Adult E-Poster

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THE VOICE WITHIN: ADULT LARYNGOMALACIA AS A RARE COMPLICATION OF ACROMEGALY

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INTRODUCTION/BACKGROUND
Acromegaly is a chronic disorder caused by excess growth hormone (GH) and insulin-like growth factor 1 (IGF-1), most often due to a GH-secreting pituitary adenoma. Adult-onset laryngomalacia is rarely reported.

CASE
A 69-year-old male with a history of hyperthyroidism and colon cancer presented with progressive left eye blurring. He was initially treated for herpetic keratouveitis. During follow-up, coarse facial features suggestive of acromegaly—thickened skin, enlarged jaw, tongue, hands, and feet—were noted. He had noisy breathing, prompting ENT referral. Flexible laryngoscopy revealed redundant mucosa over the arytenoids prolapsing into the laryngeal inlet during inspiration, consistent with adult-onset laryngomalacia. Biochemical evaluation confirmed acromegaly (GH >50 ng/mL; IGF-1: 973.5 ng/mL) with secondary hypogonadism. MRI showed a 1.6 × 2.2 × 1.6 cm pituitary macroadenoma compressing the left optic nerve. He was started on intramuscular Octreotide LAR and underwent supraglottoplasty.

Laryngomalacia is typically a pediatric condition caused by dynamic supraglottic collapse during inspiration. In adults, it is uncommon and may result from structural abnormalities or acquired soft tissue redundancy, as seen in acromegaly. Chronic GH and IGF-1 excess leads to hypertrophy of soft tissues, including the larynx, epiglottis, aryepiglottic folds, and arytenoids, contributing to narrowing of the upper airway. Awake fiberoptic laryngoscopy is the diagnostic gold standard. Findings include inspiratory collapse of supraglottic structures, which may cause stridor, dysphonia, or sleep-disordered breathing. In acromegaly, cartilage overgrowth and mucosal thickening reduce airway diameter and alter tissue compliance. The hyoepiglottic ligament may also lose tensile strength, further predisposing to dynamic airway obstruction. Laryngomalacia may be misdiagnosed or attributed to obstructive sleep apnea, a common comorbidity in acromegaly and distinct anatomical distortion should prompt ENT evaluation.

CONCLUSION
Laryngomalacia should be considered in acromegalic patients presenting with stridor or noisy breathing. Early recognition and surgical management can prevent airway complications and improve patient outcomes.

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HYPERTHYROIDISM MASQUERADING AS ACUTE MYOCARDIAL INFARCTION

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INTRODUCTION/BACKGROUND
Troponin-Positive Non-Obstructive Coronary Arteries (TpNOCA) are conditions characterized by elevated troponin levels accompanied by absent obstructive coronary artery disease (CAD) as observed on coronary angiography. It encompasses both coronary and noncoronary causes of myocardial injury.

CASE
A 31-year-old female with no known medical illness presented with fever, vomiting and diarrhea for 4 days. She did not have any features or family history of Graves' Disease. She had no goiter and denied any biotin supplements or illicit drugs. On arrival she had a fever of 38.5°C, palpitations with a pulse ranging between 110-130 beats/min, and a Blood Pressure of 89/47. She was intubated due to impending respiratory distress. Initial Electrocardiogram (ECG) done showed Atrial Fibrillation (AF). Repeated ECG showed ST-segment Elevation over the Lateral leads with reciprocal ST depression. High-Sensitivity Troponin-T taken on arrival was markedly raised at 957 ng/L, and repeated 2 hours later was 3089 ng/L. Patient was rushed for an emergency angiogram by the cardiology team which revealed unobstructed coronaries. Echocardiography performed was normal.

A thyroid function test (TFT) taken due to AF revealed Free T4 of 33 pmol/L with a suppressed TSH of 0.02 mIU/L. Alanine Aminotransferase (ALT) taken was 383 U/L attributable to ischemic hepatitis. Patient was commenced on carbimazole 10 mg daily with careful daily ALT monitoring. Lugol's Iodine 10 drops TDS and IV hydrocortisone 100 mg TDS were given for 5 days. Patient eventually improved with normalization of TFT and liver profile with tapering

carbimazole dose. TSH-receptor Antibodies (TRAb) taken was negative and we referred this patient for an outpatient Thyroid Ultrasonography to rule out toxic adenoma.

