Effect of Glycoprotein IIb/IIIa Inhibition on Acute Ischemic Stroke Injuries

Neda Kamandi, Niloufar Akhgari, Sajad Sahab Negah

Department of Neuroscience, Medicine Faculty, Mashhad University of medical sciences, Mashhad, Iran

Published: 11 April, 2017

Abstract

Ischemic stroke accounts for about 87 percent of all cases. It occurs as a result of an obstruction within a vessel of the brain and sudden loss of blood circulation to the corresponding area resulting in the loss of brain function. It is caused by thrombotic or embolic occlusion of an artery and is more common than hemorrhagic stroke. We know that most of the injuries after an acute ischemic stroke are due to thrombosis formation and the following neuroinflammation (thromboinflammation). So by blocking this pathway we can ameliorate the injuries and the infarct size and improve the brain function after an acute stroke. Platelet von willebrand factor (VWF) is a glycoprotein involved in hemostasis. It is released from the platelet alpha granules and binds to glycoprotein IIb/IIIa complex which forms a bridge between the sub endothelial surface and the platelet and promotes thrombosis formation. On the basis of this information, we hypothesize that we can alleviate the injuries of acute ischemic stroke by blocking the glycoprotein IIb/IIIa complex via a specific antagonist antibody and so preventing thrombosis formation. It can be a potential therapeutic approach in the treatment of acute ischemic stroke.

Keywords: Acute ischemic stroke, Neuroinflammation, Thromboinflammation, platelet von willebrand factor, glycoprotein IIb/IIIa

*Corresponding Author: Sajjad Sahab Negah

Email: Sahabsajad@yahoo.com