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Serum Levels of CD4, CD8, CD25, and Anti-VZV Antibodies in Patients with Varicella-Zoster Virus Infection

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ABSTRACT

Immune activation has been shown in primary and recurrent infections of varicella-zoster virus (VZV), but little is known about the detailed mechanism. The aim of this study was to determine serum profiles of soluble CD4 (sCD4), soluble CD8 (sCD8), and soluble CD25 (sCD25) in patients, and the relationship between T cell activation and pathogenesis after VZV infection. Serum concentrations of selected immune activation antigens were measured in patients with primary (varicella) and reactivated varicella (zoster) infections of the varicella-zoster virus (VZV) by enzyme-linked immunosorbent assay (ELISA) and enzyme immunoassay (EIA). Patients with the zoster infection showed significant increases of T cell-derived sCD4 and sCD25, but not of sCD8, suggesting T helper cell activation in reactivated varicella infection. In contrast, significant increases of these 3 antigens (sCD4, sCD8, and sCD25) were found in varicella infection, which suggests activation of both helper and cytotoxic cells in primary VZV infections. Anti-VZV-specific IgM could be detected in 8 of 10 patients with varicella and in 4 of 18 patients with zoster. However, anti-VZV IgG titers were apparently unchanged in patients with varicella compared to control subjects (1090.63 ± 166.87), but significantly increased in patients with zoster (4585.30 ± 774.91, $p = 0.030$). Therefore, activation of T cells and induction of sCD4, sCD8, and sCD25 contribute to the pathogenesis of VZV infection. (N. Taipei J. Med. 2000; 2:251-256)

INTRODUCTION

Primary infection with the varicella-zoster virus (VZV), a human herpes virus, causes varicella, or chickenpox. Varicella is characterized by widely distributed, discrete cutaneous vesicles that contain infectious VZV, indicating that viremia is critical in the pathogenesis of primary VZV infection. After primary

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