Chapter 19
Secondary Mania of HIV/AIDS

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Abstract Mania has for long been associated with HIV/AIDS and in many cases acts as a barrier to attaining best treatment outcomes in patients both conditions. Like many psychiatric disorders, mania was initially thought to be a psychological reaction to having HIV infection. Subsequent research has now proven that psychiatric disorders including mania could arise from the direct effects of HIV disease on the brain. Indeed research has shown secondary mania of HIV/AIDS to be different from primary mania in terms of clinical presentation, course, management and prognosis. This chapter deals with HIV mania and how it presents in the Ugandan setting. A case report helps to highlight these points. HIV mania and secondary mania of HIV/AIDS are used interchangeably in this chapter.

Keywords HIV/AIDS • Secondary mania • People living with HIV/AIDS (PLWHA)

Abbreviations

DSM IV TR	Diagnostic and Statistical Manual of Mental Disorders Fourth Edition Text Revised
GCS	Glascow Coma Score
HAART	Highly Active Antiretroviral Therapy
ART	Antiretroviral Therapy
HIV/AIDS	Human Immunodeficiency Virus/Acquired Immune Deficiency Syndrome
PLWHA	People Living with HIV/AIDS

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Introduction

Mania or manic episodes have been described for centuries. The earliest descriptions of mania can be found as far back as in the Old Testament texts of the Christian bible. In many societies all over the world, accounts of mania go back centuries. HIV/AIDS, on the other hand, is a relatively new disease with the first cases first described in the early 1980s [1]. Both diseases however have the ability to bring about a lot of despair and suffering and are often stigmatized. HIV/AIDS, for example, has been one of the greatest health challenges of Sub-Saharan Africa for the last 30 years. In this time the disease has claimed over 15 million lives, devastated economies, families and the region as a whole with billions of dollars being spent in trying to curb the scourge [2].

Early in the pandemic it was noted that mental health disorders were closely related with HIV/AIDS with many psychiatric disorders having increased prevalence in HIV positive populations [2, 3]. People living with HIV/AIDS (PLWHA) have been found to be at a greater risk of having a psychiatric illness in the same way people with psychiatric disorders are at a higher risk of contracting HIV/AIDS [3, 4, 5].

The majority of the described HIV related mental disorders are affective disorders including mania and depression. However, HIV-associated neurocognitive disorders, HAND, are increasingly being documented. Many of the psychiatric disorders in HIV/AIDS have thus been studied and it is now becoming clearer that these conditions are not just psychological reactions to having the disease but are sequalae of the neuropathogenesis of HIV/AIDS.

With the introduction of Highly Active Antiretroviral Therapy (HAART), people living with HIV/AIDS are able to live longer lives and have improved livelihoods. Current studies however show that mental conditions are in some ways responsible for failure to reach desired targets especially in the area of condom use and drug adherence [6]. Various studies have shown worse outcomes in HIV/AIDS patients that are co morbid with mental disorders. Early diagnosis and treatment of metal disorders in HIV/AIDS is one of the ways in which meeting treatment goals especially in areas of adherence and condom use can be attained.

Primary Mania Versus Secondary Mania

Classification of mood disorders has changed over time through improvements in case definitions and scientific breakthroughs. From [7] who defined manic depressive illness to the unipolar- bipolar classification by [8] or the spectrum of affective disorders by Akiskal, the classification of affective disorders has kept on getting broader and better defined [9].

Today DSM IV TR [10] provides the standard for classification of mood disorders. The proposed DSM IV TR classification of mood disorders is listed in the table below (see Table 19.1).

Table 19.1 DSM IV classification of mood disorders

Major depressive disorders
Dysthmic disorder
Depressive disorders not otherwise specified
Bipolar 1 disorder
Bipolar II disorder
Cyclothymic disorder
Bipolar disorder not otherwise specified
Mood disorder due to a medical condition
Substance induced mood disorder
Mood disorder not otherwise specified

Each of these disorders may involve a depressive episode, manic episode, mixed episode, hypomanic episode or a combination of any two different episodes. Thus a Bipolar I disorder is characterized by one or more manic or mixed episodes usually accompanied by a depressive episode while a Bipolar II disorder is characterized by one or more Major depressive episodes accompanied by at least one hypomanic episode.

