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Anaphylaxis treatment: the details

Having read the review article by Anne Ellis and James Day,[1] I have several questions about drug therapy for anaphylaxis.

Ellis and Day[1] report that patients seen in their unit are usually discharged with a 4-day prescription for prednisone and diphenhydramine, a relatively common approach. However, given that many patients must drive or go to work, I wonder why the authors do not advocate one of the newer nonsedative antihistamines. Similarly, would it be appropriate to recommend the addition of ranitidine for 48 hours, on the basis of the experimental evidence presented by Ellis and Day[1] and given the risk of a biphasic reaction? Since the second-phase reaction may be more severe than the primary reaction,[1] this approach might be safer, although it is as yet unproved. I also wondered what dosage of prednisone is recommended for postdischarge therapy and whether the dose should be tapered.

Ellis and Day[1] mention the cross-reactivity between cephalosporin and penicillin, but there have been conflicting recommendations as to whether this applies to the third-generation cephalosporins. Kelkar and Li[2] recommended against prescribing third-generation cephalosporins to patients allergic to penicillin, but their review was based on extrapolation and inference. Anne and Reisman[3] concluded that it is safe to administer cephalosporin antibiotics, especially third-generation drugs, to penicillin-allergic patients. Pumphrey and Davis[4] reported 6 anaphylactic deaths after a first cephalosporin dose, which occurred over a 5-year period in the United Kingdom. Three of these patients had a penicillin allergy, but the generation of the cephalosporins in these cases was not indicated. In my own experience, many physicians in France are not reluctant to use third-generation cephalosporins, when indicated, for penicillin-allergic patients (in the hospital environment).

Finally, prescribing epinephrine as volumes of a 1:1000 solution is a potentially dangerous dosing system. Administering epinephrine measured in micrograms (or milligrams), as pumped from clearly labelled ampoules, might avoid inadvertent ventricular tachycardia.

Axel Ellrodt
Emergency Department
American Hospital of Paris
Paris, France

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In their excellent review of the diagnosis and management of anaphylaxis, Anne Ellis and James Day[1] mention that anaphylactic patients who use β-blockers should be given glucagon. I was not aware of this use of glucagon.

In my own experience as a family physician, the most significant case of anaphylaxis that I remember involved a patient who had not previously been seen in our clinic and whose medical history was unknown to us. He walked into the clinic, bypassed the receptionist and entered an examination room, where he lost consciousness. Resuscitation required multiple intravenous doses of epinephrine. The patient's condition was eventually stabilized in hospital with administration of corticosteroids.

We later learned that this patient, who was taking β-blockers and who had not previously been aware of any allergies, had been stung by an insect while walking along a street leading toward the clinic. Fortunately, he was able to reach the clinic before losing consciousness.

Although this incident happened 20 years ago, it remains applicable, reminding us that patients with anaphylaxis often do not present to their own physician, and a history of β-blocker therapy may not be evident. In this situation, would Ellis and Day recommend a combination of epinephrine and glucagon?

Patrick J. Potter
Department of Physical Medicine and Rehabilitation
University of Western Ontario
London, Ont.

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[The authors respond:]

Axel Ellrodt raises several questions regarding discharge therapy after anaphylaxis. The first relates to alternatives to diphenhydramine prophylaxis. Diphenhydramine has been established as an effective agent in the treatment and prevention of anaphylactic and anaphylactoid reactions, where its sedative properties are an advantage.[1] Given orally at doses of 25 to 50 mg every 4 to 6 hours, it remains the antihistamine of choice to prevent and manage these episodes. A second-generation antihistamine could be substituted if sedation were a concern. However, because biphasic reactivity may be delayed for up to 24 hours, the patient should be advised to minimize activity (including driving) during this interval, and sedative effects may therefore be unimportant. After this interval, treatment with