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Anxious Depression is Followed by Pronounced Biochemical Disturbances

Uzbekov M[1]*, Maximova N[1], Shikhov S[1] and Syrejshchikova T[2]

[1]*Department of Affective Disorders, Moscow Research Institute of Psychiatry, Moscow, Russia*

[2]*Senior Researcher, Lebedev Physical Institute, Moscow, Russia*

***Corresponding author:** Uzbekov MG, Moscow Research Institute of Psychiatry, Head of the Laboratory of Brain Pathology, Moscow, Russia

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ABSTRACT

Keywords: Anxious Depression; Monoamine Oxidase; Semi carbazide-Sensitive Amine Oxidase; Middle-Mass Endotoxic Molecules

Introduction

Data from the World Health Organization indicate that depression and anxiety are the most common concomitant disorders in the primary healthcare system [1]. These two states significantly overlap in terms of clinical symptoms and various pathophysiological mechanisms of depression and anxiety can be detected in 42-100% of patients with depression [2]. Many aspects of pathogenetic and pathophysiological mechanisms of depressions need further investigations. It is especially important regarding biochemical investigations. The aim of the study was to investigate the state of aminergic metabolism and some parameters reflecting the disturbances of homeostasis in patients with anxious depression.

Material and Methods

There were investigated 21 patients with anxious depression. The patient's state according to ICD-10 criteria [according to International Classifications of Diseases, tenth edition, clinical modification (ICD-10-CM)] [1] was defined as a depressive episode as an independent disease (F32.1) and together with recurrent depressive disease (F33.1). The presence of anxiety together with depression was the main indication for the inclusion into the investigation. The clinical severity of the illness was assessed using the Hamilton Rating Scale for depression (HAM-D) (21 items) and the Hamilton Rating Scale for anxiety (HAM-A) [3].

Control group consists of 15 healthy volunteers. Investigation was performed in accordance with the permission of the local ethical committee of Moscow Research Institute of Psychiatry (N 16, 13.03.2017). Biochemical parameters were estimated at the admission. The activities of platelet monoamine oxidase (MAO) and serum semi carbazide-sensitive amine oxidase (SSAO) were estimated by methods of [4] and [5], respectively. Concentration of middle-mass endotoxic molecules (MMEM) in blood plasma was estimated by method of [6]. The significance of the differences was assessed using the Wilcoxon test Data for patients with depression were presented as the mean ± the error of the mean. Statistical significance was implied by $p<0.05$.

Results and Discussion

Data from the Hamilton scale (HAM-D) showed that at admission the total score was 21.83, which is consistent with severe depressive disorder. HAM-A data gave a total score of 18.4 which is also consistent with severe anxiety. At admission, all patients were

characterized by the significant increase in platelet MAO activity (by 95%), decrease of serum SSAO activity (by 43%) and increase of MMEM concentration (by 86%) in comparison with control values (Table 1). This implies that anxious depression is characterized by the strongly pronounced disturbances of aminergic metabolism and homeostasis.

Table 1: Activity and level of investigated parameters in patients with anxious depression.

	MAO (nmol benzaldehyde/mg protein in hour)	SSAO (nmol benzaldehyde/ml plasma in hour)	MMEM (g/L serum)
Control	8.68 ± 1.63	10.84 ± 0.65	0.45 ± 0.04
At admission	16.91 ± 3.50*	6.22 ± 0.82**	0.84 ± 0.08**

*p<0.05 **p<0.001 – in comparison with controls; MAO - monoamine oxidase; SSAO - semi carbazide-sensitive amine oxidase; MMEM - middle-mass endotoxic molecules

The disturbance of MAO activity in anxiously depressed patients may be indicative of damage of membrane structures (this enzyme is an integral component of the external mitochondrial membrane), resulting in the appearance in the blood of different toxic end products (aldehyde and ammonia). Changes in MAO activity can disturb the equilibrium between serotonin and epinephrine that play important role in the pathophysiology of the condition [2]. Hydrogen peroxide formed in the reaction of deamination catalyzed by MAO represents the main source of free radicals in the brain [7]. Thus, the elevation of MAO activity in these patients can induce the activation of free radical processes and lipid peroxidation that leads to the activation of oxidative stress. From the other side activation of oxidative stress is followed by the disturbances of mitochondria and the damage of blood-brain barrier. This statement is supported by data that depression is followed by damage of blood-brain barrier structures [8]. Taken together, these results suggest that an increased MAO activity precipitates a cascade of negative biochemical events.

Serum SSAO is an enzyme that is involved in the oxidation both xenobiotics and endogenous amino-containing metabolites [9]. The precise physiological and pathophysiological role of this enzyme is not known. SSAO can convert some endogenous amines, such as methylamine and aminoacetone, into highly toxic compounds - formaldehyde, methylglyoxal and acrolein [9]. Therefore, it is possible that an increase in the blood concentrations of these substances following stressful life events, as a depression, can exert harmful influences. It is supposed that increase of toxic metabolite concentrations in depressed patients promotes the intensification of endogenous intoxication. In previous studies, we proposed that the degree of endogenous intoxication can serve as a parameter of intensity of the disturbances of homoeostasis [6, 10].

Endogenous intoxication (endotoxicosis) is a pathophysiological process that is characterized by the formation and accumulation in tissues and body fluids of different substances and metabolites (endotoxins), in excessive concentrations or in forms that are not characteristic for the normal metabolism [6,10]. Increased level of MMEM, activation of free radical processes and lipid peroxidation, reactions catalyzed by MAOs and SSAO and so on make a major contribution to the development of endotoxicosis [10]. In patients with anxious depression MMEM concentration was elevated more than twofold support this thesis. It is postulated that, during the development of the mental disorders, there is an activation of catabolic processes that increase the concentration of different MMEM components. The increase of the MMEM concentration in blood plasma is indicative of the aggravation of the degree of endogenous intoxication as well as patient's clinical status.

Conclusion

These findings point out that anxious depression is followed by the profound biochemical and metabolic disturbances. We suppose that investigated parameters can serve as biomarkers of the severity of the condition.

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