Further classification could involve determining whether the cause is primary or secondary and from here the terms primary mania or secondary mania evolve [11]. One can differentiate mania based on the cause by dividing it into primary or idiopathic mania and secondary mania (where there is an identifiable cause.) This classification of primary and secondary mania was first suggested by Robins and Guze in 1972 [12] and improved by Klerman and Barret and later Krauthemmer in 1978 [13]. However the distinction of primary versus secondary disorders was first muted by Alistair Munro.

Initially secondary mania was described by Robins and Guze as mania occurring in patients who had no previous or concurrent psychiatric illness. Klermman and Barret later described it as an illness following past history of medical, traumatic or pharmacological complaints and use. Krauthemmer took the concept further to describe secondary mania as mania that can occur in association with organic dysfunction – medical and pharmacological toxic – in patients with no history of affective disorder. He was also the first to describe the clinical characteristics of secondary mania being different from primary mania where he noticed that patients of secondary mania were older with no family history of mania. The concept of secondary mania is that a patient can present with symptoms that are similar to the diagnostic criteria for primary mania but with an identifiable cause. In some cases the clinical characteristics are different. Thus HIV secondary mania became to be recognized as a disorder of HIV infective brain degeneration.

Secondary Mania of HIV/AIDS

Over the last 10 years research done in Africa and Uganda in particular has shown that mental health issues in HIV are not just psychological reactions to having the disease but rather a consequence of the HIV virus attacking brain tissue [2].

Dementia, major depression, psychosis, and mania are some of the mental health conditions that have been studied quite extensively by Ugandan researchers and have been shown to be different entities from the same conditions in HIV negative populations [2, 3, 14–19]. Mental health disorders in HIV/AIDS seem to be a sequalae of the neuropathogenesis of the virus itself which leads to presentations of some mental health conditions to be different between HIV positive and HIV negative groups.

In the case of mania this has led to the development of the term secondary mania of HIV/AIDS also referred to as HIV mania which was well described by Nakimuli-Mpungu et al. [18]. They described secondary mania of HIV/AIDS as a distinct syndrome with different clinical presentation from patients with primary mania without HIV and bipolar mania in HIV. This differing clinical presentation impacts on treatment modalities prognosis and in low resource settings investigations and as such clinicians need to be aware of this syndrome.

Etiology

In primary mania there have been various studies on the different aetiologies of mania. It should be noted that they are usually multifactorial and as such a biopsychosocial model is usually adopted to define the different causes of primary mania. The main theories of mania include increased neurotransmitter function especially dopamine and serotonin; genetics proved through family and twin studies and psychodynamic factors as described by Abraham, Lewin and Klein [20]. From the late 1970s Krauthemmer suggested organic causes of mania. He noted that drugs metabolic disturbances and infections were all associated with developing manic episodes. Since then a large number of organic substances have been associated with development of manic symptoms (see Table 19.2).

Pathogenesis

As is the case for many psychiatric conditions that are co morbid with HIV, the pathogenesis is not entirely understood. Five main theories have been postulated to determine how HIV causes mania in particular. These include the following:

i. Direct effects of the virus on the central nervous system.
ii. Opportunistic infections and the metabolic effects associated with them.
iii. Drugs used in the treatment of HIV/AIDS like HAART especially the non nucleoside reverse transcriptase inhibitor Efavirenz and steroids.
iv. Psychological challenges associated with having HIV/AIDS.
v. HIV associated neuro cognitive disorders (HAND) causing brain degenerations
vi. HIV associated CNS neoplasms.

