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Induction of cell surface, but not soluble HLA-G expression in pandemic H1N1 2009 and seasonal H1N1 influenza virus infected patients

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A novel H1N1 virus of swine origin (H1N1v) currently caused a pandemic. The immunotolerant HLA-G was speculated play critical roles in viral infection; however, the clinical relevance of HLA-G in H1N1 infection remains elusive. In this study, HLA-G expression in peripheral T lymphocytes, monocytes and CD4+CD25+FoxP3+ regulatory T (Treg) cells (in 50 H1N1v, 41 seasonal H1N1 infected patients and 27 control subjects) were analyzed with flow cytometry. Plasma soluble HLA-G (sHLA-G) levels (in 28 H1N1v, 29 seasonal H1N1 infected patients and 85 control subjects) were determined with enzyme-linked immunosorbent assay. Data showed that HLA-G-positive T lymphocytes and monocytes among patients with H1N1v infected patients (26.19%±18.30% and 65.29%±27.22%, respectively) and H1N1 infected patients (25.58%±21.18% and 58.57%±28.33%, respectively) was dramatically increased compared with that among healthy controls (0.96%±0.75% and 1.79%±0.78%, respectively; all $p<0.001$). Treg was increased only among H1N1v infected patients (5.54%±3.34%) compared to normal controls (3.85%±2.62%, $p=0.041$), but not for the seasonal H1N1 infected patients (4.28%±2.16%). However, no significant difference was observed for the sHLA-G levels between the groups (median: 34.32 U/mL, 33.72 U/mL and 35.72 U/mL for H1N1v, H1N1 infected patients and controls, respectively). Together, only cell surface HLA-G expression was induced in H1N1v and in H1N1 infected patients. Moreover, an increased Treg was observed only in H1N1v infected patients. Our findings suggested that induction of HLA-G expression may help the virus escaping from host immune responses and contribute to the pathogenesis of infection.

Keywords: H1N1, Immunology, HLA-G, Treg