Weight and waist circumference differences pre- and post-Ramadan were not significant; there was more weight reduction (-1.48 vs -0.53 kg) and waist circumference reduction (-1.07 vs +1.50 cm) with NovoMix 30.

CONCLUSION
Humalog Mix 50 demonstrated similar efficacy and safety compared to NovoMix 30 during Ramadan in T2DM with a potential benefit on fructosamine reduction.

PA-A-51

EUGLYCAEMIC DKA CASES: TWO CAUTIONARY TALES

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Abdullah Shamshir Abd Mokti and Miza Hiryanti Zakaria
Endocrine Unit, Medical Department, Hospital Tengku Ampuan Afzan, Kuantan, Malaysia

INTRODUCTION
Euglycaemic diabetic ketoacidosis (euDKA) presents with a triad of high anion gap metabolic acidosis, ketonemia and normal blood glucose. It has been increasingly reported with the increased use of SGLT-2 inhibitors. Here, we describe two euDKA cases of different aetiologies.

CASES
The first case is a 29-year-old pregnant female with type 1 diabetes diagnosed at 8 years old with good control pre-partum (HbA1c 6.8%) and only 1 previous DKA at diagnosis. During pregnancy, glycaemic control worsened, requiring high doses of insulin. She presented at 33 weeks of gestation with reduced fetal movement alongside epigastric discomfort, vomiting and dyspnoea. Ultrasound by her obstetrician revealed no fetal movement and investigations showed high anion gap metabolic acidosis with pH 6.9, bicarbonate 12 nmol/l while capillary blood glucose (CBG) was 10.3 mmol/l and serum ketone was 6.5 mmol/l. EuDKA precipitated by intrauterine death was diagnosed, treatment was started and patient underwent C-section. Postoperatively, acidosis and ketonaemia resolved and CBG was controlled with low dose insulin.

The second case is a 56-year-old female with poorly-controlled type 2 diabetes (HbA1c 12%) who was diagnosed with upper gastrointestinal bleeding. Blood investigations showed CBG 8.5 mmol/l, normal blood gasses and disproportionate urea:creatinine ratio. She was kept nil by mouth while waiting for gastroscopy. Eight hours later she developed tachypnoea and worsening epigastric pain; repeat blood investigations showed pH 7.0, bicarbonate 17 nmol/l, CBG 11 mmol/l and serum ketone 4.3 mmol/l. EuDKA was diagnosed and she was treated promptly and acidosis and ketonaemia resolved.

CONCLUSION
These two cases illustrate the need for a high index of suspicion for euDKA in diabetics undergoing stressful conditions and the importance of measuring serum ketone in metabolic acidosis even in patients with normal blood glucose.

PA-A-52

METASTATIC BLADDER PARAGANGLIOMA WITH UNDERLYING SHDB MUTATION

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Ooi Xin Yi
Hospital Kuala Lumpur, Malaysia

INTRODUCTION
Phaeochromocytoma and paraganglioma (PPGL) are rare tumors with up to 40% associated with inherited germline mutations. SHDB mutation is associated with an increased risk of metastasis.

CASE
A 36-year-old male presented with hypertensive emergency. He was diagnosed to have a bladder paraganglioma at age 32 when he presented with hypertensive crisis. Ga-68 DOTANOC PET/CT scan then showed a localized 4.7 x 5.3 cm bladder paraganglioma and he underwent complete surgical resection with resolution of his symptoms. Genetic testing done showed SHDB, deletion (exon 1), heterogenous pathogenic variant. He remained asymptomatic and was lost to follow-up due to COVID-19 until his recent admission.

During this admission, he had labile blood pressure with symptoms of palpitations and lethargy. He was found to have a 4.3x elevated urine normetanephrine (1639 ug/day, N<374.7). Metanephrine and 3-methoxytyramine levels were normal. His blood pressure was controlled with phenoxybenzamine 20 mg TDS (1 mg/kg), telmisartan 40 mg OM and carvedilol 25 mg BD with improvement in his symptoms. Subsequent anatomical imaging with CT and functional imaging with Ga-68 DOTATATE showed a small recurrence at the bladder wall with metastatic lesions at the left sacral ala measuring 4.5 x 5.1 cm, and multiple lytic lesions over the spine, ribs and also the left acetabulum with the highest uptake of Ga-68 DOTATATE at the C2 vertebra (SUV max 93). He is now planned for peptide receptor radionuclide therapy (PRRT).

SHDB mutation is associated with a higher risk of metastatic disease which has remained unexplained. Treatment for metastatic disease include surgical resection where possible, targeted therapy such as PRRT, meta-iodobenzylguanidine (MIBG) therapy, radiotherapy and also systemic therapy such as chemotherapy and tyrosine kinase inhibitors.