2020

Resident Clinical Vignettes/ Research Project Abstracts

Diabetes Canada/CSEM Virtual Professional Conference
October 28–30, 2020

Schedule

CSEM Resident Clinical Vignettes • Oral Presentations

Friday, October 30, 2020 • 2:00–3:45 p.m.

Oral presentations

2:00 p.m. Introduction — **Dr. Breay Paty, Chair**

2:05 p.m. Speaker: **Anna Liu**

A Rare Case of Prolactin-Secreting Pituitary Carcinoma with Epidural and Thecal Metastases

Discussion

2:15 p.m. Speaker: **Nisha Gupta**

Carney Complex: A Novel Pathogenic Variant in the PRKAR1A Gene

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2:25 p.m. Speaker: **Ameena Meerasa**

A de novo Mutation in the Melanocortin-4 Receptor (MC4R) Gene & A Lifelong Battle with Obesity

Discussion

2:35 p.m. Speaker: **Noelle Ma**

A Novel Mutation in P450 Oxidoreductase and its Role in Infertility

Discussion

2:45 p.m. Speaker: **Warren Brown**

Neurosarcoidosis Presenting with Polyuria: A Rare Cause of DI

Discussion

2:55 p.m. Speaker: **Ridha Ali**

Case Report of Undiagnosed Acromegaly Causing Dilated Cardiomyopathy Presenting with Electrical Storm and Heart Failure Requiring LVAD Implantation

Discussion

3:05 p.m. Speaker: **Cassandra Hawco**

KIT Mutation in a Gastric GIST in Patient with Familial Paraganglioma Syndrome Type IV

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3:15 p.m. Speaker: **Lurdes Tse-Agha**

A Case of Late Presentation of Central Diabetes Insipidus in a Patient with Immunotherapy-Related Hypophysitis

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A Rare Case of Prolactin-Secreting Pituitary Carcinoma with Epidural and Thecal Metastases

ANNA LIU*, STAN VAN UUM, DONALD LEE, ROBERT HAMMOND, KRISTIN CLEMENS

Western University

Background: Pituitary carcinomas are rare (only 0.2% of pituitary tumours) but important due to their association with high morbidity and mortality. In this case, we highlight the signs and symptoms that should prompt a higher index of suspicion for this condition and review the treatment of prolactin-secreting pituitary carcinomas.

Case: A 56-year-old man, with a history of well-controlled T2DM, presented with erectile dysfunction and binocular vertical diplopia. He was found to have central hypogonadism, secondary adrenal insufficiency and central hypothyroidism. His prolactin level was 1517 (reference range 4–15) mcg/L and his pituitary MRI showed a $2 \times 2.2 \times 3.1$ cm macroadenoma.

Results: Despite treatment with high dose cabergoline, two transsphenoidal resections and one course of radiation, his prolactin level continued to rise. Pathology showed a prolactin-secreting tumour with a Ki-67 proliferation index of 20–25% and methylguanine-DNA methyltransferase (MGMT) of $< 10\%$. Three years after diagnosis, he presented with lower extremity weakness and urinary incontinence caused by metastases to the epidural space and thecal sac extending from the thoracic to the sacral spine, and a new 5.5 mm nodule inferior to the cerebellar tonsil. He received eight cycles of temozolomide chemotherapy over eight months and has shown clinical and biochemical response.

Discussion: Features that may help distinguish a malignant prolactinoma from a benign prolactinoma include its presentation in males > 50 years old, lack of response to dopamine agonists, visual symptoms, anterior hypopituitarism at presentation, and a high Ki-67 proliferation index. Low MGMT on pathology might predict a good response to temozolomide.

Carney Complex: A Novel Pathogenic Variant in the PRKAR1A Gene

NISHA GUPTA*, THOMAS KITZLER, STEFFEN ALBRECHT, VINCENT LAROUCHE

McGill University

Background: Carney complex (CNC) is a rare autosomal dominant inherited disorder characterised by a constellation of skin pigmentary abnormalities, myxomas and endocrine tumours. In 70% of cases, a pathogenic variant in the PRKAR1A gene can be identified. There are established genotype-phenotype relationships for some variants, but usually, genotype does not consistently predict phenotype. Therefore, genetic testing should be conducted routinely to fully grasp the clinical significance of these novel pathogenic variants.

Case: A 39-year-old woman was referred to Medical Genetics because of a clinical diagnosis of CNC in her deceased brother. Although not fully suggestive of CNC, the proband had some of the characteristic features, such as periorbital lentigines. Previous extensive investigations revealed a mild IGF-1 elevation, a stable left adrenal gland adenoma, and a slightly enlarged pituitary gland. Diagnostic single-gene testing identified a novel heterozygous pathogenic frameshift variant in PRKAR1A, creating a premature translational stop signal expected to result in an absent or disrupted protein product (c.134_135dup, p.Pro46Aspfs*84; NM_002734.4). This variant has not been previously reported and is absent in large population databases (gnomAD). This result is consistent with a diagnosis of CNC and the recommended clinical surveillance measures were initiated for this patient.

Discussion: This is the first report of this mutation identified in French-Canadian siblings. These findings broaden the understanding of the genotypic and phenotypic spectrum of this rare disease. Furthermore, our report highlights the significance of a multidisciplinary approach, as genetic testing facilitated a timely diagnosis in our patient and enabled appropriate surveillance of CNC manifestations.

