

Case Report

Anaphylaxis shock with laryngeal edema: a case report

Anak A. N. S. Pranata^{1*}, Dewi C. Wulandari¹, Ni P. Setiawathi², Ketut Suryana¹

¹Department of Internal Medicine, Wangaya Public Hospital, Denpasar, Bali Indonesia

²Department of Otolaryngology, Wangaya Public Hospital, Denpasar, Bali, Indonesia

Received: 24 May 2024

Accepted: 18 June 2024

*Correspondence:

Dr. Anak A. N. S. Pranata,

E-mail: satyapranata41@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Anaphylaxis is a serious systemic or hypersensitivity reaction that rapid in onset and affect airway, breathing, and/or circulatory problems, and that is usually associated with skin and mucosal changes. Laryngeal edema is observed in 40-50% of death cases. A 49-year-old Asian female came to emergency department with chief complaint of difficulty to breath since 30 minutes ago after took cefixime and also common cold drug (Combination of paracetamol, glyceryl guaifenesin, phenylephrine, and Chlorpheniramine maleate). She also complained difficulty to speak and feeling strangulated on her neck. She ever had an allergic reaction, when she was child after suffering fever and common cold, but didn't remember the name of the medication. History of food allergies was denied. On physical examination revealed, dyspnea and minimal tachycardia. Present of laryngeal edema, on auscultation stridor and wheezing on both lungs. Diagnosis of anaphylactic shock was made, and immediately giving epinephrin and followed by corticosteroid and antihistamine. The signs and symptoms of anaphylaxis might differ from patient to patient and impact the skin, gastrointestinal tract, respiratory system, and heart. Anaphylaxis fatal cases rise when laryngeal edema and cardiovascular organ involvement occur. The most frequent triggers are produced by food, venom from insects, and prescription drugs. The risk of death in anaphylaxis patients is decreased with prompt treatment. It is necessary to assess and maintain breathing, circulation, and airways in addition to determining the cause. Anaphylaxis can be potentially lethal or life-threatening, thus prompt identification and appropriate treatment are necessary.

Keywords: Anaphylaxis shock, Laryngeal edema, Emergency, Hypersensitivity

INTRODUCTION

Anaphylaxis is a serious systemic or hypersensitivity reaction that rapid in onset and potentially endangers life through involvement of airway, breathing, and circulatory problems. Anaphylaxis can cause death in all ages, with a mortality rate is less than 1 per million per year however, this estimate is likely to be lower than the true rate of fatal anaphylaxis and under-notification. Its heterogenous clinical presentation but mostly associated with skin and mucosal changes.¹ In case of anaphylaxis, 90% of cases present with mucocutaneous manifestation but death are secondary due to laryngeal edema, observed in 40-50% of cases.² Several risk factors may cause anaphylaxis, including exposure to certain medications, food, or insect stings.³ Immediate identification and proper treatment are

needed in anaphylaxis cases when presenting laryngeal edema because anaphylaxis can potentially be life-threatening or fatal.⁴ In This case, we reported an anaphylaxis shock with manifestation of laryngeal edema.

CASE REPORT

A 49-year-old Asian female came to emergency department with a chief complaint of difficulty to breath since 30 minutes ago after took cefixime and also a common cold drug (combination of paracetamol, glyceryl guaifenesin, phenylephrine, and chlorpheniramine maleate). She also complained difficulty to speak due to strangulated feelings, changing in voice, and hoarseness. She was prescribed with that medication due to fever and

common cold since 3 days before she came to the emergency department.

2 weeks before being admitted to the hospital, she didn't take any other medication. She didn't have history of food allergies. History of taking other medication and food allergies was denied.

When she was a child, she ever had an allergic reaction, after having fever and common cold. However, she didn't remember the medication and she also didn't remember whether she got antibiotics at that time.

Due to her condition, she rarely takes any medication when she gets sick. She only takes herbal medication. Furthermore, she said that she had an allergic history with natrium diclofenac, allopurinol, antacids, and ranitidine. She said that if she takes the medication, around on her eyes and lips will become swollen, and also shortness of breath.

She was admitted to hospital on October 2023, due to unstable angina pectoris, and myalgia. She was prescribed with Natrium Diclofenac to relieve her pain, but she was getting an allergic reaction, such as erythema and swelling around her eyes. Then the pain reliever switched to paracetamol but she refused to take it due to afraid of an allergic reaction. After discharged, being afraid of an allergic reaction, she decided to stop the medication by herself.

