PARANOID PSYCHOSIS IN MYXOEDEMA: A CASE REPORT

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SUMMARY

A case of paranoid psychosis in myxoedema is described. Though depression is commonly encountered in myxoedema, the disorder can also manifest itself with schizophrenic features; this can lead to problems in diagnosis and management.

INTRODUCTION

Psychiatric symptoms associated with hypothyroidism are well recognised. The common mental manifestations are slowness and difficulty in apprehension, thought and action; psychomotor retardation with indecision and listlessness suggestive of a depressive syndrome.[1,2] What is perhaps less well recognised is that patients with hypothyroidism may also present with marked irritability, paranoid ideations, delusions of persecutions, and hallucinations; a picture that suggests paranoid psychosis. A number of cases were first described by Asher under the heading "myxodematous madness".[3]

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In this report we describe a patient with primary hypothyroidism who manifested a paranoid psychotic state. Our purpose is to illustrate that a paranoid state (psychosis) can be seen in patients with hypothyroidism and to suggest to clinicians that women presenting with paranoid psychosis be screened carefully to exclude hypothyroidism.

CASE REPORT

Miss K., a 32-year-old unemployed Indian woman, began to feel depressed with occasional aggressive outbursts following an operation for (L) ovarian cyst in 1970. The surgery was uncomplicated and recovery uneventful. Because of her mental symptoms she stopped work as a salesgirl and stayed home with her mother.

In 1972 she was noticed to have gradually become more depressed, irritable, withdrawn and for the first time she began to entertain persecutory (paranoid) ideas involving members of her family. In 1977 on account of worsening behaviour problems she was admitted to the University Hospital, Kuala Lumpur. A diagnosis of primary hypothyroidism with psychosis was made and she was treated with L– thyroxine and Haloperidol. Her condition was reported to have improved following treatment but follow-up was erratic and she subsequently defaulted medication.

For about a year before she was admitted to the General Hospital, Kuala Lumpur in June 1982, she became more withdrawn, neglected her personal

hygiene, agitated, combative and paranoid; she gained considerable weight and menstrual flow was said to be heavy, at times lasting for two weeks.

She presently stays with her elderly mother who is described as overprotective, overindulgent and prevents patient from social contacts. Her father with whom she was very close died following a short illness in 1962 when she was about 12-years-old. Miss K. was described as a quiet person, somewhat reserved with no close friends.

The physical examination revealed that Miss K. was obese, had marked pallor and had very dry and coarse skin. There were patches of vitiligo and xanthomata at both achilles tendons. Her pulse was 70/min, regular and the blood pressure was 100/60 mm Hg. Her ankle reflexes were delayed; otherwise, the examination findings were unremarkable. The results of all laboratory tests were within normal limits, except for the haemoglobin level and the thyroid function tests. Her haemoglobin level was 6.2 g% and peripheral smear showed a picture of mixed iron and folate deficiency anemia. Her serum thyroxine was 19 nmol/L (normal range, 64 – 167 nmol/L) and her serum thyroid stimulating hormone (TSH) level was 24.5 μTU/ml (normal range, 1 – 7.3 μTU/ml). An ECG recording showed low voltage QRS complexes and a chest x-ray revealed slight enlargement of the heart.

On mental status examination, Miss K. was noted to be hostile, irritable and harbouring persecutory delusions. She felt that her family members, in particular her elder brother, were planning to harm her. She claimed that she had frequent visions of her dead father. She felf that there was nothing wrong with her, showed no insight and judgement was impaired.

DISCUSSION

The clinical manifestations in this patient which were predominantly psychiatric may present difficulties in accurate diagnosis. The insidious onset of the mental changes may lead one to attribute the altered mental state to environmental (psychosocial) causes.[4] Patients may experience fluctuations in their mental state for long periods before the diagnosis of hypothyroidism is established.[4] In addition to a careful physical examination thyroid hormone studies, including TSH levels and specific evaluation for autosomal and microsomal antibody titers are necessary to establish the diagnosis.

The management includes gradual replacement of thyroid hormone and neuroleptics to control psychotic behavior. Most patient will respond favourably; however a small proportion may show residual cognitive impairment and continued irritability.[4] Therefore persistent psychiatric symptoms cannot be assumed to be psychogenic in origin and no longer related to CNS impairment by thyroid deficiency. In this patient replacement thyroid hormone was gradually increased from 100 to 300 mg daily and neuroleptic medication had to be given to control the psychotic process. She responded to the treatment within two to three weeks and made substantial progress, both physically and mentally. As to the cause of the hypothyroidism, the endocrinologist who reviewed this patient was not certain but felt it could be autoimmune.

Miss K. was also severely anemic (Hb was 6.2 g% on admission). This was perhaps as a result of severe dietary restrictions placed on the patient upon the advise of traditional healers and also secondary to chronic hypothyroid state. The haematologist felt that the anemia was mainly due to iron deficiency. The patient responded favourably to iron preparation and folic acid.

In view of the increasing incidence of thyroiditis[5,6] clinicians are more likely to encounter patients with this disorder who present with mental changes.[4]

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