Letters to the Editor

Allergy Evaluation and Desensitization Standards for Radiocontrast Media

To the Editor:
We reviewed with interest the article by Khan et al. outlining their use of rapid drug desensitization (RDD) to radiocontrast media (RCM).1 While we commend their use of RDD, there are some points that warrant clarification.

The term anaphylactoid has been supplanted by non-immunologic or non-IgE-mediated anaphylaxis. Anaphylactoid reactions had been perceived as non-IgE-mediated reactions, but current evidence suggests that mast cells can be triggered to release the same mediators through non-IgE mechanisms, either directly through other receptors (such as complement receptors for anaphylotoxins C3a and C5a) and others. Mast cell degranulation (MCD) by RCM is inducible through both IgE and non-IgE mechanisms, and, regardless of the trigger mechanism, all forms of anaphylaxis can be severe or fatal. The reaction history is suggestive of IgE-mediated allergy to RCM, and skin testing should have been done to demonstrate sensitization and select alternate RCM agents.2 The patient’s preceding reaction despite pretreatment likely demonstrates sensitization and that it was not a refractory event as the title indicates. This was an important clue of the IgE-mediated nature of his allergy, for which RDD was appropriately undertaken.

The authors further assert that desensitization is sometimes dose dependent, thereby rationalising their administration of a higher dose of RCM than others have cited in the literature. In fact, desensitization is always dose and time dependent, as supported by in vitro data.3 The total amount of exposed drug during RDD should always achieve the desired therapeutic or diagnostic dose required.

Moreover, although we agree that there is much to learn regarding mechanisms, RDD does not induce subclinical histamine depletion. Instead, desensitization serves to prevent IgE-antigen internalization and transmembrane calcium influx, both of which are prerequisites for MCD.3 This diverts intracellular signalling cascades away from those leading to degranulation, and induces what might be comparable to a cardiomyocyte-like refractory state of the mast cell. There is no evidence that patients would remain in their desensitized state for 24-48 hours following the procedure, because the enduring effect is influenced by an individual drug’s half-life.

Finally, although the safety and efficacy of RDD is well established, cardiac patients, owing to underlying coronary artery disease and/or coadministration of medications such as beta-blockers and angiotensin-converting enzyme inhibitors (which may induce resistance to epinephrine if required or possibly increase reaction severity), represent a higher-risk population.4 This case thereby highlights that availability of expertise in drug desensitization and capability to offer it allows for optimal care of the cardiac patient with RCM allergy.

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