Besides host factor, interferon previously used as a standard treatment in HCV, is known to cause thyroid dysfunction either by direct inhibitory effect on the thyroid gland or immune activation particularly in those with genetic predisposition to autoimmune disease

Direct-acting Antivirals (DAA) targeting specific non-structural proteins of the virus, hinders viral replication. Since it was introduced, there are few studies demonstrating the effect of DAA on thyroid dysfunction.

We report a case of new-onset hypothyroidism in a patient with HCV soon after commencement of DAA.

CASE

A 53-year-old male, former intravenous drug user with Chronic Hepatitis C Child Pugh A, was treated with DAAs (sofosbuvir and daclatasvir) and ribavirin based on viral load and genotyping. He denied preceding hypothyroid symptoms or family history of thyroid disorder. Two months into treatment, he complained of facial puffiness, weight gain and was eventually admitted for heart failure. Biochemical investigations revealed overt hypothyroidism with FT4 <3.2 pmol/L (7.86 - 14.41) and TSH >300 uIU/mL (0.38 - 5.33) with positive thyroid peroxidase antibody. Levothyroxine was started subsequently, however due to poor compliance, he showed poor clinical and biochemical response in HCV viral suppression and thyroid disorder.

CONCLUSION

Hypothyroidism related to HCV infection is a relatively uncommon association but an important one to diagnose, nonetheless. The condition can be part of the extrahepatic viral manifestation or may be treatment related. Supported by previous study that demonstrated similar effect of DAA on the thyroid gland, further larger RCTs are needed to substantiate this association.

EP_A184

CHALLENGES IN THE DIAGNOSIS AND DIFFERENTIATION OF THYROID HORMONE RESISTANCE FROM TSHOMA

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INTRODUCTION

An elevated fT4 with non-suppressed TSH levels may present as a diagnostic challenge resulting in inappropriate treatment. Although rare, resistance to thyroid hormone (RTH) and TSHomas can present in this manner and diagnosis is important to guide management.

CASE

A 43-year-old male was referred from a district hospital for evaluation of atypical chest pain. He was noted to have an elevated fT4 with normal TSH for the past four years and had received carbimazole previously. He reported symptoms of hyperthyroidism including intermittent palpitations, tremor and anxiety. No goitre was noted clinically. Both his mother and maternal aunt had undergone thyroid surgery. After stopping treatment, he had elevated fT4 (28.31 and 19.39 pmol/L) and normal TSH (1.55 and 1.619 m IU/L) performed on two different platforms. Sex hormone binding globulin (22.6 nmol/L), alpha-subunit (0.22 IU/L) and neck ultrasound were normal. Pituitary MRI showed an ill-defined hypo-enhancing nodule measuring 2.0 mm x 2.2 mm x 1.9 mm. Other pituitary hormones were unremarkable. He went on to have a thyrotropin stimulation test which showed an exaggerated TSH response with an 11-fold increase at 20 minutes, supporting the diagnosis of RTH. Genetic testing was not performed due to resource limitations. Subsequently, he was managed symptomatically with beta blockers.

CONCLUSION

When managing discordant thyroid function tests, a high index of suspicion and proper clinical assessment, including laboratory and imaging studies, are needed to ensure precise diagnosis and avoid potentially harmful or unnecessary treatment such as radioactive iodine, anti-thyroid medication or pituitary surgery. Small non-functioning pituitary adenomas are not uncommon. Abnormal imaging needs to be correlated carefully.

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UNVEILING PRETIBIAL MYXEDEMA: A CASE REPORT OF GRAVES' DISEASE

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INTRODUCTION

Pretibial myxedema (PTM), a rare manifestation of Graves' disease, holds importance as it constitutes a component of the classical triad associated with the condition. Historically observed in up to 5% of Graves' disease patients, the incidence of pretibial myxedema has notably decreased, likely attributed to advancements in early diagnosis and prompt initiation of antithyroid therapy.