



## CASE REPORT

# Assessing Iodine Status in Frontline Healthcare

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## Abstract

**Introduction:** Iodine is essential for the synthesis of thyroid hormones and both hypothyroidism and iodine deficiency are prevalent worldwide. Assessing iodine status in the individual is difficult. Spot urine iodine measurement, while readily available, is not accurate. However, combining the clinical picture with a brief dietary history along with this simple measurement may give further insight into the likely iodine requirements of an individual. Supporting nutritional deficiency in subclinical hypothyroidism is proposed to be helpful in reducing possible requirements for pharmacological intervention in the future although further studies are needed in this area. This is of particular importance in high-risk groups such as those trying to conceive, or patients with subfertility or recurrent miscarriage.

**Case description:** In this case report a 66-yr-old woman with symptomatic subclinical hypothyroidism presents with a low dietary intake of iodine and a correlating low urine iodine. These three factors taken together suggested a possible iodine deficiency. Replacement of iodine with a safe and moderate amount of iodine (75 µg/day, 50% RDA) via supplement and counselling the patient to increase simple sources of iodine in the diet restored euthyroidism and resolved all symptoms.

**Conclusions:** We propose a simple strategy for the frontline healthcare provider to estimate iodine requirements in an individual-correlating a brief dietary history, low urine iodine and suboptimal thyroid function tests. We recommend that supplementation to increase iodine is conservative and in the short-term only to avoid iodine excess. Dietary intake of iodine should be encouraged to maintain levels thereafter.

## Keywords

Iodine, Hypothyroid, Subclinical hypothyroidism, Pre-conception

[1] while a meta-analysis of European studies has identified a prevalence of 3.05% with over 85% being subclinical [2]. Iodine is essential for thyroid hormone synthesis and hypothyroidism is recognised by the WHO as an iodine-deficiency disorder and a major cause of goitre [3]. Iodine deficiency is prevalent worldwide and not restricted to developing countries. In fact, Europe has the highest percentage of iodine deficiency with 52% of the population having inadequate iodine intake.

It is important to note that the most common cause of hypothyroidism is autoimmune in nature for which anti-TPO levels can be used to support diagnosis in the clinical setting [4]. Iodine excess in autoimmune thyroid conditions can aggravate and so should be avoided in those positive for antibodies [5]. Iodine excess should be avoided in general as this may increase incidence of hypo or hyperthyroidism but this is likely linked to autoimmune pathogenesis or in cases where severe deficiency is treated with a sudden load of iodine [6]. However, restoration of iodine status and nutritional support in clinical practice represents a modifiable risk for disease progression, or an adjuvant to pharmacological treatment in non-autoimmune thyroiditis. Further studies are needed here however.

There are particular groups that may benefit from nutritional support. Pharmacological treatment of subclinical hypothyroidism is not always warranted and may depend on additional risk factors such as age of patient, presentation of symptoms and likelihood of pregnancy [7]. We propose that correcting nutritional deficiencies which may be reducing thyroid function in subclinical non-autoimmune hypothyroidism may slow the progression to overt hypothyroidism and therefore reduce the need for pharmacological intervention, or in any case, be supportive of overall thyroid function.

## Introduction

Hypothyroidism affects 4.6% of the US population



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**Table 1:** Epidemiological criteria from the WHO for assessment of iodine status in a population using urine iodine measurement [3].

Urine iodine µg/L	Iodine Intake	Iodine Status
< 20	Insufficient	Severe deficiency
20-49	Insufficient	Moderate deficiency
50-99	Insufficient	Mild deficiency
100-199	Adequate	Adequate iodine nutrition
200-299	Above requirements	Slight risk of more than adequate intake in the overall population
> 300	Excessive	Risk of adverse health consequences (iodine-induced hyperthyroidism, autoimmune thyroid diseases)
<b>Pregnant Women</b>		
Urine iodine µg/L	Iodine Intake	Iodine Status
< 150	Insufficient	
150-249	Adequate	
250-449	Above requirements	
> 500	Excessive	

Patients trying to conceive or those with subfertility or recurrent miscarriage might be considered a high-risk group. Thyroid dysfunction has been shown to cause sex hormone imbalances, elevated prolactin, anovulation, immune dysregulation, reduced oocyte quality and fertilisation rates in assisted reproduction and increased risk of miscarriage [8]. The American Thyroid Association (ATA) set the TSH threshold in the first trimester of pregnancy at a reduced level of 4 mIU/L although this might be lower in cases of sub-optimal iodine status or if TPO antibodies are present where a threshold of 2.5 mIU/L is recommended [9]. Optimization of iodine status prior to pregnancy may be beneficial to reduce risk of subfertility and recurrent miscarriage and support thyroid function in the ensuing pregnancy.

