Idiopathic Basal Ganglia Calcification with Bipolar Mood Disorder Presentation

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Fahr’s disease is a progressive and idiopathic basal ganglia calcification with normal metabolism of calcium and phosphor with motor and psychiatric signs and symptoms. Dementia, chorea athetosis, psychosis and depression due to Fahr’s disease are frequently reported, but Fahr’s disease with bipolar mood disorder manifestation is very rare and we found only 3 case in review of literature from 1955 to 2005. In this case report, a 21-year old girl is presented who was admitted to Sari-Zare psychiatric hospital for aggression, restlessness and insomnia. After mental status examination and Para clinical investigation, bipolar disorder due to Fahr’s disease was detected. To date no specific treatment was found for this disease. This point is important that the patients with Fahr’s disease are sensitive to neuroleptic malignant syndrome.

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Introduction

Fahr’s disease, first described by Karl Theodore Fahr in 1930, refers to sporadic or familial idiopathic basal ganglia calcification that is associated with many neurological and psychiatric abnormalities (1). Patients with Fahr’s disease often appear with movement disorders, such as parkinsonism, paresis, dystonia and speech impairment. Other neurological features can include stroke-like events, often combined with psychiatric conditions, such as psychosis, mood disorders, and dementia (2). Although Fahr’s disease appears most commonly with motor deficits, about 40% of the patients with Fahr’s disease are seen with primarily cognitive and other psychiatric findings (3). Organic mood disorder has been reported less often, and mania secondary to idiopathic basal ganglia calcification has been noted rarely (4). The definition requires bilateral calcifications with neuropsychiatry and extra pyramidal disorders attended by normal calcium and phosphorus metabolism CT-Scan is an easy exam and has greater diagnostic specificity for basal ganglia calcification. About 40% of patients with BGC present initially with psychiatric features (5). Cognitive, psychotic, and mood disorders are common. Clinical features are important because BGC may be viewed as an incidental finding. Headache, vertigo, movement disorders, paresis, stroke like events, cognitive impairment, psychiatric disorders, pyramidal signals and seizures are the most common manifestations (5,6). Symptomatic features may change over time (7,8).

Case presentation

A 21-year-old girl was admitted in Zare-psychiatric hospital with 2-month history of insomnia, irritability, restlessness and ritualistic behavior. In previous psychiatric history, she had two episodes of illness, the first, in 3 years ago she suffered from depress mood, insomnia, decreased appetite that was be changed to the second episode with manic presentation and she was admitted in a psychiatric hospital. During the mental statuses examination, psychomotor agitation, pressure of speech, irritable mood and appropriate affect, poor concentration and obsessive thinking were be detected. In laboratory investigation, serum concentration of calcium and phosphorus, are within normal limits. Routine hematological and biochemical investigations, as well as workup for metabolic,
inflammatory, and infectious conditions, do not disclose specific abnormalities. In computed tomographic scan, bilateral calcification of basal ganglia was detected (fig 1).

**Discussion**

Mood disorders include mania and depression are the most common behavioral changes in BGC, initially present in one-fifth, eventually occurring in about two-thirds, and occurring more commonly than in neuropsychiatry control subjects (9). Of 58 patients, mania eventually occurred in up to 31% (10). Perhaps up to one-half of patients develop depression at some point over the illness course. One study found DSM-III depressive disorders in 37% (5), and another found DSM-III-R minor depressive symptomatology in 16.7% (10). More extensive calcification and subarachnoid space dilatation correlate with the presence of psychiatric manifestations (5, 10, 11), but calcify distribution and etiology does not (5). Both the symptoms of mood disorders and biological research findings support the hypothesis that mood disorders involve pathology of the limbic system, the basal ganglia, and the hypothalamus. The limbic system and the basal ganglia are intimately connected, and the limbic system may well play a major role in the production of emotion. Structural brain imaging studies support the notion that mood disorders are associated with regional structural brain abnormalities; in particular regions involved in mood regulation, such as the limbic portion of the basal ganglia and brainstem structures (12).

**References**


**Fig 1** : Bilateral calcification of basal ganglia in the CT Scan


