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Case report

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Olanzapine Induced Hypersexuality

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Abstract

Hypersexuality is a recognized symptom of psychiatric disorders, namely mood and psychotic disorders. It has been known to accompany other personality changes in frontal lobe dementia. Dopaminergic therapy used to alleviate manifestations of Parkinson's disease may cause hypersexuality. Similarly, albeit less commonly, a paradoxical reaction to antipsychotic therapy could also result in hypersexualized behaviours. Dopamine receptor therapy has also been reported to worsen hypersexuality. It has also been observed to occur with impulse control disorders such as excessive eating, gambling, compulsive shopping and hypersexuality. Dopaminergic pathways play a vital role in the brain's reward mechanism and pathological interference could result in addictive behaviours. Dopaminergic neurotransmission has been associated with drug abuse. These commonalities between impulse control behaviours and drug addictions cannot be overlooked. Hypersexuality resulting from second generation antipsychotic (SGA) treatment is poorly understood. In recent years there have been case reports of Risperidone induced hypersexuality. Aripiprazole is slowly gaining recognition for its partial dopamine agonism causing hypersexuality. However, only a handful of cases with Olanzapine induced hypersexuality have been reported globally. We report hypersexuality in a young man with Paranoid Schizophrenia shortly after starting Olanzapine, with evidence possibly linking Olanzapine therapy to hypersexualized behaviours.

Keywords: Hypersexuality; second generation antipsychotic treatment; olanzapine; paranoid schizophrenia

Introduction

Hypersexuality is a recognized symptom of psychiatric disorders, namely mood and psychotic disorders. It has been known to accompany other personality changes in frontal lobe dementia. Dopaminergic therapy used to alleviate manifestations of Parkinson's disease may cause hypersexuality. Similarly, albeit less commonly, a paradoxical reaction to antipsychotic therapy could also result in hypersexualized behaviours. Dopamine receptor therapy has also been reported to worsen hypersexuality. It has also been observed to occur with impulse control disorders such as excessive eating, gambling, compulsive shopping and hypersexuality [1]. Dopaminergic pathways play a vital role in the brain's reward mechanism and pathological interference could result in addictive behaviours [2].

Dopaminergic neurotransmission has been associated with drug abuse [3]. These commonalities between impulse control behaviours and drug addictions cannot be overlooked. Hypersexuality resulting from second generation antipsychotic (SGA) treatment is poorly understood. In recent years there have been case reports of Risperidone induced hypersexuality. Aripiprazole is slowly gaining recognition for its partial dopamine agonism causing hypersexuality. However, only a handful of cases with Olanzapine induced hypersexuality have been reported globally [4-6]. We report hypersexuality in a young man with Paranoid Schizophrenia shortly after starting Olanzapine, with evidence possibly linking Olanzapine therapy to hypersexualized behaviours.

Case Presentation

We present a case of Olanzapine induced hypersexuality in a young man with first episode psychosis with no other identifiable organic, psychiatric, or precipitating conditions. Hypersexual

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behaviours of our patient resolved fully within 48 hours of discontinuation of Olanzapine. A rechallenge with Olanzapine was not attempted. Our patient was a single unemployed Caucasian man in his mid-30s. His initial psychiatric presentation was a decade prior to his referral to Early Intervention Psychosis service (EIS). At the time, he was referred to and diagnosed with Asperger's Syndrome by the local community adult Asperger's service during which he received solution focused therapy and was treated for depression by primary care services. He declined longer term psychological interventions but was deemed to be low risk and was subsequently discharged back to primary care services. He was a regular user of stimulant substances such as cannabis and heroin. Following his initial therapy sessions with the community Asperger's service, his usage reduced, and he was determined to make lifestyle changes, focus on his health and had considered paid work.