Table 19.2 Causes of secondary mania

Drugs of abuse
Alcohol abuse
Amphetamine abuse
Cocaine abuse
Hallucinogen abuse
Opiate abuse
Collagen vascular disease
Systemic Lupus Erythematosus
Infectious disease
Neurosyphilis
Herpes Encephalitis
Influenza
St. Louis Encephalitis
HIV/AIDS
Endocrine disease
Hyperthyroidism
Hypothyroidism
Neurologic disease
Multiple Sclerosis
Huntington's Chorea
Wilson disease
Head trauma
Complex partial seizures
Cerebrovascular accidents
Migraine headache
Neoplasms (esp. diencephalic or third ventricle)
Medications
Neuropsychiatric
Monoamine oxidase inhibitors
Heterocyclic antidepressants, SSRI
Methylphenidate
Disulfiram
Levodopa
Cardiovascular
Captopril
Hydralazine
Endocrine
Bromocriptine
Steroids
Miscellaneous
Baclofen
Bromide
Procarbazine

(continued)

Table 19.2 (continued)

Yohimbine
Cimetidine
Isoniazid
Vitamin deficiency
Vitamin B12 deficiency
Folate deficiency
Niacin deficiency
Thiamine deficiency

Adopted from Mania secondary causes in family practice notebook, http://www.fpnotebook.com/psych/Bipolar/MnScndryCs.htm. Accessed 10/30/2014

Previous studies have tried to determine the pathogenesis of HIV-related mania. HIV preferentially affects sub cortical gray matter such as the caudate nuclei and cortical white matter, both of which are important in the regulation of mood, thus manic symptoms indicating CNS HIV infection. HIV-related mania may be caused by accumulation of intracellular free calcium, which has been implicated in the pathogenesis of bipolar disorder [21] and has similarly been shown to be increased in HIV infected neurones [22, 23]. On the other hand, El-Mallakh suggested that mania or hypomania appeared to be related to immunosuppression and progression of HIV disease.

Clinical Features

Making a diagnosis of mania ideally entails following the set criteria as laid out in the DSM IV TR. In summary the classification states that the symptoms should last for at least 1 week (or less if hospitalization is required); should not involve a mixed episode, not be due to the effects of a substance and should affect social occupational functioning.

A patient with HIV mania usually meets the criteria set out for a manic episode. Primary mania is highly hereditary but in the case of HIV mania the patient usually does not have previous history of this illness or family history of the disease [16–19]. Patients with secondary mania of HIV/AIDS however present with irritability more than euphoria; are more over talkative with decreased need for sleep. They are more cognitively impaired with more perceptual disturbances [16–18, 24]. The DSM classification of a manic episode does not report any cognitive or perceptual disturbances.

In cases of primary mania or bipolar mania the patient will usually be in the late teens or early 1920s and usually has prior depressive episodes or manic episodes and family history suggestive of a mental illness. The clinical features of secondary

mania are distinct especially in terms of demographic characteristics [25]. Patients of secondary mania of HIV/AIDS in Uganda were usually uneducated older females of poor socioeconomic status as described by Nakimuli-Mpungu et al. [18]. In Caucasian populations patients of HIV mania were found to be older gay males who were well educated and of good socioeconomic status. The main demographic characteristic was age which supports the theory that secondary mania was more prevalent in older populations. This implies that a first episode manic episode in HIV populations usually occurs with a later age of onset. This differs from the secondary mania after a brain lesion where there is no noted age differences between patients who developed mania after a brain lesion and those without a brain lesion but who had mania [26]. In high HIV endemic populations, it is prudent to suspect a first episode manic episode occurring in later ages to be due to HIV/AIDS making routine HIV screening mandatory in all late onset manias [2].

In earlier case presentations of HIV mania patients were noted to present with AIDS defining illnesses like HIV wasting syndrome. Current case presentations however describe patients presenting with stage II symptoms of the World Health Organization Staging characteristics of HIV/AIDS like oral candidiasis. Indeed the occurrence of late onset first episode mania may herald HIV infection in the absence of other symptoms.

Investigations

Investigations usually involve a work up for initiation of HAART since Secondary mania of HIV/AIDS has often been found to be associated with lower immune status [16, 18]. Nakimuli-Mpungu et al. [18] showed that a CD4 count of less than 350 was associated with HIV mania while Lyketsos et al. [27] noted that patients without previous family history or personal history of a manic disorder presented much later in the infection presumably when the immunity was low. In any patient suspected to have secondary mania of HIV/AIDS an HIV test to confirm the diagnosis and immune function tests by CD4 count are paramount. There is also need to rule out co-infections like syphilis and cryptococcal meningitis which can present with manic symptoms as well. There are no specific radiological investigations for HIV mania but they might show concurrent AIDS defining illnesses like toxoplasmosis or cryptococcal meningitis. Use of rating scales like the Young Mania Rating Scale is necessary to gauge severity and monitor response to treatment.

