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.

## EP_A137

### 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