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To the Editor:

We read with interest the article by Hession, et al. that described a 59-year-old woman who developed systemic lupus erythematosus (SLE) after parvovirus B19 infection; they wondered whether this phenomenon might be due to clinical mimicry or autoimmune induction.

Although not extensively studied to date, some reports show Th1-type cytokines might be increased during parvovirus B19 infection. Isa, et al. reported that elevated levels of the Th1 cytokines such as interleukin 12 (IL-12) and IL-15 were evident at the time of the initial peak of parvovirus B19 viral load during acute infection, and some of these patients had a sustained Th1 cytokine response during followup (20 to 130 weeks after acute infection). They also showed that the Th1 cytokine response correlated with the previously identified sustained CD8+ T cell response and viremia.

Also, there have been reports showing Th1-type cytokines might be involved in the development of systemic lupus erythematosus (SLE). Tokano, et al. reported that the levels of IL-12 in SLE patients were significantly higher than those in normal subjects; Aringer, et al. showed that IL-15 was elevated in SLE sera and was correlated significantly with Bcl-2 expression. Segal, et al. showed that administration of IL-12 to aging mice reversed their Th1/Th2 cytokine profile and thus rendered them vulnerable to induction of experimental SLE; and Bo, et al. demonstrated that elevated expression of transmembrane IL-15 in immune cells correlated with the development of murine lupus, suggesting that Th1-associated cytokines might play a crucial role in the pathogenesis and development of SLE.

Thus there is a possibility that persistently increased Th1-associated cytokines, especially IL-12 and IL-15, after parvovirus B19 infection might be involved not only in transient symptoms of SLE but also in chronic SLE autoimmunity. Further studies should be performed to evaluate whether Th1-associated cytokine levels might correlate with the IgM/IgG antibody titers or viremia in parvovirus B19 infection-associated SLE.

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