Severity of asthma : the role of CD25+, CD30+, NF-kappaB, and apoptotic markers.

ABSTRACT

OBJECTIVES:We studied the role of the regulatory T cells CD4+CD25+ (Treg) and activated CD4+CD30+ cells in the pathogenesis of asthma and their association with apoptosis and NF-kappaB in patients with mild intermittent asthma (MA), severe persistent asthma (SA), and healthy volunteers (HV).METHODS:Peripheral blood lymphocytes (PBL) were extracted from asthmatic patients during exacerbations, and CD4+ cells were separated using Dynal beads. Immunostaining of whole PBL for NF-kappaB, Bax, and Bcl-2, and immunostaining of CD4+ cells for CD25+ and CD30+ cells were performed using immunocytochemistry. RESULTS:Treg cells were expressed at higher levels in MA than in HV and SA ($P < .05$), while CD30+ T cells were expressed at higher levels in both SA and MA than in HV ($P < .05$), although there was no remarkable difference between SA and MA ($P>.05$). Levels of NF-kappaB, Bcl-2, and Bcl-2/Bax increased, whereas those of Bax decreased, progressively, from MA to SA ($P < .05$). NF-kappaB levels correlated directly with the Bcl-2/Bax ratio and with CD4+CD30+ cells in SA and MA, whereas CD4+CD30+ cells correlated inversely with the Bcl-2/Bax ratio.CONCLUSIONS:Unregulated Treg cells probably return inflammatory responses to normal values during exacerbations in MA; however, expression of Treg cells was extensively diminished in SA, leading to probable loss of suppressive control over underlying immune reactions. CD4+CD30+ cells were associated with the pathogenesis of asthma but not with severity. NF-kappaB seems to be the central inflammatory factor in SA, with a remarkable loss of PBL apoptosis, diminished Treg levels, and high CD30+ cell levels that probably induce NF-kappaB, which in turn blocks the proapoptotic potential of CD30 induction itself.

Keyword: Asthma; Apoptosis; Memory cells; CD45RO; TH1; TH2; IL-4; IFN-ã.