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Anaphylactic Shock Due to Wasp Stings

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Abstract: ***Anaphylaxis is the most serious manifestation of an immediate allergic reaction and the most common emergency event in allergology. Hymenoptera venom allergy ranks among the top three causes of anaphylaxis worldwide, and approximately one-quarter of sting-induced reactions are classified as severe. Fatal sting reactions are exceedingly rare, but certain factors may entail a considerably higher risk. The occurrence of anaphylaxis has increased in recent years, and subsequently, there is a need to continue disseminating knowledge on the diagnosis and management, so every healthcare professional is prepared to deal with such emergencies. A case of a 64-year-old man has been reported with complaints of shortness of breath and weakness since 30 minutes after being stung by a wasp. On physical examination, it was found that the patient had a blood pressure drop of 80/60 mmHg, a pulse rate of 120 beats per minute, regular with insufficient volume and a respiratory rate of 24 times per minute. The diagnosis based on Samson's criteria is that the patient presents with a shock condition which is one of the three criteria, namely the patient comes with a decrease in blood pressure of more than 90 mmHg or a decrease of more than 30% of the previous blood pressure immediately after the patient is exposed to allergens without other causes of shock. The manifestations that appear on the organs are reactive transaminitis et causa and stress hyperglycemia with the management of anaphylactic shock is the administration of epinephrine 2 times, fluid resuscitation with infusion of 0.9% NaCl and given a hepatoprotector.***

Keywords: anaphylactic shock, wasp stings, transaminitis, hyperglycemia

1.Introduction

Anaphylaxis is the maximal variant of an acute life-threatening immediate-type allergy and represents the most common and often life-threatening emergency situation in allergology. Recent publications show a global incidence of anaphylaxis between 50 and 112 episodes per 100.000 person-years while the estimated lifetime prevalence is 0.3–5.1%, variations depending on the definitions used, study methodology and geographical areas. Worrying data indicate that recurrence of reactions occurs in 26.5-54.0% of anaphylaxis patients during a follow up time of 1.5 years-25 years. Along with food and drug allergy, Hymenoptera venom allergy ranks among the top three causes of anaphylaxis worldwide. Prevalence rates are estimated to reach up to 7.5% in the adult European population and up to 3.3% in the United States. Venom-induced anaphylaxis also displays regional patterns. A recent report suggested bee venom as the most frequent elicitor in South Korea, whereas, in central Europe (Austria, Germany and Switzerland) wasp is the predominating insect inducing anaphylaxis. In other regions, different stinging or biting insects have been reported to induce anaphylaxis, e.g., red ants in America and Asia and parts of Australia, antivenom used for snake bites in Australia are not uncommon causes of anaphylaxis. Sting-induced systemic reactions cover a broad clinical spectrum, ranging from urticaria/angioedema as the only manifestation to full-blown anaphylaxis with near-fatal and fatal outcomes. Investigations have revealed that many patients who present to the emergency room (ER) with anaphylaxis are misdiagnosed. Physicians mostly face difficulty in diagnosing anaphylaxis due to its vague presentation and differing symptoms. In addition, there is a difficulty in predicting the severity of a reaction. Death may happen in fatal anaphylactic shocks within minutes of the reaction. Hence, it is essential to emphasize the importance of efficient emergency management.[1, 2, 3, 4] This case report is intended to provide information on establishing the correct diagnosis of anaphylaxis and management, especially for emergencies.

2.Cases

Male patient Mr. IWS aged 64 years, a Balinese tribe came to the ER at the hospital on January 5, 2022, with the main complaint of shortness of breath. Shortness of breath was complained of since 2 hours before admission to the hospital, the shortness of breath was felt to arise suddenly and did not improve with a change in position. The shortness of breath was mild at first, but within half an hour it was getting worse. The patient said that the shortness of breath occurred 30 minutes after being stung by a wasp in the field, at that time the patient was working in the fields and suddenly the patient was stung by a wasp before he could escape. The patient said stinging all over the body such as the face, trunk, hands and feet. Another complaint is that the patient feels pain and feels hot all over the patient's body which is felt a few minutes after being stung by a wasp and the patient also feels his body getting weaker. The patient had vomited once at home and felt nauseous. At the time of observation in the ER, the patient's left ear was bleeding and felt pain.