On arrival at the emergency department, even though she felt very weak, she presented with fully alert with GCS E4V5M6. Based on physical examination, we found minimal tachycardia (102/min) and increased respiration rate (RR: 28/min), with blood pressure 126/82 mmHg and oxygen saturation (SpO₂) was 92% on room air, and given oxygenation with nasal cannula 3% litres per minute; the SpO₂ become 99%. There is no edema in the eyes and lips and face. On ENT examination: nose: nasal cavity patent/patent; mucosa: pink/pink. Concha decongestion/decongestion, secret -/-, no septum deviation; on throat; no tongue edema, uvula still in the middle, and no edema; pallatum mole no edema; laryngoscopy indirect was performed with result: edema of right and left vocal cord, plica ventricularis, epiglottis, and arytenoid but didn't cover the entire of rima glottis; no nodule or mass found. On laboratory examination, there was leucocytosis WBC: $12.51 \times 10^3/\text{ul}$, Hb 13.2 g/dl, hematocrit 42.7%, and platelet $196 \times 10^3/\text{ul}$. Increase of urea (67 mg/dL) and creatinine (1.4mg/dL), electrolyte level within normal limit. Auscultation on lung revealed: vesicular +/+/, rhonchi +/+/, wheezing +/+/, and stridor +/+/.

The patient was treated in emergency department with NaCl 0.9% 20 drips per minute, epinephrine 0.3 cc IM, diphenhydramine 1×10 mg IV, methylprednisolone 62.5 mg IV, and ventolin nebulization. She was transferred to ICU for close monitoring with blood pressure 87/63, heart

rate 110 bpm, RR 26/min and oxygen saturation was 100% with nasal cannula 3 lpm. In ICU diphenhydramine was stopped.



Figure 1: Chest X-ray showed cor prominent and suspected pneumonia.

From echocardiography normal dimension, no LVH, systolic LV function normal with EF 62%, TAPSE normal 30 mm, global normal, TR mild.

The patient was diagnosed with anaphylaxis reaction (larynx edema), CAD HF with history of UAP, RBBB, AKI, and pneumonia. She was treated by an internist, ENT, cardiologist, and pulmonologist.

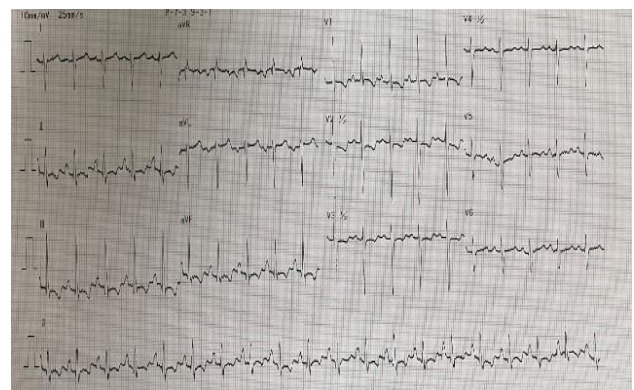


Figure 2: ECG showed sinus tachycardia with RBBB ischemia.

After being given initial treatment at emergency department, the patient was given methylprednisolone 2×62.5 mg for three consecutive days as well as ceftriaxone 1×2 gr every day and levofloxacin 750 mg every 48 hours and combiven (ipratropium bromide and salbutamol) nebulization three times daily. From cardiologist, she was given atorvastatin 1×20 mg every night, and clopidogrel 1×75 mg.

After three days, the patient showed response to the treatment. After that, she moves to general ward. She said that she didn't feel any shortness of breath and strangulated feeling anymore. On the ward, methylprednisolone dose

was tapered off to 2×31.25 mg on the fourth day and then given orally 3×4 mg on day five until day seven.

After 6 days in hospital, she didn't have any complaints anymore and no allergic reaction was observed. After that, she was discharged from hospital and was prescribed

methylprednisolone 3×4 mg orally until the seventh day, levofloxacin 750 mg every 2 days until the tenth day, codeine 3×10 mg, bisoprolol 1×1.25 mg, atorvastatin 1×20 mg and clopidogrel 1×75 mg.

Table 1: Monitoring of patient on hospital admission.

