

## DEATH FROM AN INSECT STING: A CASE REPORT AND LITERATURE REVIEW

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**Abstract:** Postmortem diagnosis of anaphylaxis remains a challenge for pathologists due to the complexity of pathogenetic factors and the absence of pathognomonic data. We describe the case of a 57-year-old man where the combination of autopsy findings, immunohistochemical investigation, laboratory examinations with serum tryptase dosage, supported by anamnestic-circumstantial data, provided the necessary elements for postmortem diagnosis of fatal respiratory arrest due to anaphylactic shock caused by a hornet sting.

In addition we illustrate the results of a literature review of the last ten years in which cases subjects died from anaphylactic shock caused by an insect sting.

**Key words:** anaphylaxis, hornet sting, postmortem diagnosis.

### INTRODUCTION

Anaphylaxis is an acute and potentially life-threatening IgE-mediated hypersensitivity reaction that results from the systemic release of vasoactive and inflammatory mediators from mast cells and basophils and occurs after contact with specific allergenic antigens in previously sensitized subjects.

Mast cell degranulation lead to systemic vasodilatation that is associated with a sudden drop in blood pressure and bronchial mucosa edema, culminating in bronchoconstriction, dyspnea, urticaria and unspecific gastrointestinal symptoms such as abdominal pain, cramps, vomiting and diarrhea [1].

Death can occur within minutes, resulting in most cases, from circulatory collapse, respiratory arrest, or a combination of the two.

Postmortem diagnosis of anaphylaxis remains a significant challenge for pathologists due to the complexity of pathogenic factors and the lack of pathognomonic data [2].

In fact, macroscopic and microscopic changes are not specific and often absent [3, 4].

Laboratory parameters for anaphylaxis, such as postmortem serum tryptase, a neural protease released during mast cell degranulation,

and immunoglobulin E, are commonly used in the investigation of suspected anaphylactic deaths, to assist the pathologist in determining of the cause of death. However neither tryptase dosage nor IgE concentration are pathognomonic, since serum elevated tryptase levels are reported in postmortem blood samples also in non anaphylactic deaths [5]. In addition, the effect of the postmortem interval on serum tryptase is not fully understood.

In many cases, the cause of death is often identified on the basis of circumstantial data [6].

We present the case of a 57-year-old man where the combination of autopsy findings, immunohistochemical investigation, laboratory examinations with serum tryptase dosage, supported by anamnestic-circumstantial data, provided the necessary elements for postmortem diagnosis of fatal respiratory arrest due to anaphylactic shock caused by a hornet sting.

### CASE REPORT

#### Case History

A 57-year-old man was gardening with some friends when he claimed to have been stung by an insect.

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He went into his house and after some minutes his friends found him cyanotic on the floor soaked in urine and foaming from his mouth.

Rescuers that arrived later on found him cyanotic, with midriasis and no central pulse, administered cortisone, intramuscular adrenaline and performed active resuscitation for 30 minutes. Despite resuscitative efforts, the subject was declared dead.

The victim had never suffered from allergy or anaphylaxis and he used to work outside as gardener or handyman. Based on what was reported by the fire brigade, that destroyed the insects nest found near the house, the insect that stung the man was a hornet, *Vespa crabro*.

### **Postmortem Findings**

The body did not show any signs of decomposition and, was refrigerated during transport and storage at 2°C. The autopsy was performed 42 hours after death.

During the external examination, a small wheal (5 mm in diameter) with surrounding redness (10 mm in diameter) was observed on the right parietal region of the head.

The autopsy showed edema of brain, pharynx, larynx and trachea with reddish mucus and whitish foam.

Upon opening the chest, the lungs were completely covered the pericardium. The lungs had a regular shape and consistency, increased volume and reddish color. Large and medium bronchi contained abundant whitish foam.

