

# Severe scombroid poisoning complicated by distributive shock, acute respiratory distress syndrome and Kounis syndrome: a case report

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## Abstract

Scombroid syndrome is a histamine-induced foodborne illness that can mimic anaphylaxis. Although typically self-limiting, scombroid syndrome can, in rare cases, lead to severe complications. The uniqueness of this clinical case lies in the

unusually severe presentation of scombroid syndrome, which typically follows a benign course. This is particularly noteworthy given the concurrent presence of severe respiratory symptoms, shock, and Kounis syndrome. We present a case of a 59-year-old woman who developed severe scombroid poisoning after ingesting improperly stored canned tuna. Her presentation was complicated by distributive shock, acute respiratory distress, and ECG changes suggestive of Kounis syndrome. Management required high-dose intravenous epinephrine, non-invasive ventilation, and intensive hemodynamic monitoring. Coronary CT angiography later excluded obstructive coronary disease. This case highlights the potential severity of scombroid syndrome, its overlap with allergic coronary syndromes, and the diagnostic challenges they pose. Prompt recognition and tailored therapy are essential. Clinicians should be aware that improperly stored fish can precipitate life-threatening systemic reactions, requiring management beyond standard antihistamine treatment.

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## Introduction

Scombroid syndrome, also known as scombroid poisoning, is a foodborne illness resulting from the ingestion of improperly stored fish, particularly from the Scombridae family such as tuna and mackerel. This condition is caused by high levels of histamine and other biogenic amines produced through bacterial decarboxylation of histidine in the flesh of spoiled fish.<sup>1</sup> Clinically, scombroid syndrome can mimic a type I hypersensitivity reaction, with symptoms ranging from flushing, urticaria, and gastrointestinal distress to more severe manifestations such as bronchospasm, hypotension, and cardiovascular compromise.<sup>2</sup>

Although most cases are self-limiting and resolve with antihistamine therapy, severe presentations may require advanced supportive measures. Rarely, complications such as distributive shock and myocardial involvement can occur, raising diagnostic and therapeutic challenges in the emergency setting. One such complication is Kounis syndrome, an underrecognized but increasingly reported condition defined as the concurrence of acute coronary syndromes with hypersensitivity reactions.<sup>3</sup> Kounis syndrome can be triggered by various allergens, including drugs, insect stings, and food, leading to coronary vasospasm or plaque rupture through mast cell activation and inflammatory mediator release.<sup>4</sup>

We report the case of a 59-year-old woman with history of arterial hypertension and chronic headache, who developed severe scombroid syndrome after consuming improperly stored tuna, complicated by distributive shock and features consistent with Kounis syndrome. The clinical severity of this case necessitated intensive care management with intravenous epinephrine and non-invasive ventilation, followed by admission to an emergency sub-intensive care unit. To our knowledge, this is one of the few

reported cases illustrating the intersection of scombroid poisoning, distributive shock, and Kounis syndrome, highlighting the importance of prompt recognition and multidisciplinary management.

