

CASE REPORT

FRONTAL LOBE ABNORMALITY AND PSYCHOSIS IN TRAUMATIC BRAIN INJURY AND CANNABIS ABUSE

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Abstract

Objective: Traumatic brain injury (TBI) and Cannabis has been implicated as a risk factor for the development of psychosis. There is lacked of literature on the link between risk factors such as brain injury and cannabis use in development of schizophrenia. **Method:** We reported a case with history of traumatic brain injury, who later developed schizophrenia like symptoms soon after initiation of cannabis uses. Further the symptoms persisted despite abstinence on cannabis but flared up after relapse of cannabis used. **Results:** Present case helps to understand common neurobiological mechanism behind schizophrenia and risk factors such as TBI and Cannabis abuse. **Conclusion:** Researches in this area are important for management and further understanding neurobiology of schizophrenia. *ASEAN Journal of Psychiatry, Vol. 18 (1): January – June 2017: XX XX.*

Keywords: Schizophrenia, Traumatic Brain Injury, Marijuana Abuse, Neuropsychiatry

Introduction

Traumatic brain injuries (TBI) are a worldwide public health problem and known as “silent epidemic.” It increases the risk of several neuropsychiatric problems such as mood disorders, anxiety disorders. Substance use disorders, psychosis, personality change, cognitive impairment, etc. TBI had been implicated as a risk factor for the development of schizophrenia-like symptoms for many decades but there is very little systematic research [1]. Although substance abuse is a well-known risk factor for TBI, recent literature reported its bidirectional association [2]. Cannabis use is most widely available and abused illicit substance in India [3]. Further, cannabis is also a well-known risk factor for development of schizophrenia. In this case, we found that patient with a long history of traumatic brain injury was vulnerable for development of schizophrenia-like symptoms soon after the initiation of cannabis use.

Case Report

Mr. A, an 18-year-old young adult male brought into the psychiatry OPD with complaints of cannabis use, disturbed sleep, hearing of voice, fearfulness, abnormal agitated behaviour, since three years ago with current exacerbation of symptoms since the past three months. Patient started using bhang at the age of 15 years old in 2013 when he was studying in 9th standard. He started taking bhang with his friend out of curiosity. He was told by his friends about appetite stimulating property of bhang and will make his physical stronger. Considering this, he started using bhang at the average dose of one ball (about 50 gram) 2 to three times a day regularly for about 10 – 15 days. He discontinued bhang and shifted to ganja (smoking form of cannabis) to get more high along with the average dose about two balls/day. After about one month of daily use of ganja, he started experiencing auditory hallucination of his old childhood friend who died within 10 years

back. This voice would be of commenting and commanding type; he continued hearing voices at every 2-3-day interval, lasting for about 10-15 seconds, 3-4 times/day, more during night times. He quit the cannabis after treatment but even after two months of quitting ganja, he developed symptoms as decreased need for sleep, elevated psychomotor activity, elated mood with intermittent lability, elevated self-esteem and authoritative. He would be found singing songs loudly and worshipping God "Bajarang Bali" excessively for about 4-5hours/day. He developed ideas of grandiosity and would claim to own crores of rupees and often expressed his wishes to become the richest person in the world. He also developed delusion of reference that person around him was talking about him due to which he would often become abusive and assault towards strangers. Auditory hallucinations also increased in frequency occurring about 15-20 times a day, every day but more during the night before sleeping. After about 15 days of onset of manic symptoms, he was advised Olanzapine 10 mg tablets by a psychiatrist. Gradually, his manic symptoms improved completely in 3 months and he discontinued medications by himself in about 6 months of starting. He kept on experiencing hallucinations but at previous frequency. About 1 year later, at the age of 16 he developed similar episode for 2 – 3 months which improved after the same treatment and he discontinued treatment after 6 months. About 1 year later, at age 17, he restarted smoking ganja regularly with an average dose of 1 ball (approx.. 50g) per day. After about 4

months of regular use, he developed delusion of made volition and somatic passivity as somebody holding his head from behind and turning around. He would also experience visual hallucination as black terrifying man with long hairs and red eyes trying to hit him with a bamboo stick. He would be agitated, fearful and assault in order to protect himself. Auditory hallucinations of his dead friend also increased in frequency and severity. He decreased ganja use from daily to 4-6 times per month. He visited our Institute after three months of current exacerbation and was started on tablet olanzapine, and dose was increased to 15 mg with complete remission of symptoms for three months of treatment.

History suggested patient had a traumatic brain injury at the age of 3 in the year 2001 after fell down from the terrace of his home. After falling down, he developed approximately 30 minutes of unconsciousness and an open wound at the frontal region of the scalp. His CT Scan Brain revealed a fracture of frontal bone with underlying bilateral frontal lobe involvement (Figure 1). He underwent surgical repairing of wound and thereafter doing well in his life.

There was no history of psychiatric illness among his family. Birth history and early development were normal. He was an intelligent student; his schooling performance was normal and would rank first in class until 8th standard. His behaviour was the good. Therefore, often family member and others would appreciate him for his good behaviour.

