

(56%) were due to infection, mostly pneumonia, followed by 22% (5 subjects) due to undiagnosed hyperthyroidism and 17% (4 subjects) were due to defaulted treatment. There were two mortalities (8.7%). Both mortalities required intubation and presented with pulmonary oedema on arrival, and had Burch and Wartofsky scores of 50 and 60 respectively. Mortality rate for thyroid storm in 2023 was 8.7%.

CONCLUSION

Death from thyroid storm is not as common as in the past owing to its prompt recognition and aggressive treatment in an intensive care unit, but mortality is still approximately 10-25%. Early detection and understanding of hyperthyroidism symptoms among the public are critically important. Analysing the outcomes of thyroid storms at Hospital Teluk Intan highlights the need for increased public education to prevent future deaths caused by thyroid storms.

EP_A148

SOMEBODY CALL 9-1-1: HYPOTHYROIDISM MIMICKING WELLENS SYNDROME

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Gerard Jason Mathews,¹ Seetha Devi Subramanian,¹ Teh When Yee,¹ Joel Mathews,¹ Noor Rafhati Adyani Abdullah,¹ Shartiyah Ismail,¹ Nor Shaffinaz Yusoff Azmi Merican,¹ Dharmaraj Kartikesan²

¹Endocrinology Unit, Department of Medicine, Hospital Sultanah Bahiyah, Malaysia

²Department of Cardiology, Hospital Sultanah Bahiyah, Malaysia

INTRODUCTION/BACKGROUND

Wellens syndrome is characterized by a distinctive pattern of electrocardiographic (ECG) pattern, specifically deep symmetrical inverted T-waves or biphasic T-waves in leads V2-V3. This condition is highly indicative of critical stenosis in the left anterior descending artery (LAD) which poses a significant risk of mortality, hence its nickname "widow maker."

CASE

A 38-year-old police officer was under Endocrine clinic follow-up for Graves' disease. He underwent radioactive iodine (RAI) treatment in June 2022 and subsequently did not require thyroxine replacement for 1 year as he was clinically and biochemically euthyroid. During a clinic review in August 2023, he reported symptoms of cold intolerance, weight gain and reduced effort tolerance which hindered his ability to do his daily work. Blood investigation showed a free T4 of 7 pmol/L, and TSH was 13.6 m IU/L. The patient was diagnosed with overt hypothyroidism and was given thyroxine replacement.

Electrocardiogram (ECG) done revealed deep symmetrical T-wave inversions in leads V2-V5, indicative of Wellens Type B pattern, which carries a 97% specificity for LAD occlusion. The patient was referred to cardiology service and underwent an early coronary angiogram, which surprisingly revealed normal coronaries. Additionally, he did not exhibit any features of common Wellen mimics such as pulmonary embolism, pancreatitis, heart failure or acute stroke. He denied consuming alcohol or using any illegal stimulant substances. Blood analysis done revealed normal electrolytes. Patient was initiated on treatment with 12.5 mcg of L-Thyroxine tablets daily, with the dose topped up every 4 to 6 weeks. Upon subsequent clinic reviews, patient's initial symptoms have resolved, enabling him to resume his job without any difficulties.

CONCLUSION

This case highlights overt hypothyroidism as a mimicker of Wellens Syndrome. After promptly excluding critical coronary artery disease, it is imperative to evaluate and treat other potential causes of Wellens Syndrome or its mimics.

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AUTOIMMUNE/INFLAMMATORY SYNDROME INDUCED BY ADJUVANTS (ASIA): POST-VACCINATION SUBACUTE THYROIDITIS

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Gerard Jason Mathews, Seetha Devi Subramanian, Teh When Yee, Joel Mathews, Nor Shaffinaz Yusoff Azmi Merican, Shartiyah Ismail

Endocrinology Unit, Department of Medicine, Hospital Sultanah Bahiyah, Malaysia

INTRODUCTION/BACKGROUND

Autoimmune/inflammatory syndrome induced by adjuvants (ASIA), also known as Shoenfeld's syndrome, encompasses a spectrum of autoimmune conditions and responses triggered by exposure to substances with adjuvant activity such as vaccines.