During pregnancy, maternal thyroid function and delivery of thyroxine via the placenta is essential until foetal thyroxine takes over at approximately 17-19 weeks gestation [10]. Maternal thyroid function and iodine levels have been shown to markedly affect foetal neurodevelopment, learning skills and IQ of offspring, and placental growth which in turn affects foetal growth. Iodine requirements are increased in pregnancy and are unlikely to be met in any country with mild deficiency [11]. Some countries such as Australia and New Zealand have issued supplementation guidelines for all pregnant women at 150 µg per day [12]. However, there is much debate over the risks of supplementation in populations where a proportion of individuals could be replete [11].

In order to support iodine levels in the individual, assessment of iodine status is important although very difficult. Urine iodine is ultimately only reliable as a measurement of the iodine status of a population and there are WHO guidelines to indicate sufficiency and mild, moderate and severe deficiency (Table 1) [3]. For the individual, it has been shown that ten spot urine samples would be required to give an accurate measurement of iodine status [13]. In primary care, this is not practical and there is a major need for accurate biomarkers of iodine status. For the health care provider practising now and trying to estimate iodine status in a patient, a spot

urine iodine may provide some indication, but further information is required from the patient for a more reliable assessment.

To further inform the health care provider, a dietary analysis would be useful but is cumbersome; many physicians do not have access to nutritional supports nor is it practical to analyse extensive multi-day diet diaries with every patient. While there are several lengthy validated food frequency questionnaires (FFQ) which can reliably assess iodine intake [14-16], a short FFQ validated in UK females of child bearing age could arguably be used simply in a clinical setting by any health care professional [17]. However, the results of this FFQ validation showed that the contribution of dietary iodine was calculated as 12% from fish or seafood and 88% from dairy. Therefore, to simplify further, it seems reasonable that any health care provider could simply ask a patient about their consumption of fish, seafood and dairy and reach a broad opinion on their risk for iodine deficiency. Depending on the population and the culture, physicians may also question about use of iodised salt or seaweed products.

In order to more accurately assess iodine requirements in the individual, it may be useful to combine three markers of iodine status; spot urine measurement, simple and quick dietary questioning and thyroid function tests. If all three are sub-optimal, this may suggest increased need for iodine.

## Case Description

A 66-year-old woman presented with symptoms of lethargy, extreme fatigue, weight gain despite efforts to reduce calories, constipation and distension and hot flashes. Onset of symptoms was 2 years prior. She had undergone the menopause at age 50 and had no significant symptoms at that time.

Thyroid function tests indicated mild subclinical hypothyroidism with a TSH of 4.82 (0.27-4.2 mIU/L), free T4 of 14.3 (12.0-22.0 pmol/L). Anti-TPO antibodies were undetectable. Patient also had a mild neutropenia, commonly seen in hypothyroidism [18], but otherwise

normal blood count, liver function tests, kidney function tests, C-reactive protein and fasting glucose and was negative for anti-tTg antibodies.

On questioning, it was apparent that dietary intake of iodine was low. The patient ate on average one small portion of goat's cheese per week but chose not to consume any other dairy. She ate one portion of fish per week and no other seafood, seaweed products or iodised salt. A morning urine iodine was then measured at 22 µg/L.

Given the patient did not wish to include any more dairy in her diet, it was recommended that she increase her fish intake to four portions per week. In addition, a daily supplement containing 75 µg iodine (50% RDA) was recommended for three months while she improved her dietary intake. Additional dietary recommendations were given to support thyroid function such as the addition of milled nuts and seeds to supply minerals such as zinc and selenium. A multivitamin and mineral supplement containing moderate amounts of zinc, selenium, B vitamins, vitamin C and vitamin A was also included for three months. This supplement contained no iodine. Her digestion was supported by increasing fibre intake.

After three months, the patient reported that her energy and wellbeing was markedly improved, she was no longer fatigued, was beginning to lose weight and there was complete cessation of hot flashes. Her digestion had also markedly improved with a daily well-formed bowel movement. Repeat thyroid function tests indicated euthyroidism with a TSH of 2.17 mIU/L and fT4 of 14.12 pmol/L. Her supplement protocol was stopped and after a three-day wash out period, her urine iodine was measured at 132 µg/L. Compliance to the recommendations had been high and the patient was happy to maintain fish intake at 4 portions per week and discontinue supplements.

## Conclusion

Iodine measurement in the urine is unreliable but is the only tool currently available in the absence of reliable biomarkers. In this case, three factors combined allowed the healthcare provider to estimate iodine deficiency: Low urine iodine, low dietary intake based on simple questioning plus suboptimal thyroid function tests (elevated TSH in this case). Simple dietary advice restored iodine intake and a moderate supplement, much less than the RDA, was given while dietary changes were implemented but avoiding the risk of excess given that the measurement protocol described here is inaccurate, and merely an indication. At three months, a repeat assessment indicated adequate status, again using three markers: optimal urine levels, good dietary intake and optimal thyroid function tests.

This strategy is therefore proposed for the frontline health care provider which might indicate greater iodine need in a patient and a safe and conservative dosage

of supplementation in the short term is recommended while improving dietary intakes of iodine. Randomised controlled trials are warranted in this field to determine whether improving iodine status is an effective treatment support in non-autoimmune thyroid conditions.

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