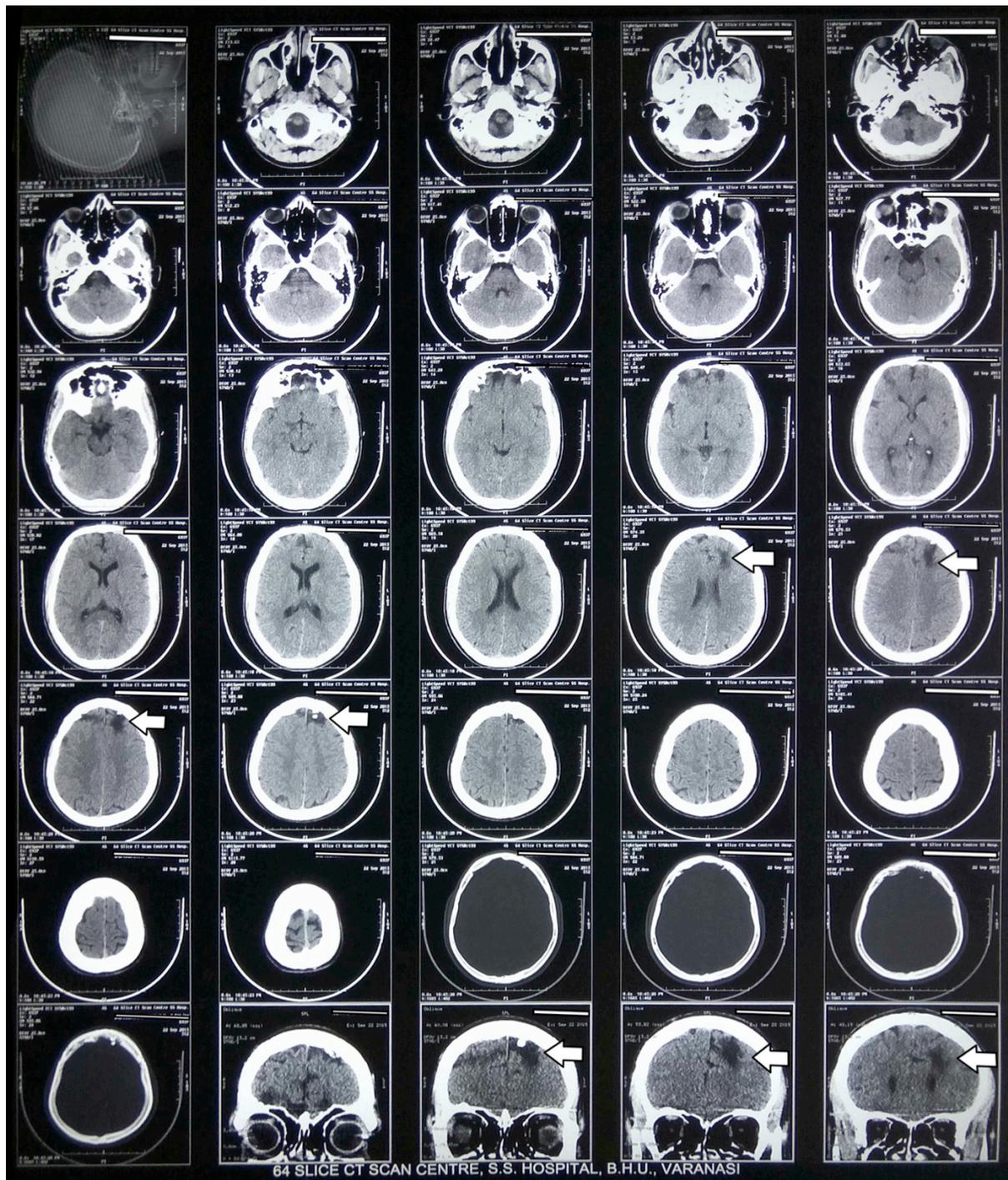


Figure 1. Computed tomography (CT) scan brain after Traumatic Head Injury (TBI) showing bilateral frontal lobe involvement

Discussion

In present case, we found that patient with a 15-year-old frontal lobe lesion developed schizophrenia symptoms after one month of cannabis use, which persisted even after quitting cannabis. Rather he developed additional two manic episode months later. Manic symptoms subsided soon after

treatment with olanzapine but schizophrenia symptom persisted and further worsened after re-initiation cannabis use. Negative family history suggested lower probability of genetic vulnerability to illness, hence we hypothesized that TBI in childhood with frontal lobe involvement took the important role in predisposition to psychopathology. Available literature has reported the risk of

schizophrenia among TBI patients was three times higher than healthy cohort [4]. Frontal, temporal lobe and left hemisphere involvement were important predictors in development of psychosis after TBI [4]. Psychosis and cognitive changes occur after TBI due to hypo cholinergic state created by involvement of cholinergic fibers in fronto-temporal regions. Hypocholinergic state further predisposes patients for smoking nicotine as self-medication. Acetylcholinesterase Inhibitor such as Galantamine has been found as effective adjuvant for treatment of psychosis in TBI [4]. Further, white matter (WM) changes in the fronto-temporal areas have been found among cannabis using patients with schizophrenia, suggesting that left fronto-temporal disconnectivity may contribute to pathophysiology of schizophrenia [5,6]. The overall findings suggest that the severity of WM changes mainly in fronto-temporal area increases vulnerability for development of severe psychotic symptoms in individual. To our best knowledge, present case is the first to report possible neurobiological link between TBI and cannabis use and occurrence of psychosis.

Conclusion

Cannabis users with TBI in the past are more vulnerable to development of schizophrenia-like symptoms due to underlying common neurological involvement affecting fronto-temporal areas.

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Received: 1 November 2016

Accepted: 17 February 2017