Adult E-Poster

CASE

A 29-year-old Malay male with a history of active smoking and drug abuse presented with a one-week history of non-productive cough and sudden onset of shortness of breath after physical exertion. On initial assessment, he exhibited marked restlessness, diaphoresis, and irregular narrow complex tachycardia on electrocardiogram (ECG). Urine toxicology was positive for amphetamine and methamphetamine, leading to an initial suspicion of substance-induced cardiomyopathy complicated by rapid atrial fibrillation. He was managed with anti-arrhythmics and non-invasive ventilation. However, persistent tachycardia and clinical deterioration necessitated intubation. A chest X-ray revealed cardiomegaly.

Interestingly, routine thyroid function tests, which were ordered due to the patient's unexplained tachycardia, returned with a significantly suppressed thyroid-stimulating hormone (TSH) of <0.008 mIU/L and an elevated free thyroxine (FT4) of 64 pmol/L. This, coupled with a Burch-Wartofsky score of 65, strongly indicated thyroid storm. The initial diagnosis was revised accordingly. Despite aggressive management for thyroid storm, including anti-thyroid medications, beta-blockers, and supportive care, the patient developed acute infarcts in the right middle cerebral artery territory with subsequent hemorrhagic transformation and significant cerebral edema on serial computed tomography (CT) scans of the brain. Neurosurgical intervention was considered but declined by the family due to the guarded prognosis. The patient eventually succumbed to death due to massive cerebral infarct.

This case highlights the importance of considering thyroid storm in the differential diagnosis of young adults presenting with acute cardiac symptoms and agitation, even in the presence of positive toxicology screens. The initial clinical picture and positive drug screen misleadingly pointed towards a primary cardiac etiology. The significantly abnormal thyroid function tests were crucial in establishing the correct diagnosis. While the exact mechanism of the cerebral infarction in this context remains unclear, it could be a rare complication of severe thyroid storm, potentially exacerbated by underlying substance abuse or other unidentified factors. This case underscores the need for a broad differential diagnosis and timely thyroid function testing in patients with unexplained acute cardiovascular symptoms, particularly when atypical features are present.

CONCLUSION

This case serves as a reminder of the protean manifestations of thyroid storm and the potential for diagnostic confusion with other acute conditions. A high index of suspicion and prompt laboratory investigations are essential for timely and accurate diagnosis, which is critical for improving patient outcomes in this life-threatening endocrine emergency.

EP_A140

DELAYED DIAGNOSIS OF LYMPHOCYTIC HYPOPHYSITIS PRESENTING AS CHRONIC HEADACHES: A CASE REPORT

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Ihsan Ismail,[1] Rabeah Md Zuki,[1] Murshidah Ainun Mukhtar[2]

[1]*Medical Department, Hospital Sultan Abdul Halim, Kedah, Malaysia*

[2]*Medical Department, Hospital Sik, Kedah, Malaysia*

INTRODUCTION/BACKGROUND

Lymphocytic hypophysitis (LH) is a rare autoimmune inflammatory disorder of the pituitary gland, often presenting with non-specific symptoms leading to diagnostic delays. This case highlights the challenges in the timely diagnosis of LH and the consequences of treatment default in a young male.

CASE

A 37-year-old male with a three-year history of chronic headaches, initially managed symptomatically, presented with recurrent episodes of worsening headaches, vomiting, and chest pain requiring multiple emergency department visits over two years. After 2 years of intermittent visits, a non-contrast computed tomography (CT) brain, performed due to persistent and escalating headaches, revealed a possible pituitary fossa mass. Subsequent urgent magnetic resonance imaging (MRI) confirmed a bulky pituitary gland with minimal suprasellar extension. Hormonal evaluation revealed panhypopituitarism. Based on clinical and radiological findings, a diagnosis of lymphocytic hypophysitis was suspected. The patient was commenced on hydrocortisone and thyroid hormone replacement. Regrettably, the patient defaulted on follow-up and discontinued his prescribed medications in favour of traditional treatment.

This case underscores the insidious presentation of LH, where chronic headaches can be the predominant initial symptom, leading to significant delays in diagnosis. The patient's repeated emergency department visits for non-specific symptoms highlight the need for a high index of suspicion for underlying endocrine disorders in patients with persistent and evolving complaints. The eventual radiological findings of a pituitary mass and subsequent confirmation of panhypopituitarism were crucial for suspecting LH. The patient's decision to discontinue conventional treatment and opt for traditional remedies emphasizes the importance of patient education, adherence strategies, and culturally sensitive approaches in managing chronic endocrine conditions. The potential long-term sequelae of untreated panhypopituitarism warrants concern.

