ARACHNOID CYST AS THE CAUSE OF BIPOLAR AFFECTIVE DISORDER: CASE REPORT

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SUMMARY – This report presents the course of diagnostic examinations and treatment of a 20-year-old man with bipolar affective disorder for which an organic basis was demonstrated. Computed tomography of the brain revealed an arachnoid cyst that was surgically treated. The patient underwent both psychiatric and neurosurgical treatment. After two-year follow-up and medicamentous treatment prescribed, the patient was symptom-free requiring no psychopharmacotherapy for the next 5.5 years. His overall life functioning is normal, with no signs of disease.

Key words: *Bipolar affective disorder, organic etiology; Computed tomography of the brain; Diagnosis; Treatment*

Introduction

Bipolar affective disorder is a category of mood disorders of a multifactor genesis. Pathologic mood and related vegetative and psychomotor symptoms make the key clinical features. The change of affect, together with the change in instinct dynamism, will and opinion, generally lead to a significant and conspicuous change of behavior in comparison to the period before the illness[1]. The manic phase of the disorder, especially when concurring with psychotic symptoms, is often not diagnosed as the affective mood disorder. Bipolar affective disorder is a mood disorder in which depressive phases occur with typical depression symptoms which are not different from unipolar depression, when only depression occurs. Hypomania or mania is a phase of elevated mood occurring in patients with bipolar affective disorder. Phases of elevated mood are characterized by hyperactivity, higher level of energy, reduced need for sleep, and other symptoms. In mania, the behavior is more conspicuously changed. Persons suffering from depression are at a risk of suicide, while in hypomania/mania there is a possibility that the person puts himself in a series of embarrassing situations relating to the person's finances, job, family relations, reputation, and the like. It is necessary to differentiate between depression and depression occurring in bipolar disorder. Although their clinical characteristics are the same, the treatment of bipolar depression differs[2,3].

Bipolar affective disorder has a greater incidence than schizophrenia. The prevalence ranges from 1% to 3%. This mood disorder appears less frequently than depression and makes up to 10%-20% of all mood disorders. Depression is most frequently diagnosed, while the hypomanic phase relatively often remains undetected. Unfortunately, too often the disorder is undiagnosed and untreated in primary health care (40%), especially in younger and older patients because of comorbidity and age specifics. A large number of patients do not seek any psychiatric help, although the suicide rate with this disorder is very high (15%-20%). In 90% of all cases, there are multiple recurrences, with unsatisfactory improvement in almost half of the total number of patients[4-6].

The etiological factors of bipolar affective disorder are multiple, including neurochemical, genetic, psychosocial and organic factors[7].

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Concerning the organic basis of bipolar affective disorder, it is well known that the following conditions increase the risk of a mental disorder: epilepsy, limbic encephalitis, Huntington's disease, head trauma, brain neoplasm, extracranial neoplasm with distant effects on the central nervous system (particularly pancreatic cancer), cerebrovascular diseases, lesions or malformations, lupus erythematosus and other collagenoses, endocrine diseases (especially hypothyroidism and hyperthyroidism, Cushing's syndrome), tropical infective and parasitic diseases (e.g., trypanosomiasis), and toxic effects of nonpsychotropic medications (propranolol, levodopa, methyldopa, steroids, antihypertensives, antimalarials)[8-10]. For a clinical syndrome to be construed as being caused by one of the above mentioned organic damages there has to be proof of a brain disease, damage or dysfunction, a systemic disease known to concur with one of the mentioned syndromes. There also needs to be a time relation between the disease development and the onset of a mental disorder. It is necessary to prove recovery from disorder after elimination of or recovery from the cause. The absence of an alternative cause of mental disorder also needs to be established (for instance, a burdened family medical history or precipitating stress)[11].

The organic origin of bipolar affective disorder has been presented by a number of authors in their case reports[12-16].

The aim of this case report of a patient with arachnoid cyst and organic bipolar affective disorder is to present our experience and approach to the psychotic condition in terms of diagnosis and treatment, as well as patient follow-up.

