內科學誌 2005 ：16 ：91-94

Bradycardia in a Patient with Anaphylactic Shock ： Case Report

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Abstract

Anaphylactic shock, as a kind of distributive shock, is considered as a consequence of histamine induced vasodilatation with increased vascular permeability. Tachycardia is the early warning sign of shock and bradycardia is a rare manifestation of shock. A 78 year-old woman presented with shrimp-induced anaphylactic shock with bradycardia. She was treated by beta-blocker currently and further Thalium-201 myocardial scan showed reversible myocardial ischemia. She had full recovery after aggressive resuscitation and discharged without any sequelae. The causes of developing bradycardia in patients with anaphylactic shock maybe multifactorial involving hypotension or histamine induced transient SA node ischemia, Benzoid-Jarish reflex, and concurrent beta-blocker usage. Prompt management with epinephrine, atropine, inotropic/vasopressor agents and intravenous fluid resuscitation is mandatory. Glucagon should be considered if there is a concern of concurrent beta-blocker intoxication. Bradycardia developed during shock status, particular in patients with severe allergic reaction, coronary artery disease and current beta-blocker usage worth particular attention. (J Intern Med Taiwan 2005; 16: 91-94)

Key Words ： Anaphylaxis, Bradycardia, Benzoid-Jarish reflex

Introduction

Anaphylaxis shock, as a kind of distributive shock, is considered as a consequences of histamine induced vasodilatation and increased vascular permeability. It has been emphasized that tachycardia is the early warning sign of shock, on the contrary to bradycardia. Bradycardia developed in patients with

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anaphylactic shock is rare[1]. Here we presented a patient who developed sinus bradycardia accompanied by seafood-induced anaphylactic shock.

Case report

A 78-year-old woman presented to the emergency department with obvious skin rash, puffy face and loss of consciousness. She has suffered from long-term hypertension and type 2 diabetes and currently was treated by amlodipine 5 mg, atenolol 25 mg for her blood pressure control and never experienced any adverse effect related to antihypertensive agents before. She had severe urticaria and angioedema after ingestion of shellfish and was warned to avoid seafood as possible. There was no fever, chills or any discomfort reported prior to admission. On arrival to the emergency department, she could only response to vigorous pain stimuli with blood pressure of 56/20 mmHg, pulse rate of 34 beats per minute and body temperature of 37.2 ℃. Physical examinations disclosed generalized urticaria and dermatographism. No stridor or wheezing was found. 12-lead electrocardiography (EKG) showed sinus bradycardia with rate of 34 beats per minute. (Fig. 1A) The laboratory data included a white blood cell count of 5400 / μL, with neutrophil 66.2 %, lymphocyte 20.3%, eosinophil 5.1 % and basophil 2.5%, a hemoglobin of 10.9 gm/dL, and a platelet count of 313,000 / μL. Her blood sugar was 189 mg /dL. Creatine phophokinase(CK) and CK-MB form were 75 and 9 u/L respectively. Troponin I level was less than 0.5 ng/mL. Other blood chemistry studies were unremarkable. Previous ingestion of chopped shrimp salad was reported by her maid. Resuscitation with intramuscular 1 : 1000 epinephrine 0.3 ml, intravenous atropine 2.0 mg and normal saline infusion were given. Dopamine was administered continuously to maintain heart rate and blood pressure. She was admitted to the intensive care unit. Her vital signs were stabilized and dopamine infusion was discontinued 2 days later. Follow-up EKG disclosed normal sinus rhythm with rate of 61 beats per minute. (Fig. 1B) The echocardiography showed normal left ventricular systolic function and minimal mitral regurgitation. She was transferred to regular ward and was discharged 8 days later without any sequelae. Thalium-201 myocardial scan disclosed reversible inferior wall myocardial ischemia. There was no evidence of pulmonary or urinary tract infection. The blood cultures obtained on admission failed to yield any pathogens. Avoidance of shellfish and seafood was advised strongly.

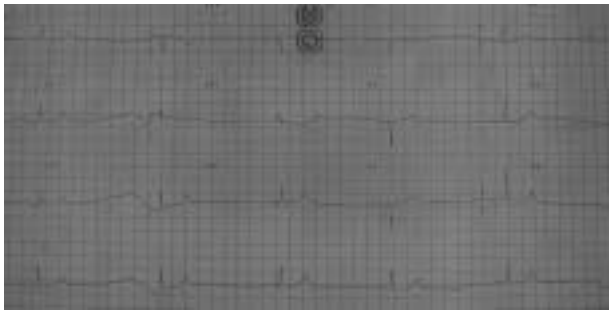

Fig.1A. EKG obtained on arrival. The EKG showed sinus rhythm with rate of 34 beats per minute.