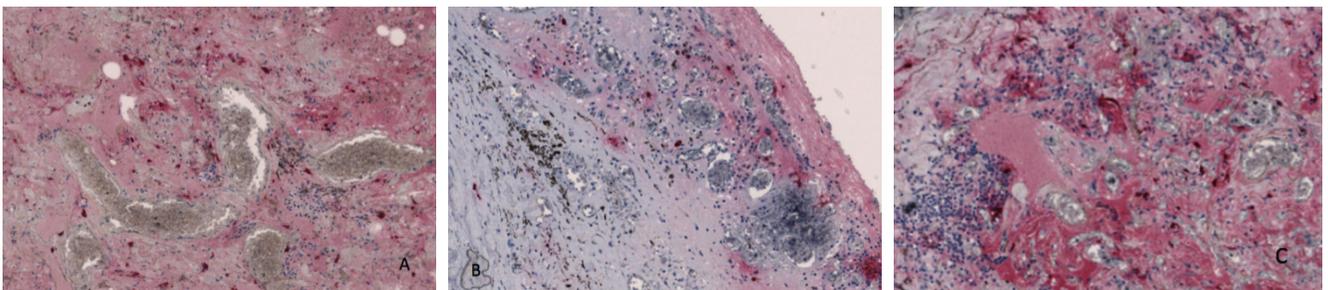
Histological examination showed severe alveolar edema mixed with areas of acute pulmonary emphysema. It also showed edema of the epiglottis, vocal cords and tracheasubmucosal site with vascular congestion (Figs 1 and 2).

Immunohistochemical investigation was performed utilizing antibodies anti-tryptase and anti-mast cells (CD117, c-kit) for lung and skin sections.

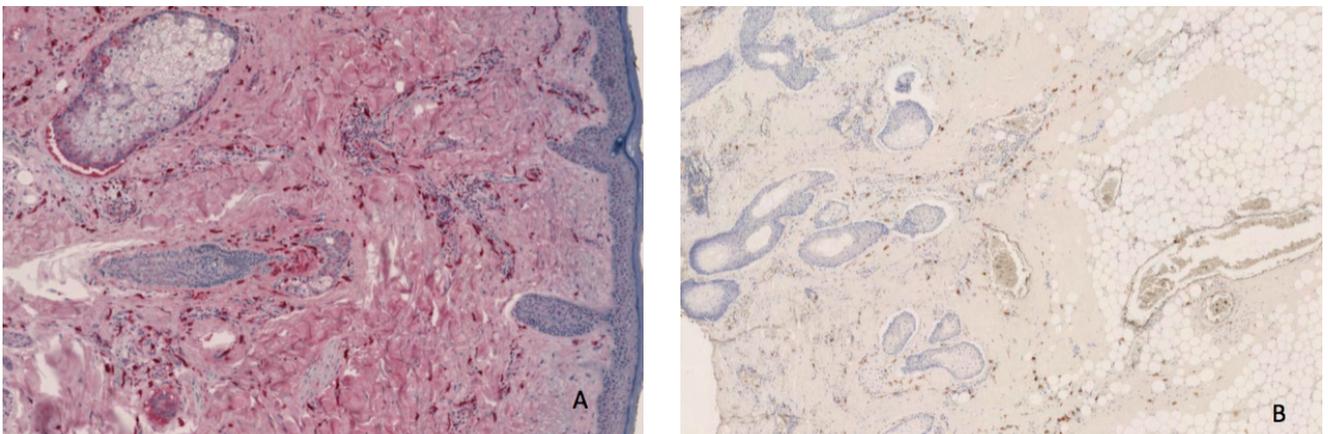
A remarkable number of degranulating mast cells with tryptase positive material outside the cells was documented in the lungs.

Skin's samples from the right parietal region of the head, showed increased interstitial and perivascular mast cells with tryptase-positive material outside the cell.

Postmortem tryptase dosage showed a concentration in blood of 142µg/L. Toxicological examinations carried out on blood taken from femoral arteries during autopsy showed a value of alcohol of 1.61 g/L.



**Figure 1.** Histological images of the lungs. Immunohistochemistry for anti-tryptase.



**Figure 2.** Histological images of the scalp. Immunohistochemistry for anti-tryptase (A) and CD117 (B).

### **Cause of death**

Anamnestic-circumstantial data, external examination evidence, autopsy findings, serum tryptase assay, histological and immunohistochemical investigations provided the necessary elements for postmortem diagnosis of respiratory arrest due to anaphylactic shock caused by a hornet sting.

A further point to consider is that the toxicological data indicated the subject, at the time of death, was in a state of alcohol intoxication; in particular at these concentrations some encephalic areas are affected (deep and midbrain motor area) with a consequent erratic state characterized by motor incoordination and delayed reactions, effects that may have reduced the subject's reactivity and also contributed to the respiratory depression caused by the shock anaphylactic.

