

## **PA-A-49**

## SERENDIPITOUS DISCOVERY OF HYPERCALCEMIA IN PREGNANCY IN A PATIENT WITH ASYMPTOMATIC BRADYCARDIA ON BETA BLOCKER

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### INTRODUCTION

Hypercalcemia in pregnancy is uncommon and the diagnosis is challenging due to its nonspecific symptoms which may mimic those of pregnancy. The risk of adverse outcomes for the mother and neonate is significant and should be identified earlier.

### CASE

We report a case of a patient with parathyroid-dependent hypercalcemia diagnosed in the third trimester of pregnancy with subsequent complications necessitating emergency caesarean section at 35 weeks of gestation.

A 30-year-old pregnant female with chronic hypertension presented at 30 weeks of gestation with asymptomatic bradycardia while on treatment with a beta blocker. She also complained of abdominal discomfort for the past week. On examination, her heart rate was 40-50 beats per minute with no other remarkable findings. Laboratory investigations revealed parathyroid hormone-dependent hypercalcemia with a serum calcium of 2.9 mmol/L and a parathyroid hormone level of 13.3 pmol. 24-hour urine calcium/creatinine clearance ratio was 0.06 and 25-hydroxyvitamin D level was adequate at 80.81 nmol/L. Neck ultrasonography was negative for parathyroid adenoma. Patient was admitted for hydration, however, due to persistent hypercalcemia, cinacalcet 25 mg daily was commenced at 33 weeks of gestation. Serial foetal growth scans demonstrated a small for gestational age (SGA) fetus. She presented at 35 weeks of gestation with severe preeclampsia, pancreatitis and serum calcium level of 3.03 mmol/L, requiring an emergency caesarean section. A male baby, weighing 1970 g, was delivered without any calcium disturbances observed. Patient's calcium levels remained elevated post-partum, requiring saline hydration and subcutaneous calcitonin. Her serum calcium level normalised over the next few days after a dose of pamidronate was given at day 4 post-partum.

### **CONCLUSION**

Hypercalcemia in pregnancy may result in significant maternal and foetal complications. A high index of suspicion is required for early diagnosis and institution of treatment to improve maternal and foetal outcomes.

### **PA-A-50**

EFFICACY OF 50% INSULIN LISPRO AND 50% INSULIN LISPRO PROTAMINE SUSPENSION(HUMALOG MIX50) VERSUS BIPHASIC INSULIN ASPART (NOVOMIX 30) IN PATIENTS WITH TYPE 2 DIABETES MELLITUS DURING RAMADAN

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#### INTRODUCTION

The month of Ramadan has always been challenging to patients and doctors due to the increased risk of hypoand hyperglycaemia attributed to large meals during the breaking of fast. Previous studies demonstrated that both Humalog Mix 50 and NovoMix 30 are safe in Ramadan. However, there is little information on their efficacy and safety in Asian populations.

The aim of this study is to evaluate the efficacy and safety of Humalog Mix 50 in comparison to NovoMix 30 in patients with type 2 diabetes mellitus (T2DM) during Ramadan.

### **METHODOLOGY**

We performed a prospective study in T2DM patients seen at the Endocrinology Clinic of UITM in the year 2018, who fasted during Ramadan. They were randomized to receive either Humalog Mix50 or NovoMix 30. Weight and fructosamine pre- and post-Ramadan, capillary glucose at 4-points per day (pre-sahur, noon, pre-iftar and post-iftar) were recorded.

### **RESULTS**

Twenty-three (23) patients (15 on NovoMix 30 and 8 on Humalog Mix 50) with an overall mean age of 57 were analysed.

NovoMix 30 showed a better glycaemic trend at pre-sahur  $(8.3 \pm 1.8 \text{ vs } 9.3 \pm 1.4)$ , during the day  $(7.4 \pm 0.9 \text{ vs } 7.7 \pm 1.5)$ , and 2-hours post-iftar  $(10.1 \pm 2.7 \text{ vs } 10.4 \pm 3.8)$ , although not significant. There were 4 patients who experienced hypoglycaemia in the NovoMix 30 arm (one with glucose 3.3 mmol/L (noon during fasting), and the others with glucose 3.8 mmol/L. Fructosamine difference pre- and post-Ramadan showed reduction of 12.62 mmol/L (HbA1c 1.82%) with Humalog Mix 50 and 7.133 mmol/L (HbA1c 1.73%) with NovoMix 30 (p=0.69).



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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### 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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### 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  $4.7 \times 5.3$  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.