



PIC QUESTION OF THE WEEK: 4/14/08

Q: Why should a history of beta-blocker administration be a consideration when treating anaphylaxis?

A: Anaphylaxis is generally defined as an immediate reaction mediated by IgE whose clinical presentation may affect multiple organ systems. Cutaneous (urticaria, angioedema, flushing, etc.), cardiovascular (palpitations, hypotension, shock, etc.), gastrointestinal (abdominal pain, nausea, etc.), and respiratory (e.g. nasal congestion, sneezing, edema of the larynx and tongue, dyspnea, wheezing) complications characterize anaphylaxis. Medications, food (peanuts, shellfish, etc.), allergen immunotherapy vaccines, insect bites, latex, and various environmental factors can trigger an anaphylactic reaction. Typical treatment of anaphylaxis includes parenteral therapy with epinephrine. Dosage ranges from 0.2 mg – 0.5 mg and injections can be repeated every 5 – 20 minutes as necessary. Intramuscular injection is preferred; however, the intravenous or even endotracheal routes may be required in life-threatening situations. Antihistamines (H₁ and H₂) and corticosteroids are generally used in conjunction with epinephrine for managing patients suffering from anaphylactic shock. Chronic use of beta-blockers has long been considered a potential factor in reducing patient response to epinephrine. There is also evidence that prior use increases the severity of anaphylaxis as well as the frequency of acute reactions to radiocontrast media. Beta-blockers increase the release of mast cell mediators such as histamine, leukotrienes, etc. that are responsible for many of the associated signs and symptoms of anaphylaxis. They also modulate adenyl cyclase activity and can diminish the stimulatory effects of epinephrine on beta receptors. These factors may contribute to apparent resistance when patients are treated with epinephrine. It has been suggested that patients with anaphylactic reactions who are receiving beta-blockers be treated with a dose of epinephrine that is 2-5 times the recommended standard. In cases of severe resistance, intravenous injection of glucagon may be of some benefit. Glucagon possesses inotropic, chronotropic, and vasoactive properties independent of beta receptors and also stimulates endogenous catecholamine release. Although there is limited data to support this indication, most authors mention the compound when discussing treatment of this potentially fatal reaction. A history of beta-blocker administration should always be considered in managing patients with anaphylaxis.

References:

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- Miller MM, Miller MM. β -blockers and anaphylaxis: are the risks overstated? *J Allergy Clin Immunol* 2005; 116:931-3; author reply 933-6.

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