### **DISCUSSION**

Death from anaphylaxis is a rare event. In fact, the incidence of fatal anaphylaxis is very low, ranging from 0.12 to 1.06 deaths per million person-years, with the prevalence of deaths in patients with severe anaphylaxis standing at 0.3%-2% [7].

The three leading causes of fatal anaphylaxis are, in descending order, drugs (50% or more), insect stings, and food.

The venom injected from stings stinging by insects is a very common allergy-causing substance and approximately 9.2 to 28.7% of the adult population shows a sensitization to Hymenoptera venom [8].

Stinging insects of the order Hymenoptera are the main cause of insect-related anaphylaxis and cause the most severe hypersensitivity reactions with a high risk of anaphylactic reactions with potentially fatal outcomes [8].

The recurrence rate of Hymenoptera anaphylaxis can be high in geographic areas where exposure to Hymenoptera anaphylaxis is increased (eg. Australia) [7]; in Europe, the most dangerous insects belong to Apidae and Vespidae family.

Fatal anaphylaxis caused by Hymenoptera sting is rare but the true number of fatalities may be underestimated due to the lack of postmortem diagnosis methods of anaphylaxis. The frequency of fatal Hymenoptera anaphylaxis peaks in spring and summer, in line with the annual life cycle of the insects and the prevalence of systemic sting reactions among adults ranges between 0.3 and 7.5% [7, 8].

Insect-induced anaphylaxis is more frequently

fatal in people older than 35-40 years [7] due to a reduced capacity to tolerate the effects of hypoxia, hypotension, and arrhythmia [7].

The diagnosis of anaphylaxis shock caused by insect sting is not easy due to the complexity of the pathogenic factors and the absence of pathognomonic data [1, 2, 4].

In cases of death suspected for anaphylactic shock, first of all, it is necessary, if possible, to collect anamnestic information to verify previous allergies.

External examination can provide useful elements for the diagnosis such as vesicular skin lesions, pustules or remnants of stingers.

The most common pathological findings are pulmonary congestion with or without edema, intra-alveolar hemorrhage, increased tracheo-bronchial secretion, visceral congestion, mucus plugging and hyperinflated lungs, pharyngeal and/or laryngeal edema and petechial hemorrhages indicating asphyxia. However, many cases do not exhibit these changes; in fact in some cases cardio-respiratory shock leads to death so quickly, that there is no time for pathological changes to develop [9].

Histologically, an increase in the number of mast cells and eosinophils may be present [2]. Immunohistochemistry (IHC) is also used for detecting the presence and the degranulation of mast cells in tissues using anti-tryptase antibodies [6].

The determination of certain blood parameters has been a main topic of recent investigations.

The tryptase analysis should be of significant help to pathologists in confirming a suggested diagnosis of anaphylaxis as the cause of death at autopsy. In fact, according to the literature, very high post-mortem values of tryptase are interpreted as a significant finding in deaths resulting from anaphylactic shock [4].

It's proper to collect a blood sample as soon as possible in cases of suspected anaphylaxis, preferably from the femoral vessels to avoid the increase of mast cell-induced tryptase due to prolonged cardiac massage or defibrillation [2, 6].

However, elevated levels of serum tryptase have been reported in blood sample also in non anaphylactic death (trauma, heroin abuse, myocardial infarction, sudden infant death syndrome) [3].

In a recent study of Garland *et al.*, the postmortem tryptase reference range in nonanaphylactic death was established as <23 µg/L (97.5<sup>th</sup> percentile) [10].

Further evidence for diagnosing death by anaphylactic shock can be obtained by IgE dosage,

which remains stable in plasma for a few weeks and provides significant indications about the subject's allergies [4].

Levels of IgE postmortem are also helpful but should be interpreted with caution: a positive IgE value does not confirm that an anaphylactic reaction had taken place, but probably showed that the person was

sensitive to the venom [11].

According to our review of the international literature (Table 1), in the last two decades there were 30 cases of fatal anaphylaxis after Hymenoptera sting. In two of these cases gender and age were not reported. In 17 cases subjects that died from fatal anaphylaxis caused by insect stings were older than 40 years.

Table 1.

