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Antidepressants induced cranial nerve 1 dysfunction

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Introduction: Multiple antidepressants have been reported to induce hyposmia including sertraline, fluoxetine, lamotrigine, clonazepam and vilazodone. A single case with multiple antidepressants independently inducing hyposmia has not heretofore been described. Such a case is presented.

Methodology: Case study: A 52 year old right handed female presented with head trauma with loss of consciousness seven years ago, followed by reduced ability to smell except for monthly olfactory windows. Coincident with this she also experienced impaired taste with ability to taste only sweet and salty foods. She denied dysgeusia, phantosmia, cacosmia and palinageusia. She also suffered from chronic depression and anxiety with complaints of easy fatigability, worry, continuous racing thoughts, obsessions and panic attacks every other day. For this she was started sequentially and independently on a variety of antidepressants. Each one induced hyposmia which resolved with discontinuation of the antidepressant.

Results: Abnormalities neurologic examination: premedication olfactory ability: subjective smell - 100%, subjective taste - 100%. On sertraline : subjective smell - 0%, subjective taste - 0%, pocket smell test - 2 (hyposmia), alcohol sniff test - 0 (anosmia), propylthiouracil disc taste test - 5 (hypogeusia); On paroxetine: subjective smell - 0%, subjective taste - 50%, pocket smell test - 2 (hyposmia), alcohol sniff test - 0 (anosmia); on lamotrigine: subjective smell - 0%, subjective taste-10%, pocket smell test - 2 (hyposmia); On vilazodone: subjective smell - 0%, subjective taste - 30%, alcohol sniff test-0 (anosmia). One antidepressant had no impact on chemosensation- vortioxetine: subjective smell - 100%, subjective taste - 100%.

Discussion: The antidepressants may have acted on neurotransmitters in the olfactory bulb, since serotonin, dopamine and acetylcholine is present in this region. Possibly, because of a preexisting mild deficit in this region, she may have been more susceptible to such changes. Alternatively, nasal mucosal engorgement may have been effected, reducing air flow to the olfactory epithelium, and thus smell. The mucosal lining of the nasal epithelium may have been altered, reducing ability of odors to dissolve and thus perceived smell. The taste deficit may have been a retronasal smell deficit, which is a result of synesthesia, misperceived as a taste loss. Why vortioxetine did not have similar effect is unclear. However, it has multimodal serotonergic actions including a 5-HT_3, 5-HT_1D, and 5-HT_7 antagonist, a 5-HT_1A agonist, and a 5-HT_1B partial agonist property. Possibly these bypass the olfactory pathway and thus the smell is not effected. In those with depression and olfactory deficits, it may be prudent to initiate therapy with vortioxetine.

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