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Case Report

Bipolar Disorder with Psychosis Featured in Patient with Systemic Lupus Erythematosus

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Abstracts

Introductions: Bipolar disorder (BD) is a mental disorder that imposes unusual shifts in mood, energy, activity levels, and the ability to carry out day-to-day tasks, characterized by biphasic mood episodes of mania or hypomania and depression. More than half of all individuals diagnosed with BD experience psychosis features in their lifetime. The cause of BD is not entirely known, study claims that BD may occur due to or associated with autoimmune diseases. Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease with various physical manifestations, including neuropsychiatric features. **Case:** This case report will discuss a 22-year-old female with BD admitted to the emergency room due to solid psychosis features. The patient was diagnosed with SLE 3 years ago but never controlled prior to the disease history. The psychiatrist's treatment in the emergency room was an extra injection of haloperidol 5 mg IM and diazepam 10 mg IM, continued with oral medication clozapine 25 mg two times a day and additional therapies such as cognitive behavioral therapy (CBT) and family therapy. The treatment from the internist was symptomatic medication due to the acute SLE symptoms, followed by autoimmune medicines such as steroids and hydroxychloroquine. **Conclusions:** BD may be associated with SLE. Psychosis features generally occur in BD, usually in the manic period. Treatment should be done for both psychiatric and internal medicine problems. As the SLE symptom was controlled, the BD symptom improved.

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Introductions

Systemic lupus erythematosus (SLE) is a worldwide chronic inflammatory autoimmune disorder of unknown cause that affects multiple organ systems [1,2]. Genetic predisposition, environmental triggers, and the hormonal milieu, interplay in disease development and activity [3]. SLE is characterized by the formation of pathogenic autoantibodies against nucleic acid and their binding proteins to their own body's components [4]. Clinical manifestations and the pattern of organ involvement are widely heterogeneous [2], including the symptoms of inflammation in the central nervous system (CNS) [1].

Neuropsychiatric symptoms in SLE develop in 20–70% of SLE patients during the disease, and bipolar disorders (BD) were found in 5.8% of SLE patients [1]. Evidence has accumulated suggesting BD was associated with immune dysfunction, such as chronic low-grade inflammatory responses, activation of cell-mediated immunity, increased oxidative stress, and autoimmune responses [5]. BD are chronic and recurrent disorders that affect >1% of the global population [6], the incidence of BD amounts to approximately 4% of the adult population, but may reach 6.5% if minor and atypical forms are included [7]. BD include several disorders of emotion, energy, and thought characterized by biphasic mood episodes of mania or hypomania and depression and are expressed as recurrent episodes of changes in energy levels and behavior [6]. The disease can lead to cognitive and functional impairment and increased mortality, particularly from suicide [6,8]. Literature suggests that executive function impairment could serve as an endophenotype for BD, especially concerning psychotic cases [9].

Psychosis features can occur in both the manic and depressive phases of bipolar disorder [10,11]. More than half of patients with BD will experience psychotic symptoms in their lifetime [10]. Psychosis in BD constitutes an important element with profound consequences for the treatment, prognosis, and overall outcome of the patients [9]. There is a common clinical assumption that BD with psychosis reflects greater severity than BD without psychosis; inversely, the presence of psychosis does not appear to be associated with poorer clinical/functional outcomes or suggests a greater degree of neuropsychological impairment [12]. Despite a considerable amount of research, the course and outcome of bipolar disorder still remain highly unpredictable [13]. Studies have shown that even after years or even decades of mood stability, most bipolar individuals will relapse within 6-12 months if maintenance therapy is discontinued [14]. Because both SLE and BD are thought of as relapsing-remitting disorders, therapy is directed to long remission and to prevent flares of disease [1].

