and for the IGD M group, it was in response to whether they felt they were a real man.

Conclusions: Men and women with IGD did not show a significant difference in their gender identity compared with controls, and gender was found to be congruent with SAAB for the majority. However, the individual question responses and the self-described gender in this small cohort may suggest that there are differences in how some individuals with IGD experience their gender development. We speculate that this may be related to how they perceive the differences in physical development that they experienced related to their condition. Larger studies in participants with IGD and other disorders that alter sex hormone production/effate are necessary to further understand the relationship between decreased sex hormone exposure during critical developmental periods and gender identity development.

References: (1) Deogracias, J.J, et al. J. Sex Res., 2007, 44:4, 370–379 (2) Singh, D. et al. J. Sex Res. 2010, 47:1, 49–58

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**Neuroendocrinology and Pituitary**

**CASE REPORTS IN UNUSUAL PATHOLOGIES IN THE PITUITARY**

**Pituitary Hyperplasia Secondary to Uncontrolled Primary Hypothyroidism**

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SUN-281

Background: There are several recognized causes of hyperplasia of the pituitary gland. These may present as sellar masses and be misdiagnosed as pituitary adenomas. Pituitary hyperplasia can occur in the presence of long standing primary hypothyroidism due to the loss of negative feedback caused by decreased secretion of thyroxine (T4) and triiodothyronine (T3) by the thyroid gland, leading to excessive thyrotropin releasing hormone (TRH) production by the hypothalamus causing Thyrotroph pituitary hyperplasia

Clinical case: 51 y/o female with a past medical history that includes anxiety & depression, obesity, pre-diabetes and uncontrolled hypothyroidism due to Hashimoto’s. Presented to the Endocrinology clinic for evaluation of acromegaly. Investigations revealed normal ACTH, markedly elevated IGF-1 levels following a 75g oral glucose tolerance test. The remainder of the pituitary panel, including TSH, prolactin, FSH, LH were all normal. An MRI sella showed a completely normal pituitary and pineal gland with no evidence of brain metastasis. She thus met clinical and biochemical criteria for acromegaly but a pituitary source could not be identified. Given her history, a paraneoplastic process or exposure during critical developmental periods and gender was found to be congruent with SAAB for the majority. However, the individual question responses and the self-described gender in this small cohort may suggest that there are differences in how some individuals with IGD experience their gender development. We speculate that this may be related to how they perceive the differences in physical development that they experienced related to their condition. Larger studies in participants with IGD and other disorders that alter sex hormone production/effate are necessary to further understand the relationship between decreased sex hormone exposure during critical developmental periods and gender identity development.

**Tumor Biology**

**ENDOCRINE NEOPLASIA CASE REPORTS I**

**A Case of Acromegaly Secondary to Ectopic Growth Hormone-Releasing Hormone (GHRH) Secretion from a Bronchial Neuroendocrine Tumour**

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SUN-930

A Case of Acromegaly Secondary to Ectopic Growth Hormone-Releasing Hormone (GHRH) Secretion from a Bronchial Neuroendocrine Tumour

Background

Acromegaly due to ectopic GHRH secretion is a rare disorder and only accounts for less than 1% of all cases of acromegaly.

Case

We present the case of a 42-year old female with acromegaly from a bronchial neuroendocrine tumour (NET) secreting GHRH. The patient presented with gradual onset of dyspnea and wheezing. Subsequent imaging (chest X-ray, CT chest) revealed a large left-sided thoracic mass. Bronchoscopy was performed and a biopsy was taken from the mass. Initial histological examination reported small cell lung carcinoma (SCLC). Therefore, she underwent chemoradiotherapy. The patient was concurrently experiencing increased digital girth, weight gain, and enlarged tongue and was therefore referred to our endocrinology clinic for evaluation of acromegaly. Investigations revealed normal ACTH, markedly elevated IGF-1 (1031 μg/L, normal 63–215 μg/L), and non-suppressed GH levels following a 75g oral glucose tolerance test. The remainder of the pituitary panel including TSH, prolactin, FSH, LH were all normal. An MRI sella showed a completely normal pituitary and pineal gland with no evidence of brain metastasis. She thus met clinical and biochemical criteria for acromegaly but a pituitary source could not be identified. Given her history, a paraneoplastic process or ectopic GHRH-producing tumour was suspected. Further workup showed a significantly elevated circulating GHRH levels of 73 pg/mL (normal 5 – 18 pg/mL). Subsequently, a second pathologist with expertise in NETs reviewed the same biopsy specimen. The specimen was found to be a GHRH-producing well differentiated pulmonary NET with a Ki67 index of 1.5%, and thus not a SCLC. Furthermore,
a somatostatin receptor scintigraphy study was done which showed evidence of a somatostatin avid receptor rich lesion in the lung, corresponding to the known tumour. Thus, what initially appeared to be a SCLC was subsequently found to be a GHRH-secreting NET, illustrating the challenges of diagnosing these rare tumours. She was treated with intramuscular sandostatin 20mg q28days and then underwent a left pneumonectomy, resulting in biochemical resolution of her acromegaly (IGF-1 219 µg/L). Genetic testing for MEN1 was also completed as there is an association between ectopic GHRH NETs and MEN-1; however, genetic testing revealed no pathogenic variants and exon deletions or duplications suggestive of MEN-1.