Art.	Country	Sex	Age	Insect	Sting site	Hospitalization	Autopsy findings	Tryptase (µg/L)	IgE (µg/L)
Mayer et al. [3]	Austria	M	57	<i>Apis mellifera</i>	One sting into the shoulder	No	Laryngeal edema	880	5.28
Mayer et al. [3]	Austria	M	48	Wasp		No		86	23.52 for <i>Vespula</i> venom, 4.03 for honeybee venom
Sravan et al. [5]	Australia	M	62	Ant		No	Increased interstitial and perivascular mast cells (C-kit positive) in the dermis, swelling and edema of the upper airways with mucosal inflammation, submucosal edema, florid mast cell infiltration microscopically	130	
Toit-Prinsloo et al. [11]	South Africa	M	58	<i>Apis mellifera</i>	5 in the skin, 3 on the upper eyelids and 2 on the cheeks	No	Mild periorbital swelling involving predominantly the upper eyelids	49	56.4
Riches et al. [16]	Australia	M	25	<i>Apis mellifera</i>		No	Minimal facial oedema and minimal laryngeal oedema, mild congestion of the lungs	174	Positive for bee venom
Riches et al. [16]	Australia	M	36	<i>Apis mellifera</i>	7 stings found	Yes	Facial and upper body cyanosis, swelling of the lips and marked laryngeal oedema	2000	Positive for bee venom
Riches et al. [16]	Australia	M	54	<i>Apis mellifera</i>	One sting on the ear	Yes	Upper body and facial cyanosis, moderate laryngeal oedema, lungs were congested and mildly oedematous	>200	Positive for bee venom

Art.	Country	Sex	Age	Insect	Sting site	Hospitalization	Autopsy findings	Tryptase (µg/L)	IgE (µg/L)
Lee <i>et al.</i> [17]	Malaysia	M	11	<i>Vespa affinis indosinensis</i>		Yes			
Perskvist and Edston. [18]	Sweden	M	50	<i>Apis mellifera</i>			Laryngeal edema and pulmonary edema	180	Positive
Rathi Sharmila <i>et al.</i> [19]	India	M	12	Wasp	Single sting	Yes			
Pratish <i>et al.</i> [20]	India	M	22	Numerous wasps (50-100)	Scalp, face, neck, upper limbs and trunk	Yes	Pulmonary, hepatic and splenic congestion		1764,96 on the 5 <sup>th</sup> day
More <i>et al.</i> [21]	USA	F	3 months	<i>Solenopsis xyloni</i>		No	Erythematous lesions without pustules, congested respiratory parenchyma and mild laryngeal edema	23.9	Positive (IgE specific for <i>Solenopsis richteri</i> )
Shen <i>et al.</i> [22]	USA			Bee				200	
Törő <i>et al.</i> [23]	Hungary	F	38	<i>Apis mellifera</i>		No	Laryngeal and tracheal subepithelial capillaries showed minimal dilatation and interstitial edema Petechiae on the skin of the head, face, neck, and limbs, massive pulmonary edema with fluid filling the alveolar spaces, along with neutrophils, macrophages and erythrocytes, cerebral edema		
Zhang <i>et al.</i> [24]	China	F	38	Wasp	Multiple sites (300 stings) all over the body	Yes			
Jung <i>et al.</i> [25]	Korea	F	65	<i>Apis mellifera</i>	Bee sting acupuncture	Yes			
Herbst <i>et al.</i> [26]	Australia	M	58	Bee	One sting the nape of his neck	No	Appreciable swelling of the dorsum of the left hand, pulmonary edema	>200	Positive for bee venom
Tekelioglu <i>et al.</i> [27]	Turkey	F	33	<i>Apis mellifera</i>	Single sting	Yes			
Anolay <i>et al.</i> [28]	Turkey	M	49	Honeybee	All over the body (>50 stings)	No	Pulmonary edema and swelling in laryngeal area		

