Blood platelet abnormalities and pharmacological modulation of platelet reactivity in patients with diabetes mellitus

Cezary Watala, Magdalena Boncler, Peter Gresner

Department of Haemostasis and Haemostatic Disorders, Medical University of Łódź, Żeromskiego 113, PL 90-549 Łódź, Poland

Correspondence: Cezary Watala, e-mail: cwatala@csk.umed.lodz.pl, cwatala@toya.net.pl; http://www.interhemostaza.pl

Abstract:
The overall picture of platelet abnormalities in diabetes mellitus (DM), including altered adhesion and aggregation, is hypersensitivity of diabetic platelets to agonists. “Primed” diabetic platelets respond more frequently even to subthreshold stimuli, sooner become exhausted, consumed and finally hyposensitive, thus contributing to accelerated thrombopoiesis and release of ‘fresh’ hyperreactive platelets. In diabetes disturbed carbohydrate and lipid metabolism may lead to physicochemical changes in cell membrane dynamics, and consequently result in altered exposure of surface membrane receptors. These phenomena, together with increased fibrinogen binding, prostanoid metabolism, phosphoinositide turnover and calcium mobilization often present in diabetic patients, contribute to enhanced risk of small vessel occlusions and accelerated development of atherothrombotic disease of coronary, cerebral and other vessels in diabetes. The paper concentrates on the role of dynamic, physico-chemical properties of platelet membrane lipid bilayer, as a major determinant of platelet hypersensitivity in diabetic patients.

As a pharmacological response to platelet hypersensitivity in DM, making a major contribution to enhanced risk of thromboembolic macroangiopathy, and consequently enhanced morbidity and mortality in diabetic individuals, we have a variety of antiplatelet agents, and acetylsalicylic acid (ASA) is no doubt most commonly used world-wide. Every-day clinical practice shows that antiplatelet pharmacological approach may not always be efficient enough in people with diabetes. Although we are at the very beginning of complete understanding of so-called ‘aspirin-resistance’, several potential molecular mechanisms of this phenomenon in diabetes have been evidenced.

Key words:
diabetes mellitus, blood platelets, platelet hypersensitivity, platelet membrane lipid fluidity, acetylsalicylic acid, aspirin resistance