

THE MECHANISM OF PSYCHOSIS IN A PATIENT WITH GRAVES DISEASE

Taha Can Tuman¹ & Akif Asdemir²

¹Birecik State Hospital, Department of Psychiatry, Birecik, Turkey

²Erciyes University, Medical Faculty, Department of Psychiatry, Kayseri, Turkey

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Dear Editor,

Graves' Disease is known as an autoimmune disorder. TSH receptor is also located in the orbital tissue and brain (Bunevicius & Prange 2006). Here, we describe a patient with psychosis associated with Graves disease and the mechanism of psychosis in this patients. The patient has provided written consent authorizing publication of this report.

A 33-years-old man with a 3-month history of psychotic symptoms. Risperidon was started at a dosage of 4 mg/day on the 1st day of treatment. In physical examination patient had tremor, palpitations and flushing. Laboratory results revealed high levels of thyroid hormones. In addition, a thyroid-scintiscan with technetium-99m showed to have diffuse hyperactivity in both thyroid lobules. Metimazole 10 mg/day and propranolol 40 mg/day were administered to the patient. The most probable diagnosis for the patient was "Psychotic Disorder due to Graves' Disease". When discharged on the 19th day with euthyroid status, he was asymptomatic. 1 month after being discharged, no psychotic symptoms was observed during control examination.

The pathogenesis of psychosis in hyperthyroidism is still unclear. In animal studies, experimentally induced hypothyroid or hyperthyroid conditions resulted in altered responsiveness of NE, 5-HT, DA and GABA systems in adult brain (Strawn et al. 2004, Bilezikian & Loeb 1983, Wiens & Trudeau 2006, Yamamoto & Hornykiewicz 2004). Autoantibodies which may have detrimental effects to the signaling, myelination, synaptic plasticity and neurotransmitters may lead to psycho-

sis (Radulescu 2009). In conclusion, it may be suggested that psychotic symptoms in this case may occur due to thyroid hormones, autoantibody formation, a possible increase in dopamin release or brain NE dysfunction that is triggered by Graves' Disease.

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Correspondence:

Taha Can Tuman, MD
Birecik State Hospital, Department of Psychiatry
Birecik, Turkey
E-mail: tahacantuman@hotmail.com