A *de novo* Mutation in the Melanocortin-4 Receptor (MC4R) Gene & A Lifelong Battle with Obesity

AMEENA MEERASA*, AMEL ARNAOUT, HEATHER LOCHNAN, ROBERT R. DENT

University of Ottawa

Severe early obesity can be caused by single gene mutations, the most common being melanocortin-4 receptor (MC4R) deficiency. We report a patient with a de novo MC4R gene mutation and severe obesity beginning in early childhood at age 1. At age 33, she weighed 122.0 kg and participated in a medically supervised meal-replacement program with a 20.5 kg weight loss. At age 36, she underwent Roux-en-Y gastric bypass surgery for weight regain to 134.7 kg. Post-surgery, her nadir weight at 6 months was 99.5 kg. Despite partaking in two community programs and another attempt at meal-replacement, she regained weight to 131.8 kg and was prescribed liraglutide. Through Sanger sequencing of 58 genes associated with weight regulation, our patient was found to have a nonsynonymous variant, Ala303Thr, in the MC4R gene. An *in-vitro* assay confirmed that this variant impaired receptor activity by $>$ 90% likely resulting in her severe phenotype. Remarkably this was a de novo mutation—absent in her parents/siblings, that was passed onto her son who also developed severe obesity despite early lifestyle interventions. This case illustrates that a negative family history does not rule out a genetic cause of obesity—onset in early childhood and resistance to multiple interventions may be sufficient clues as de novo mutations can occur. Recognizing monogenic obesity is important in supporting our patients with genetic counselling, early interventions, and emerging therapies. It has also allowed our patient to set achievable goals, reduce internalized weight bias, and counsel her son from an early age.

A Novel Mutation in P450 Oxidoreductase and its Role in Infertility

NOELLE MA*, AMEL ARNAOUT, JANINE MALCOLM

University of Ottawa

A 37-year-old woman was referred for high progesterone levels and thin endometrium while undergoing assessment for infertility. Her past medical history was significant for surgical removal of ovarian cysts and failed intrauterine insemination. She reported normal menarche, regular menstrual cycles and no clinical evidence of hyperandrogenism. Her BMI was 26.8. Her baseline 17-hydroxyprogesterone levels were persistently elevated at 20.5 nmol/L (luteal phase 2.9–15.2 nmol/L), 15.8 nmol/L (luteal phase 2.9–15.2 nmol/L), and 16.9 nmol/L (follicular phase 0.4–4.5 nmol/L). Her DHEAS and testosterone levels were within normal limits. Her cosyntropin stimulation testing demonstrated a morning cortisol level of 367 nmol/L with 17-hydroxyprogesterone level of 17.1 nmol/L (1.8 to 10.1 nmol/L) which peaked 90 minutes later to a cortisol level of 501 nmol/L and 17-hydroxyprogesterone level for 41.6 nmol/L. She was therefore referred to medical genetics for nonclassic congenital adrenal hyperplasia. Her genetic testing was negative for pathogenic variants in the CYP21A2 gene that causes the most common form of nonclassic congenital adrenal hyperplasia. Subsequently, she underwent broader genetic testing which demonstrated a novel homozygous mutation in P450 oxidoreductase (POR). POR acts a cofactor and supports the activity of multiple enzymes in the steroid biosynthetic pathway including 21-hydroxylase and 17α-hydroxylase/17,20-lyase. We are describing a new case of nonclassic congenital adrenal hyperplasia presenting with elevated 17-hydroxyprogesterone levels without clinical evidence of hyperandrogenism and infertility caused by a novel mutation in POR. Further management would include a trial of steroid treatment to see if there is an improvement in her endometrial lining thickness.

Neurosarcoidosis Presenting with Polyuria: A Rare Cause of DI

WARREN BROWN*, AMEL ARNAOUT, HEIDI DUTTON

University of Ottawa

A 63-year-old woman was referred to Endocrinology for a 1-year history of worsening polyuria, polydipsia and confusion. Initial investigations revealed: serum sodium 138 mmol/L (135–145 mmol/L), serum osmolality 285 mmol/kg (280–295 mmol/kg) and urine osmolality 78 mmol/kg. 24-hour urine volume was 6.1L with normal renal function. Supervised water deprivation test confirmed central diabetes insipidus (DI) with a final serum sodium of 145 mmol/L, serum osmolality of 310 mmol/kg and urine osmolality of 103 mmol/kg. She was started on DDAVP with immediate improvement of her urinary symptoms. Work up for causes of central DI showed an elevated prolactin of 84.6 ug/L (4.8–23.3 ug/L). All other pituitary hormones were normal. Infiltrative processes including IgG4 disease and hemochromatosis were ruled out. Pituitary and spine MRIs revealed pituitary stalk thickening and diffuse nodular leptomeningeal enhancement involving the supra and infratentorial regions extending down the entire spinal column. Lumbar puncture ruled out infectious, inflammatory and malignant causes. Cerebrospinal fluid ACE (Angiotensin Converting Enzyme) was 5 U/L, with no provided reference range. Serum ACE was 42 U/L (9–63 U/L). Lumbar spine leptomeningeal biopsy demonstrated granulomatous inflammation consistent with neurosarcoidosis. The patient's delirium resolved with steroids, though DI persists. This case identifies neurosarcoidosis as a rare cause of DI. We found that CSF and plasma ACE levels were unhelpful in making this diagnosis, highlighting the need for biopsy. Prior literature demonstrates that DI is typically permanent in these patients, though complete resolution has been described. Thus far, we have successfully reduced our patients' DDAVP dose with steroids.

Case Report of Undiagnosed Acromegaly Causing Dilated Cardiomyopathy Presenting with Electrical Storm and Heart Failure Requiring LVAD Implantation

RIDHA ALI*, RUDY UNNI, ROUPEN ODABASHIAN, LINDA WANG, CHRISTOPHER TRAN, HADI TOEG

University of Ottawa

Case Presentation: A 56-year-old male was transferred to our quaternary centre for ventricular tachycardia and electrical storm. Four years prior to presentation, he was diagnosed with non-ischemic dilated cardiomyopathy after developing heart failure during a respiratory tract infection. Upon presentation, the patient was in severe cardiogenic shock. Coronary angiography showed non-obstructive coronary artery disease and transthoracic echocardiography revealed a left ventricular ejection fraction of 20%. Despite multiple days of treatment, he remained inotrope dependent and was referred for left ventricular assist device (LVAD) placement.

