pain medications. The patient used the massage bed at work, where it was available to employees. Approximately 1 hour 21 minutes after symptom onset, her blood pressure was 64/32 mm Hg, heart rate was 59/min, oxygen saturation was 96% on room air, respiratory rate was 20/min, and temperature was 36.4°C. She had a witnessed syncopal episode and emesis while in the ED. Her physical examination revealed diffuse skin flushing and 1-mm papules in the interdigit spaces, but findings were otherwise unremarkable. She had a history of pustular psoriasis vs dermatitis on her hands, which may have facilitated allergen entry across the epithelial barrier. She was treated with intramuscular epinephrine, 0.3 mg; intravenous methylprednisolone, 125 mg; intravenous diphenhydramine, 25 mg; intravenous

ondansetron, 4 mg, and 2 L of intravenous 0.9% sodium chloride for suspected anaphylaxis. She responded to treatment appropriately with an increase in blood pressure and improvement of symptoms. Her heart rate was low on presentation at 59/min, with a subsequent increase to 91/min with treatment. Her baseline rate was 64/min. It is unknown whether there was any tachycardia preceding her presenting heart rate of 59/min. The patient was observed overnight in the intensive care unit without recurrence of symptoms. Treatment with lisinopril was discontinued, and hydrochlorothiazide therapy was used for hypertension management.

On the basis of the National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network anaphylaxis diagnostic criteria, this patient met the clinical criteria for anaphylaxis.⁵ She had acute onset of skin involvement (flushing) associated with persistent gastrointestinal symptoms and documented hypotension.

Her serum tryptase level 8 hours after the event was 8.29 ng/mL (reference range, <11.5 ng/mL). Subsequent tryptase levels at 3 and 9 months were normal at 4.25 ng/mL and 2.98 ng/mL, respectively. In view of the 2011 consensus proposal regarding the definitions, criteria and global classification of mast cell disorders with special reference to mast cell activation syndromes, which specified a significant change in serum tryptase as an “increase in serum total tryptase by at least 20% above baseline plus 2 ng/mL,” the serum tryptase level of 8.29 ng/mL was considered significantly elevated when compared with the convalescent baseline values.⁶

On further workup, the results of serum specific IgE antibodies to all foods ingested the evening of her anaphylaxis episode and latex were negative. Skin prick test results to a panel of foods and latex were negative 3 months after the anaphylactic event. These results did not reveal food or latex as a cause of anaphylaxis. No Hymenoptera sting was reported. The levels of *N*-methylhistamine and β -prostaglandin $F_{2\alpha}$ collected in the urine 9 months after the anaphylaxis episode were within normal limits. Mastocytosis was considered to be of a low probability based on these results.

The patient experienced 2 subsequent episodes of vibration-induced angioedema with symptoms that included hand and foot edema, pruritus, and lightheadedness. One of the subsequent episodes also occurred after use of the massage bed. These episodes possibly meet the definition of anaphylaxis⁵ because there was cutaneous involvement and symptoms of reduced blood pressure (lightheadedness). However, these episodes were less severe, and there was no documented reduced blood pressure. The patient was instructed to avoid vibratory stimuli and carry epinephrine. With vibration avoidance, she had no reported episodes of anaphylaxis in the following 3 years.

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The anaphylactic episode occurred after the patient had gone for a short walk followed by use of the massage bed; therefore, exercise-induced anaphylaxis with vibration as a cotrigger could be considered. However, the patient's walk was short and minimally strenuous. Exercise-induced anaphylaxis is triggered most frequently by jogging (78%) or brisk walking (42%), according to a study of 279 patients.⁷ The patient's activities were not further modified beyond vibration avoidance. She continued to exercise after the episode of anaphylaxis for an hour and a half 3 days per week, avoiding vibratory stimuli, without any further episodes in the following 3 years.

In total, this patient had 1 episode of anaphylaxis and 2 episodes of angioedema associated with vibration. The results of workup for other potential triggers and mastocytosis were negative. This case is consistent with a physical trigger of anaphylaxis secondary to mast cell and possibly basophil degranulation. Recent literature revealed that vibration alone can cause significant degranulation in susceptible mast cells, suggesting that a purely mechanical trigger could cause an allergy-like response,⁸ such as angioedema or possibly anaphylaxis. Our case most likely represents a confirmed episode of anaphylaxis with vibration as the primary trigger. However, multifactorial anaphylaxis with vibration as a trigger should also be considered. The World Allergy Organization acknowledges that some episodes of anaphylaxis are multifactorial.⁹ However, the recent World Allergy Organization and Joint Task Force Practice parameters do not recognize vibration as a trigger or cofactor of anaphylaxis.^{9,10} This report provides strong evidence that vibration is a primary trigger and potential cofactor for anaphylaxis.

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