## Case Report

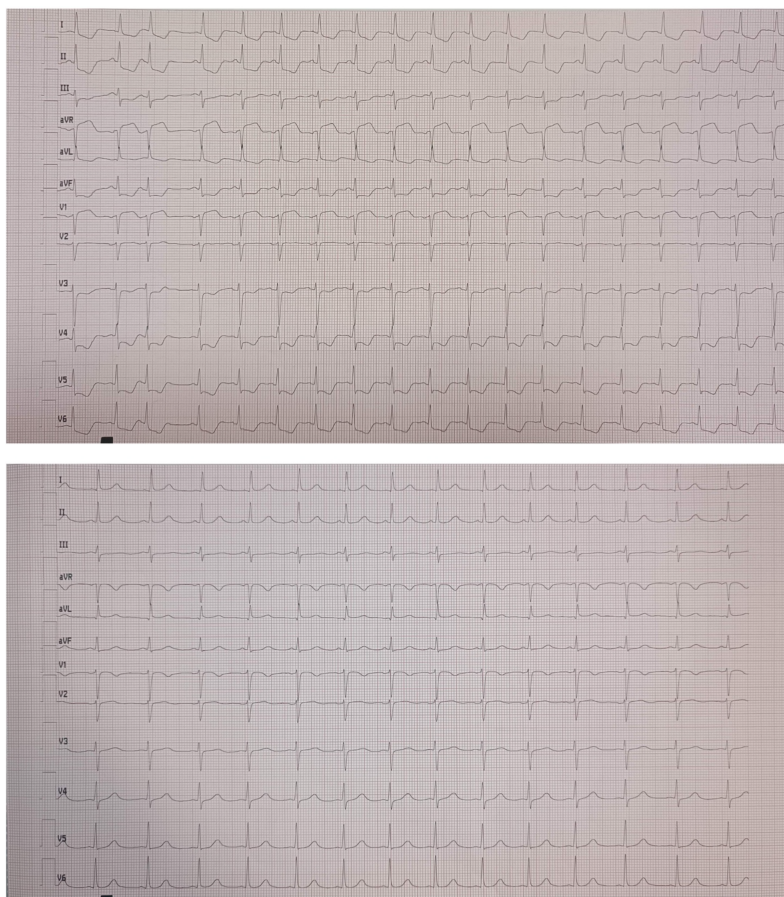
A 59-year-old Caucasian female presented to the Emergency Department (ED) via an advanced life support ambulance with medical personnel on board, due to the onset of a generalized cutaneous rash involving the entire body, one episode of diarrhea, associated with low blood pressure and sinus tachycardia, without airway compromise or respiratory distress. Emergency medical personnel administered intramuscular adrenaline (0.5 mg), intravenous chlorphenamine (10 mg), intravenous hydrocortisone (500 mg), and 1000 ml of 0.9% normal saline en route to the ED, with limited clinical benefit.

The patient's medical history was significant for arterial hypertension and migraine. Her home medications included nebivolol 5 mg once daily, rizatriptan 10 mg as needed, and vitamin D once weekly. She reported no known drug allergies. She led an active lifestyle, followed a varied diet, and was currently employed as an office worker. There was no family history of cardiovascular, cerebrovascular, or oncological diseases.

Upon arrival in the ED, the patient reported a diffuse sensation of warmth throughout the body, accompanied by widespread

erythema and oppressive chest pain radiating to the interscapular region. On further history, the patient reported having consumed approximately 200 grams of canned tuna about five hours prior to ED admission. The tuna had been opened over three weeks earlier, no longer preserved in oil, and had been stored unrefrigerated. Approximately 4.5 hours before ED arrival, she developed a generalized rash and pruritus, prompting her to consult her general practitioner. Given her recent history and hypotension, the physician contacted emergency medical services for urgent transport to the hospital. On physical examination, the patient appeared flushed, mildly tachypneic, and agitated. Cardiac auscultation revealed tachycardic and regular heart sounds. Isolated crackles were heard diffusely on lung auscultation. Peripheral pulses were weak. The airways were patent without stridor, dysphonia, or hoarseness. No wheezing or expiratory phase prolongation was detected on lung auscultation, and there were no clinical signs of bronchospasm during the ED stay. The remainder of the physical examination was unremarkable.

Upon ED admission, the following were performed: i) measurement of vital sign: blood pressure 70/50 mmHg (mean arterial pressure – MAP – 55 mmHg), heart rate 110 min R, SpO<sub>2</sub> 92% on nasal cannula 4 l/min, respiratory rate 24/min, GCS 15; ii) ABG on nasal cannula 4 l/min (Table 1): pH 7.297, pO<sub>2</sub> 71.2 mmHg, pCO<sub>2</sub> 42.5 mmHg, Hb 10.2 g/dL, meta-Hb 0.0% Hb total, K<sup>+</sup> 3 mEq/L, Na<sup>+</sup> 141 mEq/L, Ca<sup>2+</sup> 4.29 mEq/L, Cl<sup>-</sup> 108 mEq/L, Glu 251 mg/dL, Lac 4.1 mmol/L, AG 14.7 mEq/L, HCO<sub>3</sub><sup>-</sup> 20.7 mmol/L; iv) electrocardiogram (Figure 1): Sinus tachycardia with ST depression



**Figure 1.** ECG alteration on arrival in ED (upper panel) and 4 hours after therapy when haemodynamic stability was achieved (lower panel) (25 mm/s; 10 mm/mV).

in leads V3–V6, I, II, III, aVL, aVF, and ST elevation in aVR.

