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Post-infectious autoimmune syndrome (PIFAS) as a pathogenic and predictive factor to trigger chronic diseases of infectious origin (CDIO)

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Etiopathogenesis of autoimmune diseases (in particular, at its subclinical stage) is still poorly known despite intensive research of mechanisms of autoaggression. At the same time immune reactions triggered by heterogenous (i.e. microbial) Ags can also be ignored at the autoagression suppression by physiological tools of immune surveillance (apoptosis or immune suppression), because autoreactive T- and B-cells are able to survive for several reasons, including molecular mimicry phenomenon known for a variety of areas of clinical medicine. The phenomenon is based on the activation of autoreactive lymphocytes by cross-reactive epitopes of microbial agent within the confines of induced by the same agent infection. The outcome of such phenomenon are implemented during tissue auto-Ags' epitopes recognition and presentation by T-cells to result manifestations of so-called post-infectious autoimmune syndrome (PIFAS), a new combinatorial biomarkers demonstrating immune-mediated (including latent) disorders. Development of PIFAS is featured with a progression of chronic relapsing diseases of infectious origin. We have investigated the syndrome-like immunopathology as applicable to a model of chronic pyelonephritis, intracranial infectious and other chronic inflammatory processes, i.e., chronic myocardites, chronic obstructive bronchitis. The interpretation of the term molecular mimicry may stress a cross-reactivity between self-epitopes and microbial epitopes to invade the body via interplaying a network of Abs. In view of the structural homology immune response caused by a microbial pathogen to balance between two categories of epitopes (self-epitopes and microbial epitopes) is being developed (even in the absence of the pathogen) through both autoreactive T-cells and auto-Abs on account of existing autoepitopes possessing mimicking properties in point of primary microbial (triggering) Ag epitope. Wherein the evaluation of triggering role of infection in the pathogenesis of PIFAS is often difficult due to the fact that a time period for triggering the infection to be transformed into PIFAS may begin prior specific manifestations of PIFAS would form. In fact the identification of such pathogen is restricted by some difficulties, because it is practically impossible to detect autoaggressive changes before the elimination of inducing pathogen from the patient. Thus, for autoimmune myocarditis to make a bridging link with the infection is established for two-thirds of all patients, and transformation of primary (infectious) phase into PIFAS is initiated by mimicking epitopes of, for instance, Coxsackievirus (CVB3) and/or Herpesviridae (CMV), herewith presence of cardiomyosine autoreactive CTLs (CM-autoreactive CTLs) and anti-CM auto-Abs, damaging myocardium to release sequestered autoAgs and to facilitate the induction and/or development of PIFAS is required. We can stress that a strictly fixed tandem of two categories of mutually mimicking epitopes (microbial and self-epitopes) is actively implicated in the pathogenesis of PIFAS. The therapeutic strategy of such patients should be different in accordance with the results obtained: If there are signs of immunodeficiency associated with the infection (in the early stage of disease) immunomodifiers should be included in the treatment modes, whereas in case of PIFAS, a targeted immunosuppressive therapy or a treatment to target signaling biomolecules not yet defined become a valuable priority. The rational approach for solving the problems may serve enzyme-linked immunosorbent assay (ELISA) and flow cytofluorometry, the latter could be useful for features of targeted Ag presentation on processed epitopes on APC's surface rating, providing the opportunity for physicians to predict PIFAS development from both standpoints of epitope immunogenicity and related auto-Abs and TCR avidity. Moreover, clinical rating of TCR degeneracy could be invaluable for the formulation of final version of PIFAS to verify the diagnosis and propose an adequate immunotherapy protocols using higly specific (anti-TCR) MAbs with pharmacotherapeutic effect. The identification of primary microbial pathogen or microbial associate (regardless of its area) is no less important part within the boundaries of the protocol being used, for what we applied immunotechnologies combined with molecular diagnostics, special place among which occupied blot hybridization and PCR-detection in microbial gene pools being screened for. Despite a large armamentarium of approaches for the evaluation of the PIFAS and its complicative manifestations, there are still no obvious clinical and laboratory (including clinical and immunological) criteria to get the syndrome validated. An application of transgenic models to suit the aims and objectives of clinical practice will give an opportunity to reveal the sequence of events between induction and progressing of PIFAS and will allow to pre-select specific targets to be utilized as targets for pharmacotherapeutic effects to control induction and progression of PIFAS and thus chronization of the disease to prevent the latter in time.

Biography

Sergey S Suchkov, MD PhD a researcher-immunologist, a clinician, graduated from Astrakhan State Medical University, Russia, in 1980. He has been trained at the Institute for Medical Enzymology, The USSR Academy of Medical Sciences, National Center for Immunology (Russia), NIH, Bethesda, USA) and British Society for Immunology to cover 4 British university facilities. Since 2005, he has been working as faculty professor of I.M. Sechenov first Moscow State Medical University and of A.I.Evdokimov Moscow State Medical & Dental University. From 2007, he is the first Vice-president and dean of the School of PPPM Politics and Management at the University of World Politics and Law. In 1991-1995, he was a scientific secretary-in-chief of the editorial board of the international journal *"Biomedical Science"* (Russian Academy of Sciences and Royal Society of Chemistry, UK) and the international publishing bureau at the presidium of the Russian Academy of Sciences. In 1995-2005, he was a Director of the Russian-American program in immunology of the eye diseases. He is a member of EPMA (European Association of Predictive, Preventive and Personalized Medicine, Brussels-Bonn), a member of the NY Academy of Sciences, a member of the editorial board for open journal of immunology and others. He is known as an author of the concept of post-infectious clinical and immunological syndrome, co-author of a concept of abzymes and their impact into the pathogenesis of auto immunity conditions, and as one of the pioneers in promoting the concept of PPPM into a practical branch of health services.

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