

Abstract

Introduction: The recurrence of the cerebral ischemic stroke after a history of TIA or ischemic stroke is 3-4% per year. One way of reducing the risk of recurrence is using antiplatelet therapy. The aim of our study was to investigate the effect of aspirin. Even though, newer antiplatelet drugs were developed, their risk/benefit profile has not been proved to be better than aspirin. Reasons for using aspirin in secondary prevention are: the longest experience, clearly proven effect in many studies and low price. On the other hand, aspirin prevents only 25% of strokes, thus there is wide space for searching for causes of failed therapy and alternative therapeutic ways. Noncompliance of aspirin use and embolic events are usually indicated as the most common causes of an ineffective therapy. The goal of our study was to find the antiplatelet therapy effectivity in patients with history of stroke treated with aspirin in daily dose of 100mg. We assured 100% compliance among these patients and as much as possible minimalized a likelihood of embolic causes of strokes. What is more, we tried to find out whether an insufficient suppression of 11-dehydrothromboxane B₂ correlates with comorbidities, other used medication or laboratory parameters. Furthermore, whether by administrating an increased dose of aspirin, an intensified effect can be reached.

Methodology: From the group of 565 patients after stroke based on medical history, clinical and laboratory examinations, 106 patients were chosen. Preliminary inclusive and exclusive criteria were to eliminate patients with intermittently higher thromboxane level from known reasons. In involved patients their blood for assessing the concentration of 11-dehydrothromboxane in serum was taken two hours after using 100mg of aspirin, in some patients with insufficient suppression of thromboxane on 100mg the concentration was set on 300mg of aspirin.

Results: High serum level of 11- dehydroxythromboxane, which is considered a marker of an ineffective antiplatelet therapy of aspirin were present in 28% of examined patients. Higher incidence was in obese patients, patients with higher level of total and LDL cholesterol and high uric acid level. After increasing the dosage on 300mg daily in 9 patients with insufficient 11-dehydroxythromboxane suppression on the dose of 100mg, an adequate suppression was acquired in 4 of them (44%).

Conclusion: Our study proved high prevalence of ineffective suppression of 11-dehydroxythromboxane B₂ production in the selected group of patients with history of stroke using 100mg of aspirin daily. The authors discuss the causes and possible solutions to improve the therapy effectivity.

Key words: Aspirin, ischemic cerebral stroke, thromboxane