

CONSIDERATIONS OF OBSESSIVE–COMPULSIVE DISORDER PSYCHOPATHOLOGY

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Abstract:

The paper refers to the neurochemical theories explaining the etiopathogenesis of obsessive-compulsive disorder, and among them those that focus on the neurotransmission system. The existence of dysfunctions in serotonin, dopaminergic, noradrenergic systems, and most recently, cholecystokinin were reported by different authors, but without specifying whether these disorders are the cause or the effect of the installation of mental disorders. It refers to the common biological spectrum between the obsessive-compulsive disorder and other psychiatric disorders such as pathological gambling, paraphilias, dysmorphophobias, trichotillomania, hypochondria, somatization disorder, Gilles de la Tourette syndrome, autism, Asperger syndrome, kleptomania, bulimia, anorexia nervosa. It is stated that the hypothesis of the neurotransmitter system malfunction revealed some peculiarities in the therapeutic treatment of obsessive-compulsive disorder. So far no existing theory met the necessary evidence to explain the neurobiological psychiatric disorder. It can be assumed only the substantial role played by the serotonin neurotransmission in the genesis of the obsessive-compulsive disorder.

Key Words: obsessive-compulsive disorder, etiopathogenesis, neurochemical theories

THEME

The appearance of obsessive-compulsive disorders (OCD), in the context of current knowledge has as benchmark the psychosocial model which meets the personality structure and the environmental conditions of the occurrence of the disorder. On the other hand, the investigations and assessments made discover also the neurobiological conditions assumptions expressed both by genetic and also by the catecholamines imbalance at the level of the central nervous system. These assessments state that pathobiologic mechanisms are similar to other well-known disorders such as Gilles de la Tourette, pathological gambling, paraphilias, kleptomania or autism.

PURPOSE AND OBJECTIVES

This paper aims to elucidate some pathobiological mechanisms that would allow the management of this disorder.

Materials and methods

A bibliographic evaluation was performed with reference to the etiopathogenesis of the obsessive-compulsive disorder, focusing primarily on the pathobiological mechanisms at the level of neurotransmitters.

Results and discussion

In accordance with DSM-IV criteria, the obsessive-compulsive disorder is a syndrome manifested by obsessions and (or) compulsions, which, as a whole, lasts more than one hour during the day and interferes in the social and professional activity of the subject.

Several authors relate the dissimulation of the obsessive-compulsive symptoms in terms of depression or anxiety, that is why the presence of these two symptoms requires the need to examine the patient about the presence of obsessions and compulsions.

Neuroanatomic aspects

Through the PET method, there were found neural abnormalities of the cortical projections on the basal ganglia. It is also supposed the involvement of the orbital-frontal projections of the pre-frontal cortex where it has been found the increase of the metabolic activity. (4)

Genetic Aspects

We found, in some cases, genetic abnormalities which encode the information for the receptors D2, D3 and D4.

Also, there are studies claiming genetic abnormalities at synthesis level DAT (dopamine transporter protein) or catecolamintransferasis (COMT – enzyme that degrades dopamine in synaptic slot). (5)

Neurobiological hypotheses

Neurobiological hypotheses focus on serotonin neurotransmission, dopamine, norepinephrine, cholecystokinin having almost identical mechanisms to other psychiatric disorders which belong to the biological spectrum of the OCD. These are pathological gambling, paraphilias, dysmorphophobia, trichotillomania, hypochondria, somatization disorder, Gilles de la Tourette syndrome, autism, Asperger syndrome, kleptomania, bulimia, anorexia nervosa. (7). Goodman (1994) reported that obsessive-compulsive disorders can be found in schizophrenia and in about 1 – 20% of cases, and some antipsychotics like clozapine and risperidone may increase the obsessive-compulsive manifestations. Likewise, practical experience shows the presence of obsessive-compulsive disorders in head injury, carbon monoxide poisoning, cerebral ictus and other pathological conditions involving

the basal ganglia. Swedo, 1994 reported that obsessive-compulsive disorders are present in 70% cases in patients with Korea Sydenham, which appears in advanced stages of rheumatism.

Serotonin hypothesis

The hypothesis is related to the serotonergic dysfunction supposed at CNS level. Based on the effect of serotonin reuptake, medications such as fluoxetine, sertraline, paroxetine, fluvoxamine and citalopram show positive effect in this mental disorder. Improvement in OCD symptoms was consistent with parallel decrease in acid 5 – hydroxyindolacetic (5-HIAA) which is serotonin metabolite in cerebrospinal fluid. (6)

Dopamine hypothesis

It was found that about 40% of patients do not respond to treatment with serotonin reuptake inhibitors.

