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Case Report

CASE REPORT

Late onset radioiodine-induced hypothyroidism presenting with psychosis 14 years after treatment: a rare case

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Abstract

Radioiodine treatment-induced hypothyroid psychosis is uncommon. Our literature search shows only three cases of hypothyroid psychosis developed within 3 months after the radioiodine treatment. Our case represents the first case of radioiodine-induced hypothyroidism presenting as psychosis much later (14 years) after the radioiodine treatment. A 60-year-old Chinese lady, with long-standing primary hypothyroidism due to the radioiodine treatment performed 14 years ago, presented with a 1-week history of hallucination, delusion and agitation. She was not on thyroid replacement. Thyroid function test done 14 years ago and again upon her admission to our facility was consistent with primary hypothyroidism. General blood tests and brain imaging were unremarkable. Her psychotic features resolved within 1 week with thyroid replacement and 9 days of antipsychotics. No further relapse of psychosis was noted. This emphasizes that radioiodine-induced hypothyroidism can go unnoticed for many years and present much later solely as psychosis.

INTRODUCTION

About 5–15% of hypothyroidism presents as psychosis, so thyroid dysfunction should be considered in patients presenting with psychosis [1].

CASE REPORT

Madam X, a 60-year-old Chinese lady, was admitted with a history of 1 week of altered mental status after a fall. Her daughter noted her to be withdrawn, agitated, having an auditory hallucination of her children asking her to meet them at a café and a visual hallucination of a doctor pressing the call bell. She was a housewife who managed housework and grocery shopping well before the fall. She had no past history or a family history of psychiatric disorders. There was no history of substance abuse.

Her physical examination was unremarkable. Initial investigations showed a normocytic, normochromic anaemia, normal liver enzymes and renal function with no gross electrolyte abnormalities. Computed tomography of the brain, chest X-ray and urinalysis were unremarkable.

Subsequent investigations (Table 1) included a normal magnetic resonance imaging of her brain and thyroid function test that showed primary hypothyroidism.

Further history from the patient's daughter revealed that Madam X had received radioiodine therapy for hyperthyroidism 14 years ago. She has since then not taken any thyroxine replacement or had any follow-up. The last available thyroid function test done 14 years ago showed primary hypothyroidism: free thyroxine 9.2 pmol/l (9.6–19.1) and thyroid stimulating hormone (TSH) 38.7 mU/l (0.36–3.25). She was not noted to have hypothyroid symptoms by her daughter.

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Table 1: Summary of laboratory and imaging result of our patient during admission

Investigation	Result	Reference
Free thyroxine (pmol/l)	<2	8–21
TSH (mIU/l)	45.54	0.34–5.6
Free T3 (pmol/l)	2.3	3.5–6.0
Anti-thyroperoxidase antibody	Negative	<50 IU/ml
Anti-thyroglobulin	<0.9 IU/ml	
CT brain	No acute intracranial haemorrhage or skull fracture	
MRI brain	No acute infarct or intracranial haemorrhage	
0 min cortisol	161	nmol/l
30 min cortisol	573	nmol/l
60 min cortisol	704	nmol/l
ACTH	6.6	0–10.2 pmol/l

Table 2: Thyroid function test trending

	Free thyroxine (pmol/l)	TSH (mIU/l)
12 July 2014 (on admission)	<2	45.54
4 September 2014	7	22.58
20 November 2014	9	15.43
16 February 2015	19	2.03

Our psychiatrist concluded that Madam X was psychotic with delusions, thought disorder and hallucinations. Olanzapine and lorazepam were started for behaviour control. Levothyroxine was started at a dose of 25 µg daily and after 3 days, it was increased to a dose of 50 µg daily. Her psychotic features resolved within a week.

Upon discharge, she was advised to continue taking the olanzapine and thyroxine. She however did not take olanzapine at all. She was reviewed by the same psychiatrist twice (2 weeks and 2 months) after her discharge. No relapse of psychosis was reported by the patient and her daughter without antipsychotics. She was discharged from the psychiatrist follow-up.

Six weeks after discharge, the thyroid function test showed improvement and levothyroxine dose was increased to 75 µg daily and to 100 µg daily after another 3 months. The thyroid function test subsequently became normal (see Table 2 for thyroid function test trending).

