CASE REPORT: GRAVE'S DISEASE PRESENTING AS PARANOID SCHIZOPHRENIA

S. K. SINGH 1
A. HATWAL 1
J. K. AGARWAL 1
H. S. BAJPAI 2
I. SHARMA 1

SUMMARY

The case of a 37 year old male is described who initially presented as paranoid schizophrenia unresponsive to anti-psychotic drug treatment and subsequently developed features of Grave's disease. Treatment with carbimazole alone improved his psychiatric symptoms.

Psychological stress has been implicated in the pathogenesis of thyrotoxicosis (Brom, 1936) and some patients of thyrotoxicosis have been reported to develop frank paranoid schizophrenia without previous history of psychosis (Greer and Parsons, 1968, Kamalana and Holms, 1968). Rarely it may be the primary presenting feature of thyrotoxicosis (Champman and Maloff, 1956; Wohl and Shuman, 1957). A case of thyrotoxicosis is being reported whose primary presentation was schizophrenia and who improved with antithyroid treatment alone.

Case Report

A 57 year old male presented with a history of psychotic symptoms of two years duration at the out-patient clinic of the university hospital. He was diagnosed paranoid schizophrenia according to ICD—9. For the past one year he was being treated, with haloperidol and chlorpromazine, without any significant improvement. He then developed prominence of eyes and weight loss. On direct enquiry he admitted having increased perspiration and difficulty in getting up from sitting posture. Patient was then referred to the endocrinology unit for re-evaluation.

On examination the patient was afebrile with a regular pulse rate of 104/minute and blood pressure 120/80 mmHg. He was thin built (weight 48 Kg, height 1.60 meters), had bilateral ophthalmopathy with chemosis, proptosis and asymmetrical restriction of eye movement in all the directions of gaze. Visual acuity, fields and optic fundi were normal. He had a soft diffuse goitre without bruit. Neurological examination revealed proximal muscle weakness in all the extremities and fine tremors of tongue and hands. On psychiatric evaluation he had a depressed affect persecutory delusions, some decrease in psychomotor activity and impaired insight. There was no past or family history of psychiatric illness.

Investigations revealed a normal leukocyte count and a corrected erythro-
cyte sedimentation rate of 38 mm per hour (Westergren). Electrocardiogram showed sinus tachycardia and chest roentgenogram was normal. His serum thyroxine \( (T_4) \) level was 303.8 nmol/l (normal: 51—142), triiodothyronine \( (T_3) \) level was 4.91 nmol/l (normal: 1.2-3.4) and thyroid stimulating hormone (TSH) level was undetectable (normal: 10 IU/l). Fine needle aspiration cytology of thyroid revealed follicular cell hyperplasia with lymphocytic infiltration, suggestive of hyperactivity of the gland. Computed tomography (CT) scan of the orbit showed hypertrophy of the extraocular muscle.

Antipsychotic drugs were withdrawn and the patient was treated with carbimazole 45 mg daily and propranolol 40 mg twice a day. Ophthalmopathy was treated with oral prednisolone 40 mg daily (in divided dosage) along with other supportive measures. A substantial improvement was observed in the symptoms of thyrotoxicosis as well as in psychosis within a fortnight of starting the treatment. Further improvement was observed at 6 weeks follow up and at the end of 12 weeks there was total remission of thyrotoxicosis and psychosis.

**Discussion**

Many investigators (Chapman and Maloff, 1956; Wohl and Shuman, 1957) have noted the appearance of acute brain syndrome (toxic exhaustion psychosis and acute delirium state) in hyperthyroid patients. The concept of latent psychosis precipitated by thyrotoxic stress is interesting and plausible but the underlying pathophysiology is not well understood (Burslen, 1961). Alternatively, psychological stress may precipitate thyrotoxicosis (Brom, 1936).

In the present case, parallel improvement of psychiatric symptoms and thyrotoxicosis with antithyroid treatment suggests that initiality subclinical thyrotoxicosis was responsible for the manifestation of psychosis. The question whether emotional disturbance precipitate thyrotoxicosis or vice-versa needs to be re-evaluated in this light. This case report highlights the importance of careful screening of patients attending psychiatric clinics to detect subclinical thyrotoxicosis.

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