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Remission of Obsessive-Compulsive Symptoms in Hypomanic Period in a Patient with Comorbid Bipolar Affective Disorder and Obsessive Compulsive Disorder: A Case Report

Bipolar Affektif Bozukluk ve Obsesif Kompulsif Bozukluk Eştanılı Bir Olguda Hipomanik Dönemlerde Obsesif-Kompulsif Belirtilerin Düzelmesi: Bir Olgu Sunumu

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Abstract

Given the prevalence rates of obsessive compulsive disorder and the bipolar affective disorders in the general population, one would expect the co-occurrence of these syndromes to be rare. Yet, findings have revealed extremely high rates of comorbidity in obsessive compulsive disorder with both depressive disorders (50%) and bipolar disorder (10%) and shown that obsessive compulsive disorder is the most common anxiety disorder in patients with bipolar disorder. This situation makes it difficult to recognize the changing clinical disease and prognosis while response to treatment can also be adversely affected. Clarifying the phenomenological features of obsessive compulsive disorder-bipolar affective disorder comorbidity has important etiological and treatment implications. In this article, we discuss the pathophysiological importance of the improvement of obsessive compulsive symptoms during hypomanic episodes in a bipolar disorder patient, who had comorbid obsessive-compulsive disorder.

Keywords: Bipolar Affective Disorders; Obsessive Compulsive Disorders; Comorbidity.

Öz

Bipolar affektif bozukluk ve obsesif kompulsif bozukluğun genel nüfustaki yaygınlık oranları göz önüne alındığında iki sendromun birlikteliğinin nadir olması beklenebilir. Ancak çalışmalar obsesif kompulsif bozukluğun hem depresif bozuklukla (%50) hem de bipolar affektif bozuklukla (%10) birlikteliğinin yüksek oranlarda görüldüğünü ortaya koymuştur. Bipolar bozuklukta en sık görülen anksiyete bozukluğunun obsesif kompulsif bozukluk olduğu gösterilmiştir. Bu durum, hastalığın kliniğini değiştirerek tanınmasını güçleştirmekte, prognozu ve tedaviye yanıtı olumsuz olarak etkileyebilmektedir. Obsesif kompulsif bozukluk ve bipolar affektif bozukluk birlikteliğinin fenomenolojik özelliklerinin netleştirilmesi etyolojisini aydınlatma ve tedavi yaklaşımı açısından önemlidir. Biz de bu yazıda bipolar affektif bozukluk ve obsesif kompulsif bozukluk eş tanılı bir olguda hipomanik dönemlerde obsesif kompulsif belirtilerinin düzelmesinin fizyopatolojik önemini tartıştık.

Anahtar Kelimeler: Bipolar Affektif Bozukluk; Obsesif Kompulsif Bozukluk; Eş Tanı.

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INTRODUCTION

Studies show that Bipolar Affective Disorder (BAD) and anxiety disorders usually manifest themselves simultaneously (1). Although it has been suggested that this association was coincidental (2), large-scale epidemiological studies have demonstrated that this is more than a coincidence (3, 4). It has even been suggested that these two may be related pathophysiologically.The common biological mechanisms involved in their etiologies and the overlapping neurotransmitter and neuromodulator systems are signs of the reliability of this model as reports show. There are also some studies that assess generalized anxiety, panic, social phobias, obsessions, and compulsions as pathological symptoms of bipolar disorder (1).

The majority of these patients receive comorbid multiple anxiety disorder diagnosis. It has been shown that the most common anxiety disorder is obsessive-compulsive disorder (OCD) (2). This changes the clinical picture of the patient and makes the disease difficult to recognise while also adversely affecting the prognosis and response to treatment (5). It has been suggested that OCD comorbidity increases the risk of suicide in bipolar patients and influences the quality of life negatively (6).

Recent epidemiological and clinical studies point to a high incidence of association between BAD and OCD; however, the number of studies concerning socio-demographic and clinical characteristics of this association is limited. Moreover, comorbidity of these two disorders make diagnosis and treatment difficult. With the aim to contribute to this topic, we aim to present the case of a patient with comorbid BAD and OCD by studying the mechanisms underlying the improvement of OCD during hypomanic episodes.

CASE REPORT

A 23-year-old male student, E.D, was brought to our clinic with complaints of talking too much along with excessive mobility and spending. His medical history showed that the complaints had started 15 days ago accompanied by decreased sleep at night. Despite sleeping less, he had constantly been on the move and taken up the habit of purchasing expensive items that he did not need and storing them in his drawer. The initial mental state examination revealed that he was conscious, cooperative, and had full orientation with accelerated association. His mood was euphoric and did not have insight into his disease. At the start of his hospital stay, his Young Mania Rating Scale score was 20.

