

CASE

Our patient was a 73-year-old male diagnosed with recurrent transglottic squamous cell carcinoma (T2N0M0) and had total laryngectomy, total thyroidectomy and bilateral neck dissection done. Oral thyroxine replacement 100 mcg (1.3 mcg/kg/day) daily was started 3 days post operatively. Patient's post operative recovery was complicated with neck wound breakdown with suspicion of pharyngo-cutaneous fistula and was subsequently started on total parental nutrition by day 12 post operation. Patient was referred to endocrine team due to worsening hypothyroidism FT4 7.68 pmol/L (12.0-22.0) TSH 12.8 mIU/L (0.27-4.20). On examination, patient was conscious, alert, GCS full, BP 127/75 mmHg, pulse rate 86 beats per minute, on vacuum dressing over neck wound, reflexes normal, clinically asymptomatic of hypothyroidism. Patient was converted to per rectal administration of levothyroxine. Levothyroxine tablet was crushed and mix with 10 mls of water and 20 mls of lignocaine gel, pushed into rectum with nasogastric tube.

Patient was initially started on 2.6 mcg/kg/day per rectal thyroxine and subsequently increased to 4 mcg/kg/day 5 days later due to the lack of adequate biochemical response. Thyroid function normalized after 3 weeks of therapy, with FT4 18.4 pmol/L (12.0-22.0) TSH 2.80 mIU/L (0.27-4.20).

CONCLUSION

In conclusion, per rectal administration of levothyroxine can be a useful, safe, and effective alternative to oral levothyroxine in conditions precluding oral administration. We should advocate for increased availability of rectal levothyroxine preparations worldwide.

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A CASE OF TRIPTORELIN-INDUCED THYROIDITIS

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INTRODUCTION/BACKGROUND

Gonadotrophin-releasing hormone agonists (GnRHa) therapy has been associated with thyroid dysfunction, including thyroiditis. Triptorelin, a GnRHa, is used as androgen deprivation therapy (ADT) in men with prostate cancer. We present a case of triptorelin-induced thyroiditis in a patient with locally advanced prostate cancer.

CASE

An 83-year-old male with underlying stable prostate cancer presented with an acute transient episode of abnormal behaviour. After completed radiotherapy, he was on 3-monthly SC leuprorelin before recently changing to 3-monthly SC triptorelin. He had received the second dose of SC triptorelin 4 weeks prior to presentation. A plain brain CT ruled out a space-occupying lesion. His renal profile, serum calcium, glucose and dementia workup were normal, except for a deranged TFT [suppressed TSH (0.01 mIU/L, N:0.55-4.78), high fT4 (55.1 pmol/L, N:11.5-22.7), high fT3 (13.0 pmol/L, N:3.5-6.5)]. Baseline TFT taken two years ago was normal. He had no fever, neck pain, dysphagia, respiratory or thyrotoxicosis symptoms. He denied any family history of thyroid disease, recent vaccination, or supplement use, including biotin. He was clinically euthyroid, and there was no evidence of Graves' ophthalmopathy, tremor, atrial fibrillation, or a goiter. Serum anti-TPO antibody was raised (67.2 iu/mL, N<35.0). Lumbar puncture findings were normal and the cerebrospinal fluid anti-TPO antibody was not detected. Technetium-99m uptake scan reported reduced uptake in both thyroid lobes, suggestive of thyroiditis. Hence, a diagnosis of triptorelin-induced thyroiditis was made. Upon discharge, his behaviour normalized and his TFT improved (TSH 0.02 mIU/L, fT4 32.3 pmol/L, fT3 6.7 pmol/L) without antithyroid drugs or glucocorticoids. During clinic review two months after he completed ADT, he was clinically euthyroid and his TFT had normalized (TSH 1.96m IU/L, fT4 12.0 pmol/L).

CONCLUSION

Patients with thyroid autoimmunity are more susceptible to thyroid dysfunction after GnRHa administration, probably due to GnRHa immunostimulatory actions, emphasizing need for TFT monitoring during GnRHa treatment. Those with persistent thyroid dysfunction after discontinuation of GnRHa therapy may require treatment.

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ONE-YEAR AUDIT OF PATIENTS ADMITTED WITH THYROID DISORDERS TO THE MEDICAL DEPARTMENT, SIBU HOSPITAL

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INTRODUCTION

The prevalence of thyroid disorders in Malaysia is around 3.4%. It is the second most common endocrine disorder after diabetes. Organ dysfunction related to thyroid disorders such as hyperthyroidism and hypothyroidism may result in significant morbidity and mortality.