Case

A 22-year-old female was brought to Mutiara Sukma Mental Hospital at the emergency room due to a psychosis feature. The patient got manic symptoms, paranoid delusions, and visual and auditory hallucinations, so she tried to kill her mother. The symptoms started one week before and got worse two days before being admitted to the hospital. The patient also constantly had physical symptoms such as general weakness, dizziness, arthralgia, dyspepsia, and malar rush for more than three years and got worse in a month. According to the previous history, the patient was diagnosed with systemic lupus erythematosus in 2018 and was hospitalized for two months in Dr. Soetomo Hospital, Surabaya. After a month, the patient had psychosis during the treatment, so she was transferred from the internal medicine ward to the psychiatric ward and given anti-psychosis therapy. After recharging from the hospital, the patient never controlled and only took symptomatic medications such as methylprednisolone, mefenamic acid, and paracetamol.

Two years later, in late 2020, the patient got manic and psychotic again and was admitted to the hospital. The patient was diagnosed with bipolar disorder with psychosis features. At this moment, the physical examination was not thoroughly evaluated. According to a recent physical examination, the Glasgow Coma Scale (GCS) was 15, and the general condition was weak, blood pressure was 91/60mmHg, pulse 109x/m, respiration 20x/m, temperature 36.5 degrees Celsius, saturation 99%, body mass index 20.7. We found mild malar rush, epigastric pain, and moderate arthritis in the dextra and sinistra of the higher and lower extremities. From the mental status, we found clear consciousness, psychomotor hyperactivity (average PANSS EC score: 5), mood irritable, narrow affect, non-realistic thought, paranoid delusion, visual and auditory hallucination, and insomnia, with an insight score of 5.

The laboratory examination result was Hb 8.7 gr%, RBC 3.89x10^6/mm3, WBC 6450/mm3, PLT 238000/mm3, blood sedimentation rate (LED) 65 mm/hour. Peripheral blood morphology indicated anemia microcytic hypochromic suspect iron deficiency anemia and/or anemia due to chronic disease. Blood glucose, liver function, and renal function were normal. The radiology examination showed heart and lung functions were normal.

The patient received an extra injection of haloperidol 5 mg IM and diazepam 10 mg IM and continued with oral medication clozapine 25 mg two times a day in the emergency room. The patient was also being consulted to internal medicine division, and the treatment was methylprednisolone 2 mg once a day, HiD 5000 ui once a day, caviplex tablet once a day, omeprazole 20 mg once a day, ibuprofen 400 mg twice a day, and observed for another worsening or additional sign and symptom of the SLE.

After two days of treatment inward, psychosis symptom was minimal, but BD and SLE symptoms had no improvement. The psychiatrist did the MMPI-II test. The result indicated that the patient had impaired psychological function, heavy stress, a meager ability to make good interpersonal relations, and severe behavioral and thought problems that lead to difficulty doing daily activities. There was no additional therapy from the psychiatrist. The internist re-evaluated the laboratory test, and the result was Hb 8.4 gr%, and blood sedimentation rate (LED) 93 mm/hour. Therefore, another treatment was added. The patient received a transfusion of a kolf of packed red cells with premedication and another autoimmune therapy, such as increasing the dose of methylprednisolone from 2 mg to 4 mg once a day and hydroxychloroquine tablet 200 mg once a day. Another evaluation was added, and the result was normal.

A day after additional therapy, the physical symptoms improved. The mental status got better. Mood euthymic, psychomotor normo-active, no delusion, and no hallucination. The patient slept and ate well and became very cooperative. At this time, the psychiatrist also started cognitive behavioral therapy (CBT) and family therapy. After a week of treatment and observation, the patient was discharged from the hospital.

Discussions

In this case, BD was diagnosed based on the Diagnostic and Statistical Manual of Mental Disorder Fifth Edition (DSM-V) criteria by doing anamnesis and examining physical and mental status. SLE was diagnosed based on the new European League Against Rheumatism (EULAR) / American College of Rheumatology (ACR) 2019 classification criteria for SLE, where we found that there was a history of Anti-Nuclear Antibody-Indirect Immunofluorescence (ANA-IF) titer strongly positive and score for other clinical criteria was 11; fever (2), arthritis (6) and psychosis (3) [4,15].

From the current physical examination,

we found that the patient had anemia microcytic hypochromic suspected due to chronic disease. A study claimed the prevalence of anemia among chronic psychiatric patients is higher than the mean prevalence in the general population, anemia in the depressive disorder patient group (22%), and bipolar patients (25%) is higher than in the general population. In addition, anemia itself could cause aggravation of psychiatric symptoms such as cognitive function disorders and depression or could deteriorate an existing psychiatric condition when it is untreated [16].

