potential role in the neuropsychiatric effects of absinthe. α-thujone's pharmacodynamic profile is that of an antagonist at gamma amino butyric acid (GABA) receptors.[3]

The psychological effects of such compounds (GABA receptors blockers) include symptoms described in association with absinthe use, such as dysphoria, seizures and anxiety. Consistent with the latter actions are the findings of a recent study demonstrating that higher concentrations of thujone consumed with alcohol are associated with a reversal of alcohol's intrinsic anxiogenic effects.[4]

To further explore this issue, recent toxicological studies have attempted to measure the levels of thujone in both commercially available and vintage absinthe.[5] The findings of low (average 1.5mg/l) levels of absinthe in both samples have lead to assertions that thujone may play no, or at best a minor, role in the effects of absinthe[6] and that the previously held effects of absinthe in effect represent an 'urban legend'.[7]

Summary

This clinical case reflects some of the issues in the debate over whether the consumption of absinthe is associated with any particular risks over and above those associated with the consumption of alcohol.

Our patient developed marked acute suicidal feelings in the context of intoxication. While compounds (such as thujone) found in absinthe may have putative mood altering effects, nevertheless the role of alcohol in the development of suicidality is prominent.

Alcohol's potential for precipitating suicidal behaviour is well established,[8] and acute alcohol use may be an important factor in suicides among individuals with no psychiatric history.[9] Use of illicit substances may also lead to emergence of suicidal behaviour and it is important to exclude this possibility by doing a urinary drug screen in addition to full blood count, urea, electrolytes, and liver function tests.

However, the emergence in this case of acute suicidality exclusively in the context of absinthe consumption, but not in previous or subsequent episodes of alcohol misuse, is intriguing.

Healthcare providers, and those who drink absinthe, should remain alert to the putative psychological risks of absinthe consumption which are intriguing and concerning.

To end with a cautionary note, the words of Oscar Wilde regarding absinthe merit reflection: *"After the first drink, you see things as you wish they were. After the second you see them as they are not. Finally, you see things as they really are, which is the most horrible thing in the world."*

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Case report

Tardive dyskinesia on low dose risperidone

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Abstract

Tardive dyskinesia is a neurological disorder characterised by involuntary and purposeless movements affecting any part of the body. These movements typically occur in the oro-facial area and the patient is usually unaware of them. There are inconsistent findings in the literature on the risk factors for developing tardive dyskinesia. Nevertheless, previous reports indicate that tardive dyskinesia is more common in female patients, patients with a history of alcohol and substance misuse, affective disorders, and intellectual disability. The dose, class and duration of antipsychotic medication may also be independent risk factors. We report on the case of a patient who developed tardive dyskinesia on a low dose of the second generation antipsychotic risperidone.

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Case

BT, a 42 year old single male was referred by his general practitioner with a history of low mood, poor sleep, decreased concentration and anhedonia. He also described delusions of reference believing that programmes on the television were relaying messages to him and that others were talking about him in a derogatory manner behind his back. There was no history of illicit drug use or alcohol abuse. There was no history of elation. On mental state examination, he was subjectively and objectively depressed. He had ongoing paranoid delusions and no perceptual disturbances. He had similar symptoms in the past triggered by difficulties at work and had suicidal ideation.

A diagnosis of depression with psychotic features was made. He was commenced on oral citalopram 20mg mane and oral risperidone 4mg nocte. He was treated as an outpatient and an improvement in his mental state was noted during follow-up appointments. He was examined by the consultant psychiatrist at regular intervals in the outpatients department. No evidence of abnormal involuntary movements was noted at the initial starting dose of risperidone 4mg. There was a resolution in his symptoms and he no longer expressed paranoid ideation and he returned to work. There was a gradual reduction in the dose of oral risperidone from 4mg nocte to 1mg nocte over the subsequent 18 months. Over this 18 month period of dose reduction no abnormal involuntary movements were noted at follow up appointments.

He remained stable on 1mg of risperidone and was regularly reviewed in the outpatient department. At a follow up appointment six months later, he was noted to have abnormal involuntary movements in the oro-facial area. Risperidone was discontinued and at a review two months later the abnormal oro-facial movements had improved but not completely resolved. At a subsequent review, four months after Risperidone was discontinued, these movements had completely resolved.

Discussion

Risperidone, a benzisoxasole derivative is a second generation antipsychotic and a potent blocker of both serotonin (5HT2) and dopamine (D2) receptors. Cases of tardive dyskinesia have been reported among patients on higher doses of between 6mg to 9mg of risperidone.[1-3] However, there have been no reports of tardive dyskinesia on doses lower than this.

One possible explanation for the development of the involuntary movements in BT is withdrawal emergent tardive dyskinesia. This refers to the emergence of tardive dyskinesia following reduction in dose or abrupt withdrawal of risperidone.[4] In one report, a 26 year old male with schizophrenia developed tardive dyskinesia during a dose reduction of risperidone.[5] Two further cases also reported in the first few days after abrupt withdrawal of risperidone.[6,7] The case we are reporting does not appear to fit the description of withdrawal emergent tardive dyskinesia as there was a six month interval between reduction of the dose of risperidone and development of tardive dyskinesia.

This case highlights the fact that second generation antipsychotics still carry a risk of tardive dyskinesia. It also suggests that even low doses of the atypical antipsychotic risperidone might be associated with the onset of tardive dyskinesia. The routine use of standardised rating scales such as the Abnormal Involuntary Movement Scale (AIMS)[8] can help identify tardive dyskinesia in its early stages when involuntary movements may be reversible.

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