A Case of Thyroid Disease
A Lesson in Orthomolecular Medicine

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Abstract

A patient is presented who became psychiatrically ill without the classical findings of hypothyroidism and yet who was suffering from thyroid deficiency. The lesson to be drawn from this case is that Orthomolecular physicians are physicians first, and practice Orthomolecular medicine within the bounds of what should be general medicine.

Introduction

Thyroid disease has long been known as a cause of psychiatric disease[1]. What is less well known is that the diagnosis may be far from obvious on clinical grounds and can often be missed, sometimes for considerable periods of time[2]. Yet the diagnosis can be easily made if thyroid screening tests are routinely used on psychiatric patients[3].

An illustrative case is presented.

Case Report

A 14 year old girl, from a large city nearby, presented to my office with a six month history of talking to herself, becoming increasingly withdrawn, accompanied by laughing and crying inappropriately, a lot of anger, insomnia, fatigue, and panic attacks in crowds. Two weeks prior to being seen she went into a profound weeping spell, at which point she was taken by her mother to a Crisis Centre where the staff were "not able to help her too much".

The patient herself complained of being "pushed", but could not explain what she meant by this. She had bad memories of physical abuse by her deceased father, whom she hated, and yet had guilt feelings about this. She denied sexual abuse. She asked: "Is it alright if I cry on my own". She expressed many paranoid ideas. She was insomnic and "dyssomnic" (would waken through the night after falling asleep), with a poor appetite, although liking fruit.

Her memory was highly variable and her ability to concentrate had markedly declined. She admitted to both auditory and visual hallucinations. She felt as if she was being poked in her genitals, and felt "confused about morals". She felt frightened all the time. She exhibited marked dyschronia. She was indecisive. If she tried to work out a problem a voice would argue with her.

Her general health was quite good with no complaints of intolerance of extremes of temperature. She had a mild degree of achondroplastic dwarfism. There had been a suspicion of hydrocephalus at one stage. She was not taking any drugs, licit or illicit. There was no history of allergies.

Her past history included a congenital fine motor dyscoordination (which had led to her being teased by her peers in childhood). A tonsillectomy had been carried out three years before.

Both paternal and maternal grandmothers were allegedly schizophrenic and her father had been an alcoholic.

She lived at home with her mother. There were no siblings. Her school performance was described as "outstanding". Neither she or her mother smoked or consumed alcohol.

Examination revealed a strikingly attractive looking girl with very mild achondroplastic dwarfism. In particular, her eyebrows were thick, and luxuriant. There was no swelling of her limbs. Heart and lungs were normal, as was her abdomen. She was fully conscious and oriented in Time, Space, and of Person. Cranial nerves were intact. Muscle power was normal, but there was some fine motor dysco-ordination. No tremor was observable. Sensation was normal, as was proprioception. She would not co-operate with assessment

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of her reflexes.

But she was clearly psychotic with flights of ideas and neologisms. She would frequently burst into tears for no apparent reason. She showed a lot of acquired mannerisms.

I administered the Hoffer-Osmond Diagnostic Test which gave the following scores:

Total Score	185
Perceptual Score	42
Paranoid Score	14
Depression Score	17
Short Form	17

Feeling that this gave ample justification for the diagnosis of schizophrenia, and having no opportunity for further laboratory testing at the time, I placed her upon a hypoglycaemia type diet, and prescribed Niacinamide and Ascorbic acid, both 1,000 milligrams three times per day, and Pyridoxine 500mg per day. I felt at the time her prognosis was good and advised her and her mother accordingly.

They returned to their home city the same day.

Some weeks later, however, I was telephoned by her mother who informed me that she was making no progress at all and was not complying with the treatment. I saw her again a month after the initial visit and confirmed this account. A complicating factor was a chelazion, for which her own doctor had prescribed "Duricef" 500mg twice daily, and which was almost clear.

I admitted her to our local community hospital right away.

I placed her upon a strict hypoglycaemia diet, continued the niacinamide, ascorbic acid and Pyridoxine as before, added thiamine (vitamin B_1) 500mg/day, and temporarily introduced thioridazine (the most benign of the major tranquilizers) at a dose of 100mg three times per day.

And now I could investigate her further.

Haematology showed a haemoglobin of 13.6 g/dl., ESR of 44 mm/hr., and normal white cell count and differential count. Liver function tests were normal. Serum magnesium, zinc and copper were normal. A six hour glucose tolerance test gave a fasting level of 75 mg/dl., rising to 119 at the half hour, 151 at one hour, 197 at two hours, 152 at three hours, 136 at four hours, 105 at five hours, and 84 at six hours, which I interpreted as showing a lag curve in early transition to diabetes mellitus.

Total T4 (tetraiodothyronine, one of the thyroid hormones) was 0.5 micrograms per decilitre (normal range 4.5 - 12).

