Rom J Leg Med [33] 124-127 [2025]
DOI: 10.4323/rjlm.2025.124

SUDDEN DEMISE AFTER HORNET ENVENOMATION: A DUAL CASE REPORT

RunWu Chen[1], Di Liang[2], Hongyu Su[3,4], FeiFeng Chen[1], LiJie Su[1], Xian Ju[2,*]

[1]Nanning Zhongyi Forensic Science Institute, Nanning, Guangxi, [2]School of Forensic Medicine, Shanxi Medical University, Jinzhong, Shanxi, [3]Department of Human Anatomy, West China School of Basic Medical Sciences and Forensic Medicine, West China Second University Hospital, Sichuan University, Chengdu, Sichuan, [4]West China School of Basic Medical Sciences and Forensic Medicine, Sichuan University, Chengdu, Sichuan, China

Abstract: Bee venom is a biological substance produced by bees, consisting of various components. Once these components penetrate the human body, their numerous constituents complicate the detection and characterization of each individual component. Given the complexity of the legal and clinical implications of bee sting incidents, this report presents two cases of fatal anaphylactic shock following bee stings. Through the analysis of these cases, we aim to provide forensic pathological examination and biochemical indicators that can serve as references for handling similar causes of death.

Keywords: forensic case, bee venom, anaphylactic shock, allergy.

INTRODUCTION

Bee venom, a multifaceted biological substance crafted by honeybees, presents considerable health risks to humans, particularly due to its intricate array of components, which encompass proteins, peptides, enzymes, and amines (1). The composition of bee venom is not only species-specific but is also subject to the influences of environmental factors, rendering the establishment of a standardized profile for its constituents a formidable challenge. Among these constituents, melittin, phospholipase A, and apamine assume critical roles in provoking inflammatory responses, disrupting cellular membranes, and modulating immune functions (2-4).

The perils associated with bee venom are especially pronounced, stemming from two principal factors. First, the complex composition of bee venom complicates the detection and characterization of its myriad components once they infiltrate the human body (5). Current diagnostic methodologies fall short in their ability to effectively quantify or identify the diverse constituents of bee venom, presenting significant challenges in clinical settings when managing cases of envenomation. Second, individuals subjected to bee stings may endure severe, life-threatening allergic reactions, most notably anaphylactic shock (6). This hypersensitivity often manifests with alarming rapidity, necessitating immediate medical intervention to alleviate its potentially fatal ramifications.

The pathophysiological mechanisms that govern the hypersensitivity induced by bee venom, are intricate and not wholly understood. Upon exposure, individuals who have been sensitized may find themselves ensnared in a cascade of immune responses, wherein histamines and various mediators are unleashed, collectively orchestrating a symphony of cardiovascular collapse and respiratory distress (5). This complexity complicates the timely diagnosis and treatment of reactions and highlights the urgent need for increased awareness of bee venom's effects. Given the intricacies surrounding the legal and clinical implications of bee sting incidents, this report presents two fatal cases of anaphylactic shock following bee stings. Through the analysis of these cases, we aim to enhance the understanding of the underlying

*Correspondence to: Xian Ju, School of Forensic Medicine, Shanxi Medical University, Jinzhong, Shanxi, China, E-mail: rainbowx1024@163.com

mechanisms by which bee venom induces severe allergic reactions, thereby contributing to the field of forensic medicine.

CASE REPORT

According to the coroner's report, a male decedent suddenly collapsed, became unconscious, and displayed foaming at the mouth while applying fertilizer to a tree. He was subsequently transported to the hospital, where resuscitation efforts proved ineffective, leading to his death. The deceased exhibited multiple red spots on the facial region, accompanied by surrounding tissue edema (Fig. 1). Significant swelling was also observed from the area behind the left ear extending down to the neck.

The autopsy revealed an enlarged thyroid gland on the right side. The larynx exhibited congestion and edema, with a small amount of bloody fluid present in the trachea and bronchi (Fig. 2). Pathological examination demonstrated inflammatory cell infiltration within the dermal layer of the right cheek, accompanied by vascular congestion and the presence of multinucleated giant cells (Fig. 3). In the subcutaneous tissue, focal lymphocyte infiltration was observed, along with degranulation of mast cells. The lamina propria of the laryngeal mucosa showed loosening and a mild infiltration of lymphocytes, while the submucosal layer exhibited mast cell degranulation (Fig. 4). Alveolar interstitial edema accompanied by the degranulation of mast cells. Vacuolar changes in the proximal convoluted tubules associated with the engorgement of the interstitial capillaries in the kidneys. The total serum IgE level was determined to be 234.30 IU/mL (reference range: adults 0.00-100.00 IU/mL).