Case Report

A young man at the age of 20 was urgently hospitalized for acute psychotic condition. A few days before, he had been admitted to the psychiatric ward of a hospital in the town where he did his regular military service, where he was administered high dosages of haloperidol (30 mg daily), so that his clinical manifestation, along with the dominant signs of psychomotor agitation, elevated mood, racing thoughts, disorganized behavior, elevated instinct dynamisms, also showed pronounced extrapyramidal symptoms. From his medical history, we could learn that he was born as the second of four children in a family with no previous psychiatric disorder or disease history. The pregnancy, early growth and development were normal. He finished his schooling normally, with good results. He was hospitalized during his regular military service term. The family provided heterohistory data on his occasional cannabis consumption over the past three years, so his mental state was seen as symptomatic for cannabis abuse. However, we also found that he had been showing behavior changes and mood swings in the past few years: for months he had been in bad mood, had no energy, stayed mostly in bed and slept a lot. In subsequent medical history data the patient provided, he rationalized cannabis and alcohol intake, explaining that he did not feel well, that he was sad, depressed, often thinking how life was meaningless, thought of death, and that, after smoking marihuana or drinking alcohol his mental state would be better. About three weeks before he was first admitted, he was at home on a regular free weekend from the military, at which time his behavior was very conspicuous, exhibiting rapid talking, hyperactivity, an extremely good mood, he was exhilarated, full of plans, he did not sleep, none of the family members could follow his racing thoughts and behavior, and at that time, the family associated his condition with alcohol intake.

During his hospital stay, after detoxification and alleviating the side effects of haloperidol, he was started on antipsychotic treatment with olanzapine and the mood stabilizer carbamazepine in order to alleviate acute psychotic symptoms. Indicated examinations revealed changes in his electroencephalogram (EEG): left frontal temporal dysrhythmic irritation changes. Computed tomography (CT) scan showed an arachnoid cyst located in the middle cranial fossa on the left, stretching cranially along the fissure of Sylvius frontally about 7-8 cm, and with a wide basis adhering to the bone and discretely exerting pressure on the frontal part of the left temporal lobe, the frontal lobe and the insular cortex with mild compression and dislocation of the left lateral ventricle medially, and dislocation of the left middle cerebral artery flow, laterolaterally by about 5 cm and with an anteroposterior diameter of 5 cm.

The diagnosis was established on the basis of clinical evidence, in compliance with the DSM-IV and ICD-10 classification criteria, as well as psychological examination and organic examination results: EEG and CT brain scan.

After reducing the acute psychotic state of a manic character, in agreement with neurosurgeons, we did the indicated surgery, cystoperitoneostomy. The patient was subsequently discharged from the hospital in good physical and mental condition. After his inpatient stay was over, the patient received regular follow-ups and took the prescribed treatment. For months he complained of headaches and dizziness, he would rest for most part of the day, lying in bed because it made him feel physically better. Five months later, he fell into depressive state and attempted suicide by overdose drug ingestion. He was readmitted to the hospital, this time for depressive decompensation and a high risk of suicide. Along with his previous treatment, because of dominant symptoms of depression, we introduced the antidepressant clomipramine. For four months, the patient underwent inpatient care treatment, during which time he had two very serious suicide attempts: by slashing his cubital veins on both forearms while he was out on a pass and by drug intoxication and hypothermia, when he left the ward on his own will and, heavily intoxicated from alcohol, fell asleep in the snow. Upon agreement of the entire medical team and with parental consent, he was discharged in a relatively good mental and physical condition. In the months that followed, he regularly took all his medications as prescribed, he followed the instructions he was given, he functioned increasingly better and with better quality. The follow-ups showed no signs of disease relapse; on the contrary, with time his condition improved. So much better, actually, that he started working only three months after being discharged from hospital. In parallel to psychiatric follow-ups, he also received neurosurgical follow-ups, which showed normal postoperative results. The psychiatric medications prescribed were gradually reduced as months went by, and after two years, due to the lack of any signs of psychopathology, they were discontinued. Before psychopharmacotherapy was discontinued, we ran psychological tests, which revealed a person of solid cognitive abilities and a balanced personality profile. In the meantime, the patient has changed jobs, makes his own living, has had several emotional relationships and an adequate social life for his age. A year ago, the family suffered a trauma of the mother's sudden death, with which the patient managed to cope without the need for any psychiatric support. His current state is normal; his mental examination results show no signs of pathology. Five and a half years have elapsed since he last took any medication, and the last clinical follow-up in spring 2010 showed no signs of the disease.