Accordingly I prescribed 1-thyroxine 0.1 mg/day, a small dose to avoid precipitating her into a deterioration of her psychosis[4 5], after blood was drawn for further thyroid tests.

These tests showed T3 uptake of 37.4% (normal 30-40), thyroxine was 1 micro-gram/dl. (normal 4.5-11.5), free thyroid index was 1.1 (normal 5-11), and thyroid stimulating hormone was greater than 144 international units/ml (normal 1.0-5.5). This confirmed a profound hypothyroidism with a failure of the thyroid gland to respond adequately to thyroid stimulating hormone.

By the time these results had come in it was clear that the patient was having no adverse response to the small initial dose of thyroid hormone, and I doubled the dose. In the meantime I had tailed off the thioridazine and stopped it eight days after admission because it was no longer necessary.

Four days later I discussed the situation with her in great detail and attempted to place her in the "Medical Model". I also encouraged the nurses to direct their attention to her behaviour, and asked them to encourage and commend behaviour and activity appropriate to a fourteen year old girl. I asked them to express gentle disapproval of inappropriate behaviour.

Eighteen days after admission she was behaving much more appropriately but was still not clear of her symptoms. I doubled the thyroxine dose again to 4.0 mg/day.

Four days later she was obviously improving further and accordingly I discharged her with a prescription to continue her thyroxine at the above dose, and also to continue the megavitamins. The dietician gave her and her mother detailed dietary instructions. And I requested them to get in touch with their regular doctor to secure follow-up of the treatment of her

hypothyroidism.

That Christmas I received a card from her mother thanking me "for restoring my reason for living".

Discussion

Jagadeesh, et al, stated: "Myxoedema as a cause of psychosis is well known, though not commonly seen"[6]. They went on to discuss some of the possible mechanisms for this effect. For example, in myxoedema there is a reduction in cerebral blood flow, and an accompanying reduction in oxygen and glucose consumption. There is also a decreased receptor sensitivity to catecholamines with a compensatory increase in catecholamine concentrations. This latter effect could explain the onset of psychosis when thyroid hormone replacement therapy is begun[4 5].

Myxoedema presents as motor, emotional and cognitive retardation, with speech changes, intolerance of cold, slow reaction times and slowing of reflex function. It is commonly accompanied by obesity, non pitting oedema (usually of the lower limbs, but can also involve the upper limbs, the trunk and the head, particularly the features) — the origin of the name "myxoedema" — dry, scaly skin, thinning of the hair, particularly the eyebrows, anorexia, constipation, bradycardia, and hypotension. I did not consider this patient's fine motor dysco-ordination to be a sign of hypothyroidism since it had been a life long problem, nor was it of the character to be expected in thyroid deficiency.

While atypical cases, not presenting as above, are known to occur, they tend to be of a relatively mild degree of underactivity of the thyroid gland. The more severe the thyroid deficiency the more likely the case is to present as myxoedema.

The girl in point was severely thyroid deficient, but showed none of the hallmarks of myxoedema, and, indeed, presented as a florid case of schizophrenia with a disordered glucose metabolism as a contributory factor. If anything she could have been said to possibly present as hyperthyroidism. It was only the thyroid screening tests[7] that revealed the underlying problem. Perhaps the accumulation of catecholamines was the confounding factor in her picture. Certainly such an accumulation would explain both the psychotic features and the disordered glucose metabolism.

In any event, once the true diagnosis was established it was relatively easy to treat her by replacing the missing hormone in progressively increasing doses[6 8]. I believe that her management was made easier by giving the other, more typically Orthomolecular, aspects of therapy as well. A particular effort was taken to place her within the "Medical Model", the "Sick Role" in particular, in order to ensure her co-operation, compliance and responsible participation in her management. The change of her mental status from abnormal to normal was likely to be a severe stress. The diet and "megavitamins" are well known to help with coping with stress. But they also stabilize the metabolism of catecholamines, and, I believe, helped to prevent an exacerbation of her psychosis[4 5] which might have come about through the change in the metabolism of these agents as her thyroid function returned to normal.

No other case of atypical hypothyroidism, that is without myxoedema, presenting as a psychotic, schizophrenia-like illness is recorded in the medical literature to which I have access. Without screening for thyroid disease the diagnosis could not have been established since her presentation was so atypical. The question that arises is: How many patients, with psychiatric disease, remain ill because of failure to screen even when they do not appear, on clinical grounds, to have thyroid disease?

McGaffee, et al, made the plea: "thyroid function screening is recommended for patients presenting with depression, psychosis or organic mental disorder."[9] All too often this is ignored as mental health workers, including most psychiatrists, pursue their own paradigms and models of mental illness in the patients that they see, and resort to rationalization to explain why their patients do not improve.

The Orthomolecular physician, dedicated more than any other kind of mental health worker, to making as accurate a differential diagnosis as possible, is the ideal person to seek out and find such cases. For this reason the Orthomolecular

physician is, contrary to the perception of detractors, firmly in the mainstream of medicine.

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