Another male victim was stung on the head by a bee while working outdoors. Within minutes, the individual experienced dizziness, chest tightness, and difficulty breathing. He was subsequently transported to the hospital; nevertheless, despite attempts at resuscitation, he succumbed.

The autopsy revealed a red spot on the left side of the neck, surrounded by slightly swollen soft tissue (Fig. 5). Edema of the epiglottis and larynx was

Figure 1. Erythema on the right cheek of the deceased.

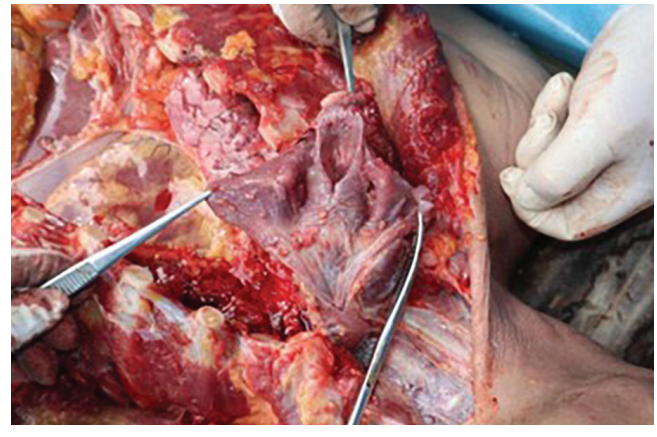

Figure 2. Anatomy of the deceased's larynx.

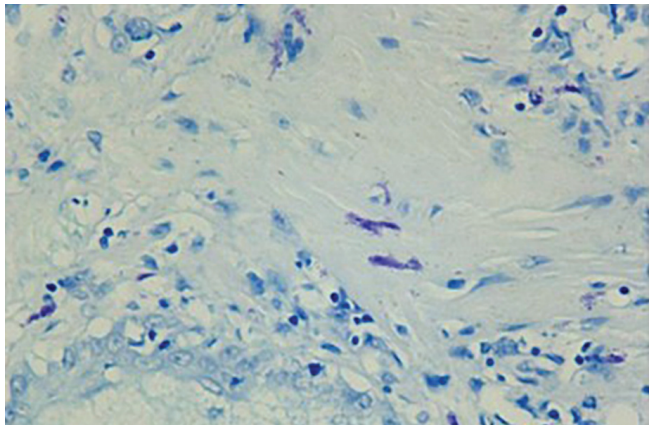

Figure 3. Degranulation of mast cells in the submucosal layer of the right cheek skin.

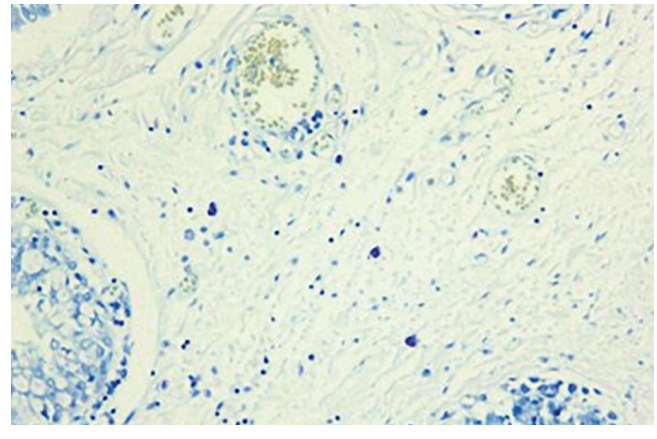

Figure 4. Degranulation of mast cells in the submucosa of the larynx.

observed, alongside hemorrhaging of the pharyngeal mucosa, and a small amount of sanguineous fluid was noted in the trachea and bronchial lumens (Fig. 6). Microscopic examination showed loosening of the lamina propria in the laryngeal mucosa, congestion of small blood vessels, and degranulation of mast cells (Fig. 7). The lamina propria of the tracheal mucosa appeared slightly loosened, with congestion in the small blood vessels within both the mucosal and submucosal layers. Vascular congestion was also apparent within the alveolar septa. The gastric submucosa exhibited congestion of small blood vessels, with a few mast cells undergoing degranulation, while a limited number of mast cells within the intestinal wall also showed degranulation. There was notable vascular congestion in the neck region, with considerable degranulation of mast cells evident (Fig. 8). The serum sent for analysis revealed a total IgE level of 35.37 IU/mL.