Investigations: Prior to LVAD surgery, a CT head incidentally revealed prominence of the pituitary gland. Subsequent MRI confirmed a left lateral pituitary macroadenoma measuring 15×14×10 mm with mass effect to surrounding structures. An initial pituitary panel revealed an elevated IGF-1 of 339 ng/mL (reference range 62–214). Other pituitary hormones were within normal limits. A 75 g oral glucose load test showed a paradoxical rise in GH to a peak of 31.1 ng/mL while nadir GH was 28.4 ng/mL (normal response GH $\leq$ 1 mcg/L), consistent with acromegaly. Re-examination of the patient showed clear frontal bossing, prognathism and macroglossia. Pathology from cardiac biopsy ruled out amyloidosis, hemochromatosis and glycogen storage disease. Ultimately, the etiology of heart failure was thought to be untreated acromegaly.

Treatment & Discussion: The patient underwent successful LVAD implantation and is currently being treated with an octreotide analogue (sandostatin) while awaiting transsphenoidal pituitary resection. Acromegaly is a rare cause of cardiomyopathy that can go undiagnosed for several years and requires high clinical suspicion to establish diagnosis.

KIT Mutation in a Gastric GIST in Patient with Familial Paraganglioma Syndrome Type 4

CASSANDRA HAWCO*, ROBYN HOULDEN

Queen's University

Objective: Familial paraganglioma syndrome type 4 is associated with mutations in the succinate dehydrogenase complex, subunit B gene (SDHB). We report a case of a patient with familial paraganglioma syndrome type 4 with the mutation c.600G>T; p.Trp200Cys who developed a gastric gastrointestinal stromal tumour (GIST) with a KIT mutation.

Methods: Clinical, radiographic and genetic data are presented.

Results: A 40-year-old man with familial paraganglioma syndrome type 4 and recurrent paraganglioma presented with recurrent epigastric pain. He had undergone resection of a paraganglioma superior to the right adrenal gland at age 19 years, resection of two para-aoritc paraganglioma at age 39 years, and resection of a paraganglioma in the interatrial septum at age 40 years. CT scan showed a 3.2 by 3.8 cm gastric body intraluminal polypoid mass. A partial gastrectomy was performed and revealed a GIST with a KIT (CD117) mutation.

Conclusion: This case provides further evidence that mutations in SDHB and KIT are not mutually exclusive with GISTs. It also identifies the need for endoscopic evaluation for GIST in patients with familial paraganglioma syndrome type 4 with unexplained gastrointestinal symptoms.

A Case of Late Presentation of Central Diabetes Insipidus in a Patient with Immunotherapy-Related Hypophysitis

LURDES TSE-AGHA*, ANGELA F. ASSAL, MARTIN SMORAGIEWICZ

University of Toronto

Background: Hypophysitis is a common sequela of immune checkpoint inhibitors (ICI) like anti-PD-1/PD-L1 and anti-CTLA4 inhibitors. Posterior pituitary hypophysitis manifesting as central diabetes insipidus (cDI) has only been reported in a few case studies.

Case Presentation: A previously healthy 54-year-old man was diagnosed with metastatic renal cell carcinoma in November 2019. After 3 cycles of combined nivolumab (anti-PD-1) and ipilimumab (anti-CTLA4), he developed central hypothyroidism, adrenal insufficiency and hypogonadism, and required glucocorticoids and levothyroxine. Notably, he had marked ongoing response in his cancer. He was then switched to single-agent nivolumab. Four months later, he noticed polyuria and polydipsia. Serum osmolality was 314 mmol/Kg, with 24-hour urine osmolality of 127 mmol/Kg (a fasting urine sample was not obtained to determine urine concentrating ability). The 24-hour urine output was 4536 mL. Creatinine was elevated at 171 μmol/L with a bland urinalysis. Serum sodium was 147 mmoL. A brain MRI showed interval development of empty sella. The high urine volume, AKI, elevated serum osmolality, hypernatremia and MRI findings all suggested a diagnosis of cDI. He was treated with oral desmopressin.

Discussion: Central DI is a rarely reported immune-related adverse event. The cDI associated with anti-CTLA4 ICI in case studies occurred within 12 weeks of administration, while cDI associated with anti-PD-1/PD-L1 ICI occurred on the order of months. The late onset of cDI in this case suggests that nivolumab is more likely to be the causative agent.

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Thyroid Hormone-Based Nutraceuticals: A Rare Cause of Thyrotoxicosis and Thyrotoxic Periodic Paralysis

SHIRLEY SHUSTER*, SARA AWAD, CAITLYN VLASSCHAERT

Queen's University

Background/Objective: Thyrotoxic periodic paralysis (TPP) is a rare disorder characterized by muscle weakness, areflexia, and hypokalemia in the setting of thyrotoxicosis. This case aims to illustrate the use of nutraceuticals as a cause of thyrotoxicosis and TPP.

Methods: Clinical and laboratory data are presented.

Results: A 32-year-old Caucasian male presented to the emergency department with lower limb muscle weakness. Physical examination revealed tremors, diaphoresis, sinus tachycardia (110–120 bpm), and positive lid lag. Thyroid exam was normal and there were no bruits. TSH was $<$ 0.01 mIU/L (reference range (RR) 0.40–4.50), fT4 6 pmol/L (RR 9.0–19), and fT3 15 pmol/L (RR 4.0–6.8). Potassium was 3.1 mmol/L (RR 3.7–5.3) which normalized with supplementation. He was suspected to have Graves' disease (GD) and was discharged home on bisoprolol 2.5 mg and methimazole 10 mg, pending TRAb levels and outpatient follow-up. He re-presented four days later with generalized paralysis, areflexia, and sinus tachycardia (100 bpm). Investigations revealed potassium 1.7 mmol/L, TSH 0.07 mIU/L, fT4 6 pmol/L, and fT3 2.2 pmol/L. Treatment of TPP with intravenous fluids and potassium supplementation resulted in rebound hyperkalemia to 6.1 mmol/L which then normalized, along with resolution of paralysis. Methimazole was also discontinued. Thyroid radioiodine uptake and scan was normal at 23%, however the patient had been ingesting weight loss supplements containing kelp, iodine, licorice, and likely undeclared thyroid hormone or mimics. Following discontinuation of all supplements, bloodwork revealed TSH 3.07 mIU/L, fT4 10pmol/L, fT3 4.4 pmol/L, and potassium 4.2 mmol/L. Symptoms of thyrotoxicosis also resolved.

