



Case Report

Copy Right@ Armand Ntchana

A Case of Brief Psychotic Disorder: An Unintended Consequence of COVID-19 Virus

Armand Ntchana*

Oceania University of Medicine, USA

*Corresponding author: Armand Ntchana, Oceania University of Medicine, USA.

To Cite This Article: Armand Ntchana. A Case of Brief Psychotic Disorder: An Unintended Consequence of COVID-19 Virus. *Am J Biomed Sci & Res.* 2022 - 16(3). *AJBSR.MS.ID.002239*. DOI: [10.34297/AJBSR.2022.16.002239](https://doi.org/10.34297/AJBSR.2022.16.002239)

Received: 📅 May 04, 2022; Published: 📅 June 03, 2022

Abstract

Brief psychotic disorder is a sudden onset of psychosis in a patient for a duration of more than a day and less than a month. The DSM-5 defines brief psychotic disorder as a psychotic condition involving the sudden onset of at least one psychotic symptoms such as delusions, hallucinations, disorganized thought processes, disorganized speech and/or behaviors. This condition can also be associated with impaired memory of recent events, screaming, mutism, outlandish dressing, or behaviors. Acute psychotic symptoms can be associated with emotional lability often triggered by substance abuse or severe and acute stress such as the loss of a loved one. Patients ultimately receive the diagnosis of schizophrenia if the symptoms persist for a duration longer than six months. Brief psychotic disorder is often diagnosed in teenage patients, but it is less commonly seen in the females. Although the exact pathophysiology of brief psychotic disorder has not been determined, historically and similarly to schizophrenia it has been strongly linked to familial history. In some cases, it has been preceded by a traumatic and stressful event. Brief psychotic disorder has also been theorized to be the cause of maladaptive defense mechanism to escape a stressor. In this case report, we present a fifteen-year-old female patient who abruptly lost her mother to COVID-19, her adaptation few months following the loss and her eventual reactive psychosis as well as management challenges.

Introduction

Brief psychotic disorder is an acute condition with heterogenous clinical presentations. This condition is often seen in adolescents. According to the DSM-5, it is a transient condition of short duration, but it can be recurring. Gross deficit in reality testing is the hallmark of this condition often with the presence of delusions and/or hallucinations. The condition can be reversible with treatment. However, in many cases symptoms may persist beyond the guidelines of this diagnosis as set forth by the DSM-5. It is more prevalent in females with a ratio of 2:1 and, according to Freeman and Fowler [1], it is more likely to occur in adolescents and adults between ages twenty to thirty. Research shows that the duration of this illness, presentation, severity, and prognosis varies based on age of onset. Kao & Liu [2] found that the age of onset

is significantly related to the cognitive elements of the Positive and Negative Syndrome Scale (PANSS) and Barratt Impulsiveness Scale (BIS) score. In particular, their research demonstrated that patients with psychotic spectrum disorder with earlier onset had significantly greater cognitive impairments. In view of these findings, we will explore the effects of the traumatic event on a susceptible young female brain, the clinical challenges to treatment, and explore an early intervention that could have potentially prevented the condition.

Case Report

Patient in this case is a fifteen-year-old Hispanic female, admitted to an intensive inpatient psychiatric program due to acute onset of suicide ideation with psychotic symptoms including



delusions and severe paranoid thoughts. She was alert and oriented to person and place at the time of admission and denied any homicidal ideations. Prior to the admission, patient had not slept for three consecutive days. Patient's sister with whom she lived with reported noticing some "strange behaviors" such as increase aggression toward her children, severe confusion, and persecutory delusions. Her concerns led her to monitor patient's sleep more closely. On the third day of absolutely no sleep despite using over-the-counter remedies, the sister took patient to the hospital. On initial admission patient presented with a broad range of affects. She was very talkative with flight of ideas, disorganized and tangential thought process. Multiple persecutory delusions and visual hallucinations were present. Patient had no insight in her condition, was extremely distrusting of everyone without any ability to process information or discern reality from fictional intrusive thought. On admission, patient tested positive for tetrahydrocannabinol (THC). She presented with very labile mood, she was quiet at time and cooperative even though her account of events was incoherent. Later, she was pacing the halls of the unit, shouting, and screaming about her distrust of people, "I don't know if I'm safe, I'm paranoid, maybe autistic no one will tell me the truth, I can't trust anyone" and "I don't have my social security number, I can't find it, it's with my passport". Few days later during history taking, we were able to gather that while enroute for the hospital with her sister, they stopped at the eldest sister's house to pick up her passport and social security card, thus the random shouting about the social security number. This pattern of behavior became noticeable as days went by. Patient would recollect some information or hear a comment which she would later relay in a different nonsensical context and sometimes mixing different stories as if she was trying to make two pieces of different puzzles fit. Patient also talked about her mother's illness with COVID-19 constantly, and her best friend K. who, according to patient, was an undocumented immigrant who had been trying to steal her identity, betrayed her and made her do things she did not want to do. We wondered at the time if this K person was even real based on some outlandish allegation's patient was making. As it turns out, K was real and some of the allegation had been verified to be accurate. Her confabulations gradually escalated for few days. One striking example was noted when patient asked for a cup to drink tap water on the unit on one day. The nurses would educate the patient about the importance of drinking filtered water instead. Later that day, when questioned by the treating physician about other incendiary allegations or behaviors from the previous night, the patient would respond by discussing how tap water was so insanitary for one's health. It appears that unconsciously with her psychosis, patient's brain had developed maladaptive coping mechanism. Patient would simply ignore some questions, especially if they related to her previous misbehaviors on the unit and would instead respond