Treatment

Pharmacological treatment does not greatly differ between management of secondary mania of HIV/AIDS and primary mania. There is however need to watch for side effects since there is documented evidence of worse extra pyramidal side effects

in HIV especially when typical antipsychotics are used. Most studies show a better side effect profile when atypical antipsychotics like risperidone are used but this also depends on which antiretroviral is being used. A case series by Kelly et al. [20] showed worse side effects when risperidone was given specifically with the protease inhibitor ritinovar/idinavir. In our setting nucleoside and non nucleoside reverse transcriptase inhibitors are our first line drugs for HIV/AIDS so atypical antipsychotics are preferred in HIV mania. For prophylaxis of recurrences, Sodium valproate is the preferred mood stabilizer as it has less drug-drug interaction with HAART regimens and a safer blood level.

Prognosis

Time to recovery is shorter in secondary mania of HIV/AIDS than primary mania [28]. DSM gives a range time of 4 weeks to several months for a typical manic episode to resolve but in secondary mania the time is usually shorter. This quick recovery seems to be irrespective of whether or not the individual is on HAART [28]. There is however a high risk of relapse even when there is good adherence to the psychotropic drugs. This necessitates need for mood stabilizers usually sodium valproate, carbamazepine or lamotrigine. Lithium carbonate in this population is not preferred because of a narrow therapeutic window and associated renal toxicity especially in wasted HIV positive patients.

Case Report

LJ was a 33 year old male from one of the central districts in Uganda. He was a taxi driver and of the protestant Church of Uganda by faith. He was newly married to NJ and the couple had no children. He had never attended any form of formal education.

LJ presented to the outpatient Mental Health Clinic of Mulago National Referral Hospital with complaints of headache, confusion and over talkativeness over 1 day. He had also been complaining of easy irritability, decreased sleep and restlessness for the week prior to admission. He was brought in by his wife who reported two episodes of vomiting and confusion on the morning of admission. He was also more talkative than usual and got easily angered. He had been sleeping poorly in the week prior to admission characterized by terminal insomnia associated with pacing around the house. LJ reported complaints of headache which had been on for the last 3 months and which “could just not go away.” The headache was associated with occasional blurred vision. He also accepted that he had been having difficulty sleeping for the last 1 month and was irritable. He denied having hallucinations of any sort during this episode. He did not report feeling sad or decreased interest in activities of daily living and he had a good appetite. He had

no thoughts of feeling worthless or committing suicide for the 2 weeks prior to this onset of this episode.

LJ had been diagnosed HIV positive 4 months prior to this admission at which time his baseline CD4 count was ten cells per microlitre. He had been started on HAART of Truvada and Nevirapine 1 month after diagnosis of HIV. He reported good adherence to HAART which was confirmed by his wife. There was no history of trauma or seizures. He denied using alcohol or any other substances of abuse and he denied any history of mental illness in the family.

Past psychiatric history revealed that this was his second admission in a 4 month period. On the first episode, he had had similar symptomatology but including auditory and visual hallucinations. He had improved after a 10 day admission and discharged on a low dose of Haloperidol which he had been on since discharge and he was adherent to it too.

Family and social history revealed a marriage to one wife with no children between them. His wife was also seropositive for HIV and on cotrimoxazole prophylaxis. LJ had a child born out of this marriage who stayed with the mother and he did not know this girlfriend's or child's HIV serological status or their whereabouts. He had not worked on his job as a taxi driver for sometime due to illness.

Physical examination revealed a Glascow coma score (GCS) of 15/15 with pupils equal and reactive to light and accommodation. The neck was soft with a negative Kerning's sign. He had decreased hearing sensation but all the other cranial nerves were intact. He had normal muscle power tone and strength with no increased deep tendon reflexes and normal sensation.

His Mental Status Examination (MSE) revealed a young man who was restless and pacing. He was not wasted, not febrile, not anemic and had no oral thrush. His speech was loud but not pressured. His mood was irritable but without suicidal ideas. He was preoccupied with his headache, had no delusions and no perceptual disturbances. He was disoriented in time but not in place and person. He had poor concentration and attention but good insight. His memory and judgment were intact.