Conclusion: This case illustrates that ingestion of thyroid hormone-based nutraceuticals should be considered as a cause of thyrotoxicosis and TPP.

Long-Term Outcomes of High Dose Fat Soluble Vitamins in Disorders of Lipoprotein Metabolism

RYAN LE*, ROBERT HEGELE

Western University

Abetalipoproteinemia (ABL) is a rare autosomal recessive disorder affecting the MTTP (microsomal triglyceride transfer protein) gene. MTTP is crucial in the assembly and secretion of normal apo-B containing lipoproteins such as LDL, VLDL, and chylomicrons. ABL patients have almost undetectable levels of these lipoproteins, and suffer from lipid malabsorption. As a result, fat-soluble vitamin deficiencies develop, subsequent complications including neuropathy, myopathy, coagulopathy, atypical retinitis pigmentosa, and osteopenia. In addition, patients also present with pathognomonic peripheral acanthocytosis. Homozygous familial hypobetalipoproteinemia (FHBL) is an autosomal codominant condition that involves the APOB gene, and phenotypically is indistinguishable from ABL. We describe a case series of 3 patients (2 with ABL, 1 with FHBL); each diagnosed and started on high dose vitamin supplementation at a young age, and report on their clinical status. On presentation, each of these patients similarly had undetectable levels of apo-B lipoproteins, neuropathy and peripheral acanthocytosis. Each of these patients on high dose vitamin therapy are now in their 5th to 6th decade of life, well beyond the previously well-established lifespan of 20–30 years before vitamin supplementation, and with the main complication being peripheral neuropathy that has remained remarkably stable despite barely detectable vitamin E levels on high dose supplementation. As minimal data exists in the current literature on long-term outcomes, we review the literature on ABL and FHBL, summarize clinical manifestations, diagnosis, treatment, and report on the outcomes of these patients to further provide data on long-term management of this condition and further characterize disease trajectory.

Graves' Orbitopathy and Subclinical Hyperthyroidism Induced by External Beam Radiation for Nasopharyngeal Carcinoma

ALEXA CLARK*, MARSHALL DAHL

University of British Columbia

Objective: We report a case of Graves' orbitopathy that was diagnosed 2 years after external beam therapy for nasopharyngeal carcinoma. Mantle radiation for Hodgkin's lymphoma has previously been reported as a rare cause of Graves' orbitopathy, however this has seldom been reported from external beam radiotherapy for nasopharyngeal carcinoma. Our case report adds to the growing literature alerting clinicians to the association between radiation for nasopharyngeal cancer and development of Graves' orbitopathy.

Methods: A 51-year-old woman presented to her family physician with a 4-month history of periorbital swelling. Past medical history included uterine myomectomy, depression and external beam radiation for T1N1 nasopharyngeal squamous cell carcinoma 2 years prior. There was no personal or family history of autoimmune disease.

Results: A magnetic resonance imaging scan of the neck done for post radiotherapy nasopharyngeal cancer surveillance showed new extraocular muscle enlargement and proptosis suspicious for thyroid disease related orbitopathy. Laboratory investigations revealed elevated thyroid-stimulating hormone receptor antibodies (TSHR), elevated thyroid peroxidase antibodies (TPO), a low thyroid-stimulating hormone (TSH) level, and normal free thyroxine (FT4) and free triiodothyronine (FT3) levels. She was diagnosed with subclinical hyperthyroidism and Graves' orbitopathy, started on selenium therapy and monitored with regular thyroid function tests.

Conclusion: Graves' orbitopathy is a rare side effect of radiation therapy for nasopharyngeal carcinoma. Physical examination of the orbits and thyroid function tests should be monitored after the completion of radiotherapy in the head and neck region.

A Rare Case of Atypical Teratoid/Rhaboid Tumour With Germinoma Differentiation in a 59-Year-Old Woman

MELISSA-ROSINA PASQUA*, HUDA ALTOUKHI, VALERIE PANET-RAYMOND, DENIS SIRHAN, JASON KARAMCHANDANI, MARIE-CHRISTINE GUIOT, NATASHA GARFIELD

McGill University

Atypical teratoid/rhabdoid tumours (ATRT) are a rare class of central nervous system malignant tumours comprised of elements of ectoderm and mesoderm germ-cell layers, more commonly seen in pediatric patients. We present the case of a previously healthy 59-year-old woman who was found incidentally to have a pituitary mass on CT head, with retrospective symptoms of headaches, polyuria, polydipsia, diplopia, and low blood pressure. At presentation, she was found biochemically to have pan-hypopituitarism with a left cranial nerve six deficit, with an MRI depicting a 19.5×22×11 mm suprasellar mass extending into the infundibulum and hypothalamus, with displacement of the optic chiasm. She was started on pituitary hormone replacement therapy, and sent for urgent transsphenoidal resection. Preliminary reports were suggestive of germinoma given the diffuse presence of Oct 3/4 and C-kit; however upon further pathological analysis, her tumour demonstrated loss of INI-1 expression, which is diagnostic of ATRT. Given the mixed features on immunohistochemistry, the final diagnosis was concluded as an atypical teratoid/rhabdoid tumour of the sella turcica with germinoma differentiation. A multi-disciplinary approach consisted of initial radiotherapy, with chemotherapy targeted towards a germinoma-type tumour, and ongoing pituitary hormone replacement. Though her treatment resulted in a significant reduction in tumour size, she eventually passed away from leptomeningeal dissemination. This represents a unique case of a rare tumour with germinoma differentiation in an older patient that has not been previously reported, and the clinical course of her treatment that currently does not have established guidelines.