Date	09 December 2023 D-1	10 December 2023 D-2	11 December 2023 D-3	12 December 2023 D-4	13 December 2023 D-5	14 December 2023 D-6
General condition	Weak	Weak	Weak	Moderate	Moderate	Good
Blood pressure (mmHg)	126/82	87/63	97/65	92/56	100/64	100/70
Heart rate (bpm)	102	99	89	60	74	75
Respiration rate (/min)	28	26	23	18	20	20
Temp (°C)	36.8	36	36	36.2	36	36.5
Room	ER	ICU	ICU	GW	GW	GW

DISCUSSION

Anaphylaxis is a severe systemic allergic reaction, that is rapid in onset and potentially fatal. The prevalence of anaphylaxis ranges between 0.05% to 2% in lifetime.⁵ It occurs more frequently in children and adolescents, but severe cases are more common in adults. The mortality rate is estimated at 0.65–2% of severe anaphylaxis cases. Involvement of cardiovascular and laryngeal edema are the main causes of death.⁶ The most common trigger of anaphylaxis in adults is, food-induced such shellfish is the most common in Asia whereas tree nuts and peanuts are in North America and Australia.⁷ the second cause is insect venom, red ants, and bee venom which are the most common causes in Asia and America.^{7,8} Drug-induced anaphylaxis also reported, most caused by antibiotics and analgesics.⁹ In this case, presented a 49-year-old woman, with laryngeal edema and angioedema grade IV; the cause was suspected to be paracetamol.

Anaphylaxis symptoms may vary from patient to patient including angioedema, abdominal pain, systemic urticaria, dyspnea, and hypotension.¹ skin symptoms are the most common manifestation, it begins from flushing and pruritus and develops into hives and/or angioedema. Eyes and mucous membranes can acquire a congestive-like aspect. Gastrointestinal symptoms include abdominal pain, nausea, vomiting and diarrhea. In respiratory organs, tightness in the throat, dysphagia, dysphonia, inspiratory stridor, and even signs of asphyxia, which is caused by the generation of laryngeal edema. When bronchoconstriction happens, patient feels cough, dyspnea, wheezing, and a feeling of tightness in the chest occurring hypoxemia and cyanosis. Cardiovascular manifestation may tachycardia and feeling of dizziness or instability and progress until the loss of consciousness. Hypotension and shock were caused by peripheral vasodilation and an increase in vascular permeability, which increases heart rate and reduces coronary perfusion. In some cases, anaphylaxis can be abrupt, like syncope, or even cause sudden death. Other

manifestations that may occur during anaphylaxis are disorientation, anxiety, seizures, and profuse sweating.^{1,5,8} The patient presented with dyspnea, stridor, laryngeal edema, hypotension, tachycardia, urticaria, angioedema, and with a history of taking paracetamol 30 minutes before symptoms appeared.

Anaphylaxis is a medical emergency that requires rapid identification and treatment, either by patients or health professionals, based on the development of symptoms suggestive of allergy. Useful clinical criteria have been established for the diagnosis of anaphylaxis (Table 2). With the proper use of these criteria, 95% of anaphylaxis cases can be identified. We also need to consider another probable diagnosis when assessing the patient, and in case of moderate or severe anaphylaxis, it is convenient to perform complete blood count, and X-ray to assess patient's general condition.

The risk of death in anaphylaxis patients is decreased with prompt treatment.¹¹ The patient also exhibits laryngeal edema, a potentially fatal anaphylactic symptom that is extremely significant. Since up to 15% of patients may experience airway obstruction, definitive airway care is necessary for angioedema involving the tongue or larynx. In half of the instances, a definitive airway such as a tracheostomy or cricothyrotomy may be required, even with an appropriate head tilt and a clear pharynx (obtained by suctioning). During a physical examination, hoarseness and voice changes in the patient indicate upper respiratory tract involvement.¹² Assessing and preserving circulation, breathing, and airways as well as determining the cause are the first steps in management. In situations of laryngeal edema, after stabilizing the airway, provide 0.3–0.05 ml of intramuscular epinephrine at a 1:1000 dilution. followed by administration of a histamine-blocker and corticosteroid. Adrenaline 0.3 cc IV, NaCl 0.9% 20 drips/min, diphenhydramine 1×10 mg IV, methylprednisolone 62.5 mg IV, and ventolin nebulization were used as the patient's initial course of treatment. If

required, administering epinephrin again every five to fifteen minutes is possible. Only when continuous cardiac monitoring is feasible, intravenous (IV) epinephrin can be administered to a patient who has not responded to intramuscular (IM) epinephrin and intravenous fluid.^{5,10-12}

