BioNanoSci. (2017) 7:636–639
DOI 10.1007/s12668-017-0441-z

Structural Alterations of Monocytes in Systemic Lupus Erythematosus

I. A. Andrianova[1] · **A. A. Ponomareva**[1,2] · **R. I. Litvinov**[1,3]

Published online: 12 August 2017

Abstract Systemic lupus erythematosus (SLE) is an autoimmune disease in which the immune system mistakenly attacks multiple organs and tissues of the body. To elucidate the involvement of blood cells in the pathogenesis of SLE, we used transmission electron microscopy to study ultrastructure of monocytes isolated from the blood of SLE patients. We found that in the SLE patients, a substantial fraction of monocytes had abnormal morphology that corresponded to the structural signs of either necrosis or apoptosis. The number of altered monocytes in the SLE patients was significantly higher than in healthy subjects and related directly with the level of anti-dsDNA autoantibodies in the blood. Our results suggest that monocytes are involved in the pathogenesis of SLE and undergo adverse necrotic and/or apoptotic changes, likely induced by autoantibodies.

Keywords Systemic lupus erythematosus · Monocyte · Transmission electron microscopy

1 Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by systemic inflammation, affecting various organs and systems, including the joints, kidney, skin and central nervous system. Inflammation in SLE is associated with production of autoantibodies, generation of circulating immune complexes and activation of the complement system [1]. Monocytes play a pathogenic role in this disease as a source of secreted immunomodulators that promote activation of the key immunoreactive T and B cells [2]. A high level of interferon I, which correlates directly with the disease activity [3], can promote maturation of monocytes into dendritic cells, whose primary function is to process and present antigens to T cells [4]. In addition, monocytes can play a role in promoting thrombotic complications of SLE. It is known that SLE patients are predisposed to venous or arterial thrombosis [5] and activated monocytes that express tissue factor on their surface can contribute to the (peo)thrombotic status [6]. Also, monocytes from the blood of SLE patients have been shown to expose high levels of the procoagulant phosphatidylserine on the cell surface that also may enhance the prothrombotic tendency in SLE [7].

To provide a structural basis for the monocyte alterations in SLE, we investigated morphology of monocytes isolated from the blood of SLE patients and healthy donors using transmission electron microscopy.

2 Material and Methods

The study was approved by the Ethical Committee of Kazan State Medical University. SLE patients were included in the study based on the diagnostic criteria of the American College of Rheumatology. Citrated blood was collected by venipuncture from six SLE patients and three healthy aspirin-free donors and centrifuged at 200*g* for 10 min at room temperature. Platelet-rich plasma (PRP) was removed and the pellet was resuspended in a Tyrode-EDTA buffer added in the volume

✉ R. I. Litvinov
litvinov@mail.med.upenn.edu

[1] Kazan Federal University, 18 Kremlyovskaya St., Kazan, Russian Federation 42008

[2] Kazan Institute of Biochemistry and Biophysics, 2/31 Lobachevsky St., Kazan, Russian Federation 420111

[3] University of Pennsylvania School of Medicine, 421 Curie Blvd., Philadelphia, PA 19104, USA