or ask some utterly unrelated question, thus her incoherence and disorganization.

Psychiatric History

Patient had no prior history of psychiatric illness or hospitalization for any psychiatric condition. However, she had a prior history of recreational cannabis use but she denied smoking cigarettes, consuming alcohol, or doing any other illicit drugs. Her family reported awareness that patient had used cannabis recreationally a few times over the past five years. Her family described her as a healthy, smart, and kind adolescent who did well in school and never got into any troubles. Patient has three siblings, two older sisters and one older brother who has a history of substance abuse and suffered from depression. He was hospitalized in 2014 for major depressive disorder with suicidal ideation. Her father lives and works mostly out of state. He has a history of alcoholism. Patient's mother passed away from COVID-19 five months ago. Patient was living with her mother and her brother before she passed. Patient's family described her as a "good kid who did well in school". Patient has a history of smoking cannabis recreationally with her friends. Her eldest sister and her father both reported some drastic changes in the patient's behavior since her mother's passing. They both reported that patient appeared emotionally blunted after her mother passed away. The sister noted that she thought it was weird that patient was not grieving. According to patient's eldest sister, patient had similar reaction in 2017 when their grandmother passed away, which was surprising to patient as she was very close to the grandmother. The eldest sister further added that the patient reacted "weirdly" given that the grandmother raised her. In 2018, patient's cousin committed suicide by gunshot and left a letter in which he apologized for his actions. The cousin had a history of severe depression for which he was treated briefly but unsuccessfully. Furthermore, the review of the family history revealed that several of its members suffered from various mental disorders. Specifically, patient's maternal grandmother suffered from schizophrenia and patient's maternal great-aunt suffered from bipolar disorder. Patient's paternal cousin committed suicide in 2006. Patient's paternal uncle has a history of severe depression and schizophrenia.

Treatment challenges

Patient was initially treated for her depressive and anxiety symptoms with sertraline 50mg daily. Aripiprazole 2.5mg was added for her psychotic symptoms. Patient showed no improvement of her psychotic symptoms with the prescribed medication. Her paranoid thoughts were escalating and affecting others on the unit. Patient alleged that she was raped overnight by one of the providers, she was pregnant and was obsessing about taking a pregnancy test. She believed people were out to get her and they were trying to give her medication to poison her.

Aripiprazole was then increased to 5mg twice a day with no improvement. The night dose was then increased to 7mg then to 10mg after no improvement was noted or reported. Unfortunately, the treatment was still not effective as patient continued to display very paranoid with gross deficit in reality testing. Patient's sleep had also worsened as she was sleeping less than two hours at night. She reported having her menses that morning, which was not accurate. When questioned about her alleged pregnancy patient stated, "it's impossible, I'm a virgin". Patient's short-term memory showed multiple deficits. She did not recall if she had had breakfast that morning or if she had spoken to her family the night before. She was not able to recall recent memories in the proper context and would instead retrieve various information and create connections and associations most time out of context.

