

Intracortical Inhibition and HPA Axis Function in MDD

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Background

Major depressive disorder is one of the most common psychiatric disorder worldwide and contributes to large medical, societal and economic burden. Previous studies suggested stress and HPA axis, the hormonal response system to stress of human beings, cause damage of several brain regions and therefore lead to depression. However, the exact mechanism remained unclear so far.

Aims & Objectives

The present study aimed to find out whether there are differences in the correlation of intercortical inhibition and HPA axis or the severities of depression, anxiety, pain and somatic symptoms between patients of major depressive disorder and healthy subjects.

Methods

Paired-pulse transcranial magnetic stimulation (ppTMS) was used to measure cortical inhibitory function including GABAA- and GABAB-receptor-related CI and cortical excitatory function including glutamate-receptor-related intracortical facilitation (ICF). Basal cortisol level was measured in represent of baseline stress while the result of dexamethasone suppression test represented stress reactivity. We recruited 22 healthy controls (HC) and 40 patients with MDD. All participants received evaluations for severities of depression, anxiety, pain and somatic symptoms. Using SPSS 22.0 software, Student t-test, Pearson χ^2 test and Pearson's correlation analysis was used to examine the results. A p value of <0.05 was considered to be significant.

Results

Significant correlation of basal ; p=0.509) while abnormally positive correlation between SICI and LICI was observed in patients of the MDD group (r=0.562; p<0.001) but not in subjects of the HC group (r=-0.014; p=0.954). However, there was neither significant difference between the results of dexamethasone suppression test of HC group and MDD group (p=0.628) nor significant correlation between DST result and SICI in HC group (r=-0.87, p=0.715) or MDD group (r=-.050, p=0.748). Furthermore, reduced SICI have a significant correlation with the severity of anxiety in both subjects of the HC group (r=0.499; p=0.025) and patients of the MDD group (r=0.382; p=0.010) while it also significantly correlates with the severity of somatic symptoms among the MDD group (r=0.302; p=0.047) but not the HC group (r=0.254; p=0.280). And reduced LICI correlates with the severity of depressive symptom in MDD patients (r=0.328; p=0.030) while such correlation wasn't seen in the HC group (r=-0.187; p=0.431).

Discussion & Conclusion

Loss of normal correlation between basal cortisol level and SICI and abnormal correlation between SICI and LICI were noticed in MDD patients. These findings suggest that stress may lead to loss of intracortical inhibitory function and end up with MDD. With the non-significant difference of the result of DST between the HC group and the MDD group and the non-significant correlation between the result of DST and SICI in both groups, the aforementioned findings suggest a failure of PFC related SICI reactivity to stress in MDD. Furthermore, changes of LICI may be a compensatory alternative coping for loss of SICI in MDD under stress and weaker LICI compensation could result in higher depressive symptoms.