Past medical history such as hypertension, diabetes, heart and kidney disease was denied by the patient. The patient also denied a history of atopy and allergy. The patient denied a family history of hypertension, diabetes, heart and kidney disease. The existence of a history of atopy such as rhinitis, asthma, and dermatitis in the family was also denied.

On physical examination, the patient's consciousness was composmentis, general condition was weak, GCS E4V5M6 with blood pressure 80/60 mmHg, pulse rate 120 times per minute, respiratory rate 24 times per minute, temperature 36.8 C, oxygen saturation 96% room water and VAS 3 on the face, trunk, upper and lower limbs. General status of the head, neck found no abnormalities,

no enlarged lymph nodes, JVP ± 2 cmH2O. Examination of the thorax and abdomen was found to be normal. On examination of the patient's local status, no angioedema or urticaria were found.

Complete blood count, white blood cells 18.1 x103/µL, hemoglobin 11.6 g/dL and platelets 226 x103/µL, hematocrit 39.3%, absolute neutrophils 11.7 x103/µL, absolute lymphocytes 5.3x103/µL. Blood chemistry Urea N 25 mg/dL, blood creatinine 0.93 mg/dL, ALT 19 U/L, AST 46 U/L, sodium 136.2 mmol/L and potassium 3.54 mmol/L, Blood sugar 323 g/dl, HbA1C 4.3%. Electrocardiography with the impression of sinus rhythm tachycardia with a frequency of 110 times per minute, and the results of the patient's chest X-ray readings are within normal limits. While in treatment there was an increase in the liver transaminase enzyme, namely ALT 95 U/L, AST 141 U/L, but after 2 days it was evaluated that there was an correction in the liver transaminase enzyme.

This patient was diagnosed with anaphylactic shock et causa insect sting with transaminitis et causa reactive and hyperglycemia et causa stress hyperglycemia. While being treated, to treat an emergency in the form of anaphylactic shock, the patient was given an injection of adrenaline 0.3 mg (im), twice with an improvement in blood pressure from 80/60 mmHg to 90/60 mmHg initially. Followed by fluid resuscitation of 0.9% NaCl 1000cc, blood pressure increased to 100/70 mmHg, followed by infusion of 0.9% NaCl 20 drops per minute, then hydrocortisone 2x200mg (iv), pantoprazole 2x40mg (iv), ondansetron 3x4mg (iv) if vomiting, cetirizine 2x10 mg (po) and hepatoprotector.

