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411. Inflammation and remodelling in lung disease

PA3915

Prooxidant-antioxidant balance in mustard airway disease with different severity

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Introduction: Sulfur mustard (SM) is a strong alkylating agent that primarily targets the skin, eye, and lung. Mustard airway is a major late complication of SM exposure. **Aims and objectives:** Current study evaluated long-term effect of SM on *oxidizing and reducing agents* values using new method prooxidant-antioxidant balance (PAB) assay in human plasma of SM-injured patients. **Methods:** sera of 35 SM-exposed patients and 19 healthy controls were recruited. Both groups had nonsmoker and nonalcoholic people with no diseases. The PAB was measured by a new method, recently developed by hamidi, by using 3,3′,5,5′-Tetramethylbenzidine (TMB) and two different kinds of reactions; one enzymatic reaction and a chemical reaction that noted prooxidant– antioxidant balance (PAB) assay. **Results:** Mean PAB values in control group were 48.74±21.07 HK (Hamidi and Koliakos), PAB levels were 101.45±32.68 HK in SM-exposed patients with normal pulmonary function test and 120.23±31.55 HK in severe obstruction pulmonary disease. . **Conclusions:** the level of oxidation is not related to the severity of disease defined by spirometry findings and a significant negative correlation was established between the PAB value and FEV_1. The increased PAB value in chemical casualties showed that these patients are exposed to oxidative stress.Likely, at first cytotoxic levels of SM can directly induce oxidative stress (OS), but many years after exposure the levels of OS were elevated through indirectly effects of inflammatory process.

PA3925

Serum metabolomic analysis of mustard airway diseases by nuclear magnetic resonance spectrometry: A pilot study

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Introduction: Nuclear magnetic resonance (NMR) spectroscopy is an ideal platform for the metabolic analysis of biofluids. Sulfur mustard (SM) is a alkylating chemical warfare agent that cause lung injury and despite years of research there is no common consensus about the pathophysiological basis of mustard airway disease. **Aims and objectives:** we used ^{1}H NMR spectroscopy to explore the metabolic profile of sera from mustard airway disease patients. **Methods:** A total of twenty nine serum samples obtained from SM-injured patients (n=17) and healthy controls (n=12) were investigated. Random forest (RF) analysis was capable of distinguishing SM-exposed patients from controls. **Results:** A total of 18 metabolites differed significantly in intensity between the two groups. Serum samples from SM-injured patients were characterized by increased concentrations of amino acid metabolism (L-glutamine, L-glutamate, L-asparagine, L-lysine, L-glycine, L-proline, and 4-hydroxyproline), lipid metabolism, citrate, ketone bodies, organic acid (lactate and acetate), urea pathway (citrulline), and creatine, relative to control subjects. **Discussion:** Our study reveals the metabolic profile of sera from SM-injured patients and indicates that NMR-based methods can distinguish these patients from healthy controls. .Also, serum Metabolomic profile of COPD patients differ with mustard airway disease by increased levels of methylhistidine (a muscle protein) and reduced levels of branched-chain amino acids as well as 4-hydroxyproline and proline. It suggests that Metabolomic-based diagnostics not only can distinguish mustard airway disease with healthy controls, but also can diagnose from COPD.