Psychosis Following Carbimazole-induced Acute Alteration of Hyperthyroid Status

Guru S. Gowda, Mallikarjun Rao Sagi, Sai Komal, T. S. Jaisoorya

ABSTRACT

Abrupt normalization of hyperthyroid state with antithyroid drugs is reported to precipitate psychosis. We report the development of acute psychosis in a 20-year-old woman, following 30 days use of tablet carbimazole 10 mg for hyperthyroidism due to the multinodular goiter. At the time of presentation, she was euthyroid with a resolution of hyperthyroid status both clinically and biochemically. After 20 days treatment with tablet olanzapine 10 mg/day and stoppage of carbimazole, psychotic symptoms remitted completely and she has since remained in a euthyroid state and free from psychotic symptoms. This case highlights the development of psychosis in individuals, following rapid restoration to normal serum thyroid hormone levels in hyperthyroid individuals with carbimazole.

Key words: Carbimazole, euthyroid state, multinodular goiter, psychosis

INTRODUCTION

Carbimazole is a prodrug and its active metabolite acts by inhibiting thyroid peroxidase enzyme, hence reduces the thyroid hormone production. It is used in a treatment of hyperthyroid status due to grave disease, thyroiditis, and multinodular goiter. Most people reach a euthyroid state within 4–6 weeks with antithyroid drugs treatment for hyperthyroidism. Abrupt normalization of serum thyroid hormones or hypothyroidism state with antithyroid treatment is reported to precipitate psychosis\(^1\)\(^\text{[1-4]}\) and mood disorder.\(^5\) We describe a case of acute psychosis in young female, following rapid normalization of thyroid hormone with carbimazole. Issues of possible pathophysiology, diagnosis, and management are discussed.

CASE REPORT

Ms. S, a 20-year-old single female, from a South Indian family of upper socioeconomic status urban background, was premorbidly well adjusted, with a family history of hypothyroidism in mother and no significant personal history. She had no significant psychiatric history. She was seen by an endocrinologist for the symptoms of sweats,
headaches, tremor, palpitations, irregular cycles, and weight loss of approximately 4 kg over the previous 3–4 months. She underwent biochemical investigation [Table 1] and ultrasonography of the neck and was diagnosed with hyperthyroidism due to multinodular goiter and started on tablet carbimazole 10 mg/day.

After 30 days of initiation of carbimazole, she started experiencing somatic, tactile, kinesthetic, cenesthetic, and occasional second person auditory hallucinations. She attributed that her abnormal perceptual experiences are due to electronic radiation produced by gadgets used by her, so she started avoiding mobile, computer, and electronic gadgets. These symptoms were also associated with decreased sleep, restlessness, and socio‑occupational dysfunction. When she presented to our hospital, the symptoms were present for 3 weeks; hence, a provisional diagnosis of acute psychosis was made.

On admission, detailed physical and neurological examinations did not suggest hyper/hypothyroidism features. On the mental status examination, she was restless, anxious and reported somatic, tactile, kinesthetic, cenesthetic, and occasional second person auditory hallucinations. Diagnostic laboratory investigations such as noncontrast computerized tomography - brain, electrocardiogram, electrolytes, renal and liver profile, blood sugar, thyroid hormone function [Table 1], folate, cyanocobalamin, lipid profile, and complete hemogram investigations were normal. Ultrasonography of the thyroid gland shows multinodular goiter feature. In liaison with the endocrinologist, the patient was started on tablet olanzapine 10 mg/day and carbimazole was stopped. All abnormal perceptual experiences, hallucination, fear, sleep difficulties resolved with full recovery in psychosocial functioning in 20 days [Table 1]. She has remained in a euthyroid state and free from psychotic symptoms on follow-up.

**DISCUSSION**

In our case, the patient developed psychosis around 30 days after initiation of carbimazole. At the onset of psychosis, she had a euthyroid status both clinically and biochemically; hence, it is possibly unlikely that her hyperthyroid status or initiation of carbimazole tablets could have led to precipitation of psychosis. One of the possibilities could be the abrupt alteration of thyroid hormone level in the body and brain precipitated psychosis in our patient. This possibility has been reported prior where an abrupt alteration in thyroid hormone level has been reported to precipitate psychosis.[2‑5] Her psychotic symptoms are resolved completely after with olanzapine treatment and improved to premorbid level.

**CONCLUSION**

An abrupt alteration of thyroid hormone homeostasis in the brain and body may lead acute psychotic illness individuals, so there needs to exercise caution while rapidly adjusting serum thyroid hormone levels in individual with a thyroid disorder.

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**Conflicts of interest**

There are no conflicts of interest.

**REFERENCES**

1. Brewer C. Psychosis due to acute hypothyroidism during the administration of carbimazole. Br J Psychiatry 1969;115:1181-3.
2. Bewsher PD, Gardiner AQ, Hedley AJ, Maclean HC.

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**Table 1: Relation between thyroid hormone and psychosis**

| Date         | Reference range | $T_i$ (87-178 ng/dl) | $T_i$ (6.09-12.3 ug/dl) | TSH (0.34-5.6 uIU/ml) | Treatment with carbimazole | Psychotic symptoms experience | Treatment with olanzapine (mg/day) |
|--------------|----------------|----------------------|------------------------|----------------------|---------------------------|-------------------------------|-----------------------------------|
| April 16, 2016 | 122         | 8.3 (ug/dl)         | -0.001                 | -                    | 10 (mg/day)                | Sleep disturbance              | 10 (mg/day)                       |
| April 23, 2016 | 155         | 11.06 (ug/dl)       | -0.001                | -                    | 10 (mg/day)               | Present                        | Present                          |
| May 03, 2016  | 120         | 10.1 (ng/dl)        | 0.19                   | -                    | 10 (mg/day)               | Present                        | Present                          |
| May 28, 2016  | 96.64       | 8.3 (ug/dl)         | 4.89                   | -                    | 10 (mg/day)               | Present                        | Present                          |
| June 20, 2016 | 94.54       | 7.12 (ug/dl)        | 4.82                   | -                    | 10 (mg/day)               | Present                        | Present                          |
| June 21, 2016 | -           | -                    | -                      | -                    | Stopped                    | Present                        | 10                                |
| August 07, 2016 | -            | -                    | -                      | -                    | Stopped                    | Absent                         | 10                                |
| August 15, 2016 | 90.54   | 8.6 (ug/dl)         | 4.60                   | -                    | Stopped                    | Absent                         | 10                                |

TSH – Thyroid-stimulating hormone
Psychosis after acute alteration of thyroid status. Psychol Med 1971;1:260-2.
3. Irwin R, Ellis PM, Delahunt J. Psychosis following acute alteration of thyroid status. Aust NZ J Psychiatry 1997;31:762-4.
4. Furuhashi Y, Ishikawa M. Acute normalization of thyroxine induced hallucinations and delusions. Int J Clin Med 2012;03:341-3.
5. Iga J, Taniguchi T, Ohmori T. Mood swing from severe depression to mania following acute alteration of thyroid status. Gen Hosp Psychiatry 2006;27:451-3.