Learning points
Diagnosis of acromegaly due to ectopic GHRH secretion requires high clinical suspicion by the treating clinicians (e.g., endocrinologist, oncologist) as well as review of histology by expert pathologists. Our case highlights that ectopic GHRH secretion is a rare but important cause of acromegaly, which should always be suspected when a clear pituitary cause is not identified.

Cardiovascular Endocrinology

HYPERTRIGLYCERIDEMIA: INFLAMMATION AND MUSCLE METABOLISM IN OBESITY AND WEIGHT LOSS I

Should Severe Hypertriglyceridemia Also Be Considered as a Contraindication for Use of Glucagon like Peptide 1 (GLP-1) Agonists?

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SAT-570

Background: Reported cases of acute pancreatitis have been associated with the use of GLP-1 agonists for treatment of diabetes mellitus. Hypertriglyceridemia is a well-established but underestimated cause of acute and recurrent pancreatitis. At the present time, there is insufficient data to know if there is a casual relationship.

Clinical Case: A 46 y.o. male with past medical history of coronary artery disease, hyperlipidemia, type 2 diabetes mellitus, hypertension, and morbid obesity, was admitted to the hospital with severe abdominal pain radiating to the back associated with non-bilious vomiting, for 1 day. Patient endorsed that 4 years ago he was diagnosed with hypertriglyceridemia. Physical exam findings were notable for a distended abdomen with mild epigastric tenderness, heart rate at 120 bpm, and a body mass index of 37 kg/m². Active medications included: atorvastatin 40 mg PO daily, fenofibrate 45 mg PO daily, metformin 1,000 mg PO twice a day, glipizide 5 mg PO daily, levetiracetam 60 units SQ twice a day, and most recently he had been started on dulaglutide 0.75 mg SQ weekly. Initial tests were consistent with acute pancreatitis and diabetic ketoacidosis: lipase 944 U/L (n 8.0 - 78 U/L), anion gap 18 mEq/L (n 5 - 13 mEq/L), creatinine 1.6 mg/dL (n 0.72 - 1.25 mg/dL), glucose 479 (n 60–100 mg/dL), β-Hydroxybutyrate 5.3 mmol/L (n <0.3mmol/L), uric acid >1,000 mg/dL (n Negative mg/dL), urine ketones 20 mg/dL (n Negative), Triglycerides (TG) 5,374 mg/dL (n <150 mg/dL) and Hgb A1C 11.9% (n <5.7%). CT abdomen and pelvis without contrast revealed moderate acute pancreatitis. Patient was admitted to the intensive care unit and was started on intravenous insulin, atorvastatin 80 mg PO daily and fenofibrate 145 mg PO daily. Despite optimization of lipid-lowering agents, TG remained above 2,000 mg/dL. Decision was made to start patient on plasmapheresis until TG was <500 mg/dL. Patient’s TG improved to 370 mg/dL after second treatment. Patient’s dulaglutide was discontinued and patient was advised to avoid GLP-1 agonist use, indefinitely. One-month post discharge patient’s TG level was 370 mg/dL. Conclusion: Pancreatitis should be considered in patients on GLP-1 agonists, that present with persistent severe abdominal pain (with or without nausea), and its use should be discontinued in such patients. Use of GLP-1 agonists should be avoided in subjects with severe hypertriglyceridemia. Further research should be made in order to determine if GLP-1 agonists should be contraindicated in patients with severe hypertriglyceridemia, as both increase risk for pancreatitis.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS I

Diagnosis of Autosomal Dominant Hypocalcemia Type 1 Following the Initiation of Imatinib for Treatment of Chronic Myeloid Leukemia

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SAT-347

Background: Autosomal Dominant Hypocalcemia Type 1 is an underdiagnosed condition due to the vast majority of patients being asymptomatic and having mild hypocalcemia. Imatinib has been associated with hypophosphatemia and hypocalcemia.

Clinical Case: 69-year-old man who had a long history of asymptomatic mild hypocalcemia, calcium level of 7.9 mg/dL (8.4 – 10.2 mg/dL) diagnosed with chronic myeloid leukemia and developed symptoms of hypocalcemia within 2 months of treatment with imatinib. After the initiation of imatinib, his calcium reduced to 6.8 mg/dL (8.4 – 10.2 mg/dL) with a corresponding ionized calcium of 0.98 mmol/L (1.12–1.32 mmol/L) within 2 months. He developed tetany. With the reduced calcium level his PTH was unexpectedly low-normal at 34 pg/mL (16–62 pg/mL). He also had a higher than expected urinary calcium of 342 mg/24 hours. The PTH and 24-hour urinary calcium levels raised concern for an underlying diagnosis of autosomal dominant hypocalcemia type 1. His phosphorous was normal at 3.3 mg/dL (2.6–4.9 mg/dL). He never had hypophosphatemia, which is common with imatinib. After two doses of IV calcium and initiation of oral calcium replacement, 1000 mg BID, his level continued to be reduced with symptoms. Given his symptoms, laboratory results, and continued hypocalcemia he underwent genetic testing. Results of his genetic testing showed a p.Thr151Met mutation in the CASR gene consistent with autosomal dominant hypocalcemia type 1. With this diagnosis, there is concern for nephrolithiasis with over treatment. His symptoms resolved with treatment with calcium 1000mg TID, calcitriol 0.25 mg BID and vitamin D3 2000 IU daily. He has not developed nephrolithiasis and his urinary calcium increased.