Aripiprazole seemed to be a good first option for our medication naïve patient as it historically has fewer side effects as compared to other atypical antipsychotics. As reported by Komossa et al. [3], aripiprazole is less effective in treating primary psychotic disorders, like schizophrenia, than another second-generation antipsychotic. However, aripiprazole has fewer metabolic and sedative adverse effects. It is also associated with less dystonia as compared to other atypical antipsychotics with usually a smaller increase in prolactin as well as QTc prolongation. Aripiprazole is approved for the treatment of psychosis related illnesses, in addition, it is also used to treat depression, which initially made this medication an ideal choice. Unfortunately, the patient showed no improvement of her psychosis on aripiprazole for three consecutive days. Her detachment to reality was escalating with constantly changing and more outlandish stories. Patient would at time question the clinicians to the truthfulness and validity of her stories as a measure of reality testing.

On the fourth day of admission, we decided to change medication and proceed more aggressively with treatment. Patient had not developed any adverse reaction or side effects. Patient was started on olanzapine 5mg twice daily with trazadone 100mg at bedtime for sleep support and sertraline 50mg daily for her anxiety and depressive symptoms. Due to the severity of her symptoms and her lack of sleep, which was exacerbating her psychosis, patient could not participate in other treatment modalities. Shortly after treatment initiation, patient displayed some psychomotor retardation, with slower and calm tone of voice. She was most likely benefiting from the sedative effect of olanzapine. For the first time she slept more than six hours which was an improvement from less than two hours nights prior. As we continued to monitor the patient closely on the unit for the next four days, we noted a drastic improvement of her anxiety and sleep pattern and a slower improvement of her psychotic symptoms. On day eight,

she appeared rested, calm, and much less confused. She was self-aware for the very first time since her admission and she started making coherent sentences without outlandish allegations. Patient was able to recognize that she was very paranoid and anxious for the past few days and spoke clearly to her level of confusion which was ameliorating. Her display of gratitude to the staff for being patient with her and trying to make her better showed appropriate executive functions and insight; something that was not seen since admission eight days prior.

Discussion

This case of brief psychotic disorder in a patient with a pervasive family history of psychiatric illnesses begs the question of etiology. Given the positive THC test on admission, one might immediately conclude that this is a simple case of cannabis induced psychosis. However, a closer look at the family history in collateral information makes us wonder if there might be more to this case. Is this a case of early onset childhood schizophrenia or a case of acute psychosis outlasting a period of acute intoxication? Better yet, are we possibly dealing with a case of complicated or delayed grieving, or a case of psychosis induced trauma?

Early onset childhood schizophrenia is rare and present with potentially more severe symptoms and worst long-term outcome as discussed by Kyriakopoulos and Frangou [4]. This case does not appear to have an insidious onset. One might wonder if this is a case of cannabis induced psychosis with a positive test for THC on admission. However, patient prior history of use and use pattern would eliminate this theory. Cannabis induced psychosis (CIP) is usually seen in patients with chronic and heavy use. Grewal and George [5], found that symptoms of a primary psychotic disorder overlap with symptoms of CIP, however, in CIP patients have more mood symptoms. The risk of CIP is associated with longer exposure, duration and severity is typically dose dependent. A diagnosis of primary psychosis such as brief psychotic disorder is given in the absence of heavy cannabis use or withdrawal as noted by Grewal and George [5]. At the same time, there have been recent reports linking the surge of acute psychotic disorders to the increased use of newer and synthetic cannabinoids usually sold as Spice or K2. According to Wilkinson, et al. [6], these synthetic drugs are more potent CB1 agonists than THC. CB1 function is the modulation of gamma-aminobutyric acid (GABA) and glutamate, both suppressive neurotransmitters present in large amounts in the cerebral cortex and hippocampus in the psychoactive brain. The link between cannabis uses and acute psychosis or transient changes in behaviors have been seen in practice and well documented. Wilkinson, et al. [6], further reported that in both cannabis use and synthetic cannabinoid products a full range of positive and

negative symptoms have been seen. These are consistent with the psychotogenic effects of cannabis. Positive symptoms observed in our case included delusions, fragmented thinking, perceptual alteration suspiciousness. Other positive symptoms can include grandiose delusions, hallucinations. As for negative symptoms in our case, patient presented with psychomotor retardation, flattening of affect, emotional withdrawal, cognitive impairments. Other negative symptoms may include short-term memory loss, poor attention, and lack of spontaneity. Acute psychotic symptoms, however, appear commonly during the period of intoxication with a severity proportional to the amount of toxins present. In contrast, in our case patient had not been exposed to any illicit substances for at least three days prior to admission. Because patient's psychosis continued to persist and increased for a week into admission, we can rule out the possibility of a simple drug induced psychosis. The concentration of the toxin in question in an acute intoxication would have gradually decreased as well as corresponding symptoms. Our case presentation did not reflect the pathophysiology of cannabis induces psychosis.