Diphenhydramine was not given to the patient in the ICU since they were experiencing anaphylaxis. Being a first-generation H1-antihistamine, diphenhydramine may have adverse cardiovascular effects, such as tachycardia and widening of the QRS complex. These effects may be brought on by diphenhydramine's anticholinergic action. It works by blocking potassium channels, which has a negative inotropic effect and causes the T-wave to flatten and the QT interval to lengthen.¹³ As a result, people are more likely to experience potentially lethal arrhythmias such as torsade de pointes.¹⁴ When taken at the authorized dosages, it also prevents histamine from interfering with neurotransmission at CNS H1 receptors. This may result in somnolence, drowsiness, sedation, and a decline in cognitive and memory abilities as well as psychomotor function.¹⁵

In the emergency room, observation of the first 4-6 hours as the initial phase is reasonable, but monitoring until 72

hours of the initial phase is needed because there can be biphasic reaction.^{16,17} Biphasic reactions is the recurrence of anaphylactic symptoms without repeated exposure to an elicitor. The biphasic reaction can be present ranging from 1.4% to 20% of the case. The risk of this late reaction still cannot be predicted thus data is still limited.¹⁸ However, patients who present severe initial reactions may have a greater risk of suffering biphasic reactions.^{1,19} Thus we administer this patient to the intensive care unit (ICU) for close monitoring, because there was involvement of skin and respiratory organs.

Patients are advised to self-administer epinephrine in the event of a new anaphylactic episode before being discharged from the hospital. It is advised to utilize auto-injector devices whenever feasible; give the patient a written emergency plan that outlines what to do in the event that anaphylactic symptoms manifest; Instructions and sufficient details about the nature of the issue should be given, together with advice on how to keep away from allergies and possible triggers; send the patient to an allergist so that they can investigate and potentially determine the precise cause of the anaphylactic reaction. They can also assess the viability of desensitization therapy or targeted allergen immunotherapy.⁸

Table 2: Clinical criteria for anaphylaxis diagnosis.¹⁰

S. no.	Anaphylaxis is highly likely when any one of the following criteria
1	Acute onset (from a few minutes to a few hours) of a condition characterized by affectation of the skin and/or mucous (ex. hives, itching, or generalized flushing/edema of the lips, tongue, and/or uvula) and at least one of the following
a	Compromised breathing (ex. dyspnea, wheezing [bronchospasm], stridor, reduced PEF, hypoxemia)
b	Arterial hypotension or symptoms associated with circulatory compromise (e.g. hypotonia [collapse], syncope, incontinence)
2	Two or more of the following situations that occur rapidly after exposure to a probable allergen (a few minutes to a few hours)
a	Affectation of the skin and/or mucous (ex. hives, itching, or generalized flushing/edema of the lips, tongue, and/or uvula)
b	Compromised breathing (ex. dyspnea, wheezing [bronchospasm], stridor, reduced PEF, hypoxemia)
c	Arterial hypotension or symptoms associated with circulatory compromise (e.g. hypotonia [collapse], syncope, incontinence)
d	Persistent gastrointestinal symptoms (ex. crampy abdominal pain, vomiting)
3	Hypotension after exposure to a known allergen for this patient (a few minutes to a few hours)
a	Children: low systolic BP (according to age) or a reduction greater than 30% in the systolic BP
b	Adults: systolic BP less than 90 mm Hg or a decrease greater than 30% in the systolic BP

PEF: peak expiratory flow, BP: blood pressure, *low systolic blood pressure in children is defined as less than 70 mm Hg in children from 1 month to 1 year old; less than 70 mm, Hg + 2× years of age, in children from 1 to 10 years old, and less than 90 mm Hg in patients from 11 to 17 years old

CONCLUSION

Anaphylaxis is a potentially fatal systemic reaction with a quick start. The venom of hymenoptera, meals, and pharmaceuticals are the most common causes. Clinical criteria are used to make the diagnosis. Skin, respiratory, cardiovascular, and gastrointestinal signs and symptoms are examples of clinical manifestations. Laryngeal edema is a life-threatening; injectable epinephrine, should be

started as soon as anaphylaxis is diagnosed. The extra treatment given in addition to epinephrine will be determined by each case's clinical presentation. Depending on the severity of the anaphylaxis, the patient's observation duration following the early phase of care should be customized. In the event that the patient experiences anaphylaxis in the future, it is imperative to offer a documented action plan and send them to an allergist for ongoing care.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

