Drug-induced Hypersensitivity Syndrome (DIHS): A Reaction Induced by a Complex Interplay among Herpesviruses and Antiviral and Antidrug Immune Responses

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A relationship between viral infections and the simultaneous or subsequent development of allergic inflammation has often been observed in various clinical situations. Recent studies suggest an intimate relationship between reactivations of herpesviruses including human herpesvirus 6 (HHV-6) and the development of a severe systemic hypersensitivity reaction referred to as drug-induced hypersensitivity syndrome (DIHS).This syndrome has several important clinical features that cannot be solely explained by drug antigen-driven oligoclonal expansion of T cells: they include paradoxical worsening of clinical symptoms after discontinuation of the causative drug. In view of the similarity to GVHD or immune reconstitution syndrome (IRS) in clinical manifestations and emergence of viral infections, the clinical symptoms observed during the course of DIHS and GVHD are likely to be mediated by antiviral T cells that can cross-react with the drug and alloantigens, respectively. In considering common intrinsic properties of the causative drugs to potentially induce immunosuppression, reconstitution of a valid immune response to these viruses, which is typically observed in IRS, may be the most crucial process that takes place after withdrawal of the causative drug in patients with DIHS. Thus, this syndrome should be regarded as a reaction induced by a complex interplay among several herpesviruses (EB virus, HHV-6, HHV-7, and cytomegalovirus), antiviral immune responses, and drug-specific immune responses. This review includes discussion of the pathomechanism, the clinical symptoms, laboratory findings, pathological findings and therapy.

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