Art.	Country	Sex	Age	Insect	Sting site	Hospita- lization	Autopsy findings	Tryptase (µg/L)	IgE (µg/L)
<b>Bridi et al.</b> [29]	Brazil	M	56	<i>Apis mellifera scutellata</i>	>1000 stings	Yes	Widespread erythematous and papules		
<b>Douglas and Zanganeh.</b> [30]	Canada	M	45	Wasp		Yes			357.6
<b>Dharma Rao et al.</b> [31]	India	M	40	Honey bee	Multiple sting sites predominantly on the face, neck, trunk and arms	Yes	Liver and lungs congested, inflammatory cells on the brain		
<b>Kularatne et al.</b> [32]	Sri Lanka	F	46	<i>Vespa affinis</i>	40 circumscribed and punctated sting marks on the face, neck, chest, abdomen and limbs	Yes	Blood-stained fluid pleural space, lungs were oedematous and with frothy fluids, kidneys congested, dermal oedema, congested postvenular capillaries and extravasated mast cells, eosinophils and neutrophils		
<b>Kularatne et al.</b> [32]	Sri Lanka	M	48	<i>Vespa affinis</i>	130 punctated sting marks all over the body	Yes	Blood-stained fluid on the chest cavity, lungs congested, dermal oedema, congested postvenular capillaries and extravasated mast cells, eosinophils and neutrophils		
<b>Akyildiz et al.</b> [33]	Turkey	M	10	Honey bee	5989 sting marks all over the body and more prominent on the face	Yes	Severe petechia and ecchymosis on the body		
<b>Kularatne et al.</b> [34]	Sri Lanka	M	59	<i>Xylocopa tranquebarica</i>	In the right malar region of the face	No	Lungs slightly congested and secretions in the bronchi		

Art.	Country	Sex	Age	Insect	Sting site	Hospita- lization	Autopsy findings	Tryptase (µg/L)	IgE (µg/L)
<b>Kanchan et al. [35]</b>	India	F	65	Bee	Circular reddish lesions with black-brown area in the centre distributed over the entire body (approximately 23 in number)	Yes	Cerebral oedema, lungs and kidneys congestion, pulmonary oedema, intra-alveolar eosinophilic fluid		
<b>Vos et al. [36]</b>	Netherlands	F	58	<i>Yellow jacket</i>	Sting marks in the neck, chest and wrist	Yes		1836	1,22 against YJ venom, 5,54 against <i>Vespa vulgaris</i>
<b>Hughes [37]</b>	USA	M	61	<i>Apis mellifera scutellata</i>	500 sting marks on the chest, abdomen, back, shoulders, arms, forearms, hands, face, neck and buttock	Yes	Edema of the aryepiglottic folds and laryngeal mucosa, lungs congested and edematous	71	

In addition, in the included cases 20 decedents were men, according to Simon and Mulla [12] and Jerschow et al. [13] studies found an increased risk for insect-induced anaphylaxis in men, since they are more frequently exposed to insects than women.

The sting site was variable. In 13 of the 30 cases it was not reported.

In our case, the subject was a 57-year-old man. The sting was on the right parietal region of the head, similar to some of the reported cases in which the stings were on the cheeks and on the right malar region of the face.

The cases included in the tabulation of autopsies showed the involvement of the respiratory system with asphyxial signs. The autopsy findings in our case report coincided with those reported in the literature: edema of brain, pharynx, larynx and trachea with reddish mucus and whitish foam were the most common.

The datum to underline is that, in most cases included in our review, the time interval between death and autopsy and blood collection for laboratory investigations was not indicated, making difficult comparisons between cases.

In 16 of the 30 described fatal anaphylactic cases (53.3%) no clear information about tryptase levels was indicated. In the others tryptase levels were very

different ranging from 23.9 to 2000 µg/L and in 13 cases tryptase levels were beyond the cut-off point proposed by some authors (45 µg/L) [3, 14, 15] remembering that there is no cut off point universally accepted by the forensic community.

In our case tryptase dosage was performed and the results showed that the tryptase level was beyond the stated cut-off point (142µg/L).

Twelve of the 30 reported cases showed positive presence of IgE antibodies but also in these cases IgE levels detected in blood were very different ranging from 1.22 to 1764.95 µg/L.

In the articles examined, toxicological investigations were not always carried out. In the cases in which they were performed drugs and alcohol were always negative.

In our case report a further point to consider is that the toxicological data indicated that the subject, at the time of death, was in a state of alcohol intoxication that may have reduced the subject's reactivity and also contributed to the respiratory depression caused by shock anaphylactic underlining that in suspected cases of anaphylactic shock it would always be advisable to carry out investigations aimed at identifying the action of other potentially fatal substances.

**In conclusion,** death from anaphylaxis caused by insect sting is a rare event. Postmortem serum

tryptase dosage could be a helpful parameter in the diagnosis of fatal anaphylaxis but given the great variety of serum tryptase levels in cases of fatal intoxication it must not be considered alone, but it must always be accompanied by the results of an autopsy examination and analysis of circumstantial data.

### Conflict of interest

The authors declare that they have no conflict of interest.

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