European Respiratory Society
Annual Congress 2012

Abstract Number: 1621

Publication Number: P2632

Abstract Group: 10.2. Tuberculosis

Keyword 1: Tuberculosis - diagnosis **Keyword 2:** Sarcoidosis **Keyword 3:** Immunology

Title: FcγR and CR on blood monocytes in differentiation between sarcoidosis and tuberculosis

Dr. Anna 8957 Dubaniewicz aduban@gumed.edu.pl MD [1], Ms. Marta 8956 Szadurska m_szadurska@gumed.edu.pl MD [1], Ms. Monika 8958 Wybieralska mwybieralska@o2.pl MD [1], Mrs. Katarzyna 14430 Rogoza kr0610@o2.pl MD [1], Prof. Jan M. 14427 Slominski jmslomin@gumed.edu.pl MD [1], Mr. Piotr 14461 Kmiec piotrkmiec@gumed.edu.pl MD [4], Prof. Piotr 8960 Trzonkowski ptrzon@gumed.edu.pl MD [2] and Dr. Adam 8962 Sternau aster@gumed.edu.pl MD [3]. [1] Department of Pneumonology, Medical University of Gdansk, Poland, 80-211 ; [2] Department of Clinical Immunology and Transplantology, Medical University of Gdansk, Poland ; [3] Department of Thoracic Surgery, Medical University of Gdansk, Poland and [4] Department of Endocrinology and Internal Diseses, Medical University of Gdansk, Poland .

Body: Genetically different patients with sarcoidosis (SA) and tuberculosis (TB) induce dissimilar immune responses to the same mycobacterial heat shock proteins, which are implicated in forming of immune complexes (CIs). The complexemia in both diseases may result from a different function disorder of receptors for Fc of immunoglobulin G (FcγR) and complement receptors (CR) on monocytes in the phagocytosis and clearance of CIs with following persistent antigenemia and granuloma formation. Therefore, we analyzed the occurrence of FcγRI, FcγRII, FcγRIII and CR1, CR3, CR4 on blood CD14+ monocytes in 24 SA patients, 20 TB patients and 20 healthy volunteers using flow cytometry. Our results revealed significantly increased monocytes' presence with FcγRI-III and decreased with CR1 and CR4 in SA than controls. Analysis of monocytes' phenotypes revealed increased FcγRIII+CR1- and decreased FcγRII-CR1, FcγRII-CR3+, FcγRII-CR4+ occurrence in SA vs controls. In TB, higher presence of monocytes with particular FcγRI+, FcγRII+, CR1+ and CR3+ than in controls was detected. In SA vs TB, the occurrence of monocytes with FcγRIII+ receptor was significantly higher and with CR1+ was less frequent. The monocytes' phenotype FcγRIII+CR1- was increased in SA vs TB. In summary, there are increased frequencies of FcγRI+ and FcγRII+ monocytes in both SA and TB but in contrast to TB, sarcoid monocytes had increased FcγRIII occurrence with CR1 and CR4 deficiency. In SA, increased FcγR presence but CR deficiency on surface of blood monocytes may explain persistent antigenemia and complexemia in our patients with SA. This study may be useful for differentiation of both diseases.