Adult E-Poster

CONCLUSION
This case serves as a reminder of the diverse clinical mani-festations of lymphocytic hypophysitis and the importance of considering pituitary pathology in patients with chronic headaches. Early radiological investigation in cases of persistent or worsening headaches, coupled with hormonal evaluation, is crucial for timely diagnosis and management. Furthermore, addressing factors influencing treatment adherence, including patient beliefs and prefe-rences, is essential for optimal outcomes in chronic endocrine disorders.

EP_A141

CUTANEOUS TUMOUR IN MULTIPLE ENDOCRINE NEOPLASIA TYPE 1

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Afifah Kamarudin, Jiun Yan Tan, Wee Mee Cheng, Lit Sin Yong, Nor Afidah binti Karim, Noor Lita Adam
Endocrinology Unit, Department of Internal Medicine, Hospital Tuanku Ja'afar Seremban, Malaysia

INTRODUCTION/BACKGROUND
Multiple Endocrine Neoplasia (MEN) 1 syndrome is a genetic disease characterized by pituitary adenoma, parathyroid hyperplasia, and pancreatic tumors. Cutaneous manifestations of the syndrome are rare. We report a case of MEN 1 syndrome with collagenoma, initially misdiagnosed as neurofibromatosis.

CASE
A 48-year-old male with hypertension and gouty arthritis was diagnosed with Neurofibromatosis type 1 10 years ago based on neurofibromas on his neck and abdomen. He defaulted follow-up until admission in June 2024 for hypertensive emergency. During admission, incidental findings of asymptomatic moderate PTH-dependent hypercalcemia (corrected calcium: 3.02 mmol/L, phosphate: 0.37 mmol/L, iPTH: 31.9 pmol/L [Normal range 1.58 - 6.03]) and a suspicious right hilar mass on chest X-ray, prompting further workup. Calcium-creatinine clearance ratio was 0.046. His tumor marker levels, including B-HCG, were normal. CT of the thorax, abdomen, and pelvis showed a solid anterior mediastinal mass with a superior mediastinal lesion and an enhancing pancreatic mass. A DOTATATE scan confirmed somatostatin receptor avid disease in the pancreas and anterior mediastinal mass with nodal, liver, and bone metastases. CT-guided biopsy of anterior mediastinal mass followed by anterior mediastinal mass resection and endoscopic guided biopsy of pancreatic mass confirmed neuroendocrine tumor. His clinical condition was suggestive of MEN 1. It is exceptionally rare for two autosomal dominant syndromes to coexist. There was no other diagnostic manifestation for neurofibromatosis type 1 except for skin lesions. Skin biopsy was done and reported as collagenoma, supporting MEN 1. Genetic study is ideal but limited by financial constraints.

CONCLUSION
Without the luxury of genetic testing, tactful correlations of clinical manifestations are essential to diagnose rare inheritable syndromes. Relevant investigations may help identify clinical signs and their association with the syndrome. In our patient, the initial diagnosis of neurofibromatosis was later revised to collagenoma with MEN 1 after HPE result from skin biopsy.

EP_A142

MYXOEDEMA MADNESS: WHEN HYPOTHYROIDISM TURNS PSYCHOTIC

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Nurul Syamimi Yahaya and Marisa Khatijah Borhan
Department of Medicine, Hospital Raja Perempuan Zainab II, Kelantan, Malaysia

INTRODUCTION/BACKGROUND
Myxoedema madness is a rare but serious neuropsychiatric manifestation of severe hypothyroidism. We report a case of overt hypothyroidism following radioactive iodine (RAI) therapy, presenting with paradoxical psychotic symptoms.

CASE
A 43-year-old Malay male with underlying ischemic heart disease, severe mitral regurgitation, and toxic multinodular goiter (MNG) was admitted for acute behavioral changes. Diagnosed with toxic MNG in 2019, he was initially treated with carbimazole before undergoing his first RAI therapy in January 2024. He reported mood swings that improved after starting carbimazole, not needing psychiatric evaluation. A second RAI therapy was administered in January 2025 after failed first therapy. Two months post-RAI therapy, his thyroid function test (TFT) showed a TSH of 7.6 mIU/L and a free T4 of 7.8 pmol/L; hence, carbimazole 2.5 mg daily was withheld, and he was scheduled for follow-up. One month after stopping carbimazole, he presented to the Emergency Department with a five-day history of disorganized behavior, irrelevant speech, and bizarre ideations. Clinically, he was restless, requiring physical restraint. There were no signs of meningism or hypothyroidism. His inflammatory markers and brain CT were normal, and both urine drug toxicology and infective screening were negative. A repeat TFT confirmed overt