mutations in phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha, which induce hyperactivation of phosphatidylinositol 3-kinase (PI3K) contributing to resistance to endocrine therapy. The use of PI3K inhibitor (alpelisib) in combination with fulvestrant has been approved for the treatment of postmenopausal women with HR+/HER2-, PIK3CA- mutated advanced breast cancer. Hyperglycemia is the most common side effect of Alpelisib.

CASE
We describe a 54-year-old female with diabetes who developed uncontrolled hyperglycaemia after initiation of Alpelisib despite taking Vildagliptin and basal-bolus insulin (total daily dose: 52 units per day). Before alpelisib initiation, this patient had good glycaemic control with HbA1c of 6.7% while on Metformin 500 mg BD. Her oncologist discontinued Metformin and started the patient on Vildagliptin 50 mg OD due to renal impairment. Her blood glucose levels (monitored by a continuous glucose monitoring device) significantly worsened once alpelisib was started. On day 1 of treatment, her sugar increased to more than 10 mmol/L, thus basal-bolus insulin was started. Despite basal-bolus insulin (S/C Glulisine 12 units TDS, S/C Insulatard 16 units ON), her glucose remained in the range of 10 to 17 mmol/L. Empagliflozin was started on day 8 of Alpelisib treatment. With Empagliflozin, blood glucose levels improved, ranging between 6 to 10 mmol/L, and we were able to discontinue insulin therapy.

CONCLUSION
We report the successful management of alpelisib-induced hyperglycaemia with the use of SGLT-2 inhibitor.

EP_A013

SEVERE HYPERTRIGLYCERIDEMIA IN A NEWLY DIAGNOSED TYPE 1 DIABETES PATIENT WITH DIABETIC KETOACIDOSIS

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INTRODUCTION/BACKGROUND
Elevated triglycerides are often noticed during periods of insulin deficiency. Severe hypertriglyceridemia (Triglyceride >10 mmol/L) is an uncommon complication of diabetic ketoacidosis (DKA) and is associated with an increased risk of acute pancreatitis.

CASE
A 14-year-old female student with a history of COVID-19, one month prior, presented with a one-day history of severe abdominal pain and breathlessness. This was preceded by a 2-month history of weight loss of 5 kg. She had severe metabolic acidosis and was intubated due to respiratory distress.

Laboratory results showed blood glucose of 19.8 mmol/L, serum ketones of 6.2 mmol/L, pH 6.99 and serum bicarbonate of 5.6 mmol/L. Serum amylase and urine diastase were normal. Her plasma had a "milky" appearance, and her total cholesterol level was 41 mmol/L with a triglyceride (TG) of 199 mmol/L. She was managed in the intensive care unit with fluid resuscitation, dietary restriction, fenofibrate and high-dose insulin infusion of up to 0.2 U/kg/hour. She responded well with TG levels reduced to 7.37 mmol/L on day 2 of admission. Subsequently, she was transitioned to subcutaneous insulin. Her HbA1c reduced from 15.8% to 7.3% over four months, and her TG improved to 0.5 mmol/L. Her anti-islet cell, anti-GAD and anti-insulin IA2 autoantibodies were strongly positive. Thyroid function test and screening for diabetic complications were negative.

CONCLUSION
Severe hypertriglyceridemia can be effectively managed in the acute situation with high-dose insulin to bring down the triglyceride level. Optimal glycaemic control also plays an important role in maintaining suppressed triglyceride levels.

EP_A014

EUGLYCAEMIC DIABETIC KETOACIDOSIS AS A CAUSE OF REFRACTORY METABOLIC ACIDOSIS IN A PREGNANT PATIENT

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INTRODUCTION/BACKGROUND
Euglycemic diabetic ketoacidosis (DKA) in pregnancy is a rare obstetric emergency that may lead to substantial morbidity and mortality to both the mother and foetus. Prompt recognition is challenging due to misleading euglycemic state. The risk for euglycaemic DKA increases during the second half of pregnancy due to the higher levels of hormones with anti-insulin effects, increase in insulin demand, combined with exhausted glycogen stores.

CASE
We report a case of a 33-year-old female G3P2 at 33 weeks AOG, admitted for fever, cough, vomiting and poor oral intake for three days. Antenatally, she had GDM and was well-controlled on metformin. On arrival she was