3.Discussion

Anaphylaxis is most commonly defined as an acute, severe, potentially life-threatening systemic hypersensitivity reaction.[5] Despite expressing common clinical features, the underlying mechanisms of anaphylaxis may vary. Nevertheless, some of the activated pathways may be common to different types of anaphylaxis reactions or be present simultaneously. IgE-mediated anaphylaxis is considered the classic and most frequent mechanism. In this type, anaphylaxis is triggered by the interaction of an allergen (usually a protein) interacting with the allergen-specific IgE/high-affinity receptor (FcεRI) complex expressed on effector cells, predominantly mast cells and basophils. This initiates intracellular signaling resulting in the release of preformed and de novo synthesis of mediators. Non-IgE-mediated anaphylaxis may be immunologic or non-immunologic. The most relevant non-IgE-mediated immunologic mechanisms may involve the activation of pathways such as the complement system (anaphylatoxins, C3a, and C5a), the contact and coagulation system activation, or immunoglobulin G (IgG)-mediated anaphylaxis. Non-immunologic mechanisms have been described for some drugs (opioids). Ethanol and physical factors, such as exercise, may be involved in triggering anaphylaxis through mechanisms which are not fully elucidated. Mast cells may be activated through receptors such as Mas-related G-protein coupled receptor member X2 (MRGPRX2) by certain drugs such as neuromuscular blocking agents and fluoroquinolones. Anaphylaxis is classified as idiopathic when no trigger can be identified and currently represents between 6.5 and 35.0% of cases, depending on the studies. In such cases, mast cell disorders should be ruled out. Excluding urticaria pigmentosa does not exclude mastocytosis, neither does a normal baseline tryptase. Detecting KIT mutation in peripheral blood or in bone marrow may be necessary. Also, the role of allergens previously unrecognized (such as alpha-Gal) or less straightforward to identify (omega-5-gliadin, oleosins) has to be considered.[1] The diagnosis of anaphylaxis is fundamentally clinical. Clinical history constitutes the most important tool in the identification of a patient who is suffering from anaphylaxis and it is also of great help to identify its cause. In addition to the detailed description of signs and symptoms present in the patient, it is important to obtain information about the moment of onset of the reaction, medication used to treat it, the duration of the episode and exposure to allergens or potential triggers. Whenever possible, we ought to question the people who witnessed the event.[6] The degree of sensitisation does not indicate the severity of a previous sting reaction. Therefore, a comprehensive clinical history with focus on indicators for severe sting reactions is indispensable. The absence of skin symptoms and a short latency time are associated with severe systemic sting reaction (SSR) in both wasp and honeybee venom allergic patients. The patient's age and BST levels significantly correlate with the grade in patients allergic to wasp venom only.[7] Older age is a very potent risk factor for severe anaphylactic reactions in general, recent data from the European Anaphylaxis Registry actually suggest that it has an even stronger impact on the incidence of severe and near-fatal anaphylaxis than mastocytosis, though this may depend on the eliciting allergen. Whereas an age-dependent predisposition to fatal food-induced anaphylaxis has been described for the second and third decades of life, severe and fatal outcomes of venom-induced reactions are clearly more common in the elderly. A continuous and strictly linear association between increasing age and the frequency of severe sting reactions was detected in the 2009 European multicenter study and other groups confirmed an increase in severe reactions from 40 years onwards. The increasing prevalence of cardiovascular comorbidities in senior age may in part explain the growing vulnerability to severe sting reactions. A male predominance is almost invariably observed in cohorts of venom-allergic patients and male sex was identified as a predictor of more severe reactions in some, but not all studies. Moreover, there is an increased risk of sting-related death in men compared with women. In accordance with these findings, male sex was recently identified as a risk factor for severe anaphylactic reactions independent of the elicitor. These observations stand in some contrast to previous concepts assuming an augmenting effect of female sex hormones on the incidence and/or the severity of anaphylactic reactions. The effect of male sex as a risk factor in venom-allergic patients, however, was mainly attributed to a higher degree of exposure to stinging insects. Detailed instructions on how to avoid Hymenoptera stings might thus be an adequate strategy to prevent severe reactions in venom-allergic males, especially if the patient's history is

suggestive of high exposure to the culprit insect due to occupational or recreational outdoor activities.[3]

In our case, the patient is a male at the age of 64 years, where there are already 2 risk factors that can cause the patient to experience severe anaphylaxis, the first is male gender and the second is age, where age >40 years is a risk factor. For severe anaphylaxis to wasp stings. Then on physical examination the absence of skin symptoms were found where this finding is also one of the factors for the occurrence of severe anaphylactic shock.