DISCUSSION

Our literature search identified only three cases of radioiodine-induced hypothyroid psychosis. Catherine [2] reported the first case in 1970. She described a hypothyroid patient: FT4 5.1 pmol/l (normal 9–19 pmol/l), TSH 38 mU/l (normal range 0.35–4.94 mU/l), presented with 2 weeks of psychosis that started nearly 3 months after the radioiodine treatment for her Graves' disease [2]. Bethell [2] reported a similar case in 1970, but the thyroid function test result was not available. In both cases, psychosis developed within 3 months after the radioiodine therapy.

Freeman [2] described another case of radioiodine-induced psychosis. The patient developed 'paranoid delusions' the day after receiving radioiodine treatment for thyroid cancer (following a partial thyroidectomy) [2]. The patient's TSH was 6.7 mU/l (borderline elevation, free thyroxine level not stated) 1–2 months prior to radioiodine treatment [2]. It was therefore unclear if the delusions were related to hypothyroidism [2]. Freeman's case was interesting, but the rapid onset of psychosis post radioiodine was very different from our case.

Our patient developed hypothyroid psychosis 14 years after the radioiodine treatment without other hypothyroid symptoms. Collateral history from the patient's daughter who lived with her did not reveal pre-existing cognitive impairment or other psychiatric symptoms prior to the onset of psychosis. The patient had been visiting her general practitioner for intercurrent minor illnesses, but was not noted to have hypothyroid features. This showed that hypothyroid psychosis can present much later after the radioiodine treatment given for hyperthyroidism. This is different from the previous cases reported by Catherine, Bethell and Freeman [2].

Psychosis usually develops several months or years after the development of hypothyroid physical symptoms [1]. However, like our case, it is possible to have psychiatric symptoms only as the presenting complaints with no physical features [3]. Some hypothyroid patients are even first misdiagnosed with a primary psychiatric disorder [3]. Considering thyroid dysfunction as one of the causes of psychosis is therefore prudent [1].

Thought disorders can be seen in clinical and subclinical hypothyroidism, indicating that psychosis may not be related to the degree of thyroid dysfunction [1, 3]. This explains the late onset of psychosis of our patient after being hypothyroid for 14 years.

Thyroid replacement usually leads to the gradual improvement in hypothyroid psychiatric symptoms, but rapid improvement within 1 week has been described [2–4]. Moeller *et al.* [4] mentioned previously 'Elderly patients with vague hallucinations, cognitive deficits and mild depression responded better to thyroid treatment than young introverted patients with explicit delusions'. Earlier remission of psychosis may be achieved with the addition of antipsychotic to thyroid replacement [3]. Initiating atypical antipsychotics at low dose was reported to be well tolerated [3]. Our patient was given a low dose of olanzapine at the same time of thyroid replacement. Her psychotic symptoms resolved within 1 week. She stopped taking the antipsychotic after 9 days. The antipsychotic may have contributed to the rapid remission of her psychosis, but the fact that the psychosis did not recur without antipsychotic use supported the diagnosis of myxoedema madness.

Initiating at high-dose or rapid dosage titration of thyroid replacement may lead to the exacerbation of psychosis [3, 4]. Risk factors of developing psychosis following thyroid replacement initiation include female, levothyroxine dosage of >150 µg daily and a previous history of psychiatric disorder [4]. Our patient was started with a low dose of levothyroxine and titrated up gradually. No relapse of psychosis was reported.

The American guidelines recommend a dose of radioactive iodine (10–15 mCi) sufficient to cause hypothyroidism [5].

10 and 15 mCi result in hypothyroidism in 69% at 1 year and 75% at 6 months, respectively [5]. Pat [6] recommended all patients to be followed up at an endocrine clinic after radioiodine treatment until hypothyroid and they should be on a stable dose of thyroxine. They can consult their general practitioners for subsequent annual thyroid function check [6].

Radioactive iodine-induced hypothyroidism can present as psychosis solely long after treatment. Timely follow-up of post radioiodine treatment is mandatory.

CONFLICT OF INTEREST STATEMENT

None declared.

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ETHICAL APPROVAL

Both authors have contributed significantly to the writing of this manuscript. This is an original manuscript and has not been anywhere and is not under consideration elsewhere.

CONSENT

Patient's consent has been obtained.

GUARANTOR

C.E. is a guarantor of this study.

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