Methemoglobin (meta-Hb) levels were measured and found to be within normal limits. This is noteworthy given prior reports of scombroid-like poisoning due to nitrate-contaminated tuna in Italy, where elevated meta-Hb levels were documented.

Continuous vital sign monitoring was initiated. Two 16-gauge peripheral venous catheters were placed and blood tests were obtained (Table 1). Given the clinical presentation and vital signs, an additional intramuscular adrenaline 0.5 mg and 1000 mL of 0.9% normal saline were administered. After 5 minutes, due to insufficient clinical response, a further dose of adrenaline 0.5 mg IM was given. However, 5 minutes later, in the absence of therapeutic response, a continuous intravenous infusion of epinephrine was initiated at 0.1 mcg/kg/min, and titrated after approximately 10 minutes until a MAP of 65 mmHg was achieved. An additional 10 mg of intravenous chlorphenamine was administered.

Simultaneously, the following were placed: a radial arterial line

for continuous blood pressure monitoring, a Central Venous Catheter (CVC) for vasopressor administration – initially started via peripheral access – and a urinary catheter for monitoring of urine output.

Given the clinical picture and the patient's history suggestive of scombroid syndrome, a toxicology consult was requested. The diagnosis was confirmed, and the recommendation was to continue the current treatment.

Furthermore, due to the ECG findings suggestive of acute coronary syndrome occurring in the context of a hypersensitivity reaction/histamine excess—also known as Kounis syndrome—a cardiology consult was requested. The transthoracic echocardiogram revealed normal biventricular function with preserved wall motion. The cardiologist recommended continuation of the current treatment for scombroid syndrome and suggested that further investigations for coronary disease be postponed to a later stage. Although the patient had a history of migraine and was

**Table 1.** Arterial blood gas analysis and blood exams.

Arterial blood gas analysis					
	T0h	T2h	T6h	T12h	T24h
FiO <sub>2</sub> (%)	36	60	60	50	28
pH	7.29	7.32	7.32	7.35	7.45
pO <sub>2</sub> (mmHg)	71.2	59	85	113	82.2
pCO <sub>2</sub> (mmHg)	42.5	40	44	40	41
SaO <sub>2</sub> (%)	94	90	97	99	98
Hb (g/dL)	10.2	11.3	10.6	10.1	9.8
Lac (mmol/L)	4.1	0.2	5.6	3.3	0.5
AG (mEq/L)	14.7	14.9	17.2	12.7	10.6
HCO <sub>3</sub> (mmol/L)	20.7	21.7	22.6	22.2	27.2
Na <sup>+</sup> (mEq/L)	141	144	145	141	144
K <sup>+</sup> (mEq/L)	3.0	3.1	3.3	3	4
Cl <sup>-</sup> (mEq/L)	108	109	108	109	110
Ca <sup>2+</sup> (mEq/L)	4.29	4.62	3.85	4.62	4.90
PaO <sub>2</sub> /FiO <sub>2</sub>	197.8	98.3	141.7	226.0	293.6
Blood Exams					
	T0h	T1h	T12h	T36h	T60h
WBC (x10 <sup>9</sup> /L)	26.50		13.70	20.10	10.60
RBC (x10 <sup>9</sup> /L)	3.42		3.65	3.26	3.67
Hb (g/dL)	10		10.4	9.2	11.4
PLT (x10 <sup>9</sup> /L)	298		229	195	278
Neut (x10 <sup>9</sup> /L)	22.58		12.4	17.63	8.29
Eos (x10 <sup>9</sup> /L)	0.03		0.00	0.00	0.02
Bas (x10 <sup>9</sup> /L)	0.05		0.01	0.02	0.07
Lym (x10 <sup>9</sup> /L)	3.10		0.63	1.25	2.81
Mono (x10 <sup>9</sup> /L)	0.74		0.62	1.21	0.70
INR	1.0		1.0	1	0.9
PT sec	11.4		11.3	12.1	11
aPTT sec	25.2		28.2	28.7	28
GPT (U/L)	14		25	18	17
Creatinine (mg/dL)	1.34		0.72	0.68	0.71
TnT hs (pg/mL)	6.15	26.20	116	90.80	18.70
CRP (mg/L)	<5		25	12	<5

