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SHIFTING SPECTACLE OF THYROID ANTIBODIES: A UNIQUE PRESENTATION

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INTRODUCTION/BACKGROUND
Graves' disease typically manifests with hyperthyroidism. However, the switch between TSH receptor-stimulating antibodies (TSAb) and TSH receptor-blocking antibodies (TSB Ab) is uncommon. We discuss three patients with Graves' disease who switched between hypothyroidism and hyperthyroidism throughout the course of their disease.

CASE
Case A involves a 44-year-old female with initial diagnosis of hyperthyroidism. She was treated with carbimazole for two years and remained euthyroid for a few years without medication. Four years following the diagnosis, she developed overt hypothyroidism requiring levothyroxine. She remained hypothyroid for nine years until her TSH levels trended towards the lower end, hence, she was restarted on carbimazole. She had elevated TRAb [2.0 IU/L, N: <1.75] and anti-TPO [890 IU/ml, N:<9] at screening. Her neck ultrasound showed a goitre with a solitary left thyroid nodule.

Case B involves a 68-year-old male diagnosed with hyperthyroidism [FT4: 140 pmol/L, TSH:0.008 m IU/L]. Twelve months following diagnosis, he developed overt hypothyroidism while on low dose carbimazole and eventually required levothyroxine. Thyroid antibodies were elevated [TRAb 37 IU/L and anti-TPO 534 IU/ml]. His neck ultrasound revealed a small thyroid nodule with benign features.

Case C is a 43-year-old female who presented with overt hypothyroidism [Ft4 9 pmol/L,TSH 95.75 m IU/L] and was treated with levothyroxine. Initial antibodies were elevated [anti-TPO 235 IU/ml, anti-Tg 104.8 IU/ml]. Three years following diagnosis, her TSH levels trended towards the lower ranges and eventually showed overt hyperthyroidism [FT4 28, TSH <0.008]. She was commenced on oral carbimazole. Repeat antibodies were elevated [anti-Tg 30 IU/ml, anti-TPO 218.25 IU/ml, TRAb 23.8 IU/L]. Her neck ultrasound showed multiple subcentimetre thyroid nodules.

CONCLUSION
Graves' disease is characterized by the presence of TRAb, which can exhibit either TSAb or TSB Ab activity. Treatment with anti-thyroid drugs (ATD) such as carbimazole may further trigger the switch to hypothyroidism. Therefore, close monitoring and follow-up are crucial for these patients.

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A RARE CASE OF THYROTOXICOSIS PRESENTING AS HYPERBILIRUBINAEMIA

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INTRODUCTION/BACKGROUND
Hyperthyroidism affects multiple body systems, including the nervous, cardiovascular, gastrointestinal, and hepatobiliary systems. Presentation of severe cholestatic jaundice in thyrotoxicosis, although uncommon, has been described in literature.

CASE
A 28-year-old Malay male presented with 1-week history of painless jaundice, associated with tea-coloured urine and diarrhoea. He also had a significant weight loss of 12 kg over the past 10 months. Physical examination showed an underweight young male, deeply jaundiced, with fine tremors. He was normotensive and not tachycardic. He did not have a goitre, thyroid eye disease or pretibial myxoedema. He had no stigmata of chronic liver disease. Blood investigation showed transaminitis with conjugated hyperbilirubinemia, with ALT 174 IU/L, AST 112 IU/L, total bilirubin 357 µmol/L, and predominant direct bilirubin (252 µmol/L). Autoimmune, infectious, and primary hepatobiliary disorders were ruled out. Thyroid function test was taken on day 16 of admission, which showed suppressed TSH <0.01 m IU/L, and elevated free T4 at 77 pmol/L. He was started on carbimazole, prednisolone and cholestyramine. carbimazole was withheld after 1 week of treatment in view of worsening hyperbilirubinemia and transaminitis. Subsequently, he received radioactive iodine therapy after 3 weeks of treatment. He had clinical and biochemical improvement after the radioactive iodine therapy. He eventually progressed into a hypothyroid state. His bilirubin levels subsequently normalized.

