Antigen-specific sulphidoleukotriene production and histamine release in pollinic patients

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We studied suplhidoleukotriene (SLT) production, by means of CAST-ELISA (Bühlmann) in 92 atopic (54 pollinic and 38 non-pollinic) patients, and in 9 control subject, after antigenic stimulation of peripheral blood leukocytes with 20 ng/ml and 2 ng/ml of Lolium perenne pollen extract, in thee presence of IL-3. Antigen-specific stimulation of leukocytes from pollinic patients studied during the pollen season led to a SLT production significantly higher ($p = 0.003$ at 2 ng allergen/ml) than in those studied out of the pollen season. Histamine release was also significantly higher in pollen season than out of the season ($p = 0.04$ at 20 ng allergen/ml and $p < 0.001$ at 2 ng allergen/ml). There was a significant positive correlation between SLT production and histamine release ($r = 0.67$ at 2 ng allergen/ml and $r = 0.57$ at 20 ng/ml, both $p < 0.001$), and between SLT production and skin test results ($r = 0.5$ at 2 ng allergen/ml and $r = 0.46$ at 20 ng allergen/ml, both $p < 0.01$). We found that SLT production was lower, although not significantly, in patients older than 40 years, and histamine release was significantly ($p = 0.02$) higher in women than in men at 2 ng allergen/ml. We conclude that SLT production in pollinic patients is higher when antigenic pressure is increased in the environment, and that SLT quantification by CAST-ELISA might be useful for evaluation of this sensitization, with analogous results to the histamine release test.

Tumor Necrosis Factor α Gene Expression and the Response to Interferon in Chronic Hepatitis C

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Tumor necrosis factor a (TNF-α) is a cytokine with pleiotropic properties that is induced in a variety of pathological situations including viral infectios. In this work, we analyzed the expression of TNF-α gene in patients with chronic hepatitis C. Serum TNF-α levels were found to be elevated in all chronic hepatitis C patients including those cases presenting sustained biochemical remission of the disease after interferon therapy. Untreated patients with chronic hepatitis C showed increased TNF-α messenger RNA (mRNA) levels in the liver and mononuclear cells as compared with healthy controls. After completion of treatment with interferon, patients experiencing sustained complete response showed values of TNF-α mRNA, both in the liver and in peripheral mononuclear cells, within the normal range, significantly lower than patients who did not respond to interferon and that those with complete response who relapsed after interferon withdrawal. Pretreatment values of TNF-α mRNA were lower in long-term responders to interferon than in cases who failed to respond to the treatment. Values of TNF-α mRNA in the liver or in mononuclear cells were higher in specimens with positive hepatitis C virus (HCV) RNA than in those samples where the virus was undetectable. Neither the intensity of the liver damage nor the amount of HCV RNA in serum or in cells showed correlation with the levels of TNF-α transcripts in peripheral mononuclear cells but it was found that high TNF-a values were associated with genotype 1b. In conclusion, there is an enhanced expression of TNF-α in HCV infection. High levels of this cytokine may play a role in the resistance to interferon therapy.