



COPD Researchers are Still Looking for the “Holy Graal”: A Simple and Inexpensive Biomarker Helpful in Grading and Phenotyping COPD Patients

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Editorial

In recent years, red blood cell distribution width (RDW) a simple parameter of the standard full blood count and a measure of heterogeneity in the size of circulating erythrocytes, attracted the attention of researchers. It is provided by automated hematology analyzers and reflects the range of the red cell size. It is calculated by dividing the standard deviation of erythrocyte volume by the mean corpuscular volume (MCV) and multiplied by 100 to convert to a percentage. Although RDW has been traditionally used in the investigation of the etiology of anemia there is an increasing evidence linking elevated RDW with poor outcome in general population, in patients with coronary artery disease, metabolic syndrome, heart failure and rheumatoid arthritis. In a few words, RDW is an inflammatory marker with a significant predictive value of mortality in diseased and healthy populations [1-3]. Paradoxically, in English language literature there are no data regarding the role of RDW in COPD. Our group is performing a retrospective study designed to evaluate the role of RDW in COPD patients. We are analysing the medical records of patients discharged from our clinic from 2010-2016 with the main diagnosis of COPD exacerbation. Preliminary data are intriguing: RDW is higher in frequent exacerbators and in patients with higher eosinophils (>3%) in blood samples, and correlates significantly with fibrinogen, c-reactive protein and GOLD stages. Another interesting topic is Obstructive sleep apnoea syndrome (OSAS), and in particular related comorbidities. Our study group is deepening the theme of cognitive impairment. The most frequent affect domains seem to be attention, memory, and, above all, executive functions, such as problem solving, planning, goal-oriented behaviour, and mental flexibility. In some patients, the cognitive deficits associated with OSAS are irreversible leading to a cognitive pattern named “pseudodementia” [4]. Sleep fragmentation, hypoxia, and the ensuing daytime drowsiness are among the most important causes related to the decline of neurocognitive functions in OSAS patients. Some authors demonstrated, by using magnetic resonance imaging (MRI) and 18 fluoro-2 deoxy-D glucose positron emission tomography (18 FDG-PET), an association between OSAS and structural changes at cerebral level, especially in the right hemisphere, with regards to grey matter density and metabolism. In some phenotypes there is a more evident frontal damages, in others a widespread damage involving hippocampus, basal ganglia and cerebellum [4,5]. We are performing a randomized controlled trial design to elucidate the role of continuous airways pressure (CPAP) in the reversibility of the above mention clinical pattern. Preliminary data seem to demonstrate that after 6 months of CPAP therapy the vast majority of subjects have a partial regression of symptoms.

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