In conclusion, the forensic assessment for the two victims determined that their deaths were caused by anaphylactic shock induced by bee stings.

DISCUSSION

Bee venom is a complex mixture composed of various components, including peptides, amines, enzymes, amino acids, and trace elements (6). Notably, the venom contains over 55 different enzymes, with bee venom peptides accounting for approximately 40-60% of the dry venom and serving as the primary pathogenic factors (7). The venom exhibits hepatotoxicity, nephrotoxicity, and neurotoxicity, which may manifest clinically as intravascular hemolysis, acute renal failure, rhabdomyolysis, and coagulopathy (8, 9). Furthermore, the antigenic components found in bee venom can induce type I hypersensitivity reactions, which in severe cases may lead to anaphylactic shock and even multiple organ dysfunction syndrome (MODS), resulting in death (10).

Fatalities resulting from bee stings are commonly reported in everyday forensic work (11, 12). The intricate composition of bee venom poses significant challenges in establishing direct laboratory detection methods after exposure. Thus, a conclusive

Figure 5. Erythema on the left side of the neck of the corpse, accompanied by soft tissue swelling.

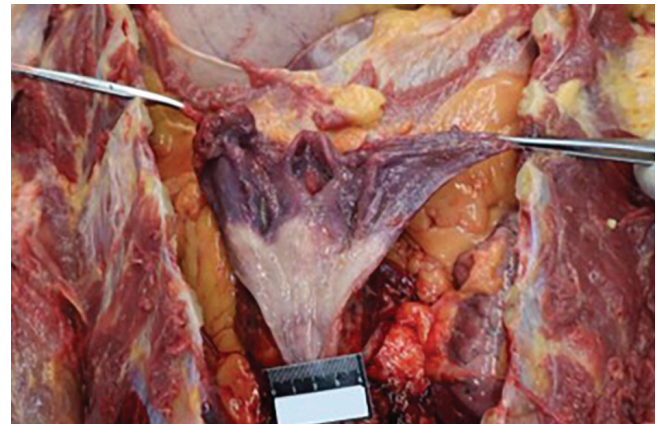

Figure 6. Edema of the epiglottis and larynx, accompanied by pharyngeal mucosal bleeding.

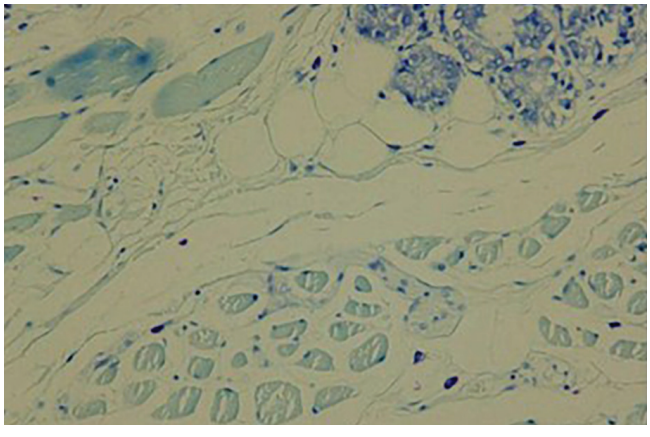

Figure 7. Degranulation of mast cells in the mucosa and muscular layers of the larynx.

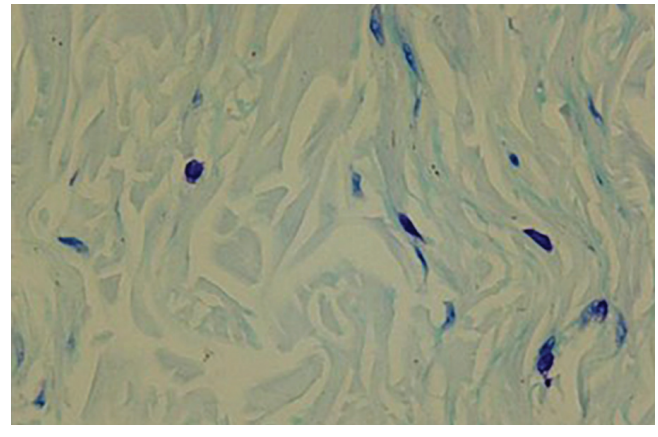

Figure 8. Degranulation of mast cells in the subcutaneous tissue of the neck.