It is important to note that although we believe cannabis exposure not to be the etiology of this patient presentation, we at the same time cannot rule out its contribution to the genesis of a primary psychotic disorder. A reasonable alternative consideration would be acute psychosis outlasting the period of intoxication. We can hypothesize that a patient early and chronic use of a substance that causes micro insults to their developing brain could trigger an acute psychosis due to the compounding effect of such insult. Yet, our patient's history and duration of drug use are not consistent with this premise. Wilkinson, et al. [6], further reported that early exposure to cannabinoid products has also been linked with increased risk of psychosis with a decline in risk when exposure occurs later in adolescents or adults. Hence, patients who use cannabis at an earlier age are at a higher risk not only for developing acute psychosis, but also for reoccurrences of such symptoms later in their lives. Also, such patients are more likely to develop schizophrenia. An array of such interrelated factors as the severity of the patient symptoms in this case, strong family history of primary psychotic disorders, patient's preoccupation about her mother's death, and inappropriate response to such trauma, leads us to explore other plausible etiology. Although an acute psychosis outlasting the period of intoxication appears to be a good possible etiology for this presentation, the patient's pattern of drug use does not appear to match her presentation. Although there is growing clinical evidence on the positive association between cannabis exposure and acute primary psychotic disorders, Wilkinson, et al. [6], posited that the claim of direct causality remains controversial. A stronger causality however exists between acute psychosis and trauma. According to Buckley, et al [7], The lifetime prevalence rates

of PTSD in the general population is estimated at seven-point eight percent (7.8%) compared to 30% in patients already diagnosed with a psychotic disorder. Because many traumata and PTSD do not get reported or are not diagnosed, it is fair to assume that the number of patients with PTSD and psychosis could be substantially higher than thirty (30%). Although the link between trauma and psychosis has been established in multiple patients accounts and literatures, the exact etiology is not well understood and appear to be more complex and multifactorial. Some of the pathways proposed by Varese, et al. [8], include psychosis as a result of childhood adversity, trauma as a result of psychotic symptoms or involuntary treatment experiences, psychosis as a dimension of PTSD resulting from trauma as well as re-traumatization as stressors that can worsened the course of a psychotic disorder.

Grief, also referred as bereavement, is a normal emotional response a patient displays after a traumatic event, for example, the loss of a loved one. According to DSM-5, patients grieving over the loss of a loved one may have a difficult time and may show symptoms like a major depressive episode. Patients may present with depressed mood, feelings of guilt as it relates to the loss of the loved one, feeling worthless, impaired psychomotor skills, lack of pleasure in activities, dysregulated sleep and even hallucinations. Grieving patient will experience the intensity of their symptoms decrease overtime. Although there is no specific time to delineate when normal grief becomes complicated grief, we can see that in complicated grief patient might not show a decrease of their symptoms overtime. Grief may also interfere with their day-to-day functioning with intense depressive symptoms, difficulties accepting the death, self-destructive behaviors, suicidal thoughts. As the name implies, delayed grief involves symptoms of grief that appear later after the loss of a loved one. Delayed grief has been described by some patients as severe and overwhelming sadness felt suddenly weeks to months after the loss of a loved one. Although hallucinations can be present in grief, mood dysregulation remains the most prominent feature either with normal, delayed, or complicated grief. In our case, patient did not display mood reactivity related to the loss of her mother. She presented with more psychotic features with severe and constant paranoid thought process with gross deficit in reality testing. Our patient appears to have developed a primary psychotic disorder also called brief reactive psychosis with symptoms manifesting shortly after a traumatic event. In our case, the earliest identifiable but very subtle signs appeared five months prior, related to a severe and traumatic event in our patient's life; the loss of her mother to COVID-19. Subtle changes in the patient's mood, and most importantly, her lack of grieving can be shown to be the trigger of her primary psychotic disorder. These issues taken together raise the question of whether given the patient predisposition to psychiatric illnesses, treating

patient's perceived dysfunctional grief earlier on could have prevented the development of this primary psychiatric disorder?

A concept that has been studied in the context of post-traumatic stress disorder (PTSD). According to Freedman, et al. [9], cognitive-behavioral therapy (CBT) is the best therapeutic modality to be used and have been shown to be an effective modality in treating patients with PTSD. Studies have shown that PTSD can be prevented when CBT is delivered earlier, shortly after the traumatic event or insult. Freedman, et al. [9]. Providing effective therapeutic interventions during the post exposure period can have lasting effects with patients remaining asymptomatic for up to four years. Freedman, et al. [9].