His current mental state was quite different in some aspects to his first presentation where he presented with physical signs of immunosuppression characterized by oral thrush, gross wasting and anemia. His speech then was loud, pressured and he was extremely irritable and aggressive. His thoughts at that time were many, grandiose and not connected. He had mood congruent auditory and visual hallucinations. He was disoriented in time place and person with poor memory, attention, concentration abstraction and judgment. His insight was also poor then.

A DSM IV-TR Multi axial diagnosis of HIV related manic psychosis with traits of antisocial behavior was recorded on axes I and II respectively. Axis III had medical conditions of primary CNS pathology of HIV opportunistic infections with Toxoplasmosis, Cryptococcus meningitis and Tuberculosis meningitis were highly suspected but laboratory investigations for them were negative. So, they were subsequently ruled out. On axis IV HIV disease was documented as a predisposing factor, precipitated by the low CD4 count. LJ being on HAART with good social support from the wife were thought to be protective factors. His global assessment of function was 45 % on that first admission. Then more biological investigations

revealed slightly raised alanine transferase. A complete blood count was unremarkable with negative serology for syphilis, serum CRAG and toxoplasmosis titers. His CD4 count was 168 cells. Neuroimaging with a brain Computer Topography Scan was unremarkable. Psychological assessment involved using the Yang Mania Rating Scale while social investigations sought to trace the child and former girlfriend in order to determine their HIV serological status.

His management involved admission into the inpatient psychiatric ward with immediate treatment to contain the high agitation using 10 mg of IM Haloperidol and 50 mg of IM promethazine as initial doses. Once less irritable he was started on oral antipsychotic medication while on the ward of haloperidol 10 mg twice a day, benzhexol 2 mg twice a day and clonazepam 2 mg at night. He was also advised to continue with HAART and cotrimoxazole 960 mg once a day.

LJ soon improved in his mental state on this management and was discharged after 1 week of hospitalization. He was discharged on oral haloperidol 5 mg at night and oral benzhexol 2 mg at night. Plans were made to start him on the prophylactic mood stabilizer, sodium valproate.

Discussion

The above case shows the salient features of secondary mania of HIV/AIDS. This is a syndrome that is different from primary mania described in DSM IV TR especially in demographic characteristics, clinical features, treatment, course and prognosis. According to Nakimuli-Mpungu et al. [18], patients with HIV mania are usually older, uneducated and of poor social economic status. This is different to Caucasian western populations where patients with HIV mania are usually educated males of good socioeconomic status [27, 29, 30]. Secondary mania of HIV/AIDS is associated with a lower immune status and patients usually present later in the infection [18, 27]. LJ's CD4 count of 10 cells/ul supports the theory that in low resource settings a first manic episode in HIV/AIDS is suggestive of a low CD4 count and may as well be an indication to start HAART. Patients with secondary mania of HIV/AIDS present with irritability more than euphoria and are over talkative with decreased need for sleep. They are also more cognitively impaired and have more perceptual disturbances like hallucinations than patients with primary mania [18, 19]. This case illustrated these features.

Pharmacological treatment does not greatly differ between management of secondary mania of HIV/AIDS and primary mania. There is however need to watch for side effects since there is documented evidence of worse extra pyramidal side effects in HIV especially when typical antipsychotics are used. The high risk of relapse may also necessitate initiating mood stabilizing medication. In the HIV positive population, sodium valproate is the preferred mood stabilizer as Lithium carbonate has a narrow therapeutic window and associated renal toxicity.

Finally, remission of symptoms is faster in patients with HIV secondary mania with an average time of 2 weeks for remission. This quick recovery seems to be irrespective of whether the individual is on HAART or not [2].

Conclusion

This case report depicts an HIV-positive patient with manic symptoms that are not dissimilar in presentation to DSM IV TR criteria for a manic episode. His clinical picture is very typical of the clinical presentation of secondary mania of HIV/AIDS as described in the literature [16, 18]. It supports the theory that secondary mania of HIV/AIDS is a clinically distinct syndrome and represents infective brain degeneration. This is not unusual in African settings.

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