CONCLUSION
Severe jaundice is a rare consequence of hyperthyroidism and can be due to various pathologies. A thorough investigation should be done to look for contributing

factors and hence, be treated accordingly. Treatment of hyperthyroid patients with liver abnormalities is rather challenging as antithyroid drugs have been associated with liver injury. Various case reports showed remission of hyperbilirubinemia after radioactive iodine therapy. Therefore, radioactive iodine therapy should be offered as early as possible for patients with severe hyperbilirubinemia that is likely due to hyperthyroidism.

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BIZARRE THYROID FUNCTION TEST IN A PATIENT WITH MULTINODULAR GOITRE: A CASE REPORT

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INTRODUCTION/BACKGROUND
Multinodular goitres may be accompanied by various thyroid function abnormalities. Assessment of thyroid status and correct interpretation of thyroid function tests (TFTs) is important to ascertain the aetiology.

CASE
A 68-year-old female was referred for re-evaluation of abnormal TFT associated with a huge multinodular goitre. She was seen 3 years ago by a private practitioner due to progressive goitre enlargement since age 30 years. TFTs then showed markedly reduced fT4 at 1.3-3.6 pmol/L (12-22), with normal TSH at 0.3-0.65 μIU/mL (0.27-4.2). She was started on L-thyroxine 100 ug daily based on these results, but she was only taking it intermittently. At the time of evaluation, there was no sign or symptom of hypothyroidism, but she complained of weight loss and irritability. On examination, she had a huge goitre with no lymphadenopathy. TFTs done showed low fT4, 8.6 pmol/L and TSH <0.005 μIU/mL. Central hypothyroidism was ruled out by a paucity of signs of hypothyroidism with no accompanying hypopituitarism. Due to the persistent and markedly suppressed TSH (<0.005) but fT4 at a low normal limit, fT3 was assessed and was found to be elevated at 11-16.7 pmol/L (3.1-6.8). L-thyroxine was stopped.

Three months later, a repeat TFT off L-thyroxine still showed a very low fT4 at 1.94 pmol/L, but normal fT3 (5.02 nmol/L) and TSH (0.291 μIU/mL). SHBG was normal at 52.4 nmol/L (16.8- 125.2) supporting euthyroidism. She remained well and euthyroid on subsequent follow-up with similar TFT but refused FNAC or surgical intervention for her goitre.

CONCLUSION
Low fT4 with normal TSH points towards central hypothyroidism but in patients with goitre and clinically euthyroid, disorders like iodine deficiency and thyroid dyshormonogenesis need to be considered. A T3 measurement should be done. A high T3/T4 ratio may be found in rarer entities such as resistance to thyroid hormone α and has also been reported in follicular thyroid cancer due to increased thyroidal deiodinase activity.

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TUBERCULOUS MENINGOENCEPHALITIS MASKING MYXOEDEMA COMA

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INTRODUCTION/BACKGROUND
Myxoedema coma is a rare but potentially lethal complication of extreme hypothyroidism. Despite its low incidence, the mortality rate may reach 60%.

CASE
A 40-year-old male presented with shortness of breath, vomiting, frontal headache and abnormal behaviour for 2 days. He also suffered from fever, chesty cough, and chronic back pain for 2 weeks. He was confused, had unequal pupils, loss of lateral 1/3rd of his eyebrows, and reduced breath sounds bilaterally. Cranial CT scan demonstrated obstructive hydrocephalus necessitating external ventricular drainage. Pus aspirated from a right exudative pleural effusion yielded an ADA value of 68.78U/L. An MRI showed intracranial hyperintense lesions and L3/L4 spondylitis. Diagnosed with disseminated TB, anti-TB treatment with tapering doses of dexamethasone was commenced. He needed tracheostomy for prolonged intubation and had poor GCS recovery. On day 28 of hospitalization, he developed hypotension with a BP of 70/50 mm Hg, warranting noradrenaline infusion.

In retrospect, he had been bradycardic (heart rate ranged 30-55 bpm), hypothermic with a temperature of 35.7°C, and had recurrent hypoglycaemic episodes 7 days prior. Blood gas demonstrated CO_2 retention. Echocardiography did not exhibit pericardial effusion. His TSH level was >48.8 m IU/L, T4 level <3.2 pmol/L, and morning cortisol 163 nmol/L. He was administered IV Hydrocortisone 100 mg including IV Thyroxine 200mcg slow bolus. IV Thyroxine was then reduced to 100 mcg OD for 2 days and subsequently switched to an oral maintenance dose of 100 mcg OD. His