FiO<sub>2</sub>, fraction of inspired oxygen; Hb, haemoglobin; Lac, serum lactate; AG, anion gap; WBC, White Blood Cells; RBC, Red Blood Cells; PLT, Platelets; Neut, Neutrophils; Eos, Eosinophils; Bas, Basophils; Lym, Lymphocytes; Mono, Monocytes; INR, International Normalized Ratio; PT, Prothrombin Time; Aptt, Activated Partial Thromboplastin Time; GTP, Gamma-Glutamyl Transpeptidase; TnT hs, High-Sensitivity Troponin T; CRP, C-Reactive Protein.

prescribed rizatriptan as needed, she denied recent use prior to symptom onset, making it an unlikely contributing factor in the development of Kounis syndrome in this case.

Following initial stabilization, a chest X-ray was performed, showing signs of pulmonary congestion. However, due to progressive deterioration in gas exchange (Table 1), non-invasive mechanical ventilation was initiated.

Approximately 3 hours after initiation of intravenous adrenaline, vasopressors were discontinued. A progressive normalization of the ECG and ST-T segment was also observed (Figure 1). However, considering the severity of the clinical presentation and the need for ongoing non-invasive ventilation, suspended after 6h, the patient was transferred to the Emergency Department's High Dependency Unit (HDU), following anaesthesiology evaluation. The anaesthesiologist confirmed the appropriateness of the therapeutic approach and did not indicate the need for Intensive Care Unit (ICU) admission.

In terms of the respiratory findings, a chest CT scan was deliberately deferred due to the patient's marked hemodynamic instability and the presence of lung ultrasound findings consistent with ARDS – namely, the absence of a clear B-line to A-line gradient, no pleural effusion, and the presence of spared areas. Given the subsequent rapid weaning from mechanical ventilation, the CT scan was ultimately not pursued.

Due to the absence of these signs and the delayed presentation – by which time nearly all of the ingested histamine had likely already been absorbed – gastrointestinal decontamination was not undertaken.

After approximately 24 hours of observation in the HDU, with hemodynamic stability and progressive weaning from non-invasive ventilation (although still requiring low-flow oxygen therapy), the patient was transferred to a general ward. Given the ECG abnormalities and troponin elevation (Table 1), a coronary CT angiography was performed, which showed no significant coronary stenoses. After approximately three days of medical therapy with antihistamines and successful weaning from low-flow oxygen, the patient was discharged home.

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## Discussion

We presented the case of a patient with severe scombroid poisoning, characterized by several of the possible manifestations of this condition occurring simultaneously: acute respiratory distress, distributive shock, and histamine-mediated coronary syndrome. The peculiarity of this case lies in its exceptional severity, which is uncommon in an otherwise healthy patient following the ingestion of improperly stored canned tuna.

This case is of particular clinical interest for several reasons. First, it describes an exceptionally severe presentation of scombroid syndrome, complicated not only by distributive shock but also by ECG abnormalities and troponin elevation suggestive of type I Kounis syndrome—a rare and often underdiagnosed cardiovascular complication triggered by massive histamine release: the exclusion of obstructive coronary artery disease through CT angiography reinforces the diagnosis and highlights the importance of considering allergic – histaminergic - coronary syndromes in the differential diagnosis of chest pain with allergic features. Secondly, the patient experienced persistent hypotension despite repeated intramuscular doses of adrenaline, ultimately requiring continuous intravenous vasopressor infusion and advanced hemodynamic support: this underscores the potential for severe cardiovascular

instability in scombroid syndrome, even in patients without pre-existing cardiac disease. Additionally, the case is remarkable for the poor presence of gastrointestinal symptoms, which are typically prominent in scombroid poisoning: this atypical presentation may have delayed suspicion of a foodborne etiology, allowing greater systemic absorption of biogenic amines and contributing to the severity of the systemic response.

