

Foreword

Drug Allergy and Primum Non-nocere



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Consulting Editor

Drug-induced allergy is the moral equivalent of the primal sin for the medical profession. Arguably, the temptation here is not pleasure but a noble goal to help a sick patient. Nonetheless, we are in conflict with our first principle of “*primum non nocere*” whenever our patients experience an adverse event because of our intervention.

Drug allergy has always been a difficult subject because of poor understanding of the underlying mechanism. This was primarily a result of the difficulty in establishing a patient cohort, lack of appropriate study reagents, and inadequate knowledge about the immune response to small molecular-weight substances. Remarkable progress has been made in recent years in this very difficult area of science. Application of pharmacogenomic approaches led to identification of the role of HLA-B*1502, HLA-B58, and HLA-B57 for sensitivity to carbamazepin, allopurinol, and abacavir, respectively.^{1,2} The screening for these HLA genotypes now saves lives. HLA-B*1502 predisposes the Chinese Han population to Stevens-Johnson syndrome when taking carbamazepin.³ Progress has also been made in understanding the immunologic basis for drug allergy. Precise binding of the offending drug to a particular HLA subtype leads to a productive CD8 T-cell immune response.⁴ This type of research has allowed better classification of various drug allergies.⁵ Better understanding has begun to result in newer diagnostic tests. Finally, drug desensitization protocols are beginning to be validated in a more systematic manner instead being presented as empirical experience.

Dr. Pichler has been performing pioneering studies in drug allergy and contributing to new knowledge in the field.⁵ He has invited a distinguished group of investigators

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and clinician-scholars, and in so doing has put together an outstanding update on drug allergy. This is a valuable asset for any practicing clinician.

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