Bipolar Disorder Recurrence in a Nonagenarian: An Uncommon and Unfortunate Health Concern for a Senior

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ABSTRACT
Bipolar Disorder (BD) may affect people of all ages. Although it was once believed that bipolar symptoms slowly disappear with age, the studies showed that the truth is not so! Untreated BD tends to worsen over time. On the other hand, people who are initially diagnosed with BD late in life may well have had unnoticed BD for decades. As populations grow older, the number of BD cases among the seniors is expected to increase. In late-onset BD, etiopathology may differ from young BD patients. Cardiovascular diseases and some other biological mechanisms may play a significant role in the onset of disease. In this case report, we present a nonagenarian admitted with recurrent BD symptoms after twenty-four years of the symptom-free period. We aimed to discuss possible mechanisms and effective treatment methods of geriatric BD in light of current literature. This is the first case of geriatric BD recurrence reported after such a prolonged symptom-free period of a nonagenarian senior.

Keywords: Bipolar disorder, nonagenarian, recurrence, treatment

INTRODUCTION
Bipolar disorder (BD) is a mood disorder that is defined by recurrent episodes of mania, hypomania, and major depression. It is usually encountered in young adults. Although BD is rarely seen in elderly patients, it still constitutes 8 to 10% of all psychiatric admissions among patients of advanced age (1). The definition of geriatric BD is used for patients older than fifty years of age (2,3). The knowledge about characteristics of BD among elderly is insufficient, and prevalence is about 7 to 25% of all bipolar patients (4). The BD affects approximately 0.1% of the population over the age of sixty-five (5). However, the number of geriatric BD patients is expected to increase due to progressive aging of the world population through forthcoming consecutive decades (6).

BD is characterized by its repetitive nature that commonly occurs within two years of remission among nearly fifty percent of patients (7). These episodes trigger a therapeutical resistance which further causes a tendency for suicide attempts by deteriorating cognitive, social and functional ability (8,9). Although the recurrences are often seen within following years after the initial episode, recurrences may also occur long after treatment of the previous attack (10). However, it is very unusual to experience a BD recurrence in a geriatric patient after an extended period of remission. In this paper, a case of BD recurrence in a nonagenarian after twenty-four years of remission is reported. According to the available literature, this is the first case of geriatric BD recurrence after such a prolonged symptom-free period of a nonagenarian senior.
CASE PRESENTATION

A 97-year-old male patient admitted to the psychiatry outpatient clinic with symptoms of increased energy and anger associated with incoherent talkativeness. According to history taken from his family, these symptoms started three weeks ago and got worse for the last ten days. The symptoms of reduced need for sleep and uncontrolled fear of getting harmed were lasting most of the daytime. In his past medical history, he had similar complaints firstly in 1994 which resulted in hospitalization for two months when he was seventy-three. The treatment with Lithium Carbonate (300 mg/day) for the next five years achieved full recovery with no recurrences or psychiatric complaints. There was no history of hypertension, diabetes mellitus, hyperlipidemia, head trauma, infection, seizure or cognitive dysfunction. Bilateral carotid artery lesions causing no impairment in cerebral perfusion were reported as medical history. According to social history, his wife passed away four years ago, and he started to live with his children.

The patient was hospitalized with the diagnosis of a BD recurrence according to these parameters. Psychiatric examination revealed unruly behavior, persecutory delusions, and functional disability. The patient was conscious, oriented but no cooperation was achieved. He was talking uninterruptedly and easily distracted. His affect was labile, and the mood was irritable. His thought content includes paranoid delusions. No hallucinations or obsessions were reported. The patient had no insight into his illness and no judgment. The intelligence and memory test (Mini-Mental State Examination-MMSE) score was 25 within reasonable limits (24-30). The Brief Psychiatric Rating Scale (BPRS) was 93 (range 0 to 108, 15-30: minor syndrome, ≥30: major syndrome), the Young Mania Rating Scale was 41 (range 0 to 44), and the Beck depression scale was 7 (0-9: normal). The physical and neurological examination revealed no pathology. Vital signs as well as biochemical tests, complete blood work, and thyroid function tests were within reasonable limits. The cranial magnetic resonance imaging (MRI) and computed tomography (CT) of the brain demonstrated no more than age-related atrophy. Bilateral carotid artery Doppler Ultrasound (DUS) revealed unimportant atheroma plaques (<30%) due to diffuse senile atherosclerosis causing no diminished cerebral perfusion. Acetylsalicylic acid (100 mg/day), Lithium Carbonate (300 mg/day) and Quetiapine Fumarate (12.5 mg/day and increased gradually up to 50 mg/day in two divided doses) were started. The symptoms were subsided in three weeks after this medical treatment. No drug-related side effects were observed. The patient was discharged with full recovery (BPRS: 5, YMRS: 0, Beck Depression Scale: 3). Lithium Carbonate (300 mg/day) was continued as a prophylactic treatment for preventing further episodes as well as cognitive decline. The follow-up visits were revealed no BD symptoms.