From an emergency medicine perspective, the case exemplifies the diagnostic and therapeutic challenges posed by toxin-mediated syndromes. The patient initially presented with non-life-threatening symptoms such as rash and mild chest discomfort, yet evolved rapidly into a state of circulatory shock and respiratory failure requiring non-invasive ventilation and high-dependency care. This highlights the importance of early recognition, prompt risk stratification, and a high index of suspicion for potentially life-threatening progression in patients with apparent allergic reactions, especially when associated with chest pain or hemodynamic instability. Scombroid poisoning is a frequently encountered foodborne illness linked to seafood consumption globally.<sup>5</sup> Although primarily associated with fish, it has also been reported following ingestion of contaminated Swiss cheese.<sup>6</sup> Clinical manifestations typically emerge rapidly and include facial and cervical flushing, erythematous or urticarial rash, gastrointestinal symptoms such as diarrhea, and headache. In rare cases patients may experience acute respiratory distress or cardiac complications, like shock or arrhythmias.<sup>7,8</sup> Owing to the similarity of symptoms, the condition is often misdiagnosed as an allergic reaction to seafood. Most cases respond well to treatment with H1 and/or H2 antihistamines, while those presenting with airway compromise or circulatory collapse should be managed as per standard anaphylaxis protocols.<sup>9</sup>

The worsening of the P/F ratio despite hemodynamic recovery suggests a possible histamine-induced capillary leak syndrome or early non-cardiogenic pulmonary edema, which has been described in the context of severe scombroid poisoning.<sup>10,11</sup> The management of these cases – beyond supportive care and treatment of scombroid syndrome – is essentially analogous to that of ARDS.

Methemoglobin (meta-Hb) levels were measured and found to be within normal limits. This is noteworthy given prior reports of scombroid-like poisoning due to nitrate-contaminated tuna in Italy, where elevated meta-Hb levels were documented.<sup>12</sup>

An additional peculiarity of the present case is that the gastrointestinal symptoms are blurred: typically those are commonly described in scombroid syndrome and are part of the typical clinical triad alongside dermatologic and neurologic findings.<sup>1,10,11,13</sup> The poor gastrointestinal manifestations may have diagnostic implications, potentially delaying the suspicion of a foodborne illness and contributing to unrecognized systemic toxin absorption. It could also reflect a differential tissue sensitivity to histamine and related amines, whereby the gastrointestinal tract remains unaffected while cardiovascular and respiratory systems are more severely involved. This observation raises the hypothesis that, in certain individuals, the absence of early “warning” symptoms like gastrointestinal discomfort might paradoxically predispose to a greater risk of systemic or cardiovascular complications, due to unopposed toxin progression before medical attention is sought.<sup>1,10,11,13</sup> Further studies would be needed to validate this potential association, but this clinical scenario emphasizes the heterogeneity of scombroid syndrome presentations and the need for a high index of suspicion even in the absence of the full classic symptom constellation.

Scombroid poisoning has a global distribution and frequently presents in clusters following ingestion of contaminated dark-fleshed fish. The condition is classically associated with species

within the Scombridae and Scomberesocidae families, including tuna, mackerel, skipjack, and bonito.<sup>14,15</sup> However, cases have also been linked to other species such as mahi-mahi (*Coryphaena hippurus*), bluefish, amberjack, swordfish, marlin, herring, sardines, anchovies, salmon, tilapia, and trout.<sup>1,14</sup>

In the United States and Europe, scombroid poisoning is responsible for up to 40% of seafood-related outbreaks.<sup>1</sup> Between 2000 and 2007, the U.S. Foodborne Disease Outbreak Surveillance System documented 223 outbreaks, involving 865 individuals; however no fatalities were recorded. Population-based incidence estimates vary, ranging from 2–5 annual outbreaks per million individuals in countries such as Denmark, New Zealand, France, and Finland, to as many as 31 outbreaks per million per year in Hawaii.<sup>1</sup> Nevertheless, the condition is believed to be underreported due to its nonspecific presentation and a usual short clinical course.

Scombroid poisoning results from the improper storage of fish at temperatures exceeding 4°C (40°F), leading to bacterial proliferation and the enzymatic decarboxylation of histidine into histamine and other biogenic amines.<sup>1,15</sup> This process may occur within 2–3 hours if the fish is kept at temperatures around 20°C (68°F). The primary bacterial agents responsible include *Escherichia coli*, halophilic *Vibrio* spp., *Proteus*, *Klebsiella*, *Clostridium*, *Salmonella*, and *Shigella* spp.<sup>1,15</sup> Notably, histamine is heat-stable and cannot be destroyed through cooking, freezing, or refrigeration.<sup>15</sup> Consequently, contaminated fish may appear and smell normal, though some patients report a distinctive “peppery,” “spicy,” or “bubbly” taste. Contamination may occur at sea if refrigeration is inadequate, but improper handling at any stage before consumption can also result in toxic accumulation.<sup>1,13,15</sup>

Although fresh fish is the most common source, improperly handled canned fish may also lead to histamine poisoning. One such case was documented by Predy *et al.*,<sup>16</sup> who described a 51-year-old woman developing symptoms including headache, nausea, palpitations, tachycardia, and hypertension within 30 minutes of consuming tuna salad as in our case report. Elevated histamine levels in the food confirmed the diagnosis.

