EP_A172

STORMY SEAS: MANAGING THYROID STORM TREATMENT-RELATED COMPLICATION WITH BETA-BLOCKER TOXICITY

https://doi.org/10.15605/jafes.039.S1.183

Siti Nabihah Mohamed Hatta,[1] Monisha A/P Murthi,[1] Lim Fang Chan,[1] Tan Jia Miao,[1] Tee Hwee Ching[2]
[1]*Medical Department, Hospital Tawau, Malaysia*
[2]*Endocrinology Unit, Hospital Queen Elizabeth II, Kota Kinabalu, Sabah, Malaysia*

INTRODUCTION/BACKGROUND
Thyroid storm is a life-threatening condition involving multiple organ systems due to thyrotoxicosis. The standard treatment often includes the preferred option of the beta blocker (BB) propranolol. However, usage of BB in thyroid storm management was linked to cardiogenic collapse due to its toxicity. We present a case of thyroid storm who was treated with a beta-blocker and developed toxicity.

CASE
A 65-year-old female presented with shortness of breath, palpitations, fever, and diarrhoea. In the Emergency Department, she was fully conscious but agitated. She had tachycardia with signs of heart failure. Urgent TFT showed suppressed TSH <0.01 m IU/L and elevated T4 level of 44.12 pmol/L. Her Burch and Wartofsky Score was 70. Thus, diagnosis of thyroid storm with thyrotoxic cardiomyopathy was made. She was started on propylthiouracil, Lugol's Iodine, steroids, Propranolol 40 mg QID.

After 8 hours of treatment, she became drowsy, developed junctional bradycardia and hypotension. Appropriate resuscitation with IV Atropine and inotropes was started. Excluding other causes of hypotension with bradycardia, we considered beta-blocker toxicity. Subcutaneous glucagon was initiated. Within one day, inotropes were weaned off.

Thyroid storm can lead to lethal complications. The presentation ranges from thermoregulatory, neurologic, gastro-hepatic, cardiac dysfunctions to circulatory collapse and shock. The treatment includes BB, anti-thyroid drugs, and potassium Iodide or Lugols Iodine, along with hydrocortisone. Second-line options may include lithium, dialysis, or plasmapheresis.

Beta blockers work by reducing hyperadrenergic states and blocking the peripheral conversion of T4 to T3. They can have adverse effects such as peripheral coldness, syncope, bradycardia, hypotension, circulatory collapse, and even cardiac arrest. Glucagon is the first-line antidote for BB toxicity.

CONCLUSION
The use of beta blockers in treating thyroid storm requires close monitoring due to the risk of devastating cardiogenic collapse.

EP_A173

NODULAR PRETIBIAL MYXEDEMA FOLLOWING TREATMENT OF GRAVES' DISEASE

https://doi.org/10.15605/jafes.039.S1.184

Wei Xiong Wong,[1] Yi Chwen Eunice Lau,[1] Yew Liang George Lau,[1] Hock Gin Teo[2]
[1]*Endocrine Unit, Department of Medicine, Hospital Sibu, Malaysia*
[2]*Dermatology Unit, Department of Medicine, Hospital Sibu, Malaysia*

INTRODUCTION/BACKGROUND
Pretibial myxoedema (PM) is an uncommon manifestation of Graves' disease (GD). Typically, thyroid dermopathy may present as non-pitting oedema with indurated skin giving a 'peau d'orange' appearance. Less commonly, patients may present with other variants such as plaques, nodules and elephantiasic type lesions.

We report a case of biopsy-proven PM in a patient with GD.

CASE
An 80-year-old woman presented with multiple painless nodules over both shins with gradual increase in size over 6 months. She had a history of hypertension and difficult-to-manage GD, complicated by thyroid storm, atrial fibrillation, and heart failure. Her thyroid function fluctuated from hypo- to hyperthyroidism within weeks. She refused radioactive iodine ablation and was subsequently controlled with a block and replace regimen. She did not have any constitutional symptoms, preceding trauma, or insect bite. Clinically, she was euthyroid and did not have any active thyroid eye disease.

On examination, there were multiple ill-defined, firm, non-tender, flesh-coloured nodules over both shins. Her FT4 was 18.12 pmol/L (12.0 - 22.00) and TSH <0.005 m IU/L (027 – 4.20). A punch biopsy revealed fragmented collagen fibres with conspicuous mucin deposits over the reticular dermis and subcutaneous layer, consistent with PM. She was started on potent topical corticosteroids with marked improvement in her skin lesions.