Long-Term Follow-up of One of the First Patients to Receive Human Growth Hormone Therapy

CASSANDRA HAWCO*, ROBYN HOULDEN

Queen's University

A 78-year-old man presented for endocrine followup. He had been one of the first patients to receive GH therapy in 1958. Growth had been normal until age 3 years and then decelerated. At age 17 years, he was 4'3" (129.5 cm) with absent sexual development. Bone age was 7 years. He was referred to Dr. Raben who initiated treatment with thyroid hormone and cortisone for 8 months. Human GH extract from the pituitary glands of deceased donors was then initiated at a dose of 2 mg 3 times a week for 2 years, and 3 mg 3 times a week for 6 months resulting in growth to a final height of 5'6½". Testosterone cyclopentylpropionate in oil 30 mg intramuscularly every 2 weeks was then added with achievement of sexual maturation over 2 years. He remained on testosterone injections until age 40 and then used transdermal testosterone until age 50 years. He received treatment with human chorionic gonadotropin and human menotropins for spermatogenesis restoration under the care of Dr. Raben at age 27 years with successful conception by his wife. At age 78 years, a MRI of the pituitary revealed a tiny amount of pituitary tissue within the floor of a normal-sized sella turcica with absent pituitary infundibulum. A combined pituitary hormone deficiency genetic panel did not reveal any mutations. These features suggest remote pituitary infarction/apoplexy rather than congenital pituitary deficiency. Sixty-two years later the patient remains in good health and is grateful to a pioneer in Endocrinology.

Rare Case of Androgen-Secreting Ovarian Luteoma and Adrenal Adenoma with Subclinical Cushing's Syndrome in a Post-Menopausal Woman with Virilization: A Diagnostic Challenge

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Background/objective: Post-menopausal virilization is caused by excess androgens from the adrenals or ovaries, and ovarian luteoma is a rare cause. Subclinical Cushing's syndrome (SCS) is a cause of metabolic syndrome and virilization, and is usually due to adrenal adenoma; however, rare cases of SCS due to ovarian etiology have been reported. This case aims to illustrate ovarian etiology as a rare cause of post-menopausal virilization and possibly SCS.

Methods: Clinical and laboratory data are presented.

Results: A 63-year-old female presented with a 3-year history of increasing dark, coarse hair growth around the face, trunk and extremities, androgenic alopecia, aggression, and voice deepening. Medical history was significant for obesity, type 2 diabetes and hypertension. Physical examination was significant for virilization, clitoromegaly and purple abdominal striae. Total testosterone was 10.7 nmol/L (RR 0.3–1.3), with bioavailable testosterone 5.74 nmol/L (RR 0.1-0.6), but normal DHEA-S 2.3 umol/L (RR 0.8–4.9). 17-(OH)-progesterone was normal. CT scan revealed a 2.1 cm left ovarian mass and 3.2 cm right adrenal adenoma. Renin, aldosterone and serum metanephrines were normal. However, cortisol failed to suppress to $<$ 50 nmol/L following 1 mg dexamethasone (cortisol 63 nmol/L), concerning for SCS. The patient underwent bilateral salpingo-oopherectomy and right adrenalectomy. Pathology revealed ovarian stromal luteoma and adrenocortical adenoma. One-month post-operatively, testosterone normalized to 0.7 nmol/L and cortisol appropriately suppressed to $<$ 28 nmol/L following dexamethasone suppression. The patient lost 20 pounds, and insulin requirements and anti-hypertensive medication doses decreased.

Conclusion: This case illustrates the importance of considering ovarian luteoma as a rare cause of post-menopausal virilization, and the rare possibility of SCS from an ovarian source.

COVID-19 Consequences: A Spike in Thyroid Storm Cases During Pandemic Lockdown

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COVID-19 has had dramatic impacts on the provision of healthcare. One model conceptualizes the destruction caused by the pandemic in waves. The first wave comprises direct consequences of SARS-CoV-2 infection. The second wave is due to the impact of the pandemic on urgent non-COVID conditions. We describe a spike in cases of thyroid storm during the pandemic lockdown between March to July 2020. A 71-year-old man had a few weeks of worsening dyspnea. His cardiologist's office was closed from the lockdown. His family hesitated to bring him to be assessed given recent international travel. He presented with uncontrolled new rapid atrial fibrillation and CHF exacerbation. He was admitted and with endocrine consultation was treated for thyroid storm. He has ongoing poor biochemical monitoring given outpatient lab restrictions. A 59-year-old man presented with dyspnea and edema. He had a month-long history of weight loss, tremors, palpitations and agitation. He had not seen a physician due to COVID-19 fears. He was admitted and treated for thyroid storm. Despite best possible treatment, he suffered a cardiac arrest and died during hospitalization. A 30-year-old woman four months post-partum had a few weeks of tremors, palpitations, anxiety and increased bowel frequency. She sought care several weeks late due to fear of exposure to SARS-CoV-2 infection. She was treated for thyroid storm during a short admission. Three cases of thyroid storm with delayed presentations during lockdown illustrate the conceptual second wave of destruction from the COVID-19 pandemic. The public needs continued reminders to seek timely care.

Thyrotoxic Periodic Paralysis Induced by Intravenous Methylprednisolone for Graves' Orbitopathy: A Case Report

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Université de Montréal

Background: Thyrotoxic periodic paralysis (TPP) is a rare complication of thyrotoxicosis that occurs primarily in Asian males. It is characterized by sudden muscle weakness resulting from an intracellular potassium shift. Potassium supplementation and non-selective beta-blockers have been shown to reverse the paralysis.

Case: We report the case of a 29-year-old Thai non-castrated transgender female with Graves' disease who presented with severe weakness in the extremities 24 hours after receiving 500 milligrams of intravenous methylprednisolone for severe orbitopathy. The patient had been off estrogen therapy for a year. Initial biochemical workup showed potassium levels of 1.9 mmol/L and marked thyrotoxicosis (FT4 37.2 pmol/L). The patient regained full strength after administration of intravenous potassium and increase of oral propranolol doses. One week later, she received a second methylprednisolone infusion under surveillance, as an inpatient. At that time, she had become biochemically euthyroid (FT4 16.3 pmol/L). Prophylactic oral potassium supplements were administered (60 milliequivalents) and propranolol was continued. Potassium levels were closely monitored for 24 hours and remained normal. There was no recurrence of muscle weakness.

