Abstract

Behcet’s disease (BD) is a chronic, inflammatory, and multisystemic condition although its pathogenesis is uncertain. Main component of St. John’s wort (Hypericum perforatum, HP) is hyperforin and induces antiinflammatory and antioxidant properties. We aimed to investigate effects of HP on oxidative stress, apoptosis, and cytosolic-free Ca\(^{2+}\) ([Ca\(^{2+}\)]\(_i\)) concentration in neutrophil of BD patients. Nine newly diagnosed active patients with BD and nine control subjects were included in the study. Disease activity was considered by clinical findings. Neutrophil samples were obtained from the patients and controls. The neutrophils from patients were divided into three subgroups and were incubated with HP, voltage-gated calcium channel (VGCC) blockers, (verapamil? diltiazem) and non-specific TRPM2 channel blocker (2-aminoethyl diphenylborinate, 2-APB), respectively. The neutrophils were stimulated by fMLP as a Ca\(^{2+}\)-concentration agonist and oxidative stress former. Caspase-3, caspase-9, apoptosis, lipid peroxidation, and [Ca\(^{2+}\)]\(_i\) values were high in the patient groups, although cell viability, glutathione (GSH), and glutathione peroxidase (GSH-Px) values were low in patient group. However, the [Ca\(^{2+}\)]\(_i\), caspase-3, and caspase-9 values decreased markedly in patient?HP group although GSH and GSH-Px values increased in the group. The [Ca\(^{2+}\)]\(_i\) concentration was also decreased in the patient group by V?D, 2-APB, and HP incubations. In conclusion, we observed the importance of neutrophil Ca\(^{2+}\) entry, apoptosis, and oxidative stress through gating VGCC and TRPM2 channels in the neutrophils in the pathogenesis and activation of the patients with BD. HP induced protective effects on oxidative stress by modulating Ca\(^{2+}\) influx in BD patients.