SMGroup

SM Journal of Pediatrics

Editorial

Milestones along the Way towards the Inflammatory Triad of obesity, Asthma and Liver Diseases

Mustafa Metin Donma[1]*

[1]*Department of Pediatrics, Faculty of Medicine, Namik Kemal University, Turkey*

Article Information

Received date: Jul 22, 2016
Accepted date: Jul 25, 2016
Published date: Aug 05, 2016

***Corresponding author**

Mustafa Metin Donma, Department of Pediatrics, Faculty of Medicine, Namik Kemal University, Turkey,
Tel: 00-90-5323548630;
Fax: 90-282-2509928;
Email: mdonma@gmail.com

Editorial

Inflammation is recognized as a pathophysiologic mechanism in obesity and associated chronic diseases such as asthma, diabetes, cardiovascular diseases. There are potential mechanisms for obesity-asthma link. A significant association between hepatitis B and asthma is detected. A synergistic interaction exists between obesity and hepatitis virus infection. Hepatotropic viruses and obesity, which is prevalent among patients with hepatitis affect lung function and respiratory system [1-5]. Among the T cell subsets with suppressive function, regulatory T cells (Tregs), defined by the expression of $CD4^+$, $CD25^+$, and the transcription factor $FoxP3^+$, control immune responses. Reduced Tregs result in liver injury by initiating inflammatory processes and are detected in obese, asthmatic and asthmatic obese children [6,7]. Low Birth Weight (LBW) is a predisposing factor for metabolic abnormalities such as atherosclerosis, hypertension, diabetes, asthma, obesity. They have all been reported to be more common among those who were small at birth. The associations of LBW with liver diseases such as hepatitis, cirrhosis, hepatoblastoma were also noted [8,9]. The nuclear factor NF-$\kappa\beta$ contributes to viral persistence, leading to the development of hepatocellular carcinoma by inhibition of apoptosis mediated by cytotoxic T cells. Obesity-related inflammatory problems may be solved by the inhibition of NF-κB signaling pathway. NF-κB inhibition may also prevent airway inflammation in asthma. Obesity impairs apoptotic cell clearance in asthma. NF-κβ activation is induced by hepatitis viruses [10,11]. Peroxisome proliferator activated receptor gamma (PPARγ) is an important signaling pathway that occurs at the crossroads of depression and obesity and involved in anti-inflammatory reactions as a modulator of inflammation. PPARγ agonists prevent both airway inflammation and remodeling in asthma. This beneficial effect is mediated by inhibition of Toll-like receptor-4 and NF-κB pathways. Also, liver inflammation and impairment may be reduced by simply activating PPARγ and inhibiting NF-κB [12-14]. Hyperinsulinemia may impair the ability of Tregs to suppress inflammatory responses and may contribute to the development of obesity-associated inflammation. Obese and asthmatic obese patients with Insulin Resistance (IR) display significantly decreased Tregs. Asthma is associated with IR and a systemic inflammatory response possibly mediated by adipokines. IR is an important prognostic factor for hepatitis C. Obesity enhances hepatitis C-induced IR. Hepatitis C associated with obesity promotes the development of hepatocellular carcinoma [7,15-17]. Common mechanisms and synergy among obesity, asthma and liver diseases, gaining importance during childhood in recent years and characterized by inflammation, are interesting and quite complicated. Many bidirectional interactions might contribute to the progression to obese state and then to steatohepatitis and eventually fibrosis, which may also contribute to respiratory dysfunction. T cells of the immune system, apoptosis, signaling molecules, LBW, IR all appear to participate in this progression. However, there is uncertainty concerning the relative contribution of each into these processes. It needs further investigation to understand how each factor affects the process and how to prevent the complications as well as the interactions.

References

1. Donma MM. Potential mechanisms for obesity-asthma link. J J Obesity. 2015; 1: 003.
2. Goh LY, Card T, Fogarty AW, McKeever TM. The association of exposure to hepatitis B and C viruses with lung function and respiratory disease: a population based study from NHANES III database. Respir Med. 108:1733-1740.
3. Hassan MM, Abdel-Wahab R, Kaseb A, Shalaby A, et al. Obesity early in adulthood increases risk but does not affect outcomes of hepatocellular carcinoma. Gastroenterology. 2015; 149:119-129.
4. Menta PL, Correia MI, Vidigal PV, Silva LD, Teixeira R. Nutrition status of patients with chronic hepatitis B or C. Nutr Clin Pract. 2015; 30: 290-296.
5. Wang CC, Tseng TC, Kao JH. Hepatitis B virus infection and metabolic syndrome: fact or fiction? JGastroenterol Hepatol. 2015; 30: 14-20.

6. Choi YS, Lee J, Lee HW, Chang DY, Sung PS, et al. Liver injury in acute hepatitis A is associated with decreased frequency of regulatory T cells caused by Fas-mediated apoptosis. Gut. 2015; 64: 1303-1313.

7. Donma M, Karasu E, Ozdilek B, Turgut B, Topcu B, et al. CD4(+), CD25(+), FOXP3(+) T regulatory cell levels in obese, asthmatic, asthmatic obese, and healthy children. Inflammation. 2015; 38: 1473-1478.

8. Donma MM, Donma O. Low birth weight: a possible risk factor also for liver diseases in adult life? Med Hypotheses. 2003; 61: 435-438.

9. Akyüz A, Alpsoy S, Akkoyun DC, Nalbantoglu B, Topcu B, et al. Low birth weight is a risk for early atherosclerosis in childhood. J Am Coll Cardiol. 2013; 628: C38-C39.

10. Paulmann D, Bortmann S, Grimm F, Berk I, Kraemer L, et al. NF-κB activation induced by hepatitis A virus and Newcastle disease virus occurs by different pathways depending on the structural pattern of viral nucleic acids. Arch Virol. 2014; 159:1723-1733.

11. Miyake T, Miyake T, Hamada H, Morishita R. Prevention of airway inflammation by simultaneous inhibition of NF-κβ and STST6 using chimeric decoy oligonucleo chictides in a mouse model of asthma. Mol Ther. 2016; 24: S231.

12. Donma MM, Donma O. Promising link between selenium and peroxisome proliferator activated receptor gamma in the treatment protocols of obesity as well as depression. Med Hypotheses. 2016; 89: 79-83.

13. Lee HY, Rhee CK, Kang JY, Park CK, Lee SY, et al. Effect of intranasal rosiglitazone on airway inflammation and remodeling in a murine model of chronic asthma. Korean J Int Med. 2016; 31: 89-97.

14. Chen K, Li JJ, Wang JS, Xia YJ, Dai WQ, et al. 15-deoxy-gamma 12, 14-prostaglandin J2 reduces liver impairment in a model of ConA-induced acute hepatic inflammation by activation of PPAR gamma and reduction in NF-kappa B activity. PPAR Res. 2014; 215631.

15. Han JM, Patterson SJ, Speck M, Ehses JA, Levings MK. Insulin inhibits IL-10-mediated regulatory T cell function: Implications for obesity. J Immunol. 2014; 192: 623-629.

16. Morishita R, Franco MC, Suano-Souza FI, Solé D, Puccini RF, et al. Body mass index, adipokines and insulin resistance in asthmatic children and adolescents. J Asthma. 2016; 53: 478-484.

17. Goossens N, Negro F. the impact of obesity and metabolic syndrome on chronic hepatitis C. Clin Liver Dis. 2014; 18: 147-156.