Conclusion: Glucocorticoids seem to be a trigger for TPP, possibly by increasing the number of Na/K-ATPase pumps and stimulating their activity through insulin secretion. Insulin resistance and hyperinsulinemia have also been observed in patients with TPP, possibly contributing to the pathogenesis. As glucocorticoids are commonly used in patients with thyrotoxicosis, clinicians should be aware of this potentially serious complication and monitor its occurrence. Glucocorticoid-triggered TPP, like other forms, does not seem to recur once euthyroidism is restored.

Navigating Diagnostic Decisions in a Hypoglycemic Patient

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Background and Case: Algorithms for assessment of non-diabetic hypoglycemia are described but may be challenging to conduct in practice. We present a case of an 87-year-old non-diabetic woman admitted with Whipple's triad (serum glucose 1.9 mmol/L, decreased level of consciousness, and improvement in level of consciousness with administration of D50). Adrenal insufficiency, sepsis, and hepatic/renal dysfunction were swiftly excluded.

Investigations: We documented two episodes of objective hypoglycemia where venous blood glucoses were 1.9 mmol/L and 2.9 mmol/L, and associated insulin and C-peptide levels were 14 pmol/L and 652 nmol/L, and 42 pmol/L and 1228 nmol/L respectively. Ketones were negative. Triphasic pancreatic CT excluded insulinoma.

Discussion: Interpretation of these results was challenging; the first set of insulin and C-peptide levels were near the lower limit of normal, whereas the second set was clearly high and consistent with endogenous hyperinsulinemia. However, the first insulin and C-peptide levels were added to a blood sample 48 hours after it had been drawn, raising questions about sample stability and therefore interpretability. Given our patient's frailty, we did not perform selective arterial calcium stimulation and a presumptive diagnosis of noninsulinoma pancreatogenous hypoglycemia syndrome (NIPHS) was made. Treatment was initiated with octreotide due to concerns with fluid retention from diazoxide. NIPHS is rare. While suggested guidelines exist for work-up and management, interpreting results is dependent on external factors such as timing of endocrinology involvement and personnel available to draw timely bloodwork; clinical decisions must also be made with a holistic view of the patient and their circumstances.

Cinacalcet for the Treatment of Humoral Hypercalcemia of Malignancy

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Humoral hypercalcemia of malignancy (HHM) is the most common cause of hypercalcemia of malignancy (HCM) and results from ectopic parathyroid hormone-related peptide (PTHrP) production. HHM is frequently refractory to standard therapy with bisphosphonates and recent case reports have described success with cinacalcet in bisphosphonate-refractory HHM. We report a case of a 57-year-old female with ovarian clear cell carcinoma and bisphosphonate-refractory HHM in whom the addition of cinacalcet was associated with a sustained reduction in serum calcium. Our case provides further support for cinacalcet as a potential therapy in bisphosphonate-refractory HHM and we discuss a variety of proposed mechanisms that may explain the efficacy of cinacalcet in this setting.

Perioperative Cardiogenic Shock Evokes Rare Diagnosis of TSH-Secreting Pituitary Adenoma

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Case: A 56-year-old male with autism spectrum disorder, type 2 diabetes, obstructive sleep apnea, hypertension, and obesity presented with a 5-year history of behavioural aggression. MRI demonstrated an invasive 28×40×27 mm pituitary mass. Initial biochemistry revealed normal IGF-1, ACTH, LH, FSH, and total testosterone. AM cortisol was 254 nmol/L, prolactin 7 ug/L, TSH 1.6 (0.34–5.6 mIU/L), and FT4 29.8 (7–17 pmol/L). Transsphenoidal surgery attempt was aborted due to cardiogenic shock following anaesthetic induction. Post-intubation, TSH was 1.94, FT4 32.2 and FT3 5.2 (3.3–6.0 pmol/L). Elevated alpha subunit of 3.8 (< 0.5 ng/mL) and high alpha subunit to TSH ratio of 3.22 confirmed a diagnosis of TSH-oma. Left ventricular ejection fraction (LVEF) was 18%. Methimazole acutely decreased FT4 prior to transition to cabergoline, which normalized thyroid function within 2 weeks. Ten months after the initial attempt, transsphenoidal resection of the pituitary mass was successful. Immunohistochemistry showed the tumour stained positive for thyrotropin and prolactin. Cabergoline was stopped postoperatively with normal thyroid indices. There was cardiac recovery with LVEF rising to 48%.

Discussion: Perioperative TSH-oma management may include thionamides, beta-blockers, dopamine agonists and/or somatostatin analogues, with somatostatin analogues being first-line. While effective initially, thionamides may result in reflexive TSH-oma growth due to loss of regulatory feedback. Cabergoline was chosen over somatostatin analogues for financial and logistical reasons. Immunohistochemical staining for prolactin is a marker for higher dopamine D2 receptor expression, which cabergoline would target and likely contributed to the effectiveness of cabergoline as monotherapy.

Tumor-Induced Osteomalacia: Overlooked and Underreported

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We describe a 47-year-old man who sustained multiple rib fractures and non-displaced left hip fracture, requiring total hip replacement due to poor healing, after slipping on ice. He failed to progress with rehabilitation, had diffuse bony pain and proximal muscle weakness. Multiple pelvic fractures and right hip stress fracture were identified on repeat imaging. He was previously healthy, with no prior fragility fractures or family history of bone disease. Bone density Z-score was −3.1; bone scan was consistent with osteomalacia; labwork showed low serum phosphate (0.63 mmol/L), increased urinary phosphate, undetectable calcitriol, and elevated ALP. Serum calcium and PTH were normal; 25-OH vitamin D was not deficient. The suspicion for tumor-induced osteomalacia (TIO) to explain renal phosphate wasting was further supported by elevated fibroblast growth factor-23 (FGF-23) > 800 pg/mL. Octreotide scan revealed increased avidity deep to the left gluteal minimus muscle; characterized as a 3.3 cm non-infiltrative mass on MRI. CT-guided biopsy confirmed a phosphaturic mesenchymal tumor (PMT). He underwent surgical resection of the tumor with normalization of serum phosphate and subsequent complete clinical and biochemical recovery. TIO is a rare acquired paraneoplastic syndrome; inappropriately elevated FGF-23 secretion from occult PMT leads to renal phosphate wasting, hypophosphatemia, low calcitriol, and debilitating fractures with poor healing. Our case highlights the importance of measuring serum phosphate in individuals presenting with unusual fractures, bone pain or proximal myopathy. The occult nature of TIO often delays its recognition. Although PMT are notoriously difficult to localize, with surgical resection, prognosis is excellent with complete resolution.