CONCLUSION
Thyroid dermopathy can present in an atypical manner, hence, physicians should be aware of this. Treatment of hyperthyroidism may not have any significant effect on the cutaneous lesions. PM may occur even after successful control of the disease.

EP_A174

NEUROLOGIC MANIFESTATION AND PERICARDIAL EFFUSION UNVEILING AN AUTOIMMUNE HYPOTHYROIDISM

https://doi.org/10.15605/jafes.039.S1.185

Shu May Chong and Lay Ang Lim
Hospital Seberang Jaya, Malaysia

INTRODUCTION/BACKGROUND
Autoimmune hypothyroidism is an antibody-mediated chronic inflammatory process. Thyroid destruction may be intermittent. Given its chronic and progressive nature, the diagnosis is often challenging since the exhibited signs and symptoms are often subtle and non-specific. We report a middle-aged male with bilateral upper and lower limb weakness and pericardial effusion. Investigation led to a diagnosis of autoimmune hypothyroidism.

CASE
A 64-year-old male presented with a two-week history of lethargy, poor appetite, and lower limb swelling. Initially, he was treated for pneumonia and cardiac failure due to chest radiography showing obscured cardio-phrenic angle. Further history revealed he had inability to walk for one year due to bilateral lower limb weakness. He had proximal muscle weakness of all four limbs. Sensation and reflexes were preserved in the upper limbs but absent in the lower limbs. His nerve conduction study and electromyography revealed myopathic changes involving all four extremities with absent neurosensory responses in both lower extremities. His cranial CT scan showed bifronto-temporal subdural effusion while his echocardiography exhibited pericardial effusion with cardiomegaly. His thyroid function tests revealed profound hypothyroidism (TSH>100, fT4 <1). Together with the presence of markedly raised anti-TPO antibodies, he was diagnosed to have autoimmune primary hypothyroidism. He also had normocytic normochromic anaemia and hypercholesterolemia consistent with severe hypothyroidism. He was eventually started on oral L-thyroxine (1.6 mcg/kg/day).

CONCLUSION
This case report highlights the potential for severe neuromuscular and cardiovascular consequences due to untreated chronic autoimmune hypothyroidism. Thyroid dysfunction is a consideration in a patient with neurologic manifestation. Early diagnosis and prompt treatment of hypothyroidism can potentially avert long-term hypothyroid sequelae.

EP_A175

THYROID FUNCTION ABNORMALITIES: CONNECTING THE DOTS BETWEEN GENETICS AND CLINICAL PRESENTATION

https://doi.org/10.15605/jafes.039.S1.186

Adilah Zulaikha Abd Latib,[1] Yik Zhi Kum,[1] Siti Nabilah Atiqah Othman,[1] Ooi Chuan Ng[2]
[1]*Department of Medicine, Hospital Sultan Abdul Aziz Shah, Universiti Putra Malaysia*
[2]*Faculty of Medicine and Health Sciences, University Putra Malaysia*

INTRODUCTION/BACKGROUND
Subclinical hyperthyroidism presents with low or undetectable levels of thyroid stimulating hormone (TSH), alongside normal levels of free thyroid hormones (fT4 and fT3). While certain individuals may not show any symptoms, others may experience hyperthyroid symptoms like palpitations, weight loss, and heat intolerance. Early recognition and prompt appropriate management are crucial to prevent potential complications, including atrial fibrillation, osteoporosis, and progression to overt hyperthyroidism.

CASE
A 59-year-old female was referred to our endocrine clinic due to abnormal thyroid function tests (TFTs) revealing subclinical hyperthyroidism. She was asymptomatic.

Her thyroid function tests 5 years ago showed similar results, however, she had not received proper consultation or treatment during that time.

She has no significant medical history. She has been in menopause since 50 years old with regular menses before that. She had five pregnancies, four of which were preterm, with her eldest child having cerebral palsy and her fourth child deceased due to prematurity. Two of her children have hyperthyroidism. One son is on carbimazole while one daughter has subclinical hyperthyroidism.

A repeat thyroid function test still showed suppressed TSH level of 0.04 m IU/L and normal FT4 level of 13.2 pmol/L and free triiodothyronine (fT3) level of 4.3 pmol/L. Molecular studies showed polymorphism of exon 3 of the TSHR gene from the son.