LETTER TO THE EDITOR
Iran J Allergy Asthma Immunol
December 2012; 11(4): 349-350

Immune-Based Pathogenesis of Sulfur Mustard; Much Still Need to Be Done!

Amin Saburi [1], Majid Shohrati, [1] and Reza Karbasi-Afshar [2]

[1] Chemical Injuries Research Center, Baqiyatallah University of Medical Sciences, Tehran, Iran
[2] Department of Cardiovascular Diseases, Faculty of Medicine, Baqiyatallah University of Medical Sciences, Tehran, Iran

Received: 29 July 2012; Accepted: 26 August 2012

To the Editor:

We read with interest a published paper entitled "Smad Molecules Expression Pattern in Human Bronchial Airway Induced by Sulfur Mustard; Iran J Allergy Asthma Immunol; 2011 Sep; 10(3):147-54."[1] Adelipour *et al.* skillfully explained pattern of Smads molecule expression in airway wall biopsies of patients affected by sulfur mustard (SM). They finally concluded that in SM-induced subjects, Smad 3 and 4 may be involved in airway remodeling processes by activation of TGF-β. This is a useful paper on molecular and proteomics studies but it seems that there are some challengeable points to consider.

Some diseases such as alpha 1 antitrypsin deficiency may be a predisposing factor (even though a weak) for pulmonary lesions in individuals affected by the mustard gas.[2] Also, some studies have previously introduced various disorders which can be similar to the SM injury in proteomic and molecular pathways. These confounding disorders will also be the cause of some other diseases such as bronchiectasis or chronic bronchitis.[3,4] It is not clear whether the effects of mustard gas in these patients are higher than others or not. Therefore, if the authors had excluded other confounding immunological and biochemical disturbances, they would have had more reliable findings about pure SM lung injury.

In addition, bronchoscopic study is a potentially dangerous procedure. Performing bronchoscopic study on the healthy subjects might be ethically debatable on the other hand, to solve the SM dilemma, we should focus on another pathway to resolve its pathogenesis. For this purpose, clinical feature of these patients is a proper guideline for this issue. One of the most important chronic complications of SM injuries is pulmonary complication which is known as Bronchiolitis Obliterans (BO).[5] There are many mechanisms which have been proposed to explain pathogenesis of long-term SM injury and finally, it seems that defect in the cell repair mechanisms is the main problem.[4] Oxidant-Antioxidant imbalance, especially Glutathione (GSH) plays a key role in this pathway.[6] Also, Previous studies demonstrated uncontrolled apoptosis (via both intrinsic and extrinsic pathways) and defective efferocytosis along with ineffective cell repair (approved by raise in tissue growth factor beta (TGF-β)) as the main cellular mechanism which were justified by imbalance in oxidative-antioxidative system.[1,4,7] There is evidence for the higher risk of neoplastic disorders among SM injured patients, immunogenomic pathway, regarding to the maintained higher level of TGF-β, enhances the hypothesis that a genetic mutation has probably occurred in one of components of repair pathways.[8] It is necessary to focus genomic studies on mutations which can explain above mentioned immunological, pathological and biological pathway.

Corresponding Author: Amin Saburi, MD;
Chemical Injuries Research Center, Baqiyatallah University of Medical Sciences, Tehran, Iran. Postal code: 14359151371. Tel/Fax: (+98 21) 8860 0067, E-mail: aminsaburi@yahoo.com

REFERENCES

1. Adelipour M, Imani Fooladi AA, Yazdani S, Vahedi E, Ghanei M, Nourani MR. Smad molecules expression

Published by Tehran University of Medical Sciences (http://ijaai.tums.ac.ir

pattern in human bronchial airway induced by sulfur mustard. Iran J Allergy Asthma Immunol 2011; 10(3):147-54.

2. Shohrati M, Shamspour N, Babaei F, Harandi AA, Mohsenifar A, Aslani J, et al. Evaluation of activity and phenotype of alpha1-antitrypsin in a civil population with respiratory complications following exposure to sulfur mustard 20 years ago. Biomarkers 2010; 15(1):47-51.
3. Ghanei M, Harandi AA. Long term consequences from exposure to sulfur mustard: a review. Inhal Toxicol 2007; 19(5):451-6.
4. Ghanei M, Harandi AA. Molecular and cellular mechanism of lung injuries due to exposure to sulfur mustard: a review. Inhal Toxicol 2011; 23(7):363-71.
5. Ghanei M, Mokhtari M, Mohammad MM, Aslani J. Bronchiolitis obliterans following exposure to sulfur mustard: chest high resolution computed tomography. Eur J Radiol 2004; 52(2):164-9.
6. Shohrati M, Ghanei M, Shamspour N, Babaei F, Abadi MN, Jafari M, et al. Glutathione and malondialdehyde levels in late pulmonary complications of sulfur mustard intoxication. Lung 2010; 188(1):77-83.
7. Mehrani H, Ghanei M, Aslani J, Tabatabaei Z. Plasma proteomic profile of sulfur mustard exposed lung diseases patients using 2-dimensional gel electrophoresis. Clin Proteomics 2011; 8(1):2.
8. Hassan ZM, Ebtekar M, Ghanei M, Taghikhani M, Noori Daloii MR, Ghazanfari T. Immunobiological consequences of sulfur mustard contamination. Iran J Allergy Asthma Immunol 2006; 5(3):101-8.