The diagnosis of anaphylaxis is based on Sampson's criteria, the first is acute onset (within minutes to several hours) involving the skin and mucosal tissue or both and at least one of the complaints is respiratory system or decreased blood pressure, collapse, syncope or incontinence. Second, clinical features were found in two or more organs immediately after exposure and third, there was a decrease in blood pressure immediately after exposure, namely systolic pressure less than 90 mmHg or a decrease of more than 30% of the previous blood pressure immediately after the patient was exposed to the allergen without other causes of shock found. Symptoms that appear during anaphylaxis vary with manifestations according to the target organ, namely the skin, mucosal system, respiratory system, cardiovascular system, gastrointestinal system and other systems.[8]

Table 1: Clinical criteria for diagnosing anaphylaxis[12]

Anaphylaxis is highly likely when any one of the following 3 criteria are fulfilled:
1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (eg, generalized hives, pruritus or flushing, swollen lips-tongue-uvula)
AND AT LEAST ONE OF THE FOLLOWING
a. Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
b. Reduced BP or associated symptoms of end-organ dysfunction (eg, hypotonia [collapse], syncope, incontinence)
2. Two or more of the following that occur rapidly after exposure *to a likely allergen for that patient* (minutes to several hours):
a. Involvement of the skin-mucosal tissue (eg, generalized hives, itch-flush, swollen lips-tongue-uvula)
b. Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
c. Reduced BP or associated symptoms (eg, hypotonia [collapse], syncope, incontinence)
d. Persistent gastrointestinal symptoms (eg, crampy abdominal pain, vomiting)
3. Reduced BP after exposure to *known allergen for that patient* (minutes to several hours):
a. Infants and children: low systolic BP (age specific) or greater than 30% decrease in systolic BP*
b. Adults: systolic BP of less than 90 mm Hg or greater than 30% decrease from that person's baseline

Anaphylactic reactions are accompanied by a multitude of symptoms affecting different organs, which sometimes occur in succession and sometimes simultaneously, but not necessarily always to the same degree. In most cases (80-90%), the reactions start with subjective general symptoms and skin manifestations (for example, urticaria/hives 62%, angioedema 53%), sometimes accompanied by formication on the palms of the hands and soles of the feet. Advanced symptoms include nausea of the gastrointestinal tract in 24% of those affected, colic-type pain in 16%, vomiting in 27%, and diarrhea in 5%. The respiratory tract is affected in 49%. Those affected experience dyspnea, either as a narrowing of the upper airway in the sense of laryngeal edema or as asthmatic bronchial constriction (35%). Anaphylaxis can affect the cardiovascular system-for example, by triggering tachycardias or blood pressure fluctuations in up to 42% of cases. These can be so comprehensive that anaphylactic shock may ensue. Anaphylaxis can affect the same patient to different degrees of intensity, which is considered in the classification into grades of clinical severity.[2]

Hymenoptera venoms are complex mixtures of biologically active peptides, enzymes, and amines. Bee venom contains melittin, phospholipase A2, mast cell-degranulating peptide (peptide 401), hyaluronidase and apamin, among other constituents. Melittin, which makes up approximately 50% of the entire bee venom mixture, powerfully disrupts cell membranes and has direct toxic effects on renal tubular cells of the host. Wasp venom lacks melittin; instead it contains antigen 5 as the main allergen. These components have direct and indirect cytotoxic (hepatic, renal and myocyte membrane), hemolytic, neurotoxic and vasoactive properties, which can cause intravascular hemolysis and rhabdomyolysis. Phospholipase A2 is believed to trigger the release of arachidonic acid from lipids in the cell membrane, which initiates the production of inflammatory eicosanoids. Hyaluronidase in the venom causes breakdown of connective tissues, thereby facilitating the spread of the venom.[9] Wasp stings can result in multi system involvement ranging from hemolysis, rhabdomyolysis, acute renal failure, disseminated intravascular coagulation, myocardial dysfunction, hepatic dysfunction and thrombocytopenia, which may occasionally become fatal. Acute kidney injury can occur due to acute tubular necrosis secondary to shock, or pigment nephropathy resulting from rhabdomyolysis and intravascular hemolysis, interstitial nephritis from a hypersensitivity reaction to the wasp venom, or direct nephrotoxicity of venom. Acute cortical necrosis has also been reported following wasp stings. Other systemic manifestations include myocardial necrosis and infarction, centrilobular necrosis of liver, and thrombocytopenia as a result of direct platelet toxicity.[10] Timely treatment considerably reduces the risk of mortality in patients with anaphylaxis. An immediate assessment utilizing an airway, disability, circulation, breathing, and exposure approach is the primary step in a patient with anaphylaxis. Patient must stay in a flat position and must not stand or walk. Then, if

