Basic Science E-Poster Presentation

EP_B001

NIRMATRELVIR/RITONAVIR-INDUCED HYPONATREMIA PRESENTING WITH SIADH AND GASTROINTESTINAL LOSS: A CASE REPORT

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INTRODUCTION/BACKGROUND
Nirmatrelvir/ritonavir (Paxlovid™) is an antiviral used to treat mild to moderate COVID-19 infection. Hyponatremia in COVID-19 has been documented in several case reports but none related to treatment with nirmatrelvir/ritonavir to date.

CASE
A 75-year-old female with diabetes mellitus, hypertension and bronchial asthma was treated as COVID-19 Category 2b with symptoms of fever, diarrhoea and poor oral intake. Serum sodium on admission was 138 mmol/L. She was given nirmatrelvir/ritonavir and intravenous drip due to ongoing gastrointestinal losses. Her symptoms resolved in the next two days. On day 7 of illness, she started having new-onset diarrhoea. Repeat Na was 112 mmol/L. Further hydration led to declining Na levels until she became symptomatic at 105 mmol/L. Her paired sample results suggested SIADH, with serum osmolarity 228 mOsm/kg, urine osmolarity 233 mOsm/kg and urine Na 84 mmol/L despite ongoing gastrointestinal losses and clinical signs of hypovolemia. Nirmatrelvir/ritonavir was already discontinued. She was treated with hypertonic 3% saline until her symptoms improved. Her diarrhoea resolved, and her Na levels normalized with fluid restriction and oral salt treatment.

CONCLUSION
Nirmatrelvir/ritonavir has been reported to cause elevated transaminases, hypertension and diarrhoea, apart from its known drug-drug interactions. This case highlights the importance of SIADH as one of the possible adverse effects of this new drug which is now widely used for the management of COVID-19. It is important for clinicians to be vigilant about drug-induced SIADH when investigating the cause of hyponatremia in patients with COVID-19.

EP_B002

INHIBITION OF LIPID ACCUMULATION AND UPREGULATION OF BROWNING GENES BY SIRT1 ACTIVATION IN 3T3-L1 CELLS

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INTRODUCTION
Obesity is the cause of over 4 million deaths each year according to the Global Burden of Disease study. It is characterized by an increase in adipocyte number and size, making adipogenesis an important target in the prevention and management of obesity. A possible approach is to induce adipose cells into becoming thermogenesis-competent, such as brown or beige adipose cells. In this study, the effect of activating Sirtuin1 (Sirt1) in 3T3-L1 and its role in promoting the browning of white adipose cells was investigated.

METHODOLOGY
White pre-adipocyte cell lines 3T3-L1 were treated with Sirt1 activator (SRT1720) 2.5 mM, Sirt1 inhibitor (EX527) 10 mM or rosiglitazone (positive control for adipogenesis) 50 mM throughout ten days of adipogenic differentiation period. The effect of these treatments was assessed by western blotting, oil red O lipid staining and real-time PCR.

RESULTS
Inducing Sirt1 activity affected intracellular lipid accumulation. This was based on the observation of lesser accumulation of lipid in Sirt1-activated cells stained with oil red O. In contrast, when Sirt1 activity was attenuated by Sirt1-specific inhibitor, lipid production increased. Further investigation showed that the expression of genes critical for brown adipose tissue regulation, such as PGC1a and Cidea, were found to be elevated when Sirt1 activity was induced.

CONCLUSION
This work shows that targeting Sirt1 activity in white adipose cells would instigate an underlying molecular network that modulates the adipogenesis process during cell differentiation by producing less lipid. It also promotes the cells to differentiate into brown-like adipose cells that

have better thermogenic capability. This can serve as a target for further investigation for therapeutic intervention in obesity.

EP_B003

CENTRAL DIABETES INSIPIDUS WITH COVID-19 PNEUMONIA: A CASE REPORT

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INTRODUCTION
Although COVID-19 is well-known for its respiratory manifestations, extrapulmonary presentations such as cardiac arrythmias, acute coronary syndrome, thrombosis, neurologic and ocular illnesses have also been reported. The mechanism of extrapulmonary complications of COVID-19 involves both the direct effects of SARS-CoV-2 via ACE2 receptors and indirect mechanisms associated with dysregulated host immune response. Apart from the respiratory system, ACE2 receptors are widely expressed in the cardiovascular, gastrointestinal, urogenital and nervous systems, which explains the multisystemic effects seen in COVID-19.

CASE
We present a case of COVID-19 pneumonia complicated by central diabetes insipidus.

A 64-year-old female with hypertension, diabetes mellitus and atrial fibrillation presented with fever and vomiting for two days. She was lethargic, with stable vitals and slight tenderness at the right upper quadrant of abdomen. She was initially treated with antibiotics in the emergency unit for ascending cholangitis. Contrast-enhanced abdominal CT showed cholelithiasis with no intraabdominal collections. COVID-19 GeneXpert tested positive. Chest radiography showed right lower zone opacities. On the ninth day of admission, she had polyuria (6000 mL in 24 hours). Test results showed serum Na 141 mmol/L, serum osmolarity 300 mOsm/kg, urine osmolarity 89 mOsm/kg and urine Na 15 mmol/L, suggestive of diabetes insipidus. She responded well to subcutaneous desmopressin, which reduced urine output to 15 to 30 mL/hour and improved results of her paired samples. While pituitary tests were normal, MRI revealed absence of T1 hyperintensity in the posterior pituitary, supporting the diagnosis of central DI. She required regular desmopressin doses for up to 3 weeks. Her polyuria resolved in her subsequent admission 4 months later for heart failure.

CONCLUSION
This case highlights self-limited diabetes insipidus as one of the extrapulmonary manifestations of COVID-19.

EP_B004

IMPACT OF COVID-19 ON THE INCIDENCE OF NEWLY DIAGNOSED TYPE 1 DIABETES MELLITUS: A SINGLE-CENTRE EXPERIENCE

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INTRODUCTION
Recent studies suggest the increasing occurrence of newly diagnosed type 1 diabetes mellitus during the COVID-19 pandemic. It is postulated that the COVID-19 virus triggers a cascade of autoimmune reactions leading to the development of antibodies against beta cells of the pancreas. This study investigates the incidence of newly diagnosed T1DM in a Malaysian tertiary centre during the COVID-19 era in comparison to the same duration prior to it.

METHODOLOGY
This is a single-centre retrospective cross-sectional study among newly diagnosed T1DM patients. Patients aged between one year to less than 18 years old, who were referred to the Paediatric Endocrine Unit of the University Malaya Medical Centre from September 2017 to August 2022 were included in this study. Data including age, gender, anthropometric measurements, diabetic ketoacidosis occurrence, biochemical results and COVID-19 status for the past three months were obtained.

RESULTS
Fifty-seven patients who fulfilled the criteria of T1DM were included. Thirty-two patients (56%) were diagnosed during the COVID-19 era. Forty-four patients (77%) presented with DKA. There is no difference in the incidence of DKA and the severity status between these two periods, (77% versus 76.7%, p=0.902; and 51.9% versus 53.3%, p=0.546, respectively. Although not statistically significant, more patients needed pediatric ICU admission (13 versus 9), with lower pH at presentation during the COVID-19 era (7.05 versus 7.12). More than a third (37.5%) needed intubation (p=0.019). Recovery was also longer (48 hours versus 36 hours).