Tapan N Dhole, J Antivir Antiretrovir 2014, 6:2
http://dx.doi.org/10.4172/1948-5964.S1.022

OMICS Group Conferences
Accelerating Scientific Discovery

4th World Congress on
Virology

October 06-08, 2014 Hilton San Antonio Airport, TX, USA

Severity of pandemic influenza response mediated through cytokine stroms

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H1N1 subtype of pandemic 2009 (pH1N1) influenza virus lineages has raised severe concerns about its pandemic potentiality. The pathogenesis of the disease and its progression as post-infectious sequelae is not well understood. As moderate inflammatory response protects against the ill effects and hyper-inflammatory response promotes the pathogenesis in disease progression. On infection in respiratory epithelium, the recognition of influenza virus RNA triggers activation of the NLRP3 inflammasome, which in turn modulates the severity of disease through cytokine storm (IL-1β and IL-18). A total of, 216 pandemic samples were screened by RT-PCR and classified pH1N1 (63) and Influenza A virus (47) infected patient. Antibody titer was analyzed by hemagglutination inhibition assay and cytokine/chemokine response by Cytometric bead array assay, 106 samples were remain negative. One hundred samples were also taken as healthy control. Lower antibody titer was found in patient infected with pH1N1/Influenza A virus and expression of cytokines (IL-6, IL-8, and IL-10) and chemokine MCP-1 was higher in patient infected with pH1N1 compare to healthy/disease control, however there was no significant difference observed in the expression of pro-inflammatory cytokines TNF-α and antiviral cytokine IFN-β in pH1N1 infected patients. The cytokine storm can be well treated either by doxycycline or through antivirals to curtail the disease process.

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