Forum Minireview


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Stroke causes 9% of all deaths worldwide and is the second most common cause of death after ischemic heart disease (1). Therefore, there is an urgent need to develop novel medications that are safe and effective for intercepting stroke and treating affected individuals. Endothelial injury is one key event, which leads to cerebral damage. Recently, it has been suggested that cerebral damage by cerebral vascular injury is not caused by the failure of barrier function of the endothelium, but by failure of neurovascular unit, including endothelium, glia cells, neurons and the like (2, 3). Accordingly, investigating the mechanisms of failure of the neurovascular unit should provide a guide for the development of novel medicines for stroke.

At the 83rd Annual Meeting of The Japanese Pharmacological Society, Osaka, 16–18 March, 2010, we held a symposium to review issues associated with endothelial injury and the impact of endothelial injury on stroke. The mission was to find insights into the causes of stroke and identify appropriate strategies for drug development. The review articles appearing here were presented at this symposium.

The aim of this JPS Forum Minireview series are to present recent findings on the mechanism of novel situations related to endothelial injury and stroke to suggest strategies for drug development.

The present review series contain “Novel mechanism of the expression and amplification of cell surface–associated fibrinolytic activity demonstrated by real-time imaging analysis” by Y. Suzuki and T. Urano (4); “Intracranial bleeding associated with the treatment of ischemic stroke: thrombolytic treatment of ischemia-affected endothelial cells with tissue-type plasminogen activator” by Y. Suzuki, N. Nagai, and K. Umemura (5); and “Protective effects of antiplatelet agents against stroke” by M. Shimazawa and H. Hara (6). We hope that this JSP Forum Minireview contributes to the development of new therapeutic strategies for the treatment of endothelial injury and stroke.

References


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