The first arguments related to the possible involvement of dopamine in OCD have been provided for Von Economo encephalitis disease, which are symptoms of obsessive-compulsive series. The presence of this disease was found in inflammatory lesions of ventral tegmental area (ATV or 10th or mesolimbic area of the CNS). Another argument in support of the theories of dopamine in OCD is the motor acts that are repetitive manner similar to compulsive rituals for patients who abuse drugs, including amphetamine, which enhances the release of dopamine in the synaptic slot. Also other substances acting as dopamine agonist induce movements of OCD type. They are bromocriptine, apomorphine, L-dopa. Cocaine can worsen the OCD symptoms considered, and also other diseases in the same spectrum, e.g. Gilles de la Tourette disease. The pharmacological arguments in favour of dopamine pathology of the OCD are based on the beneficial therapeutic effects of classical neuroleptics.

There are reports that autoimmune disease may develop at the basal ganglia disorders such as OCD. The most significant link that shows the involvement of dopamine in the basal ganglia is the identical mechanism established between the OCD and Gilles de la Tourette disease. From 45 to 90% of the patients with Gilles de la Tourette present obsessions and compulsions, and a very significant percentage of patients with Gilles de la Tourette meet the diagnostic criteria for OCD. (5) The family history was found in some OCD manifests and to others, Gilles de la Tourette's. In these cases, it seems that there is involvement of both serotonin and dopamine transmission. It is not known which neurotransmitter system has been affected first: serotonin, dopamine or serotonin-dopamine balance. Proceeding from these considerations, it was found the symptomatic improvement in some patients that are not responding to serotonin reuptake inhibitors, to who were given medicines with dual mechanism of action, namely atypical antipsychotics.

ASPECTS OF PSYCHOPHARMACOLOGY IN THE TREATMENT OF OBSESSIVE-COMPULSIVE DISORDER

Serotonin reuptake inhibitors

1. The first validated antidepressant for the treatment of OCD was melipramine. The effect of serotonin reuptake blocker has been demonstrated in the efficacy of the TOC treatment, and demonstrated the positive effect of other antidepressants inhibiting the reuptake of serotonin. Furthermore, the efficacy of clomipramine cannot be explained by the effect of noradrenaline reuptake inhibition, because the effectiveness of antidepressants reuptake of norepinephrine was very weak in the cases of administering desipramine and nortriptylin. Moreover, it shows that both antidepressants inhibiting the reuptake of serotonin and much less those of norepinephrine reuptake inhibitor for OCD are largely effective because of the effect on depression associated with OCD and less on basic symptoms. Depression in OCD seems to be of serotonergic nature and not noradrenergic. The antidepressants doses used in OCD are recommended to be much higher than in depressive states and the esti-

mated time until the appearance of the first effects will be longer, being estimated according to various authors within 6 – 12 weeks compared with the depressive moods when the effect appears much earlier.

Another important aspect is the probability of relapse after stopping the treatment, which is also much greater than depression.

It was found that depressed patients who respond well to treatment with serotonin reuptake inhibitors, at the establishment of a diet without tryptophan, which is the predecessor of 5-HT – there is a worsening condition in patients with depression return. Same deprivation of tryptophan in patients with OCD and which responded to treatment with SSRIs did not relapse of OCD symptoms. We must return to the supposed different mechanisms in the occurrence of depression and OCD: both are of serotonergic nature, the disposition being adjusted by the projection of serotonergic neurons in the raphe's area on the frontal cortex and the motility, including obsessions and compulsions are influenced by the projection of these neurons on the basal ganglia. (5)

Therefore, it is determined the efficiency, in general, of the antidepressants in the treatment of OCD. It is assumed that the positive response is caused more by the serotonergic mechanisms than the noradrenergic ones. The presence of obsessive and compulsive symptoms is less dependent on the serotonergic availability at synaptic level, compared with depressive states where this serotonin deficiency is characterized by the worsening of the depression. The risk of relapse after stopping treatment in OCD is higher, compared with depressive moods.

Adjacent solutions

It is estimated the effectiveness of partial serotonin agonists as buspirone, serotonin antagonists as trazodone, benzodiazepines, including hypnotics, classical or atypical neuroleptics. Benzodiazepines, including hypnotics will be associated in generalized anxiety or insomnia. Classical neuroleptics will be associated with comorbidity in cases of Tourette's syndrome or schizophreniform symptoms.

Psychosurgery

Would be applicable in very resistant cases through the sectioning of the frontal neural pathways by of the basal ganglia. There are currently no data in reference to these experiences.

Psycho-behavioural therapy

Would be as effective as SSRIs and relapse should be fewer than after stopping treatment with SSRIs.

In conclusion, the dysfunction hypothesis of the system of the neurotransmitter revealed some peculiarities in the treatment of OCD. So far, the existing theories do not meet the conditions necessary for conclusive statements regarding the etiology of the obsessive-compulsive disorder. It can be assumed only the substantial role played by the serotonin neurotransmission, and will determine the degree of participation of other neurotransmitters in the genesis of this disorder.

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