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A case of vermiculate tongue

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Background

Pharmacology of atypical antipsychotics is complex; where in addition to lower biding affinity, potency and occupancy to dopamine (D2) receptors these drugs also selectively antagonise mesolimbic D2 receptors more so than the nigrostriatum and prefrontal cortex.

This explains lower frequency of side effects due to poor nigrostriatal D2 blockade (especially tardive dyskinesia) in comparison to effects attributable to mesocortical orefrontal D2 blockade (like neurocognitive impairment). We present a case report where an atypical antipsychotic drug overdose induced a rather rare tardive dyskinesia in the form of reversible lingual dyskinesia.

Case Report

A 75 year old female presented to emergency department with a week history of insidious onset delirium, fatigue, lethargy, slurred speech and drowsiness. There were no other reported symptoms.

On examination, she was confused and disoriented to time, place and person. Rest of cardio-respiratory-abdominal examination were unremarkable. There were no upper or lower motor neuron signs or meningism; however, interestingly, she had bilateral tongue fasciculations without any associated tongue muscle bulk wasting. There was no other obvious skeletal muscle weakness, fasciculation or polyminimyoclonus/fine tremor. She had mild oro-pharyngeal swallowing impairment for which she was assessed by our speech and language therapist.

The term vermiculations meaning worm like movements, more aptly describes this outwardly visible tongue sign was deemed to represent a "vermiculate tongue" in absence of any other associated neurodegenerative or anterior-horn-cell disorder signs.

Investigations revealed prolonged corrected QTc (500 milliseconds) on her electrocardiogram (ECG) without any serological dyselectrolytemia.

Inflammatory markers were not raised and infective markers were normal along with normal bed-side urine-analysis. There was no family history of spino-muscular or neurodegenerative disorders.

Computerised-tomography (CT scan and magnetic radioimaging (MRI) scan of brain were unremarkable.

On reviewing her medication it was noted that she had been taking quetiapine for last 6 months.

Hence, in presence of prolonged QTc and tongue fasciculations with absence of any other neurological features a diagnosis of probable quetiapine overdose was suspected.

Her quetiapine was discontinued. Decontamination and enhanced elimination with activated charcoal was not indicated. There were no features of neuroleptic malignant syndrome.

Empirical management with intravenous fluids, conservative support and strict cardio-respiratory monitoring with frequent neuro-observations were maintained over next 48 hours.

Interestingly, these involuntary twitching of tongue muscles visually resembling movement of worms inside the tongue (like a bag of worms) improved within one week, her QTc normalised and her confusion settled with improvement of her confusion after above supportive measures.

She made a remarkable full recovery with complete symptomatic resolution and was discharged from hospital in fortnight; and she remains well at one-month follow-up with no recurrence of her symptoms.

Conclusion

Our case report describes a rather rare form of tardive dyskinesia in form of lingual fasciculations in relation to quetiapine-overdose with reversible altered mentation and prolonged QTc in a patient who made a full recovery after early-recognition, drug-discontinuation, and supportive-management resulting in favourable clinical outcome.

It also emphasises importance of medications review which in this case not only provided a correct diagnosis but also avoided otherwise costly or rather inappropriate tests like electromyography (EMG) and other neuro-muscular immunological investigations.