The psychiatric history of the patient revealed that his first complaints started as compulsive behaviours such as constantly watching his back in high school. He related that he felt he was being tracked by his family and friends and he had obsessive thoughts about religion. The patient had been followed for OCD for the last 2 year and he had not used any drugs in the last 2 months. About 1 year ago, he spent his time indoors for about 1 month during which he was obsessed with fate concept, read many books on the subject, and gave much thought on people's life goals. During this period, his condition was accompanied by OCD and depression. He defined a depressive mood in which he did not enjoy anything in that period. He stated that he thought he lost his energy although he did not attempt or think about suicide. We learnt that his obsessions continued to increase during this depressive time. At the time, he had started using fluoxetine, benzodiazepine, and clomipramine but discontinued using these drugs due to the side effects. However, we also learnt that, following the depressive episode a year ago, the patient had experienced a similar hypomanic period during which he was hyperactive, talked a lot, and spent too much money. This was repeated once again after 6 months. The patient informed us that these obsessions and compulsions disappeared during his hypomanic periods. After the medical history, mental status examination, and interview with the family, the patient was diagnosed with bipolar affective disorder type 2 accompanied by pre-diagnosed hypomanic episodes and hospitalised.

Throughout his time at the hospital, we observed that the patient was still in his period of hypomania though without any signs of OCD. We started to give the patient valproic acid 1000 mg/day and risperidone 3 mg/day. At the end of three weeks, his mobility and talking decreased. He could sleep in regular sleep patterns although, immediately after this, he developed obsessions and compulsions once again. We started anafranil 75 mg/day and discharged the patient for outpatient follow up and treatment. In his follow-ups, we observed that the predominant symptoms were of OCD when BAD-2 was in remission.

DISCUSSION

In this case report, we present the case of a 23-year-old male patient with comorbid BAB type 2 and OCD. It is stated that patients with bipolar affective disorder have higher risk to develop at least one more anxiety disorder. It has been reported that the most common anxiety disorders is OCD (1). In the past, OCD was associated with major depressions but recently it is thought that there is a strong link between OCD and BAD (2). A study has shown that 16% of the followed OCD outpatients patients had comorbid BAD and 67% of these patients had BAD 2 (7). In our case, although BAB and OCD co-existed, OCS symptoms were denser in depressive episodes whereas obsessive-compulsive symptoms were more notable during hypomanic episodes and this makes our patient a noteworthy case.

Studies on the pathophysiology of co-existing BAD and OCD are limited. Striatal hyperactivity is thought to be a common biological basis for both (8). It is known that both diseases are caused by the changes in the serotonergic, dopaminergic, GABAergic, and glutamatergic neurotransmissions (9). The fact that drugs that block serotonin reuptake bring about good results for OCD has turned the attention to OCD-serotonin

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hypothesis (10). It is known that serotonin alone is inadequate to explain the etiology of the disease. The change in balance between serotonin and other neurotransmitters or receptor changes may be more effective in explaining its etiology (11). But the high rates of hypomania and mania associated with the use of antidepressants in all anxiety disorders prevent the serotonin reuptake inhibitors in the treatment of comorbid conditions (12). The presence of OCD in patients with basal ganglia damage in which dopamine neurones are dominant has attracted attention to the idea that dopamine may play a role in OCD. Another sign that supports this idea is that dopaminergic agents can lead to the formation of symptoms and that SSRIs improve when they are added antidopaminergic agents. Antidopaminergic treatment alone is not effective. Anxiety and egodystonicity seen in OCD are not present with the symptoms that develop with dopaminergic agents only. This suggests that serotonergic dysfunction is necessary for egodystonicity (13). Norepinephrine and serotonin are more commonly mentioned in the studies analysing the biochemical etiology of bipolar disorder. The effect of dopamine has also been theoretically recognised. Data show that dopamine, serotonin, and norepinephrine levels decrease in depression while they tend to increase during manic episodes (14). These neurotransmitters, which increase during manic episodes, are the ones that decrease in OCD and may be responsible for the etiology of OCD. Therefore, the data presented above about our case may explain the biological factors concerning the onset of manic episodes which took place when the patient was not followed as well as the remission of OCD. It can be taught that neurochemical factors such as dopamine, serotonin, and norepinephrine, which are responsible for the pathophysiology of manic episodes, may be correcting the symptoms of OCD. However, as each neurotransmitter is not sufficient alone to explain the etiology, the interaction and imbalance between the two diseases suggests that the coexistence of these two disorders change the clinical course for patients.

As a result, in order to follow the prevalence of OCD in bipolar disorder patients, associating factors, and clinical processes of these disorders, more follow-up studies are required. In this article, we have aimed to draw attention to the pathophysiological importance of the disappearance of OCD symptoms during manic episodes. In this regard, further research will provide progress in the description of the pathophysiology and treatment of both diseases.

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