REFERENCES

- Shaker MS, Wallace DV, Golden DBK, Oppenheimer J, Bernstein JA, Campbell RL, et al. Anaphylaxis-a 2020 practice parameter update, systematic review, and Grading of Recommendations, Assessment, Development and Evaluation (GRADE) analysis. *J Allergy Clin Immunol*. 2020;145(4):1082-23.
- Tanno LK, Demoly P. Anaphylaxis in children. *Pediatr Allergy Immunol*. 2020;31(S26):8-10.
- Pattanaik D, Lieberman P, Lieberman J, Pongdee T, Keene AT. The changing face of anaphylaxis in adults and adolescents. *Ann Allergy, Asthma Immunol*. 2018;121(5):594-7.
- Castilano A, Sternard B, Cummings ED, Shi R, Arnold T, Bahna SL. Pitfalls in anaphylaxis diagnosis and management at a university emergency department. *Allergy Asthma Proc*. 2018;39(4):316-21.
- Simons FER. Anaphylaxis. *J Allergy Clin Immunol*. 2010;125(2):18-23.
- Arias-Cruz A. Anaphylaxis: Practical aspects of diagnosis and treatment. *Med Univ*. 2015;17(68):188-91.
- Cho H, Kwon JW. Prevalence of anaphylaxis and prescription rates of epinephrine auto-injectors in urban and rural areas of Korea. *Korean J Intern Med*. 2019;34(3):643-50.
- Cardona V, Ansotegui IJ, Ebisawa M, El-Gamal Y, Fernandez Rivas M, Fineman S, et al. World allergy organization anaphylaxis guidance 2020. *World Allergy Organ J*. 2020;13(10):100472.
- Jerschow E, Lin RY, Scaperotti MM, McGinn AP. Fatal anaphylaxis in the United States, 1999-2010: Temporal patterns and demographic associations. *J Allergy Clin Immunol*. 2014;134(6):1318-28.
- Sampson HA, Muñoz-Furlong A, Campbell RL, Adkinson NF, Bock SA, Branum A, et al. Second symposium on the definition and management of anaphylaxis: Summary report - Second National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network symposium. *J Allergy Clin Immunol*. 2006;117(2):391-7.
- Motosue MS, Bellolio MF, Van Houten HK, Shah ND, Campbell RL. Risk factors for severe anaphylaxis in the United States. *Ann Allergy, Asthma Immunol*. 2017;119(4):356-61.
- Jeppesen AN, Christiansen CF, Frøslev T, Sørensen HT. Hospitalization rates and prognosis of patients with anaphylactic shock in Denmark from 1995 through 2012. *J Allergy Clin Immunol*. 2016;137(4):1143-7.
- Nishino T, Wakai S, Aoki H, Inokuchi S. Cardiac arrest caused by diphenhydramine overdose. *Acute Med Surg*. 2018;5(4):380-3.
- Eckes L, Tsokos M, Herre S, Gapert R, Hartwig S. Toxicological identification of diphenhydramine (DPH) in suicide. *Forensic Sci Med Pathol*. 2013;9(2):145-53.
- Simons FER, Simons KJ. Review Article Of Current Status and Future Directions. *WAO J*. 2008;563:145-55.
- Kraft M, Scherer Hofmeier K, Ruëff F, Pföhler C, Renaudin JM, Bilò MB, et al. Risk Factors and Characteristics of Biphasic Anaphylaxis. *J Allergy Clin Immunol Pract*. 2020;8(10):3388-95.
- Pourmand A, Robinson C, Syed W, Mazer-Amirshahi M. Biphasic anaphylaxis: A review of the literature and implications for emergency management. *Am J Emerg Med*. 2018;36(8):1480-5.
- Ichikawa M, Kuriyama A, Urushidani S, Ikegami T. Incidence and timing of biphasic anaphylactic reactions: a retrospective cohort study. *Acute Med Surg*. 2021;8(1):1-9.
- Pflipsen MC, Vega Colon KM. Anaphylaxis: Recognition and Management. *Am Fam Physician*. 2020;102(6):355-62.

Cite this article as: Pranata AANS, Wulandari DC, Setiawathi NP, Suryana K. Anaphylaxis shock with laryngeal edema: a case report. *Int J Adv Med* 2024;11:389-93.