Massive Reticulocytosis: A Rare Cause of Artifactual Hypoglycemia

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Introduction: True hypoglycemia consists of a low serum glucose concentration in the presence of Whipple's triad. Artifactual hypoglycemia occurs when the measured serum glucose concentration is low due to lab assay factors with absence of hypoglycemic symptoms.

Case: A 53-year-old woman with a history of lupus, multiple sclerosis, sickle cell anemia, and previously normal glucose values, presented with a puzzling set of low serum glucose readings (range: 2.6–3.4 mmol/L) over the preceding year. Whipple's triad was absent. There was no history of co-morbidities or medications associated with hypoglycemia. Capillary glucose ranged 3.9–5.1 mmol/L over a 2-week period. A blood gas analyzer displayed a fasting glucose of 4.8 mmol/L and serum glucose from a fasting sample collected in a sodium fluoride tube (inhibits glycolysis) indicated a glucose of 4.6 mmol/L. She had considerable reticulocytosis (up to 594.6x109/L, normal: 22–92x109/L) due to sickle cell disease and chronic hemolysis. We concluded that the serum glucose concentrations were artificially low due to in vitro consumption by the high number of reticulocytes.

Discussion: In vitro consumption of glucose in a blood collection tube has been demonstrated rarely in other clinical settings, such as massive leukocytosis with leukemia, erythrocytosis in polycythemia vera, and in chronic hemolytic anemia. Here we document this phenomenon due to sickle cell disease and massive reticulocytosis.

Conclusion: This artifact should be considered in similar patients before extensive work-up for hypoglycemia is undertaken. Rapid processing of the sample or the use of sodium fluoride in the collection tube can address this issue.

Isolated Central Adrenal Insufficiency Induced by Durvalumab in a Patient with Metastatic Oropharyngeal Cancer: A Case Report

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Background: Immunotherapy for cancer treatment is associated with endocrine-related toxicities. Hypophysitis, characterized by the inflammation of the pituitary gland, is a common and potentially life-threatening toxicity mainly described with anti-CTLA4 therapy. New reports have been published on the adverse effects of anti-PD1 therapy on pituitary corticotroph function.

Case Presentation: We report the case of a 78-year-old man with metastatic oropharyngeal cancer treated with durvalumab (anti-PD1) every four weeks. After his 7th cycle of immunotherapy, the patient presented with general malaise and fatigue. At that time, biochemical data showed morning ACTH level increased at 27.6 pmol/L (N: 2.0–11.0 pmol/L) with an upper limit of normal cortisol level at 590 nmol/L (N: 185–624 nmol/L) and normal thyroid function. Follow-up laboratory workup was scheduled before his 9th cycle of treatment and showed biochemical signs of adrenal insufficiency (ACTH 2.6 pmol/L; cortisol 27 nmol/L) with an otherwise normal anterior pituitary axis function. His pituitary gland was described as normal on MRI. Supplementation with hydrocortisone (20 mg AM plus 10 mg PM) was initiated, significantly improving the patient's symptoms.

Conclusion: Anti-PD1-induced hypophysitis is a rare clinical entity, mainly resulting in isolated adrenocorticotrophic insufficiency. In the present case, the initial ACTH elevation could reflect the release of ACTH from the destruction of corticotrophs. This finding may be important for early detection of Immunotherapy-induced hypophysitis and subsequent onset of adrenal insufficiency. With the advent of immunotherapy, clinicians should, therefore, be familiar with this clinical entity to ensure prompt recognition and management of such potentially life-threatening toxicity.

An Eight Years Exposure of Chronic Hypoxemia in Infancy and Diagnosis of Paraganglioma in Adulthood: A Rare Case in the Setting of Cyanotic Congenital Heart Disease and Cohort Analysis

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Context: Pheochromocytomas and paragangliomas (PGLs) (PPGLs) are rare neuroendocrine tumors arising from the adrenal medulla or along the sympathetic/parasympathetic chains. Hypoxemia is a risk factor for PPGLs development that are more frequent in individuals living in high altitude. Moreover, hereditary PPLGs may be caused by mutations in the SDHx, VHL or FH genes that are linked to the pathway that activate hypoxia-inducible factors.

Case: A 44-year-old woman presented for abdominal pain. She was known for previous chronic hypoxemia in childhood secondary to congenital tricuspid atresia. At 8 years old she underwent a FONTAN operation conducting to normoxemia. A CT scan showed a 32×25×37 mm mass in the left pararenal space with T2-hyperintense at MRI and increased uptake at 18F-FDG PET/CT (SUVmax:4.5). Plasma normetanephrine were 9.73 nmol/L (N < 1.09) and methoxytyramine were 6.67 nmol/L (N < 0.16). She underwent laparoscopic resection of the mass and PGL was confirmed at pathology. A 14 multigene panel for PPGLs susceptibility genes was performed and no mutations were found. PPGL cohort analysis for CCHD: We revised the files of 350 patients with PPGLs evaluated at CHUM from 1990–2020 and no other patients had past or current medical history of CCHD. A literature review revealed that only 5 other cases of tricuspid atresia with concomitant abdominal PGLs were reported so far.

Conclusion: PPGLs may be linked to patients with CCHD. Our case underlines that less than 10 years of hypoxemia in childhood may likely confer a risk factor to develop PPGLs in adulthood.

