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ZOLPIDEM-INDUCED GALACTORRHEA VIA GABAERGIC INHIBITION OF DOPAMINE: A CASE REPORT

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Objective: To discuss a case of zolpidem-induced galactorrhea.
Introduction: The prevalence of insomnia can be as high as 32 to 33%. Non-benzodiazepines (such as zolpidem) have become more commonly used to treat insomnia.
Case report: The patient is a 29-year-old woman with a history of (PTSD) who presented with PTSD associated insomnia. She was started on zolpidem 5 mg po qhs. Two months after the initiation of zolpidem treatment, the patient presented with breast tenderness and galactorrhea. Zolpidem was discontinued and the galactorrhea resolved after two weeks. A serum prolactin level drawn shortly after discontinuation of zolpidem was 15.67 mg/ml.
Discussion: Zolpidem has a high affinity at the α1 containing GABAA receptors, with reduced affinity for those containing the α2- and α3- GABAA receptor subunits and minimal affinity for α5 receptor subunit. Psychotropic drugs have been well recognized to produce hyperprolactinemia. However, there has been no reported case of zolpidem-induced hyperprolactinemia. Specifically, zolpidem has been noted to activate GABAergic neurons within the ventral tegmental area (VTA), where there is a sizable population of GABAergic neurons. These GABAergic neurons regulate the firing of dopaminergic counterparts, also located in the VTA, which send projections throughout the brain. This inhibition results in a decrease in the dopaminergic inhibitory influence on prolactin and an increase in prolactin releasing factors which act on the anterior pituitary, leading to hyperprolactinemia and thus galactorrhea.
Conclusion: Pharmacologically induced hyperprolactinemia may be a problem of underestimated prevalence due to the lack of externally visible symptoms.