A 30-year-old woman in the UK was referred to the endocrinology team with a goiter, which had been progressively enlarging for the previous 10 months. She described symptoms of fatigue and weight gain and was found to have biochemical hypothyroidism. On examination, there was a large smooth goiter which was clinically larger on the right side.

The patient’s past medical history is complex. She developed intestinal failure in her early twenties and quickly became dependent on total parenteral nutrition (TPN). The patient has been dependent on TPN for six years. One year prior to presentation, a change was made to her TPN feeding regimen, necessitated by the development of manganese toxicity. It led to the withdrawal of a trace element supplement, Additrace.

Investigation
Thyroid function tests showed thyroid-stimulating hormone (TSH) of 8.3 IU/L and T4 of 6.1 pmol/L along with a TPO titer of <28 IU/mL. Five months later, the TSH had increased to 25.0 IU/L and free T4 was 3.1 pmol/L. A total T3 level was checked at this point, which was within normal range at 2.3 (0.9–2.5) nmol/L. A diagnosis of iodine deficiency was considered.

Treatment and outcome
After confirming that the patient was no longer suffering from manganese toxicity, her feeding regimen was changed back to include trace element supplementation with Additrace, containing 16.6 μg of potassium iodide per mL. She received 10 mL per day, which equates to 130 μg of iodine in 24 hours, (the European Society for Clinical Nutrition and Metabolism recommends 70–150 μg per day). Prior to it being stopped, the patient was receiving a lower dose of 5 mL Additrace per day, despite which she had never experienced symptoms of thyroid dysfunction.

Hypothyroidism due to iodine deficiency is rare in developed countries
Cases of hypothyroidism in adults dependent on TPN are rare in the literature (1). However, they have been reported in children. One prospective study found that the UIC of children on TPN decreased significantly as time progressed (2). Cases of severe hypothyroidism have been reported in children on TPN that have been successfully treated with parenteral iodine supplementation (3). This case highlights that adults can successfully be treated in the same manner.

Iodine deficiency in the developing world is the leading cause of hypothyroidism but is rarely encountered in the clinical setting in developed countries due to adequate nutrition thanks to iodized salt. The authors would urge other clinicians to consider iodine deficiency as a plausible diagnosis in patients dependent on artificial nutrition who present with a diffuse goiter and biochemical hypothyroidism with negative thyroid antibodies.

References

A U.S. study warns that patients on long-term TPN may be at risk for iodine deficiency
There is no current consensus among scientific societies regarding the quantity of iodine to be added in adult enteral (EN) and parenteral (PN) formulations. A recent study in the U.S. measured the iodine content of adult EN/PN nutrition solutions (10 enteral and 4 parenteral solutions). The measured and labeled EN iodine contents were similar (range 131–176 μg/L vs. 106–160 μg/L, respectively). In contrast, PN formulas were found to contain small, unlabeled amounts of iodine, averaging 27 μg/L.

Typical fluid requirements are 30 to 40 mL/kg/day for adults receiving total PN (TPN). This means that adults on long-term TPN would require on average 5.6 L PN/day to meet the recommended daily allowance of iodine of 150 μg/day. This volume of PN is far in excess of typical consumption. Thus, U.S. patients requiring long-term TPN may be at risk for iodine deficiency.