Case report

DOI: https://doi.org/10.4038/sljpsyc.v15i2.8519

A case of reversible psychosis due to myxoedema madness with apparent euthyroidism

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SL J Psychiatry 2024; 15(2): 72-74

Keywords: hypothyroidism, myxoedema madness, reversible psychosis

Introduction

Triiodothyronine (fT3), the active form of thyroid hormone, acts as a neurotransmitter and binds to nuclear receptors which are widely distributed in the brain (including the amygdala, hippocampus and limbic system). Psychiatric manifestations of hypothyroidism such as depression and anxiety can be attributed to the effects of these. Rarely does it come present with mania and psychotic symptoms as 'myxoedema madness'(1,2). This case demonstrates biochemical hypothyroidism presenting for the first time with manifestations of psychosis in an apparent physical state of euthyroidism.

Case report

A sixteen-year-old female was admitted to the adolescent psychiatry unit with the first presentation of hearing voices, suspicions, and social withdrawal in one month. There were multiple third-party whispering voices of both genders accusing and scorning her of being a demoness. There were sensations of invisible hands and bodies fondling and deep genital pain in wakefulness and sleep. The patient experienced bruxism leading to uneasiness over the spine.

Strong nonfactual beliefs of neighbours devising evil spiritual curses bonding her with an incubus demon were present. The persecutory voices also embarrassed her about her body image while beliefs that others observing such changes resulting from the incubus lingered. This resulted in social withdrawal leading to isolation.

There were neuro-vegetative symptoms of anorexia and insomnia which were attributed to impregnation of the incubus. She attempted self-exorcism by ingestion of acaricide permethrin in the absence of suicidal intent with the ideation of breaking the curse for healing. She denied persistent low mood but had been determined to fight against the voices and bodily sensations. Failed attempts of poisoning made her and the family resort to an exorcist, who during the ritual physically assaulted even burning. The patient had an absent pain reaction to these with the perception that the spirits departed her body. This was short-lived resulting in distress and helplessness leading to the consumption of permethrin with a suicidal intent. The patient was euthyroid in the absence of coarse skin, cold intolerance, lethargy etc.

There was no past medical history of endocrine or neuro-epileptic disease or psychoactive substance usage. She was the third child with no perinatal or early childhood adverse events. The patient was shorter than her siblings, peers and parents for that age. There was age-appropriate achievement of cognitive milestones. She was described as below average performance in academics. Her interactions with family members and school friends were unremarkable. There was no family history of endocrine disorders, schizophrenia or mood disorders. Her premorbid personality was described as aloof, quiet and shy with a preference to associate with only a few close associates.

The mental status examination demonstrated gross neglect of personal hygiene with apathy. She was conscious and oriented in time, place, and person. There was poor eye contact, with difficulty in establishing initial rapport. The speech was in monosyllables or gestures until rapport was established. Thereafter speech was spontaneous and rational with normal tone, volume and rate although less reactive with blunted mood. Suicidal ideation was present with delusions of persecution, passivity, possession and pseudocyesis. There was no demonstration of delusions of control or thought alienation. Second and third-person auditory, and tactile hallucinations, along with reflex hallucinations of synaesthesia were present. She did not believe she had a psychiatric disorder warranting admission or medication.

Her weight was 33 kg while her height was 1.33 m (BMI 18.75). The mid-parental height was 1.6 m. There were no dysmorphic features except for short stature. 2nd degree burns on the palms of her hands with multiple abrasions with scar formation were a pain. She was not in pain. The

pulse was regular and normal in character of 72/min while the blood pressure was 100/70 mmHg. She did not have features of hypothyroidism or goitre. Secondary sexual characteristics (including breast and external genitalia) were age-appropriate (Tanner stage IV). The basic investigations are shown in Table 1. There was evidence of primary hypothyroidism. Further, the altered lipids could be attributed to the same.

She was commenced on levothyroxine 50 mg/day. Risperidone 0.5 mg nocte commenced to alleviate her psychotic symptoms. The patient demonstrated significant improvement in mood and social interaction. She had returned to normal socialisation with family and peers. The psychotic symptoms resolved completely with biochemical euthyroidism (TSH -3.8 mU/l, fT_4 -101 ng/l). The antipsychotic was tailed off completely.

Discussion

This reports near loss of life associated with subacute psychosis, due to several gaps in identification of adolescent hypothyroidism. It further highlights the detrimental consequences of not identifying and treating hypothyroidism in a child observed with a low IQ and poor scholastic performance. Manifestation of mental and behavioural disorders due to brain damage and dysfunction and to physical disease, organic delusional (schizophrenia-like) disorder (F06.2, ICD 10) due to hypothyroidism was observed in the patient (3). Lack of parental awareness resulting in seeking traditional ritualistic remedies rather than medical treatment led to physical abuse of the child with permanent stigmata. Health service providers should be more vigilant and geared to identify hypothyroidism even in the absence

Table 1. Investigations of the patient at presentation

Investigation	Value (Normal range)
White blood cell count	9.3 x 10^9/l (4.5-11 x 10^9) Neutrophils 69.6%, lymphocytes 20.4%
Haemoglobin	11.5 g/dl (12-15)
Platelets	324 x 10^9/l (150-400 x 10^9)
Serum creatinine	78 µmol/l (74-110)
Blood Urea	3.1 mmol/l (2.8-7.2)
Serum Na	136.2 mmol/l (135-145)
Serum K	3.8 mmol/l (3.5-5.1)
Serum Calcium	2.4 mmol/l (2.02-2.6)
Aspartate aminotransferase	23.2 U/l (<50)
Alanine aminotransferase	26.3 U/l (<50)
Total Bilirubin	20 µmol/l (5-21)
Direct Bilirubin	3.2 µmol/l (0-3.4)
INR	1.01
Alkaline Phosphatase	190 U/l (<150)
Non-Contrast CT Brain	Normal
Urine toxicology screen	Negative
Random Blood Sugar	135 mg/dl (100-180)
Thyroid-stimulating hormone (TSH)	100 mU/l (0.40-4.0)
Free thyroxine (fT_4)	20 ng/l (77-155)

of physical signs and symptoms and to start treatment as early as possible, to minimize the lapses in services, and to mitigate harm.

Primary hypothyroidism requires replacement of levothyroxine 1.6 µg/kg. TSH levels should be commenced. Successful treatment in our patient was noted. Neuro-psychiatric sequelae of hypothyroidism usually take longer to resolve although psychotic symptoms of delusions and hallucinations usually remit in 1 week of commencement(4). Adding antipsychotic medications leads to early remission of psychosis(5). Atypical antipsychotics administered in low doses are well tolerated. This was compatible with our patient as with previous case reports, and we were able to stop antipsychotic medication within 3-6 months.

Conclusion

Hypothyroidism presents with various forms of neuropsychiatric signs and symptoms. It is therefore imperative that psychiatrists are aware of these myriad presentations, appropriate and early identification, assessment of thyroid function, liaison medical teams, and treatment options.

Author Contribution

CAA was a registrar/ trainee in psychiatry while SWK was the Consultant Child and Adolescent Psychiatrist of the National Institute of Mental Health, Sri Lanka at the time of the presentation of this patient. FHDSS was the Consultant Physician at the same institution. All three individuals were involved in the management of the patient and writing up this manuscript.

Acknowledgement

We extend our appreciation to the patient and her parents for providing us consent to publish this manuscript.

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