Discussion

Various mechanisms were responsible for developing bradycardia in patients with anaphylactic shock[2]. Bradycardia may develop secondary to direct ischemic effects on the SA node resulting from spasm of the right coronary or left circumflex arteries. Histamine is capable of inducing tachycardia, but coronary vasoconstriction as well[3]. Some mediators released during anaphylactic shock are potent coro-

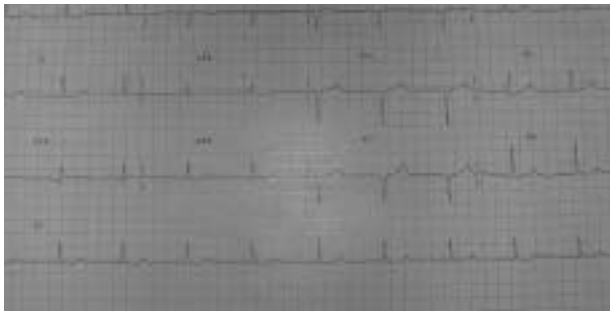

Fig.1B. EKG obtained 2 days later. The EKG showed sinus rhythm with rate of 60 beats per minute. Non-pathologic Q wave was found over lead II, III and aVF.

nary constrictors could induce coronary spasm[4]. Benzoid-Jarish reflex is an inhibitory reflex that originates in the inferioposterior wall of the left ventricle. The precise mechanism is unknown, but probably is mechanical because activation occurs concurrently with systolic bulging of the ischemic myocardium[5]. This reflex is activated by local ischemia of the myocardium which was perfused by the right coronary artery but not activated by hypoxia and hypercapnia. Stimulation of the reflex increases parasympathetic activity and inhibits sympathetic activity, producing bradycardia, further vasodilation and hypotension. This reflex mechanism may serve as a protective mechanism allowing for improved diastolic filling when venous return is critically reduced [6]. Beta-blockers are used widely in patients with cardiovascular diseases. Patients treated with beta-blockers have increased severity and incidence of anaphylaxis due to decreased intracellular cyclic adenosine monophosphate (cAMP) and lowered threshold of mediator release by mast cell and basophils[7-8]. They may not have typical symptoms or signs attributed to increased sympathetic tone. Thus such medications should be prescribed with caution in a patient with history of severe allergy.

The causes of developing bradycardia in patients with anaphylactic shock, as the case we presented here, maybe multifactorial involving hypotension or histamine induced transient SA node ischemia, Benzoid-Jarish reflex, and concurrent beta-blocker usage. Sick sinus syndrome and autonomic dysfunction should be also considered in elderly and diabetic patients. Patients with anaphylactic shock should be resuscitated with intramuscular epinephrine injection, fluid challenge and inotropic/vasopressor agents[1]. Colloid fluid may be better than crystalloid in such circumstance. Atropine is indicated for the unstable bradycardia. H2 antihistamine is sometimes useful in anaphylaxis, particular in reversing cutaneous manifestation[9] but cimentidine could decrease the clearance of beta-blockers and might prolong its effect. Patients with bronchospasm should be treated with corticosteroid and aminophyllin. Intravenous injection of glucagons has been reported effective in treating beta-blocker overdose, possibly owning to the direct effect on cAMP in the cardiac tissue[7]. Glucagon should be administered if the vaspopressor/inotropic agents fail to stabilize patients.

In conclusion, bradycardia could be the initial presentation in patients with anaphylactic shock. Invasive hemodynamic monitoring and echocardiography would elucidate complex hemodynamic status. The Importance of those alphabets A, M and P, that denote the allergy, medicines and past illness, of the "AMPLE" history worth to be emphasized again [10]. Although tachycardia is considered as the early warning sign of shock, mistaken a relative bradycardia accompanied with or without hypotension as a benign course and led to a delayed resuscitation may contribute to a negative outcome eventually.

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過敏性休克合併心搏過慢：一病例報告

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摘　要

過敏性休克爲組織胺所造成血管擴張及通透性增加使然的一種分佈性休克，心搏加速爲休克初期的徵兆而心搏過慢於過敏性休克極爲罕見。一位78歲的女性因誤食蝦類引發而引發過敏性休克並合併心搏過慢。此病患平時有服用貝他阻斷劑而其後所接受的心肌掃描亦顯示可逆性的心肌缺血現象。此病患經過積極地急救後出院無任何後遺症。於過敏性休克的病患發生心搏過慢現象似爲多重因素，可能的機轉包括有組織胺或低血壓造成的一過性的竇房結缺血，Benzoid-Jarish 氏反射及接受貝他阻斷劑的治療等。病患若是呈現休克合併心搏過慢的情況應積極地予以腎上腺素、阿托平、強心劑/血管收縮劑及輸液等治療，昇糖素應用於beta-blocker 過量之病患。若於休克的病患發生心搏過慢的現象，特別是合併有嚴重的過敏反應、冠心症及使用貝他阻斷劑者，應予以特別的注意。