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Pathogenesis of skin injury of systemic lupus erythematosus (SLE)

Guo-Min Deng
Nanjing Medical University, China

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by high levels of autoantibody and multi-organ tissue damage. Skin injury is the second most common manifestation in patients with SLE, yet the etiology and pathogenesis of skin injury in SLE remains unclear. In this review, we discuss the role of lupus serum IgG in the mechanism of skin injury of SLE based on our recent findings. Compared to skin of healthy individuals, there is a large amount of IgG deposition in skin of patients with SLE. Serum from SLE patients and lupus-prone mice induces skin inflammation following intradermal injection into normal mice. Lupus serum depleted of IgG failed to cause skin inflammation. Monocytes, but not lymphocytes, were found to be crucial in the development of lupus serum-induced skin inflammation, and lupus serum IgG induced monocyte differentiation into dendritic cells (DCs). TNF-α and TNFR1, but not TNFR2, were required for the development of lupus serum-induced skin inflammation. TNFR1 inhibitor but not TNFR2 inhibitor suppressed skin injury in lupus -prone mice. Spleen tyrosine kinase (Syk) is involved in FcγR signaling transduction, inhibition of Syk in lupus-prone mice prevented the development of skin disease and significantly reduced established skin disease. These studies demonstrated that disruption of the TNFR1-mediated signaling pathway and blockade of DC generation and IgG- FcγR signaling transduction may prove to be of therapeutic value in patients with cutaneous lupus erythematosus.

Biography

Guo-Min Deng has graduated from Tongji Medical University and Peking Union Medical College with MD in 1988 and MS degree in1994. He obtained his PhD from Gothenburg University in Sweden in 2001. He completed his Postdoctoral training in National Institutes of Health in Maryland, USA during 2002-2006. He became as an instructor in Harvard Medical School in 2006 and became Assistant Professor in Harvard University in 2011 in Boston, USA. He accepted a position of distinguished Professor in Nanjing Medical University in 2012. His research field is "pathogenesis and therapy of autoimmune diseases".

gmdeng@njmu.edu.cn