Discussion

In psychiatric clinical practice, we daily encounter patients who show psychotic symptoms, being either recurrent or first episodes of the disease. In case of first hospitalization, clinical evaluation and psychological tests are always run along with a series of other diagnostic procedures such as laboratory tests, EEG and CT brain scan. In this particular case, the patient was hospitalized at our department for the first time, and although we had clear clinical evidence, the symptoms that satisfied the criteria for establishing the diagnosis of bipolar affective disorder according to DSM-IV and ICD-10, medical history data, heterohistory data of his premorbid functioning and cannabis and alcohol abuse, we followed our standard clinical practice. As soon as we got the EEG results, which indicated a possible organic cause, in view of the left frontal temporal dysrhythmic changes, CT brain scan was indicated as soon as possible. After CT scan, it was clear that there possibly was an organic basis of the disorder. As we were dealing with manic decompensation with an abundance of glaring symptoms, we needed to detoxify the patient first because of pronounced extrapyramidal side effects, and then stabilize him by administering appropriate medications. We chose olanzapine and carbamazepine; olanzapine for its design and the patient's prior hypersensitivity to haloperidol, and carbamazepine because of organic test results, possible epileptic seizures (not only because of the arachnoid cyst, but also the administration of antipsychotics)[17,18]. From the neurosurgical aspect, surgery was indicated in order to drain the cyst and thus reduce the intracranial pressure. After reducing the acute state and prepping the patient, neurosurgery was performed and postoperative recovery was normal. Then came psychiatric and neurosurgical follow-ups, and re-hospitalization due to depressive decompensation. His state was complicated by suicide attempts. However, with administration of appropriate psychopharmacotherapy, which included olanzap-

ine, carbamazepine and clomipramine, after several months of inpatient care, the patient was discharged in good mental and physical state. On subsequent follow-ups, his medications were adjusted to his mental state. After two years, treatment was discontinued. It has now been 5.5 years since the patient took any medications. His mental functioning is normal.

We believe that this patient's bipolar affective disorder, which presented itself in a wide-ranging symptomatology of manic and depressive phases, had an organic basis. We draw this conclusion on the following facts: for a clinical syndrome to be considered as being caused by organic damage there should be proof of a brain disease that is known to concur with one of the mentioned syndromes. In our case, the existence of an arachnoid cyst was undoubtedly confirmed. There also needs to be time relation between the disease development and the onset of mental disorder. As the arachnoid cyst in our patient was taken to be congenital because there were no data on head trauma or any other brain disease experienced during his life, it is our opinion that the mentioned cyst must have grown over time, which ultimately led to pressuring certain brain structures and to mental disorder. It was necessary to prove recovery from the disorder after elimination of or recovery from the cause. This criterion was also met because after surgery, although it was followed by a depressive phase, the patient did fully recover both mentally and physically and is able to function in all segments of life. Apart from that, the absence of an alternative cause of mental disorder should also be proven (for instance, a burdened family medical history or precipitating stress). In the detailed data provided by members of the family we found no hereditary cause or a precipitating stress event.

In conclusion, based on several years of the patient's follow-up, we believe that the patient's mood disorder, the bipolar affective disorder, was of an organic genesis.

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Sažetak

ARAHNOIDNA CISTA KAO UZROK BIPOLARNOG AFEKTIVNOG POREMEĆAJA: PRIKAZ SLUČAJA

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Ovaj prikaz predstavlja tijek dijagnostičkih pregleda i liječenja 20-godišnjeg muškarca koji boluje od bipolarnog afektivnog poremećaja, a za kojega je i prikazana organska podloga same bolesti. Kompjutorska tomografija mozga otkrila je kod bolesnika arahnoidnu cistu koja je kirurški liječena. Bolesnik je podvrgnut i psihijatrijskom i neurokirurškom liječenju. Nakon dvije godine kontinuiranog praćenja stanja bolesnika uz primjenu propisane medikamentne terapije bolesnik više nije pokazivao simptome bipolarnog afektivnog poremećaja i nije bila potrebna daljnja primjena psihofarmaka slijedećih 5,5 godina. Njegova ukupna kvaliteta života je zadovoljavajuća, a bolesnik normalno funkcionira u svakodnevnom životu bez ikakvih znakova bolesti.

Ključne riječi: *Bipolarni afektivni poremećaj, organska etiologija; Kompjutorizirana tomografija mozga; Dijagnostika; Liječenje*