The clinical features of scombroid poisoning are attributable to elevated levels of histamine and typically present within one hour following ingestion of contaminated fish or dairy products. The symptomatology is variable but consistently involves dermatologic,

gastrointestinal, and neurologic components.<sup>13,16,17</sup>

The most frequently reported symptoms are reported in Table 2. The diagnosis of scombroid poisoning is primarily clinical and should be considered in patients presenting with the acute onset of flushing, rash, headache, and gastrointestinal symptoms following recent ingestion of fish—especially species from the *Scombridae* or *Scomberesocidae* families.<sup>1,13,17</sup>

The diagnostic criteria include:<sup>1,13,17</sup> i) symptom onset within 60 minutes of ingesting potentially contaminated food; ii) clinical features consistent with histamine toxicity (e.g., flushing, urticaria, diarrhea, headache); iii) occurrence of similar symptoms in other individuals who consumed the same food; iv) prompt symptom resolution following administration of antihistamines.

Although not routinely required, laboratory confirmation can be pursued in suspected outbreaks or for public health surveillance. Histamine concentration in the ingested fish can be measured using validated biochemical methods.<sup>1</sup> Allergy testing (e.g., skin prick tests or specific IgE assays) may be useful in cases where food allergy remains a differential consideration.<sup>18,19</sup> Patients with true scombroid poisoning will demonstrate negative type I hypersensitivity testing to fish allergens.<sup>18</sup>

Several conditions may present with clinical features that overlap with those of scombroid poisoning. A thorough clinical evaluation is essential to distinguish among these possibilities: Myocardial Ischemia or Infarction (most cases can be distinguished by electrocardiographic monitoring and cardiac biomarkers, histamine-mediated coronary vasospasm may cause transient subendocardial ischemia with mildly elevated troponin levels),<sup>3,4</sup> allergic reaction or anaphylaxis (key differentiating features include the absence of prior allergic reactions to fish, clustering of cases among individuals consuming the same meal and a history of improper food storage), Staphylococcal enterotoxin food poisoning (this condition may present with abrupt-onset nausea, vomiting, and abdominal cramping shortly after food ingestion) and other marine toxin syndromes (Ciguatera poisoning, neurotoxic shellfish poisoning, paralytic shellfish poisoning or pufferfish toxicity).

Among the differential diagnoses of chest pain with ECG alterations and troponin elevation, particularly in the context of allergic or anaphylactoid reactions, Kounis syndrome represents a rare but clinically significant entity.<sup>3</sup> Kounis syndrome is defined

**Table 2.** Common signs and symptoms of scombroid poisoning.<sup>9-11</sup>

System	Manifestations
Dermatologic	<ul style="list-style-type: none"> <li>- Flushing (face and neck)</li> <li>- Erythematous or urticarial rash</li> <li>- Perioral burning, itching, or edema</li> </ul>
Gastrointestinal	<ul style="list-style-type: none"> <li>- Abdominal cramping</li> <li>- Nausea and vomiting</li> <li>- Diarrhea</li> </ul>
Neurologic	<ul style="list-style-type: none"> <li>- Headache</li> <li>- Dizziness</li> <li>- Blurred vision</li> </ul>
Cardiovascular	<ul style="list-style-type: none"> <li>- Tachycardia</li> <li>- Palpitations</li> <li>- Hypotension (distributive shock) (rare)</li> <li>- Cardiac arrhythmias (rare)</li> </ul>
Respiratory	<ul style="list-style-type: none"> <li>- Chest tightness</li> <li>- Shortness of breath</li> <li>- Bronchospasm and respiratory distress (rare)</li> </ul>
Sensory/Oral	<ul style="list-style-type: none"> <li>- Peppery, spicy, or metallic taste in the mouth</li> </ul>

as the coexistence of acute coronary syndromes with hypersensitivity reactions and is typically triggered by allergens leading to mast cell activation and the release of vasoactive mediators like histamine, tryptase, and leukotrienes.<sup>20</sup> These substances can induce coronary vasospasm or, in patients with underlying atherosclerosis, promote plaque erosion or rupture, resulting in myocardial ischemia or infarction. Three variants have been described: type I (in patients with normal coronary arteries), type II (in those with pre-existing coronary artery disease), and type III (involving stent thrombosis).<sup>20</sup>

