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NOD_2 expression, DNA damage and oxido-inflammatory status in Egyptian patients with atopic bronchial asthma: Exploring their nexus to disease severity

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Abstract:

Background: Allergic asthma is a chronically relapsing inflammatory airway disease with a complex pathophysiology. **Aim:** This study was undertaken to investigate the potential contribution of NOD2 signaling, proinflammatory cytokines, chitotriosidase (CHIT1) activity, oxidative stress and DNA damage to atopic asthma pathogenesis, as well as to explore their possible role as surrogate noninvasive biomarkers for monitoring asthma severity. **Methods:** Sixty patients with atopic bronchial asthma who were divided according to asthma severity into 40 mild-moderate, 20 severe atopic asthmatics, in addition to thirty age-matched healthy controls were enrolled in this study. NOD_2 expression in PBMCs was assessed by quantitative real-time RT-PCR. DNA damage indices were assessed by alkaline comet assay. Serum IgE, IL-17, IL-8 and 3-Nitrotyrosine levels were estimated by ELISA. Serum CHIT1and GST activities, as well as MDA levels, were measured. **Results:** NOD_2 mRNA relative expression levels were significantly decreased in atopic asthmatic cases relative to controls with lower values among severe atopic asthmatics. On the other hand, IL-17 and IL-8 serum levels, CHIT1 activity, DNA damage indices and oxidative stress markers were significantly increased in atopic asthmatic cases relative to controls with higher values among severe atopic asthmatics. The change in these parameters correlated significantly with the degree of decline in lung function. **In conclusion,** the interplay between NOD_2 signaling, proinflammatory cytokines, CHIT1 activity, heightened oxidative stress and DNA damage orchestrates allergic airway inflammation and thus contributing to the pathogenesis of atopic asthma. These parameters qualified for measurement as part of new noninvasive biomarker panels for monitoring asthma severity.