Conclusion

Cognitive behavioral therapy (CBT) has been used for many years in the treatment and management of PTSD. Multiple studies have shown its effectiveness in not only reducing the severity of PTSD symptoms in some cases prevent their occurrence. According to the review by Kar [10], CBT has been shown to be not only safe but also an effective intervention when treating adults, adolescents, and children with acute and chronic PTSD. In some studies CBT has also been shown to be effective in preventing PTSD when administered sooner after a traumatic event. Freedman et al. [9]. Based on this clinical evidence, we can apply the same concept to primary psychotic disorders triggered by a traumatic event and allege that early intervention in our case could have possibly prevented the development of this condition. Increased awareness would be imperative in such circumstances to reduce incidence of primary psychotic diseases in predisposed families. Although many studies point to potential benefit for early CBT in preventing PTSD, Freedman et al [9] and possibly psychotic disorders, the lack of research prevents us to affirm or recommend early CBT as a treatment modality to prevent psychotic disorders. With the rise of the novel COVID-19 disease and its devastating effect on human lives and the U.S. health care system, we suspect that the case we presented is one of the earlier psychotic disorders diagnosed in the aftermath of COVID-19 devastation. With a considerable and growing number of individuals at risk, the need for more studies is warranted. Given the global health significance, CBT in the context of primary psychotic disorder should be rigorously evaluated.

The Pathophysiology of acute psychotic disorders is still not well understood. Regardless of the exact etiology, more research

in this area will be beneficial. Such research will help identify the factors that either mediate or moderate psychosis and based on improved knowledge to establish more effective preventative measures. Until then, it is critical for patients with a positive family history of schizophrenia or any psychotic disorders or episode of psychosis regardless of etiology to completely refrain from using any hallucinogenic substances. In addition, early treatment, debriefing or processing of stressors can help in preventing acute psychosis or chronic psychotic disorder like schizophrenia.

As our patient's primary psychotic disorder continues to improve, she will benefit from individual, group, and family therapy to facilitate her reintegration into society. Patient's maintenance medication dosage and her ability to return fully to baseline are yet to be determined, however her prognosis is encouraging three weeks after her initial evaluation.

References

1. Daniel Freeman, David Fowler (2009) Routes to psychotic symptoms: Trauma, anxiety and psychosis-like experiences. *Psychiatry Res* 169(2): 107-112.
2. Yu Chen Kao, Yia Ping Liu (2010) Effects of age of onset on clinical characteristics in schizophrenia spectrum disorders. *BMC Psychiatry* 10: 63.
3. Komossa Katja, Christine Rummel Kluge, Franziska Schmid, Heike Hunger, Sandra Schwarz, et al. (2009) Aripiprazole versus other atypical antipsychotics for schizophrenia. *Cochrane Database of Systematic Reviews* 4.
4. Marinos Kyriakopoulos, Sophia Frangou (2007) Pathophysiology of early onset schizophrenia. *Int Rev Psychiatry* 19(4): 315-324.
5. Grewal Ruby, Tony George P (2017) *Psychiatric Times* 34(7): 2-19.
6. Samuel Wilkinson T, Rajiv Radhakrishnan, Deepak Cyril Souza D (2014) Impact of cannabis use on the development of psychotic disorders. *Curr Addict Rep* 1(2): 115-128.
7. Peter Buckley F, Brian Miller J, Douglas Lehrer S, David Castle J (2009) Psychiatric comorbidities and schizophrenia. *Schizophr Bull* 35(2): 383-402.
8. Filippo Varese, Feikje Smeets, Marjan Drukker, Ritsaert Lieve, Tineke Lataster, et al. (2012) Childhood adversities increase the risk of psychosis: a meta-analysis of patient-control, prospective-and cross-sectional cohort studies. *Schizophr Bull* 38(4): 661-671.
9. Sara A Freedman, Ehud Dayan, Yael Bleich Kimelman, Heidi Weissman, Renana Eitan (2015) Early intervention for preventing posttraumatic stress disorder: An internet-based virtual reality treatment. *European Journal of Psychotraumatology* 6(1): 25608.
10. Kar, Nilamadhab (2011) Cognitive behavioral therapy for the treatment of post-traumatic stress disorder: A review. *Neuropsychiatr Dis Treat* 7(1): 167-181.