We also found that the patient never controlled and only took symptomatic medications to treat her symptoms (ex: methylprednisolone). An exciting study claimed that most bipolar individuals would relapse within 6-12 months if maintenance therapy were discontinued, and glucocorticoids also induced psychiatric problems such as depression, mania, psychosis, and delirium [10,14]. These syndromes collectively are known as steroid psychosis, and their prevalence is estimated at 0.87%. Glucocorticoid-induced mood disorders vary from bipolar manic-depression and unipolar disorders, in which either mania alone or depression alone is found [10].

Psychosis, generally defined as the occurrence of hallucinations or delusions, is a common feature across numerous psychiatric disorders [12]. Approximately 58% of patients with bipolar disorder had a lifetime history of at least one psychotic symptom, usually when manic [10]. A meta-analytic study confirmed that a history of psychosis was associated with poorer cognitive functioning in BD [17]. But another study found that the presence of psychosis was not associated with worse neuropsychological function [12].

However, the cause of BD is still not entirely known [13]. Genetic influences are believed to account for 60–80 percent of the risk of developing BD, indicating a strong hereditary component. Environmental factors play a significant role in development, and individual psychosocial variables may interact with genetic dispositions. Recent life events and interpersonal relationships probably contribute to the onset and recurrence of bipolar mood episodes [5,13]. Furthermore, BD may occur due to or associated with a neurological condition or injury such as stroke, traumatic brain injury, autoimmune diseases, and rarely temporal lobe epilepsy [5]. Evidence has suggested that inflammation and immune dysregulation play a significant role in neuroprogression in bipolar disorders [18]. The hypothesis was; 1. Chronic peripheral inflammatory process activated by Systemic Autoimmune Diseases (SADs) might cause the up-regulation of Central Nervous System (CNS) inflammation resulting in BD. 2. SADs exacerbate the BD syndrome in the presence of subclinical BD vulnerability. The mechanisms could be direct (acting on the biological substrate of BD) or indirect (acting on vulnerability to depression or other anxiety disorders that might exacerbate BD). 3. Subclinical BD acting as a nonspecific stress factor may have increased the vulnerability to SADs. 4. SADs and BD have a shared etiology. Inflammatory cascades that can support this possibility may be genetically varied in BD patients and some SADs and BD co-aggregate in families [5,18].

Management patients, in this case, are both psychiatric and internal medicine problems. After treating the emergency symptoms, we controlled BD by giving clozapine as an oral medication, also CBT, and family therapy as additional therapies. Clozapine is an atypical antipsychotic used in treatment-resistant bipolar disorder. Evidence for its anti-suicidal, anti-aggressive properties, and efficacy in substance use comorbidities suggests that clozapine exhibits strong mood-stabilizing properties [19]. CBT is recommended for the prevention of depressive or manic episodes and increasing treatment compliance of the pa-

tients. Although studies have not reached the best point targeted in BD, CBT is one of the approaches that will provide the clearest contribution to the goal of relieving the suffering of patients and improving their lives [20]. Family therapy in BD will lower the patient's risk of recurrences and functional psychosocial impairment, improve health, and reduce and optimize socioeconomic resources [21]. For the SLE, we treated the acute symptoms and then controlled the autoimmune disease by giving steroids and hydroxychloroquine. As we know, BD might be a consequence of autoimmune reactions affecting the brain, and the use of considering steroids might decrease the inflammation, resulting in lower BD incidence [5]. According to literature data, hydroxychloroquine has an excellent response to SLE treatment in a patient with BD; it is not like the usage of chloroquine, which was known to be a precipitating factor for exacerbations of both depressive and manic episodes [22].

Conclusions

Bipolar disease may be associated with Systemic Lupus Erythematosus. Psychosis features generally occur in BD, usually in the manic period. Treatment should be done for both psychiatric and internal medicine problems. As the SLE symptom was controlled, the BD symptom improved.

Acknowledgments

Not declaration

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