Moderate serotonin toxicity precipitated by a possible interaction between lavender tea and sertraline

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Consent has been obtained for the publication of this case and is held with the treating institution

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A 29-year-old Caucasian male presented to the Emergency Department reporting facial spasms, lower limb rigidity, ataxia and tremor that had developed over the previous 24 hours. This was on a background of starting sertraline 50mg daily for 5 days prior to presentation, which was commenced by a psychiatrist for anxiety. He denied using any serotonergic agents prior to this. His only other medical history included asthma for which he took Symbicort® (Budesonide/Formoterol 200/6 mcg) two puffs twice daily. On the night
before presentation at approximately 2200, he drank a cup of lavender tea (consisting of two teaspoons of dried English Lavender (Lavandula angustifolia) flowers steeped in hot water for several minutes prior to consumption). The product was purchased from a regional lavender farm. He observed that his symptoms developed early the following morning and progressed during the day prompting presentation to hospital.

On physical examination, he was a well-nourished man who appeared anxious and diaphoretic. He was alert and orientated despite this. His vital signs on presentation were: Temperature of 36.7°C, heart rate 70 bpm, blood pressure 150/90, respiratory rate 16 breaths per minute, and oxygen saturation 98%. He had non-directional nystagmus with repetitive clenching of his jaw suggestive of bruxism. A fine tremor was noted of both upper and lower limbs. Examination of the lower limbs revealed inducible clonus of up to 6 beats bilaterally, which was marginally worse in the left ankle. No sensory or motor deficits were noted. Blood tests (Full blood examination, Urea, Electrolytes and Creatinine, C-Reactive Protein, Creatine Kinase, Paracetamol, Ethanol and VBG), ECG, CT Brain and MRI brain were all reported as within normal range. A urine drug screen was performed and was positive only for benzodiazepines (specifically, amphetamines and cocaine were not detected). The urine specimen was collected after treatment with diazepam was commenced. A diagnosis of serotonin toxicity was made as the Hunter Serotonin Toxicity Criteria.1 He was admitted for ongoing management and received 20mg of oral diazepam intravenously on presentation then oral diazepam (titrated to a total of 110mg over 16 hours) and a single dose of oral cyproheptadine 8mg 13 hours after presentation. Symptoms improved subjectively after the latter however due to timing, it was unclear whether this was due to cyproheptadine. He subsequently recovered and was admitted to a private psychiatric hospital for ongoing management of his anxiety.

Serotonin Toxicity is the result of excess serotonin in the central nervous system and is typically characterised by the triad of neuromuscular excitation, autonomic hyperactivity and altered mental state. Three levels of severity are described and are usually delineated by the treatment required – mild (symptoms not distressing for the patient), moderate (symptoms causing significant distress and require treatment) and severe (rapidly increasing temperature with potential for multi-organ failure and death if not treated).1,2

The observed reaction was deemed a possible adverse drug reaction with a Naranjo score of 4- the adverse event occurring after lavender was ingested, the reaction improved following discontinuation (and cyproheptadine may have helped), and the adverse reaction was defined using the Hunter Criteria for Serotonin Toxicity.3 Despite having recently commenced sertraline days before becoming unwell, the patient’s symptoms had a closer temporal relation to his ingestion of lavender tea. Whilst sertraline is a selective serotonin reuptake inhibitor (SSRI), it is not typically associated with causing significant serotonin toxicity on its own although it should be noted that it takes 5-7 days to reach steady state.4 Typically, where a patient exposed to sertraline has more serotonin toxicity, it is usually due to concurrent exposure to other agents with serotonin-augmenting properties. Sertraline concentrations and pharmacogenomic analysis were not available to support this in our case.
In this case, the possible mechanism for lavender tea precipitating moderate serotonin toxicity is described in a rodent model, where both lavender essential oil and one of its major components found in all species of lavender, linalool, demonstrated moderate activity in an assay examining SERT inhibition with significant displacement of $^3$H-citalopram (a ligand used for assessment of serotonin reuptake) in a dose-dependent manner$^5$. Silexin, a patented lavender essential oil product, has been shown to have anxiolytic properties and has been shown to bind 5HT-1a receptors$^6$. A limitation for this case is that a clinical assay for lavender essential oil or its components is not available and thus the ideal confirmatory and quantitative analysis was not feasible. However, despite this limitation, there is evidence that can be extrapolated from analytical studies of other herbal teas that have demonstrated that essential oils were extracted from and displayed the same chemical profiles as the raw ingredients used in the teas, albeit in lesser quantities$^7$.

This case demonstrates a possible association between lavender tea, sertraline, and the manifestation of moderate serotonin toxicity with objective clinical features. Clinicians should ask about the use of complementary or herbal medications when taking a drug history given the potential for drug interactions with these agents and prescription medications.

References

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