and often delayed. Despite their size and invasiveness, macroprolactinomas in postmenopausal women generally respond well to treatment with dopamine agonists.

EP_A134

CHARCOT ARTHROPATHY IN A CONTROLLED DIABETIC PATIENT: A CASE REPORT

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INTRODUCTION/BACKGROUND

Charcot arthropathy is a severe complication of diabetes which is often diagnosed late, characterized by a red, warm, and swollen foot with bone abnormalities on imaging. Most studies report elevated HbA1c as a risk factor in Charcot patients, but there are rare cases with normal HbA1c. If not promptly diagnosed and treated, the condition can lead to deformity, foot ulcers, amputation, and death.

CASE

A 54-year-old male came to M Djamil General Hospital with complaints of ulcers around the right ankle. The patient has a history of diabetes mellitus (13 years). We found deformity with ulcers and pus in the right ankle joint. We did several examinations to confirm the diagnosis. The laboratory results are random blood glucose 152 mg/dL; fasting blood glucose 65 mg/dL; two-hours postprandial glucose 111 mg/dL; HbA1c 7.0%. CT scan of the lower extremities found osteomyelitis of the tarsal bones with cellulitis; histopathology found chronic and acute inflammation with granulation tissue. The working diagnosis was Charcot arthropathy of the right distal tibia Brodsky Type 3A, and Type 2 Diabetes Mellitus. We performed immobilization, external fixation, sequestrectomy and boot casting and controlled glycemia with medical nutrition therapy and rapid acting insulin for perioperative management. We used antibiotics and analgesics to treat infection and pain. The results were good and the patient was advised to use ankle foot orthosis.

CONCLUSION

This is a rare case report of Charcot arthropathy in a patient with normal HbA1c. This condition may be associated with rapid HbA1c normalization, which can trigger acute episodes, and the duration of diabetes. Clinicians should assess glycemic history and neuropathic risk factors. Target HbA1c between 7.0 – 8.0% during treatment can facilitate wound healing without increasing mortality.

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SEVERE HYPOTHYROIDISM-INDUCED RHABDOMYOLYSIS IN THE ABSENCE OF A TRIGGERING FACTOR

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INTRODUCTION/BACKGROUND

Thyroid disorders are among the most common endocrine diseases globally, with hypothyroidism affecting approximately 3.4% of the Malaysian population. Muscle-related symptoms, such as fatigue, cramps, and myalgia are frequently observed in hypothyroidism and usually present with mild to moderate elevations of the muscle enzymes. However, rhabdomyolysis due to hypothyroidism, particularly in the absence of other apparent causes, is rare and is more frequently associated with Hashimoto's thyroiditis. The exact mechanism remains unclear, but it is hypothesized that hypothyroidism disrupts muscle metabolism, leading to prolonged oxidative damage and subsequently rhabdomyolysis.

CASE

We report a case of a 32-year-old male with no prior medical history who presented with one month of weight gain and lethargy, associated with facial puffiness for 2 weeks. He denied systemic symptoms, strenuous activity, trauma, alcohol use, or recent medications. No family history of thyroid or autoimmune disease was noted. Examination showed mild facial puffiness, no muscle weakness, and normal reflexes. Laboratory investigations revealed elevated creatinine kinase (CK) levels of 2,527 U/L (55-170), aspartate transaminase (AST) of 130.3 U/L (8-33), alanine transaminase (ALT) of 118.1 U/L (7-56) and acute kidney injury with urea 7.3 mmol/L (7-12), creatinine 182 μmol/L,

and eGFR 37.6 mL/min. Thyroid function tests confirmed severe hypothyroidism with free T4 7.60 pmol/L (NR: 7.86-14.41), and TSH >49.40 mIU/L (NR: 0.38-5.33) with positive thyroid peroxidase antibody, confirming Hashimoto's thyroiditis. No other causes for rhabdomyolysis were identified and autoimmune hepatitis screening was negative. The patient was managed with aggressive intravenous hydration and levothyroxine replacement therapy, resulting in clinical and biochemical resolution.

CONCLUSION

This case underscores the importance of considering hypothyroidism in the differential diagnosis of unexplained rhabdomyolysis, especially in the absence of conventional triggers. Prompt recognition and early treatment are essential in preventing complications and ensuring optimal patient outcome.

EP_A136

MEMBRANOUS NEPHROPATHY IN A PATIENT WITH ELEVATED CARCINOEMBRYONIC ANTIGEN: AN UNUSUAL PRESENTATION OF MEDULLARY THYROID CARCINOMA

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INTRODUCTION/BACKGROUND

Membranous nephropathy is an important cause of nephrotic syndrome where immune complexes are deposited at the subepithelial space of the glomerular basement membrane. Membranous nephropathy is classified into primary and secondary causes, including infections, autoimmune, neoplasms, drugs or idiopathic. Medullary thyroid carcinoma (MTC) is a relatively rare malignant tumour subtype originating from the parafollicular cells of the thyroid gland, producing tumour markers such as calcitonin, carcinoembryonic antigen (CEA) and chromogranin A. The co-occurrence of membranous nephropathy with MTC is extremely uncommon, and such an association may reflect a paraneoplastic manifestation or an underlying monoclonal gammopathy.

CASE

We report the case of a 68-year-old woman who presented with progressive shortness of breath, bilateral lower limb edema, frothy urine, and periorbital puffiness. She denied orthopnea, paroxysmal nocturnal dyspnea, constitutional symptoms, or features suggestive of autoimmune disease. Initial workup revealed nephrotic-range proteinuria, and a renal biopsy demonstrated early membranous nephropathy. Notably, her CEA level was persistently elevated; however, upper and lower gastrointestinal endoscopies showed only benign findings, including a hiatal hernia and sigmoid colon diverticulum. Contrast-enhanced CT imaging revealed a retrosternal goitre, while FDG-PET scanning identified an FDG-avid lesion in the left thyroid lobe with ipsilateral cervical lymphadenopathy. Thyroid ultrasound showed a TIRADS 4 nodule, and subsequent core needle biopsy confirmed MTC. She underwent total thyroidectomy and modified neck dissection without complications. At one-month postoperative follow-up, her proteinuria had slightly improved.

CONCLUSION

This case underscores the importance of a thorough malignancy workup in atypical presentations of nephrotic syndrome and highlights a rare paraneoplastic link between MTC and glomerular disease. In the context of raised CEA with negative findings despite extensive investigations for gastrointestinal tract causes, one might need to consider other non-gastrointestinal related causes for raised CEA such as medullary thyroid carcinoma.

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NOT JUST TYPE 2 DIABETES: SEVERE INSULIN RESISTANCE WITH ATYPICAL FAT DISTRIBUTION SUGGESTS LIPODYSTROPHY SYNDROME

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INTRODUCTION/BACKGROUND

Lipodystrophy syndromes are rare disorders of adipose tissue distribution, often leading to severe insulin resistance and metabolic complications. We present a case of a young woman initially diagnosed with type 2 diabetes, unresponsive to standard insulin therapy, who was ultimately diagnosed with familial partial lipodystrophy (FPLD).

CASE

A 26-year-old female, with diabetes diagnosed 4 years ago, was referred for uncontrolled capillary blood glucose levels persistently ranging from 20–25 mmol/L despite high-dose basal-bolus insulin and oral hypoglycemic agents. Her body mass index (BMI) was 22 kg/m^2 with an HbA1c of 12.2%. Autoantibody screening (GAD, ICA, IA2 antibodies) was negative, and C-peptide was markedly elevated (2193 pmol/L). Her metabolic profile showed hypertriglyceridemia, raised liver enzymes suggestive