CASE

A healthy 43-year-old male with no known medical illness or family history of thyroid disorder developed painful thyroiditis after receiving his influenza vaccination. He undergoes regular health checkups, which have consistently shown normal results, including previous thyroid function tests (TFT). Patient received his influenza vaccine (Vaxigrip tetra) and developed left sided neck pain and severe thyrotoxicosis symptoms after 1 week. Blood investigation done showed free T4 25 pmol/L, TSH <0.01 m IU/L and a raised CRP. Physical examination revealed a tender diffuse

goitre and fine tremors. Ultrasonography of the neck was done with the impression of subacute thyroiditis. These findings fulfilled the Japanese Thyroid Association (JTA) criteria for subacute thyroiditis. A Tc-99m Pertechnetate also confirmed the findings of thyroiditis. The patient was commenced on oral prednisolone 25 mg daily (0.5 kg/BW/day) with a tapering regimen over 2 weeks. He also received oral celecoxib 200 mg daily for 5 days and oral propranolol 20 mg daily. He gradually improved upon the 2-week clinic review and all medications were discontinued. Upon re-evaluation at 3 months, thyroid function tests normalized, and the thyroid ultrasound displayed the resolution of thyroiditis characteristics, accompanied by amelioration of all symptoms.

CONCLUSION

Subacute thyroiditis is an inflammatory thyroid condition characterized distinctly by painful enlargement of the thyroid. Transient hyperthyroidism is a hallmark of subacute thyroiditis where the inflamed thyroid gland releases unregulated excessive thyroid hormone into the bloodstream, leading to thyrotoxicosis symptoms. In this case, we treated our patient with a short-term 2-week combination of steroids + NSAIDs which showed non-inferior efficacy to the traditional long-term steroids (4-8 weeks).

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HUMAN CHORIONIC GONADOTROPHIN (HCG) AND HYPERTHYROIDISM: RARE BUT PARALLEL CAUSE

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SA Baskaran, MU Tukiman, II Adam

Medical Department, Hospital Sungai Buloh, Malaysia

INTRODUCTION/BACKGROUND

Choriocarcinoma is a hCG-producing malignancy, with the beta subunit being structurally similar to TSH, allowing it to bind to TSH receptors on thyroid follicular cells and at high levels to elicit biochemical hyperthyroidism.

CASE

We report a case of choriocarcinoma-induced hyperthyroidism in a 29-year-old female. She was initially admitted for breathing difficulties and was found to have a posterior mediastinal mass, which was later histologically confirmed to be choriocarcinoma with B-HCG levels of 466,511 that peaked to 825,316. Thyroid functions measured shows TSH of 32.07, T4 of 0.009 with negative thyroid antibodies (Anti Thyroid Peroxidase <9, Anti Thyroglobulin 11.9). Ultrasonography showed bilateral thyroid nodules, ACTR TR3 (1.5 cm), however, staging CT did not pick

up an overt goitre. Patient was started on carbimazole and planned for chemotherapy with thyroid function monitoring.

CONCLUSION

TSH and Beta-HCG are highly homologous and can cross-link to produce elevated thyroid hormone levels. The prevalence of hyperthyroidism in choriocarcinoma is not known; however, prolonged exposure to high HCG levels is required for it to occur. It is generally accepted that 25,000 IU/L of HCG is equivalent to 1 mU/L of TSH activity, with suggestions to measure thyroid function in patients with HCG >50,000 IU/L. Patients with symptomatic hyperthyroidism are treated with antithyroid drugs and the primary choriocarcinoma is treated with chemotherapy. Reduction or normalisation of the beta hCG levels quickly induces euthyroidism. Beta-HCG-induced hyperthyroidism is rare; however, with high levels found in choriocarcinoma, suspicion of concurrent hyperthyroidism should be raised. Patients with HCG-secreting tumours should be evaluated for hyperthyroidism and may benefit from treatment until the underlying cause is treated.

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DOUBLE WHAMMY: CIRCULATORY COLLAPSE AND LIVER DYSFUNCTION IN THYROID STORM

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Jie En Tan, Florence Hui Sieng Tan, Yueh Chien Kuan, Pei Lin Chan

Endocrinology Unit, Medical Department, Sarawak General Hospital, Malaysia

INTRODUCTION/BACKGROUND

Liver dysfunction is not an uncommon association in patients presenting with thyroid storm and could limit the treatment armamentarium. Circulatory collapse precipitated by the use of long-acting non-cardioselective beta-blockers in certain groups of patients can complicate the course of the disease.

CASE

We report three cases of thyroid storm with circulatory collapse and ischemic hepatitis complicating the use of beta-blockers and thionamides. All were females in their 40's. Two presented with rapid atrial fibrillation (ventricular rate 158- 196 per minute) and biventricular failure, and one with acute pulmonary oedema. All developed hypotension required inotropic support; two after beta-blocker and one after intubation. Their free T4 was 53.6 pmol/L to 74.3 pmol/L, Burch-Wartofsky scores were 60-95. All received ventilatory support and were treated for