Depression is an illness characterized by sad mood and/or diminished ability to enjoy things accompanied by other symptoms such as changes in appetite, problems sleeping or excessive sleepiness, decreased energy level, slowed movements and poor concentration. At times, patients can have recurrent thoughts of death or that life is not worth living. Depression is generally diagnosed when these symptoms have been present every day for at least two weeks. It has been shown that depression in PD is a leading factor contributing to reduced quality of life (more so than the motor features). Most research suggests that depression in PD is caused by biological changes related to the underlying brain disease rather than solely a reaction to disability.

Depression occurs in 28% (PRODEST-PD study) to 60% (Parkinson's Disease Foundation) of patients with PD. There is little hard evidence of the efficacy and safety of antidepressant therapies in PD according to Cochrane Database of systematic reviews. This interventive, parallel, safety/efficacy, RCT study included 141 patients aged 36–90, with ICD 10/DSM IV criteria for PD and depression. Purpose of the study was to estimate depression, quality of life, and severity of PD symptoms after 3 months of antidepressant therapy.

Methods: we have randomly divide patients into C – control group (N=45) without antidepressants and two experimental groups in accordance with applied antidepressants: ES (sertraline 50–100 mg/24 h) group (N=51) and EM (mirtazapine 15–30 mg/24h) group (N=45). We have used HAMD for estimation of depression, QOL scale for quality of life, and UPDRS subscales I (behaviour and mood) and II (daily activities) for PD symptoms at 0 point (pretrial score) and 90 point (after 3 months) in all groups. Data were processed with SPSS for Windows (nonparametric tests: Kruskal-Wallis, Wilcoxon and Mann-Whitney test).

Results: there is no statistical significance in pretrial scores between groups, p=0.814 for HAMD, p=0.184 for QOL, and p=0.211 for UPDRS I and II. There is significant increase of HAMD score in control group after 3 months (p<0.000), median of increase where 2. There is statistical significance in decrease of HAMD scores after 3 months of antidepressant therapy in experimental groups (p<0.000), in favour of antidepressant with higher median: 8 for sertraline and 3 for mirtazapine. There is no statistical significance in QOL scores in control group after 3 months (p=0.593). There is statistical significant increase of QOL scores after 3 months of antidepressant therapy in sertraline (p<0.000) and mirtazapine group (p=0.01). Comparing the effects of antidepressants in QOL scores after 90 days of therapy there is statistical difference between sertraline and mirtazapine group in favour of sertraline (sertraline vs. mirtazapine p=0.015). There is significant difference in UPDRS I and II pretrial and after 3 months scores in experimental groups (sertraline p=0.000, mirtazapine p=0.001) but without significance among themselves (p=0.198). We had one case of severe side effects in mirtazapine group.

Conclusion: Tested antidepressants are efficient in reducing HAMD score, but sertraline improved HAMD, QOL, UPDRS I and II scores without side effects.

References