diagnosis often involves a multifaceted approach that integrates necropsy, histopathological analysis, laboratory physicochemical assessments, and field investigations. Autopsy findings in both Case 1 and Case 2 displayed multiple red punctate sting marks on the surface of the body, along with localized swelling, edema of the epiglottis and larynx, and a small quantity of blood-stained fluid present in the trachea and bronchi. Microscopic examination revealed bronchial constriction, pulmonary congestion, and edema, alongside observed degranulation of mast cells within various tissues and organs, indicative of pathological alterations characteristic of anaphylactic shock. Furthermore, histological analysis did not reveal any signs of myocardial ischemia or hypoxia. The contextual details of the cases and the medical histories were not aligned with manifestations typically seen in sudden cardiac death related to coronary heart disease, thereby excluding the possibility of death being associated with coronary atherosclerosis. Collectively, these findings confirm that the cause of death was due to anaphylactic shock resulting from bee stings.

Allergic reactions to bee stings fall under the category of IgE-mediated type I hypersensitivity reactions(13). Typically, an increase in serum IgE levels is viewed as a supportive biochemical test for allergic sudden death (14). Nevertheless, studies have shown that postmortem blood IgE concentrations can decline due to the degradation and denaturation induced by bacteria, fungi, and other decomposing factors. Consequently, it can be inferred that the lack of a notable increase in total serum IgE in Case 2 may result from the prolonged interval between death and autopsy, leading to decomposition of the cadaver. Currently, no specific biomarkers have been established to differentiate allergic sudden death from other etiologies, including cardiogenic sudden death. Therefore, the search for reliable biomarkers has become a significant focus in forensic pathology research. The application of multi-omics technologies may represent a novel approach in this field.

In conclusion, for forensic cases that present ambiguous biochemical indicators and pose difficulties in direct identification, it is essential to clearly define the events leading to the incident. A thorough collection of clinical case treatments, alongside meticulous documentation of the disease progression, should be undertaken. Additionally, it is vital to standardize the procedures for autopsies and pathological examinations to prudently determine the cause of death.

Conflict of interest

The authors declare that they have no conflict of interest.

References

1. Habermann E. Bee and wasp venoms, Science. 177(4046); (1972): 314-322.
2. Palm NW, Medzhitov R. Role of the inflammasome in defense against venoms. Proc Natl Acad Sci USA. 2013;110(5):1809-1814.
3. Khalil A, Elesawy BH, Ali TM, Ahmed OM. Bee Venom: From Venom to Drug. Molecules. 2021;26(16):4941.
4. Lee G, Bae H. Bee Venom Phospholipase A2: Yesterday's Enemy Becomes Today's Friend. Toxins (Basel). 2016;8(2):48.
5. Kim W. Bee Venom and Its Sub-Components: Characterization, Pharmacology, and Therapeutics. Toxins (Basel). 2021;13(3):191.
6. Wehbe R, Frangieh J, Rima M, El Obeid D, Sabatier JM, Fajloun Z. Bee Venom: Overview of Main Compounds and Bioactivities for Therapeutic Interests. Molecules. 2019;24(16):2997.
7. Hossen MS, Shapla UM, Gan SH, Khalil MI. Impact of Bee Venom Enzymes on Diseases and Immune Responses. Molecules. 2016;22(1):25.
8. Gong J, Yuan H, Gao Z, Hu F. Wasp venom and acute kidney injury: The mechanisms and therapeutic role of renal replacement therapy. Toxicon. 2019;163:1-7.
9. Bogdanov S. Bee venom: composition, health, medicine: a review. Peptides 1; (2015): 1-20.
10. Rayamane AP, Kumar M, Kishor D. Honey bee stings and anaphylaxis. J. Forensic Med. Sci. Law 23. (2014): 53-60.
11. Singh RR, Kumar S, Rupani R, Yadav PK, Choudhary R, Verma AK, Anwar T. Postmortem Analysis of Lethal Honeybee Stings: A Case Report, Indian Journal of Forensic Medicine and Toxicology. 2024; 18(3).
12. Sethi SS, Jena MK. Bee sting envenomation: rare fatality, Journal of Indian Academy of Forensic Medicine. 2015; 37(2): 202-203.
13. Herbst J, Heath K, Heddle R, Gilbert JD, Byard RW. Multiple bee stings, peritumoral mast cell degranulation and anaphylaxis-is there a relationship? J Forensic Leg Med. 2013;20(6):591-594.
14. Tran L, Palmiere C. Postmortem serum levels of total IgE, International Journal of Legal Medicine. 2016; 130: 1567-1573.