**CLINICAL UNDERSTANDING OF INFLAMMATION, INDUCING THROMBOGENESIS, RESPIRATORY IMPAIRMENT, MYOCARDIAL CELL APOPTOSIS AND REMODELLING, ANAEMIA AND RENAL DYSFUNCTION IN PATIENTS WITH PULMONARY AND COINCIDENT CARDIAC DYSPNEA**

I. Angomachalelis, S. Tryfon, G. Kyriazis, **N. Angomachalelis**

Aristotle University of Thessaloniki, School of Medicine, Department of Clinical Pulmonology, Heart-Lung Section, ”G. Papanickolaou” General Hospital,

Thessaloniki, Macedonia, Greece

*Purpose*: The study aims to investigate understanding of clinical pathophysiology of primary inflammation in pulmonary and coincident cardiovascular dyspnea patients (Pts).

*Methods*: 111 Pts, 67 males and 40 females, mean age 66 years and 40, matched control, normal individuals underwent: 1.Biomarkers serum evaluation (CRP and pro-inflammatory cytokines,D-dimers, NT-ProBNP, Tr-I, EPO, MMP-2,PCT, 2. Physical examination, ECG, Chest X-ray and CT 3. Echocardiography 4. PFTs and ABGs 5. Blood tests.

*Results*: Resulted abnormal values of serum Biomarkers, echocardiographic and Doppler indices, PFTs, ABGs and Blood tests presented significant correlations as follows: CRP with A) D-dimers B) PCO2 C) NT-ProBNP D) EF E) LVID. Furthermore, A) D-dimers with A1) RVSP, correlated to LAD and LVID, A2) RVSP correlated with P (A-a)O2 and –FVC and –TLC, A3) PCO2. B) PCO2 with NT-ProBNP, FEV1 and Na, correlated with left ventricular E-Wave. C) NT-ProBNP with EF, PCO2 and C1) Tr-I, C2) ASH, C3) Hb, C4) Urea and Creatinine, C5) Pleural NT-ProBNP. However, C1) Tr-I correlated with left ventricular E/A ratio, MMP-2, EPO, - FVC and –TLC, -Na and Creatinine. C2) ASH with EF, Urea and Creatinine. C3) Hb with Pleural NT-ProBNP, TLC and PCT, correlated with MMP-2, Tr-I, EPO and PCO2. D) EF with FVC and Tr-I. E) LVID with LAD, RVSP, RVID, Creatinine and Urea.

*Conclusions*: 1. Reported abnormal results and significant correlations arise originally from an inflammatory process, expressed by CRP levels, introducing various pathways of clinical pathophysiology, correlated with D-dimers, PCO2, NT-ProBNP, EF and LVID. 2. All indices lead either to thrombogenesis and/or pulmonary, myocardial and renal dysfunction, followed by disarrangement of haemopoiesis and infective complications. 3. Inflammatory Thrombogenesis also seems developing on the grounds of cardiovascular apoptosis and remodelling. 4. Molecular and interstitial disarray phenomena, combined with pulmonary and renal impairment, further worsen haemopoietic disorders and late infection, all possibly creating a dynamic early-late inflammation-infection pathophysiology, carrying on Thrombogenesis, pulmonary and cardio-renal dyspnea.