a Karyotype in fibroblasts culture from oral cavity sample. The results revealed low IGF-1 and mosaic TS in 14%. We preformed 2 provocative tests which revealed low growth hormone peak < 5 ng/ml. A brain and pituitary MRI to exclude pituitary lesions or structural abnormalities revealed glomias of the optic chiasma and the right optic nerve with characteristic NF1 “spots” (regions of signal abnormality in T2 sequences) involving the basal ganglia, cerebellum and the right temporal lobe. DNA sequencing targeted to a gene panel related to NF1 and NF2 revealed a novel de novo heterozygous NF1 gene mutation in exon 28 [3764A>G];[=] p.[Gln1255Arg].

Discussion: NF1- Glomias are most commonly seen in young children, (mean 4.5 years). Only 1/3 of affected children will require therapeutic intervention. However early diagnosis, of optic gliomas is important. Our patient was completely asymptomatic by the time of diagnosis and no other symptom or sign of NF1 was apparent. Ophthalmologic examination was normal, but visual electrophysiologic testing was abnormal as far the right optic nerve is concerned. The oncology team decided to perform chemotherapy. In TS impaired growth is related to resistance in GH. Some studies suggested that there could be a relationship between GHD and NF1 even in the absence of an organic pituitary damage. In our patient it has been decided not to treat with GH and closely track the patient’s growth.

Conclusion: Coexistence of NF1 with TS is rare. Awareness is needed as early identification and treatment of CNS gliomas can prevent visual loss and severe co-morbidities.

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Reproductive Endocrinology

MALE REPRODUCTIVE HEALTH - FROM HORMONES TO GAMETES

Testosterone Reduces Atherosclerosis and Plaque Specific Inflammatory Markers in the ApoE-/- Mouse Model

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SAT-041

Low serum testosterone in men is an established cardiovascular risk factor and epidemiological evidence demonstrates an association between low testosterone and with coronary events. Clinical evidence suggests that testosterone therapy (Tth) can improve key cardiovascular risk factors in men and surrogate measures of atherosclerosis, the chronic inflammatory process underlying cardiovascular disease. Atherosclerotic plaque-specific testosterone actions are not fully understood. The present study investigates the influence of testosterone on mediators of vascular inflammation and plaque burden in an in vivo model of atherosclerosis. ApoE-/- mice were either sham operated, castrated or castrated and received fortnightly intramuscular injections of physiological doses of testosterone (mixed testosterone esters, Sustanon 100) to create 3 experimental groups; normal testosterone, testosterone deficient and testosterone replaced respectively. All groups were fed a high-fat ‘Western’ diet for 16 weeks. Lipid deposition in the aortic root was assessed by Oil Red O as an indication of atherosclerotic burden. Plaque composition was assessed immunohistochemically for indicators of stability including collagen content via Masson’s trichrome, and α-smooth muscle actin (αSMA) as well as markers of inflammation including vascular cell adhesion molecule-1 (VCAM-1), intracellular adhesion molecule-1 (ICAM-1), endothelial-leukocyte adhesion molecule 1 (E-Selectin), and a pan monocyte/macrophage marker (MOMA2). Testosterone deficient castrated mice had significantly increased lipid accumulation in the aortic root compared to testosterone replete sham-operated littermates (48% intima-media area vs 40%, p<0.05). Tth in castrated mice reversed this effect (39%, p<0.05). Plaque stability was not altered between groups. MOMA2 staining indicated increased infiltration and localisation of monocytes/macrophages in the plaques of castrated mice compared to sham-operated (positive staining (% of plaque) 77% vs 59%, P=0.062) and Tth treatment reduced this (77% vs 63%, P=0.1). Vascular cell adhesion molecule-1 (VCAM-1) and intracellular adhesion molecule-1 (ICAM-1) expression were reduced in castrated mice receiving Tth compared to castrated mice receiving saline (33% vs 46%, p<0.05; and 39% vs 58%, P=0.084 respectively). No significant difference in expression of E-Selectin and αSMA were observed between groups. These findings demonstrate that low testosterone increases aortic root lipid deposition and inflammatory composition in a mouse model of atherosclerosis. Increasing testosterone levels through Tth reduces plaque specific inflammatory markers and atherosclerotic burden. This indicates an anti-inflammatory mechanism by which testosterone can protect against the development and progression of atherosclerosis to reduce cardiovascular risk in men.

Diabetes Mellitus and Glucose Metabolism

TYPE 1 DIABETES MELLITUS

A Case of Autoimmune Polyglandular Syndrome Type 2 and Guillain-Barré Syndrome

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SAT-683

Background: Autoimmune polyglandular syndrome type 2 (APS2) is defined by the occurrence of two or more autoimmune diseases, with Addison’s disease being most prevalent, and autoimmune thyroid disease and type 1 diabetes mellitus also being common. Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyradiculopathy that is also autoimmune in nature, resulting in ascending muscle weakness or paralysis.