The association between scombroid syndrome and Kounis syndrome, although mechanistically plausible, is exceedingly rare and scarcely reported in the literature.<sup>21</sup> In our case, the diagnosis of type I Kounis syndrome is supported by the presence of diffuse ST-segment depression and ST elevation in aVR, elevated troponin levels, and the absence of obstructive coronary lesions on CT angiography. However, unlike typical allergic reactions in Kounis syndrome, where mast cell activation occurs in response to an IgE-mediated mechanism, the pathophysiology of scombroid poisoning involves the direct exogenous ingestion of histamine and other biogenic amines.<sup>1</sup> These mediators act systemically, bypassing the classical immune pathways and mimicking an anaphylactoid response. Therefore, while the clinical and electrocardiographic manifestations may overlap, the underlying mechanisms differ substantially: endogenous mast cell degranulation in Kounis syndrome versus exogenous histamine toxicity in scombroid syndrome induced Kounis syndrome.<sup>1,13</sup>

This distinction is not only of pathophysiological interest but also has practical implications for diagnosis and treatment. The potential for coronary involvement in severe scombroid poisoning—though rare—should prompt clinicians to maintain a high index of suspicion, particularly when cardiovascular symptoms, ECG changes, or troponin elevation are present.<sup>13,21</sup> In such cases, Kounis-like presentations should be considered, even in the absence of a classical allergic context, as part of the broader spectrum of histamine-mediated cardiovascular effects.

Although scombroid poisoning is typically self-limiting, patients presenting with severe manifestations—such as upper airway edema, bronchospasm, or hypotension—should be managed according to established protocols for anaphylaxis.

For patients without life-threatening symptoms but with moderate to severe discomfort (*e.g.*, flushing, pruritus, rash, burning sensations, or perioral edema), antihistamine therapy is recommended over supportive care alone: i) H1 antihistamines: a single dose of an H1 receptor antagonist (*e.g.*, diphenhydramine or hydroxyzine) should be administered; ii) H2 antihistamines: in patients with gastrointestinal symptoms, more pronounced clinical manifestations, or incomplete response to H1 antihistamines, a single dose of an H2 antagonist (*e.g.*, famotidine or cimetidine) is advised.

Patients with significant fluid losses due to vomiting or diarrhea should receive intravenous fluid resuscitation with isotonic crystalloids.

Patients presenting with mild symptoms that resolve fully after treatment with antihistamines (H1 and/or H2 blockers) may be safely discharged from the emergency department. Although evidence is limited, the administration of oral antihistamines (*e.g.*, loratadine or cetirizine) for one to two days following discharge is a reasonable strategy to prevent symptom recurrence due to ongoing toxin absorption from the gastrointestinal tract.

Patients with severe manifestations – such as respiratory distress, hypotension, or airway edema – should be admitted or observed for several hours to ensure full clinical resolution.

Those who receive intravenous fluid therapy should demonstrate adequate oral intake and hemodynamic stability prior to discharge.

## Conclusions

Scombroid syndrome typically follows a benign and self-limiting course; however, in rare instances, it may present with a sudden and severe onset, potentially life-threatening, as in the case described. Furthermore, the association with histamine-mediated coronary vasospasm, which can lead to transient subendocardial ischemia, troponin elevation, and ECG changes, represents a variant of Kounis syndrome, adding to the peculiarity and complexity of this clinical presentation. Kounis syndrome, if not promptly recognized, may lead to misinterpretation of the clinical picture and result in inappropriate management and patient disposition.

Timely recognition, accurate diagnosis, and appropriate treatment are essential in severe forms of scombroid syndrome, although the most effective therapy remains prevention.

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