an allergen still presents, it should be removed, for example, insect stings should be flicked out, and ticks should be frozen with liquid nitrogen or ether-containing spray and allowed to drop off. Local reactions to hymenoptera stings are treated with oral antihistamines and topical corticosteroids. A short course of oral corticosteroids may be needed for large local reactions. Patients with airway obstruction or hypotension should be treated with adrenaline intramuscularly. Adrenaline is the first drug for treatment of anaphylaxis. The recommended dose is 0.01 mg/kg (maximum dose of 0.3 mg in children and 0.5 mg in adults) administered intramuscular in the antero-lateral area of the thigh, repeating every 5-15 min if necessary. However fluid replacement is required with adrenaline. High flow oxygen and beta-agonist therapy should be given for bronchospasm treatment. Intravenous administration is an option in patients with severe hypotension or cardiovascular collapse who do not respond to treatment with intramuscular epinephrine and intravenous fluids. Nevertheless, due to the risks of lethal arrhythmia, the latter form of administration should be considered only when continuous cardiac monitoring is possible. Continuous monitoring of the pulse, oxygen saturation, blood pressure, and respiratory rate is recommended.[4, 6, 11]

In our case, based on the anamnesis, the patient came with complaints of shortness of breath since 2 hours before admission to the hospital and was getting heavier and felt weak. The patient complained of shortness of breath after 30 minutes of being stung by a wasp, so the patient was diagnosed with anaphylaxis due to a wasp sting. On physical examination, the patient was found to be in shock (a drop in blood pressure of 80/60 mmHg with a pulse of 120 beats/minute, regular content less, respiratory rate 24 times/minute). The diagnosis based on Sampson's criteria was that the patient presented in shock. One of the three criteria is a decrease in blood pressure of more than 90 mmHg or a decrease of more than 30% from the previous blood pressure immediately after the patient is exposed to an allergen without other causes of shock.

In our patient there was anaphylactic shock, then epinephrine 0.3 mg intramuscularly given twice and then there was an improvement in blood pressure from 80/60 mmHg to 90/60 mmHg. Followed by corticosteroids, namely hydrocortison and crystalloid fluid resuscitation, namely NaCl 0.9% 1000cc to improve intravascular volume, followed by 20 drops/minute.

On laboratory examination found an increase in the liver transaminase enzyme, this may be caused by the poison caused by wasp stings where wasp venom contains antigen 5 as the main allergen. These components have direct and indirect cytotoxic (hepatic, renal and myocyte membrane), hemolytic, neurotoxic and vasoactive properties, which can cause intravascular hemolysis and rhabdomyolysis. Phospholipase A2 is believed to trigger the release of arachidonic acid from lipids in the cell membrane, which initiates the production of inflammatory eicosanoids. Hyaluronidase in the venom causes breakdown of connective tissues, thereby facilitating the spread of the venom. Wasp stings can result in multi system involvement ranging from hemolysis, rhabdomyolysis, acute renal failure, disseminated intravascular coagulation, myocardial dysfunction, hepatic dysfunction, and thrombocytopenia, which may occasionally become fatal.[9, 10]

4.Summary

A 64-year-old male patient has been reported with complaints of shortness of breath and weakness after being stung by a bee. The diagnosis of anaphylactic shock is based on the Sampson criteria. On laboratory examination found an increase in liver transaminases enzymes. Management by administering epinephrine and fluid resuscitation and given a hepatoprotector. After the therapy was given, the patient improved and the liver transaminase enzymes returned to normal.

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