He was assessed by local drug services and was commenced on Naltrexone which he reported was beneficial. This was 5 years prior to his presentation to EIS. In 2021 following concerns from his family, our patient was conveyed twice to emergency care. He was referred to the Home Treatment Team following his first visit but was reported to have disengaged and was non concordant with antipsychotic Olanzapine 5 mg. He was observed to have become increasingly agitated and distressed in the days leading up to his presentation. He described paranoid persecutory delusions and was convinced that he was under attack from people surrounding his home. He reported auditory hallucinations in 2nd and 3rd person that were threatening and derogatory in nature, accusing him of being a paedophile and described voices threatening to hurt him. He described voices giving a running commentary about his life and was convinced that his whereabouts were being tracked.

He was tearful and distressed, was low in mood and had contemplated suicide. He was assessed to have partial insight into his condition. He agreed to commence antipsychotic medication. Our patient was recommended on Olanzapine 5 mg followed by 10 mg a week later. He reported discontinuing cannabis but despite this his psychotic symptoms persisted. However, his overall distress and agitation improved. By the 3rd week of his treatment, he reported an unusual pattern to his thoughts and behaviours which were of a sexual nature and had coincided with him commencing antipsychotic treatment. He said that he was embarrassed to discuss his symptoms, thus indicating the delay in reporting. He reported feeling sexually aroused and that his sexual urges had increased, both of which were not in keeping with his thoughts and/ or behaviours. He described an increased desire for sex and excessive masturbation.

He recognized this change to be uncharacteristic for him and regarded his presentation to be "against morals of society". He did not report thoughts or urges or compulsions to act on these sexual urges and denied having new or random intimate sexual relationships. He denied experiencing any other repetitive ego dystonic thoughts or urges or imagery of sexual nature. He denied increased use of pornographic websites prior to or during his antipsychotic treatment. He did not report somatic passivity phenomenon in relation to his sexual urges and sexual desires. On closer observation it was determined that our patient's symptoms had started and worsened upon commencement of treatment with Olanzapine and subsequent dose increase. Olanzapine was discontinued with immediate effect and our patient reported a significant reduction in hypersexuality within 48-72 hours. He requested an alternative antipsychotic and was placed on Risperidone for psychosis. The dose increase of Risperidone from 2 mg to 4 mg was uneventful. Unfortunately, he reverted to using psychoactive substances, but this neither impacted on nor triggered hypersexual behaviours.

Conclusion

A causal relationship between Olanzapine therapy and hypersexuality is difficult to state with any degree of certainty. In our patient's case history, a temporal association has been evidenced between emergence of hypersexualized behaviours upon commencing Olanzapine and full resolution of symptoms upon discontinuation of Olanzapine. This could only suggest a potential link between Olanzapine and hypersexuality in the absence of other medical conditions. Our patient engaged polysubstance misuse but has never before experienced symptoms of hypersexuality. Our case highlights the need for direct questioning about sexual function before commencing psychotropic medication and as part of routine clinical history. This may also highlight any other issues that may not necessarily be related to psychotropic medication. Our patient did not have any pre-existing sexual issues. He did not report increased or compulsive sexual behaviours as part of his psychosis.

It is important and necessary not only as part of initial assessment of a psychiatric condition but to also rule out uncommon adverse effects of second-generation antipsychotic (SGA) treatment in patients with psychosis and mood disorders. Although we considered an alternative treatment plan to include Aripiprazole, this was subsequently dismissed because of its partial 5HT1A agonism; a possible causal link to hypersexuality. Although Risperidone has been reported to have similar adverse effects, it is not a partial agonist, and it suited our patient. A causal relationship between Olanzapine therapy and relevant demographics such as age, gender, genetic or biological predisposition is beyond the scope of our report. Our case illustrates that hypersexualized behaviours may be a rare adverse effect of olanzapine therapy and possibly second-generation antipsychotics. Considering clinicians' limited knowledge in this area we conclude that further research is necessary and guaranteed to understand, delineate, and widen treatment options for our patients.

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