CONCLUSION

TpNOCA may be induced by hyperthyroidism due to heightened oxygen demand and coronary vasospasm leading to Type-2 Myocardial Infarction in the presence of unobstructed coronary arteries. Prompt identification and management of hyperthyroidism is crucial to avert severe complications and ensuring a favourable outcome.

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A CASE OF HEART FAILURE UNVEILING HIDDEN ACROMEGALY

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INTRODUCTION/BACKGROUND

Acromegaly is a rare disease caused by hypersecretion of growth hormone. Cardiovascular disease is the most common comorbidity in acromegaly and constitutes a leading cause of mortality. However, there is currently limited direct literature addressing heart failure with preserved ejection fraction (HFpEF) in acromegaly. We present a case of acromegaly presenting with heart failure.

CASE

At a district hospital in Kedah, a 46-year-old female with a known case of hypertension since the age of 23 years old presented with dyspnoea on exertion, orthopnoea, and bilateral leg swelling. She had significant weight gain following her hypertension diagnosis. Her physical examination showed a weight of 121 kg, height of 1.75 m, and body mass index of 46.7 kg/m2. Her blood pressure was 141/89 mm Hg with a heart rate of 90 beats/min. Lung examinations revealed coarse crepitations with bilateral pitting oedema. A comprehensive physical examination revealed spade-like hands and feet, prominent supraorbital ridges, widening of teeth spaces with thick lips, and an enlarged nose. Given the characteristic clinical findings, we suspected the provisional diagnosis of acromegaly. Chest radiography showed cardiomegaly with congestive features. Echocardiogram revealed an ejection fraction of 57%, mildly dilated left atrium with grade 1 diastolic dysfunction which is consistent with HFpEF. Laboratory workup showed elevated insulin-like growth factor 1 level of 278.4ng/ml (normal 56.8-194.5 ng/ml). Subsequently, she was referred to an endocrinologist in a tertiary centre for further investigation and treatment.

CONCLUSION

This case highlights the critical importance in recognizing acromegaly as a rare underlying cause of cardiac manifestations. The clinical suspicion based on physical examination can facilitate prompt diagnosis to prevent early cardiovascular death in acromegaly patients. Clinicians should maintain a high index of suspicion for endocrine disorders that may present with cardiovascular manifestations.

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A RARE PRESENTATION OF MEDULLARY THYROID CARCINOMA: A CASE REPORT

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INTRODUCTION/BACKGROUND

Medullary thyroid carcinoma (MTC) is a rare neuroendocrine tumour arising from the parafollicular C cells of the thyroid gland, accounting for approximately 4% of all thyroid malignancies. We present a case of MTC with an unusual and life-threatening initial manifestation — cardiac tamponade — which led to the diagnosis.

CASE

A 63-year-old Kadazan male with a medical history of myocardial infarction with non-obstructive coronary arteries (MINOCA) in 2017, intracranial haemorrhage in 2018, polycythaemia rubra vera, dyslipidaemia, hypertension, and type 2 diabetes mellitus, presented with a three-day history of exertional dyspnoea and chest tightness. He also reported a gradual neck swelling and unintentional weight loss over the past year.

Initial chest radiography revealed a right lower zone lung opacity, and he was empirically treated for pneumonia. However, a neck ultrasound demonstrated a right thyroid nodule categorized as TIRADS 4, raising suspicion for malignancy. A contrast-enhanced CT (CECT) of the thorax revealed a suspicious right thyroid nodule with bilateral cervical, supraclavicular, and mediastinal lymphadenopathy, multiple pulmonary nodules, a segment VIII liver lesion, and a significant global pericardial effusion measuring 2.8 cm. Fine needle aspiration cytology (FNAC) of the right thyroid nodule and left cervical lymph node confirmed medullary thyroid carcinoma, with positive staining for calcitonin and amyloid deposits identified