DISCUSSION

In this case report, we report a nonagenarian with the recurrence of BD after remission of twenty-four years. According to the available literature, this is the first case of geriatric BD recurrence reported after an extended symptom-free period of a nonagenarian senior.

The International Society for Bipolar Disorders (ISBD) Task Force on Older-Age Bipolar Disorder suggests that older-age BD covers individuals above fifty (3). The elderly patients whose episode presented earlier in life and the patients whose episode arise for the first time in later life both constitutes a geriatric BD group (3,11). The term "geriatric BD" has replaced the previous term "older-age BD" according to ISBD Task Force (3). The calculated one-year prevalence of BD in sixty-five or older varies from 0.1 to 0.7%. The estimated lifespan rate is 1 to 2% for the same age group (12). Geriatric BD patients are mostly women (1). Although etiology is unknown, it may happen due to cerebrovascular lesions or biologic factors such as treatment with steroids for comorbid medical illnesses (arthritis, asthma), stressful life events (marital divorce or death) as well as changes in daily life, family role or economic status (13). The vascular mania hypothesis which links an association between mood disorders with cerebrovascular diseases seems to be most reasonable pathophysiological hypothesis (14). Late-onset BD has a strong association with vasculopathy-
related white matter hyperintensities (WMH) in older adults which were demonstrated on neuroimaging (15). On the other hand, WMH which are characterized as a result of vascular perfusion deficits may solely indicate causality rather than a direct association (16). However, post-stroke mania which is strongly associated with right hemispheric strokes involving limbic, limbic-related cortical (orbitofrontal and basotemporal) or subcortical (caudate nucleus and thalamus) regions has also been reported in some cases (17). As bilateral carotid artery DUS examination and neuroimaging with CT and MRI revealed no signs of cerebral hypoperfusion, infarct or ischemia, vasculopathy-related etiopathology was thus excluded in this patient. Recurrent mood episodes in geriatric BD can also be triggered by biologic factors such as treatment with certain medications (18). Pharmacological models thus suggest a probable role of an increased dopaminergic drive for mania and reverse action in depression. For example, in Parkinson’s disease, administration of dopaminergic precursors in high doses can cause state like a manic episode, which then switches conversely into a depressive mood after an instant withdrawal. It is also possible that in BD there is a cyclical nature. The high dopaminergic transmission in mania may result in subsequent down-regulation of dopaminergic receptor activity over time corresponding to the depressive phase (19). This cyclic nature of BD may be the only etiopathology in this patient.

The clinical characteristics of older age BD are distinct from younger patients. Cognitive dysfunction and comorbid medical diseases are more common in older patients than in young. Extreme sexual interest and behavior during manic or hypomanic episodes, comorbid anxiety, and substance use disorders appear to be less frequent in older age group (13). Geriatric BD patients have more general medical conditions than younger patients. The most common comorbid illnesses are cardiovascular disease, arthritis, diabetes mellitus, hypothyroidism, hypertension, and metabolic syndrome (3).

Early-onset BD and late-onset BD groups are described in geriatric BD. Early-onset BD represents patients under fifty years of age. In this group, the depressive state begins during middle-ages, and it is followed by recurrent episodes which finally convert into manic episodes during late-life. The prevalence of BD in first-degree relatives of this early-onset group is higher than the general population. In late-onset BD, the onset of disorder occurs after fifty years of age. It is described as “secondary mania” in which BD begins initially and mildly during late-life. This group has a higher prevalence of comorbid neurological disorders (dementia, neurological and cerebrovascular conditions) and a comparatively low genetic tendency for BD (5). The secondary mania is first described by Krauthammer and Klerman as a syndrome connected with multiple associations. These include drug usage (steroids), metabolic, infectious and organic brain pathologies (stroke or tumor) (20). Late-onset BD patients tend to achieve faster cure and early discharge rates (14).

Pharmacotherapy for geriatric BD needs to be more precise to prevent drug-related side effects. Therefore, drug doses should be titrated slowly to reach the aimed final dose in seven days (21). In this patient, Quetiapine Fumarate monotherapy was started with 12.5 mg/day, which then gradually increased to 50 mg/day in conjunction with the rule of "start low and go slow." After symptoms were reduced Lithium Carbonate (300 mg/day) was combined as a prophylactic treatment.

In conclusion, geriatric BD and BD in the younger population are two separate etiological entities sharing a common anatomical pathway. Vascular depression and atherosclerotic changes resulting in hypoperfusion of brain most probably associate with late-life BD as etiopathogenesis. If this vascular mania hypothesis would be considered as the valid etiopathological mechanism for geriatric BD, the entire cardiovascular system should be examined cap-a-pie to rule out any possible overlooked outcomes. The other important point is that lithium should not be discontinued in these patients as it can sustain the cognitive abilities in more elderly population by preventing cognitive deterioration associated with late-life BD.

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