Atypical Thyrotoxic Psychosis with Seizure: A Case Report

Mohd Hayat B,[a] Syed Mushtaq S,[a] Smeena S,[b] Riyaz US[c]

[a]*Dept. of Medicine, SMHS Hospital Srinager J&K India.*
[b]*Dept. of Surgery, SMHS Hospital Srinager J&K India*
[c]*Dept. of Medicine, SkIMS Soura J&K India.*

ABSTRACT

Thyrotoxicosis results in alteration in functioning of nervous system in some patients. However, these mental disturbances may be severe in rare cases in the form of manic-depressive, schizoid or paranoid reaction. The pathophysiological basis of these nervous system findings is not well understood.

KEYWORDS: Atypical hyperthyroidism, visual auditory hallucination, acute psychosis.

INTRODUCTION

Patients with acute psychosis are generally evaluated and managed by psychiatrists. Medical evaluation to rule out underlying causes of psychosis (substance-induced psychotic disorder, psychotic disorder secondary to general medical condition, mood disorder with psychotic features, psychotic disorder not otherwise specified (NOS), psychosis associated with personality disorders, epilepsy, Ganser syndrome) is an important part of the initial assessment.[1] Psychosis has been documented as a rare complication of thyrotoxicosis, with a prevalence of around 1%.[2] Hyperthyroidism is commonly associated with fatigue, irritability, insomnia, anxiety, restlessness and emotional lability; marked impairment in concentration and memory may also be evident.[3,4]

CASE REPORT

A 20-year-old unmarried female patient, without any significant past medical history was brought to a psychiatric outpatient department with auditory and visual hallucinations, bizarre behavior, disorganized speech, disorientated with poor attention and seizures (more than two times per week). There was no history of any drug intake, altered bowel habits, cold or heat intolerance, headache, diplopia or weight loss. There was also history of labile mood and suicidal thoughts. General physical examination revealed an average built patient who was conscious, irritable, agitated, with impaired concentration and disorganized speech. Patient was afebrile, blood pressure 110/80mmHg, and pulse 130 bpm regular. Grade I goitre and tremor were noted. There was no thyroid associated ophthalmopathy or dermopathy. The patient's cardiovascular, respiratory and abdominal examinations were normal. Central nervous system examination otherwise was normal with no focal neurological signs and no nystagmus. The patient was diagnosed as a case of psychosis with seizures and was put on mood stabilizers (quetiapine 100 mg bd) and anticonvulsant (phenytoin 150 mg bd). After six weeks of treatment, there was no significant relief of psychosis and seizures, and she was referred to our medical outpatient department for a medical consultation. Further history from relatives revealed that patient had menstrual disturbances, excessive sweating and tremor before the start of the present illness (mental disturbances and seizures). Investigations revealed the full complete blood count, peripheral blood film, renal and liver function tests were all normal. Electrocardiogram showed sinus tachycardia, and chest X-ray was normal. Ultrasound of the neck showed a normal-sized thyroid gland. Magnetic resonance imaging of the brain was normal. Electroencephalogram of the brain revealed a generalised spike, and a slow wave discharge at 3-5 Hz, suggestive of seizures. Thyroid function test showed TSH value <0.01mIU/L (0.5-4.5), serum T_3 value 6.18 pg/dL (2.0-4.4), serum T_4 value 5.02 ng/dL(0.7–1.85) and anti TPO antibodies level 613 U/mL(11-210).

Radioactive iodine I-131 uptake was 20% at 6 hours (6-16%) and 40% at 24 hours (8-25%). A diagnosis of Grave's disease with acute psychosis was made, and she was put on methimazole (20mg daily) in addition to optimisation of the dose of the mood stabilizer and anticonvulsant. After six weeks of the combined therapy, she was reassessed. Absence of tremor and remission of the psychiatric

Corresponding author:
Syed Mushtaq Saif Andrabi
Post graduate, Department of Medicine
SMHS Hospital Srinagar, Kashmir India
Email: dr_mushtaq012@rediffmail.com

function showed TSH value at 2.5 mIU/L (0.5 – 4.5) and T3 value 2.9 pg/dl (2.0-4.4) and T4 value 1.02 ng/dL (0.7–1.85). The patient remained under our regular follow-up and remained well with a euthyroid state clinically and biochemically, achieved by methimazole at a maintenance dose of 15 mg daily.

