triggered by multiple aetiologies including hypertension, eclampsia, cytotoxic and immunosuppressant drugs and, rarely, hypercalcaemia.

Case Report

A 64 years old woman presented with five weeks history of fatigue, poor appetite, dry mouth, constipation and abdominal discomfort and one-week history of nausea and vomiting. She was hypertensive at 177/88 mmHg with dry mucous membranes. Physical examination and neurological examination were unremarkable.

Laboratory investigation showed corrected calcium of 4.83 mmol/L (2.25-2.54) with

Ionized calcium of 2.62 mmol/L (1.15-1.27), parathyroid hormone (PTH) of 1330 ng/l (15-68), phosphate of 1.16 mmol/L(0.8-1.5), magnesium of 0.51 mmol/L (0.7-1.0) urea of 10.7 mmol/L (2.8-8.4), creatinine of 119 umol/L (49-90), potassium 3.4 mmol/L(3.5-5.1). She was aggressively rehydrated, commenced on Intravenous (IV) frusemide and was given IV zoledronic acid. Cinacalcet was commenced and titrated gradually up according to corrected calcium level (target corrected calcium level between 2.5-3.0 mmol/L).Electrolytes deficiencies corrected with replacement therapy.Ultrasound neck and parathyroid MIBG scan showed large 5.1cm heterogeneous lesion posterior to the right lobe of the thyroid extending inferiorly into the superior mediastinum consistent with parathyroid mass. Histology confirmed benign parathyroid adenoma.

38 hours after admission, the patient became intermittently confused and complained of visual symptoms followed by complete visual loss in the left eye. This was followed shortly by status epilepticus which required treatment with intravenous antiepileptic therapy and mechanical ventilation. Corrected calcium at that time was 3.82 mmol/L.Patient was noted to have left upper limb weakness.Computed tomography of the brain was normal and magnetic resonance imaging (MRI) of the brain showed bilateral symmetrical subcortical T2 hyper- intensities in the occipital- parietal lobes consistent with PRES.

By day five, corrected calcium was 2.52 mmol/L. On day six patient had successful parathyroidectomy. Post operatively PTH was 7.73 ng/L and corrected calcium 2.27 mmol/L. Repeated Brain MRI showed resolution of symmetrical subcortical T2 hyperintensities within both occipital lobes. She made a complete neurological recovery. DEXA scan showed osteoporosis (T score in left forearm of -3.8). She was commenced on bisphosphonate therapy.

In conclusion, we demonstrated hypercalcemia-induced PRES. This can be a life-threatening condition and can be reversed by proper treatment of hypercalcemia.

Cardiovascular Endocrinology

PATHOPHYSIOLOGY OF CARDIOMETABOLIC DISEASE

Simvastatin Inhibits the Pro-Inflammatory and Pro-Atherogenic Effects of Cream in Obese Subjects

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SUN-560

Our previous work has shown that the ingestion of cream induces an increase in oxidative stress and other cellular and molecular indices of inflammation and atherosclerosis and that treatment with Vytorin for 6 weeks reduced and reversed majority of the effects of cream. However, it is not clear which component of Vytorin, simvastatin or ezetimibe, is responsible for these intriguing and potent effects. Therefore, we further investigated the effects of simvastatin treatment on indices of inflammation and atherosclerosis at baseline and following intake of dairy cream. Ten obese patients with LDL >100mg/dl were given simvastatin 40mg/day for 6 weeks. Subjects were asked to ingest 33ml of cream (about 300 Calories) containing about 85% saturated fat. Fasting and post-cream intake blood samples were obtained at baseline and at 6 weeks. Total cholesterol and LDLc concentrations were lowered significantly at 6 weeks following simvastatin ($p<0.05$). Cream intake at 0 week induced significant increases in MNC expression of IL-1β (by 58±16%), TNF-α (by 79±19%), CD16 (by 103±32%), MMP-9 (by 68±17%), TLR-4 (by 68±12%) and TLR-2 (by 53±9%) over the baselines ($p<0.05$ for all). Cream intake at 0 week also induced a significant increase in IL-1β plasma concentrations by 94±18% over the baseline. Simvastatin treatment suppressed fasting levels of CD68 expression in MNC (by 38±9, $p<0.05$) and fasting plasma levels of IL-18 and MMP-9 (by 24±11% and 28±12%, respectively, $p>0.05$) compared to fasting levels at 0 week. The increase in IL-1β, TNFα, CD16 and MMP-9 expression in MNC following cream intake at end of simvastatin treatment was significantly suppressed (by 41±15%, 48±17%, 87±16% and 34±8%, respectively, $p<0.05$) compared to that before simvastatin treatment. In addition, there was a paradoxical suppression of the expression of TLR-2 and TLR-4 (by 30±11% and 24±9%, respectively) below baseline levels following cream at 6 weeks. Simvastatin treatment also suppressed cream induced increases in plasma IL-1β concentrations by 37±11% ($p<0.05$, compared to increases at 0 week). We conclude that simvastatin exerts a powerful anti-inflammatory effect and reduces expression of pro-inflammatory mediators induced by cream intake. This effect is similar in nature to that observed previously with Vytorin with some differences in the magnitude of the changes.

Diabetes Mellitus and Glucose Metabolism

ISLETS AND INSULIN SECRETION

Cornus Officinalis Promotes IGFBP2 and Autophagy in Human 1.1B4 Pancreatic Cell Line as Revealed by Employing a Global Proteomic Approach via Mass Spectrometry

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Type 1 diabetes (T1D) results in the loss of pancreatic beta cells and subsequent loss of insulin production. Exogenous