Clinical Case: A 49 year old female with past medical history of vitiligo, subclinical hyperthyroidism, and Guillain-Barré
syndrome (GBS) presented to our institution with fatigue, nausea, vomiting, polyuria, and polydipsia. She had no history of diabetes. Her history was also significant for GBS, diagnosed 5 months prior to her current admission. She was treated with intravenous immunoglobulin (IVIG) and had partial improvement of motor impairment. On exam, she was noted to have dry mucous membranes, epigastric tenderness, and patches of hypopigmented skin. Laboratory studies were consistent with diabetic ketoacidosis, and she was admitted to the ICU for management.

Labs from 5 months prior were significant for a HbA1c of 6.4% (4.0-5.6%), TSH <0.002 mIU/L (0.550-4.7 mIU/L), total T3 154.9 ng/dL (79-149 ng/dL), and free T4 1.7 ng/dL (0.7-1.9 ng/dL), and elevated thyroid stimulating immunoglobulin. During the current admission, HbA1c had risen to 13.6%, C-Peptide 0.6 ng/mL (1.1-4.4 ng/mL) and GAD-65 antibody >250 IU/mL (<5 IU/mL), consistent with a diagnosis of late-onset type 1 diabetes. Repeat thyroid function tests (TSH <0.002 mIU/L, total T3 74 ng/dL, and free T4 1.2 ng/dL), were consistent with subclinical hyperthyroidism. A 21-hydroxylase antibody level was 13 U/mL (<1 U/mL), but cortisol rose appropriately in response to cosyntropin. Based on the patient’s constellation of vitiligo, autoimmune thyroid disease, type 1 diabetes, and elevated 21-hydroxylase antibodies, she was diagnosed with APS2.

Conclusion: We present an unusual case of a patient with APS2, who was diagnosed with type 1 diabetes 5 months after developing GBS and being treated with IVIG. Prior reports demonstrate an association between GBS and other autoimmune diseases, including one case report of GBS in a patient with APS2. HLA DR3 has been associated with APS2, type 1 diabetes, Addison’s disease and Grave’s disease. Its association with GBS is less clear, although HLA DR3 was increased in one Mexican cohort with GBS. This case report adds to the literature suggesting an association between GBS and other autoimmune diseases, specifically, with APS2.

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Diabetes Mellitus and Glucose Metabolism
TYPE 2 DIABETES MELLITUS
Case Study of Nonalcoholic Steatohepatitis Reversibility in Type Two Diabetic Patient with Weight Loss Using Liraglutide
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SUN-694
Case Study of Nonalcoholic Steatohepatitis Reversibility in Type Two Diabetic Patient with Weight Loss Using Liraglutide
Introduction

Nonalcoholic fatty liver disease (NAFLD) is a common form of chronic liver disease.1 NAFLD prevalence is likely to be between 75-100% in the morbidity obese. Where Nonalcoholic steatohepatitis (NASH) in turns develops in 30% of patients with NAFLD. Obesity prevalence in Kuwait is estimated to be 39% for adults male and 52% for adults females.2 No pharmacotherapy is approved for NAFLD treatment, and the basis treatment is lifestyle modifications focusing on body fat loss.3 A study showed that may take 10% or more weight loss to have an impact on NASH Activity Scores as assessed by liver biopsy. Glucagon-like peptide-1 receptor agonists (GLP-1 RA) are approved for the treatment of T2D and obesity and have also been shown to reduce liver inflammation and fibrosis.3

Methods
A 54 years old male presented to our clinic with a history of hypothyroidism, diabetes with Hemoglobin A1C (HbA1c) of 7%, hypercholesterolemia and overweight with Body Mass Index (BMI) of 27.6 kg/m2. Patient’s investigations showed high Gamma-glutamyl transferase (GGT) and ferritin level, and due to his abnormal liver function test patient was referred for abdominal ultra sound that showed fatty liver disease. With a high NAFLD score, he was referred for Fibroscan that showed fibrosis score of F3; which indicates a severe liver scarring. GLP-1 RA was started for weight management and a better glycemic control in a setting of multidisciplinary team, including endocrinologist, diabetes educator, dietitian and a physical trainer.

Results
In a six months’ period of time, patient was able to lose 15.3% of his total body weight, with better glycemic control of HbA1c of 5.6% from 7% and his repeated Fibroscan showed improvement in his score from F3 to F0 with other clinically important outcomes.

Conclusion
GLP-1 RA seems to be safe to use for patients with NASH, and it might have benefits of reversing fibrosis in addition to other benefits such as weight reduction and HbA1c improvement.

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Genetics and Development (including Gene Regulation)
GENETICS AND DEVELOPMENT AND NON-STEROID HORMONE SIGNALING I
Custom Panel to Diagnosis Genetic Endocrine Disorders in a Tertiary Academic Hospital
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