DISCUSSION

Thyrotoxic psychosis with seizures is not a common occurrence, and not viewed as a single clinical entity. Affective psychoses are more common, with mania being as common as depression; less common forms of thyrotoxic psychosis are schizophreniform psychosis, paranoid psychosis and delirium.[3]

Briefly, these psychotic symptoms can be explained based on the increased circulating levels of thyroid hormone. pamine has been shown to inhibit secretion of TSH. This inhibition is partially blocked by the administration of metoclopramide.[3,6,7] These data would seem to support a possible role for psychosis and elevated dopamine levels, exacerbating or mimicking a hyperthyroid state by suppressing TSH.[3,8] DSM-IV diagnostic criteria require the presence of one or more of the following: delusions, hallucinations, disorganized speech, or grossly disorganized or catatonic behaviour (Table 1).

Table 1: DSM-IV Diagnostic Criteria for Psychotic Disorder Due to a General Medical Condition

A.	Prominent Hallucinations or Delusions
B.	There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct physiological consequence of a general medical condition.
C.	The disturbance is not better accounted for by another mental disorder.
D.	The disturbance does not occur exclusively during the course of a delirium.

These criteria also require an acute onset (less than two weeks). Complete recovery usually occurs within one to three months (depending on the specific disorder), often within a few weeks or days. Only a small proportion of patients with these conditions developed persistently disabling state.[1]

The phenomenon of the psychotic disorder is defined in DSM-IV by further specifying the predominant symptoms. When the diagnosis is used, both the medical condition and the predominant symptom pattern should be included in the diagnosis (e.g. psychotic disorder due to a brain tumor, with delusions).[1,9] Our patient met the criteria for DSM-IV U (auditory and visual hallucination and bizarre behavior).

A small percentage of patients presented with psychosis as first manifestation of thyroid disease.[3,4] It is important to think of thyrotoxicosis as a possible cause of acute psychosis since management will differ, and every psychiatrist needs to be more alert to this possibility. In a report of 18 cases of thyrotoxic psychosis, all the patients were treated with antithyroid drugs and all but one subsequently received radioiodine therapy; the youngest patient (23 years) opted for thyroidectomy.[3] After ablative therapies, overt hypothyroidism should be avoided since it could precipitate a psychiatric relapse.[1,3]

Lack of response to antipsychotic therapy alone and a good therapeutic response, when methimazole was added, was observed in our patient. The good response of the psychiatric features following normalisation of the thyroid function test and the small dose of antipsychotic required are also good arguments for thyrotoxicosis as the underlying cause of acute psychosis in our patient.

CONCLUSION

We recommend both clinical examination of the thyroid gland and serum TSH measurement for every patient with acute psychosis in the second week, because thyrotoxicosis is a potentially reversible cause of acute psychosis. Moreover, only combined therapy (antipsychotic drugs and antithyroid drugs) are effective in thyrotoxic psychosis. An ablative therapy (radioiodine or surgery) for thyrotoxicosis is usually recommended, but only when euthyroidism is achieved and acute psychosis is remitted.

REFERENCES

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (DSMIV-TR). 4th ed, Washington DC. American Psychiatric Press, 2000.
2. BEW Brownlie, AM Rae, JWB Walshe, JE Wells. Psychosis associated with thyrotoxicosis – thyrotoxic psychosis. A report of 18 cases, with statistical analysis of incidence. Eur J Endocrinol 2000;142:4
3. Gagliardi JP, Clary GL. Treatment of Thyrotoxicosis – Induced Psychosis. Psychopharmacol 2002;36:7-13.
4. PC Whybrow, M. Bauer. Behavioural and psychiatric aspects of thyrotoxicosis. In: Braverman, RD Utiger (Editors), Werner & Ingbar's The Thyroid, A fundamental and clinical text, Ninth edition, LE Lippincott Williams and Wilkins 2005; 644-50.

5. Besedovsky HO, Rey AD. Immune-neuro-endocrine interactions: Facts and hypotheses. Endocr Rev 1996;17:64-102.
6. Coiro V, Volpi R, Cataldo S. Dopaminergic and cholinergic involvement in the inhibitory effect of dexamethasone on the TSH response to TRH. J Investig Med 2000;48: 133-6.
7. Samuels MH, Kramer P. Effects of metoclopramide on fasting-induced TSH suppression. Thyroid 1996;6:85-9.
8. Nemeroff CB, Bissette G, Manberg PJ, et al. Neurotensin-induced hypothermia: evidence for an interaction with dopaminergic systems and the hypothalamic – pituitary-thyroid axis. Brain Res 1980;195:69-84.
9. Lazarus A, Jaffe R. Resolution of thyroid induced schizophreniform disorder follow ing subtotal thyroidectomy: a case report. Gen Hosp Psychiatry 1986;8(1):29-31.