

negative serology and the absence of a family history, along with a hyperfunctional state on imaging, likely suggests a rare presentation of sporadic, non-autoimmune, non-familial form of thyrotoxicosis. Following the initiation of anti-thyroid therapy, the CHB completely resolved without the need for further cardiological intervention. He achieved a complete clinical remission after a few months and is being planned for radioactive iodine therapy.

CONCLUSION

Most of the thyrotoxicosis-associated CHB reported in the literature was due to Graves' disease or some forms of autoimmune thyrotoxicosis, with auto-antibodies and activated lymphocytes having a role in the pathogenesis of the CHB. CHB in association with a non-autoimmune, non-familial form of thyrotoxicosis is indeed rare, and as this case illustrates, it completely went into spontaneous sinus rhythm upon initiation of conventional anti-thyroid therapy. This rare presentation of CHB is believed to have a benign clinical course.

EP_A157

STEROID-RESPONSIVE ENCEPHALOPATHY ASSOCIATED WITH THYROIDITIS

Chua Chong Yee¹, Nur Haziqah Baharum², Fadzliana Hanum Jalal³, Gunavathy Muthusamy¹

¹Department of Internal Medicine, Hospital Shah Alam

²Hospital Al-Sultan Abdullah UiTM

³Avisena Specialist Hospital

INTRODUCTION

Steroid-responsive encephalopathy associated with thyroiditis (STREAT) is a rare clinical entity. Clinical presentations of STREAT can range from a stroke-like presentation to psychiatric symptoms. STREAT is diagnosed based on four major criteria which include altered cognitive function, new or worsening psychiatric symptoms, elevated antithyroid antibodies, and exclusion of infectious, toxic, metabolic, or neoplastic causes. Corticosteroids are the cornerstone of treatment, with reported excellent response in neurocognitive symptoms resolution.

CASE

This was a case of a 48-year-old male with Graves's disease who underwent radioiodine therapy 4 months earlier and was started on levothyroxine 100 mcg 2 weeks prior to presentation. He presented with 4 days of behavioral symptoms, irritable mood with auditory and visual hallucinations. There was no history of fever, headache, limb weakness, or seizure. On general appearance, the patient was agitated, talking incoherently, and disorientated with normal vitals. There was no delayed relaxation of the reflex.

The examination of the cranial nerve, motor, sensory, and cerebellar system was normal. A lumbar puncture showed normal opening pressure with a high protein CSF content of 1,596 mg/L. The thyroid-stimulating hormone (TSH) was elevated at 72.7 uIU/mL and T4 at 9.56 pmol/L. Serum anti-thyroid peroxidase was elevated at 877.42 IU/mL, and anti-TSH receptor antibody at 21.30 IU/L. CSF oligoclonal band, serum aquaporin-4, and viral screening were negative; non-contrasted computed tomography brain revealed normal findings. Based on the clinical history and examination, a diagnosis of STREAT was made at the emergency department. The patient was initiated on hydrocortisone 100 mg three times a day. Within 24–48 hours of steroid therapy, marked improvement and subsequent resolution of the mental status and behavioral symptoms were seen.

CONCLUSION

Hashimoto's thyroiditis can rarely lead to STREAT, a condition with diverse neurological manifestations, where early recognition and prompt corticosteroid therapy are essential for favorable outcomes.

EP_A158

MUSCLE WEAKNESS IN THYROID DISEASE: WHEN IT IS NOT THYROTOXIC MYOPATHY?

Hamizah Hamzah¹, Sarojini Devi Simanchalam¹, Yap Yon Lek², Wong Poh Shean¹, Nor Afidah Karim¹, Nadiyah Mohd Noor¹, Noor Lita Adam¹

¹Endocrinology Unit, Department of Medicine, Hospital Tuanku Ja'afar Seremban

²Rheumatology Unit, Department of Medicine, Hospital Tuanku Ja'afar Seremban

INTRODUCTION

Muscle weakness in thyroid disease is commonly attributed to thyrotoxic myopathy or hypokalemic periodic paralysis. Nevertheless, autoimmune conditions such as idiopathic inflammatory myopathies (IIM) and myasthenia gravis (MG) should be considered, as they may coexist with Graves' disease.

CASE

A 54-year-old female with hypertension and Graves' disease, treated with carbimazole for 2 years, had her therapy discontinued after remission. She was restarted on low-dose carbimazole following symptom recurrence. Three weeks later, she developed progressive proximal weakness, dysphagia, hoarseness, anorexia, and weight loss. On examination, body mass index was 22 kg/m² with mild proptosis, symmetrical proximal weakness (MRC 4/5), and erythematous rashes on thighs and shins. Otorhinolaryngology evaluation confirmed bilateral vocal