Tumour-Induced Osteomalacia—A Rare Cause of Fractures

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Case: A 72-year-old woman presented with bilateral calcaneal stress fractures, left femoral neck stress fracture, worsening gait and leg pains. She had a known S2-S5 biopsy-proven calcifying pseudoneoplasm of the neuroaxis with no evidence of malignancy. Initial investigations revealed ALP 464 U/L (35–120), calcium 2.37 mmol/L (2.15–2.60), albumin 47 g/L (35–52), PTH 5.8 pmol/L (1.6–6.9), creatinine 88 umol/L, eGFR 61, TSH 1.25 mIU/L (0.32–4.0), 25-hydroxyvitamin D 89 nmol/L (75–120). Hepatic causes of elevated ALP were ruled out. Bone mineral density showed spine T-score −0.8, femoral neck T-score −1.8, total hip T-score −1.6. Whole body bone scan showed focal MDP activity in multiple ribs and vertebrae. Skeletal survey showed no evidence of Paget's disease. Given persistent ALP elevation and fracture history, she received zoledronic acid 5 mg IV. The patient subsequently developed urinary incontinence and constipation. MRI spine revealed increased size of the known sacral mass. Surgery revealed a 5×4×1 cm phosphaturic mesenchymal tumour, with incomplete resection.

Discussion: Tumour-induced osteomalacia (TIO) is a rare paraneoplastic syndrome caused by tumour overproduction of fibroblast growth factor 23 (FGF23), resulting in renal phosphate wasting. It is most commonly found in phosphaturic mesenchymal tumours (PMTs). Manifestations include muscle pain, weakness, and fragility fractures, low phosphate, low/low-normal 1,25-(OH)2D, elevated ALP, and normal calcium and PTH. Definitive management is surgery +/- radiotherapy. Medical management for residual tumours involve phosphate and calcitriol supplementation.

Conclusion: Serum phosphate is not a routinely recommended biochemical test for initial osteoporosis assessment. However, elevated ALP should prompt investigation for high bone turnover, including differential causes of osteomalacia.

Horses Not Zebras: Severe Preeclampsia Mistaken for Pheochromocytoma/Paraganglioma Recurrence in a Pregnant Patient with Von Hippel-Lindau Disease

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Background: Von Hippel-Lindau (VHL) Disease is a heritable multisystem tumor syndrome that predisposes patients to hemangioblastomas, pheochromocytomas/paragangliomas (PPGLs), and tumors of the inner ear, pancreas, kidneys, and gonads. It presents unique considerations in pregnancy.

Case: A 29-year-old nulliparous female with VHL, and PPGL resected 10 years prior, presented at 24+5 weeks gestation with palpitations, peripheral and pulmonary edema, hypoxemic respiratory failure, severe hypertension (BP 230/120), and an AKI with mild/intermittent proteinuria. Blood pressure improved significantly with nitroprusside and phentolamine. The presentation was initially not felt to be in keeping with preeclampsia; therefore, alternative diagnoses of PPGL (despite normal plasma normetanephrines 8 weeks prior), cardiomyopathy or pulmonary embolism (PE) were favoured. Left-sided heart failure and PE were ruled out. Repeat plasma normetanephrines were only mildly elevated at 0.66 nmol/l (< 0.59). Phentolamine was discontinued without significant deterioration. Additional endocrine considerations including ectopic ACTH-production or renin-production from a known pancreatic NET and renal mass, respectively, were excluded. On admission day 3, treatment for severe preeclampsia with MgSO4 infusion was initiated, and C-section was performed on admission day 4. Clinical condition improved significantly post-delivery.

Discussion: This case illustrates that common conditions should not be overlooked due to the presence of an underlying genetic syndrome. Focusing only on syndromic pathologies can lead to delays in diagnosis and treatment. Sporadic PPGL is unlikely to develop in 8 weeks, but the effects of VHL and pregnancy on PPGL growth velocity remain largely unknown. Further research is needed to develop evidence-based recommendations regarding VHL in pregnancy.

A Diagnostic Approach to a Rare ACTH-Producing Neuroendocrine Tumor Causing Cushing's Syndrome

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A 57-year-old woman presented with a three-month history of worsening lethargy, weight gain, central adiposity, bruising, proximal muscle weakness, and hypokalemic hypertension. Her initial work-up confirmed ACTH dependent Cushing's syndrome. The MRI sella was normal. Petrosal vein sampling indicated ectopic ACTH production. She required several hospital admissions for worsening lower extremity weakness and falls. Ketoconazole, spironolactone, and insulin were initiated. CT scan of chest/abdomen/pelvis showed no intrathoracic or intra-abdominal abnormality. Thyroid ultrasound and whole-body octreotide scan showed no abnormalities. A Gallium 68-dotatate PET scan showed focal tracer uptake in the pancreatic tail suspicious for a well-differentiated neuroendocrine tumor (NET). MRI pancreas showed a slightly truncated pancreatic tail with no discrete lesion. We proceeded with Endoscopic Ultrasound which revealed an irregular hypoechoic mass in the pancreatic tail. Endoscopic ultrasound-guided fine-needle biopsy (EUS-FNB) using a 22G SharkCore needle, revealed a neuroendocrine neoplasm staining positively for ACTH. She underwent distal pancreatectomy with pathology showing a 1.4 cm well-differentiated NET, staining positive for ACTH with a Ki67 index of 10%. Postoperatively, her ketoconazole, spironolactone, and insulin were discontinued. Her dysglycemia, hypertension and other clinical symptoms gradually improved. Immediately post-operatively, her AM cortisol was 403 and her 24-hour urine cortisol was elevated 959 nmol/day. One month post-surgery her Cushing's syndrome was in remission.

Discussion: Cushing's syndrome from ectopic ACTH production is associated with significant morbidity and mortality. The localization of ACTH-producing tumors can be difficult and require multiple investigations and multidisciplinary team involvement. This case discusses a systematic approach for diagnosis and management of an ACTH-producing pancreatic NET.

Thyroid Hormone Resistance with Concomitant Iatrogenic Hypothyroidism: How Should Levothyroxine be Titrated?

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Introduction: Resistance to thyroid hormone (RTH) is a genetic syndrome of impaired sensitivity to thyroid hormones. Most cases are caused by mutations in the thyroid hormone receptor β (TRβ) gene, characterized by elevated thyroid hormones with non-suppressed TSH. The thyroid hormone excess partially compensates for resistance in peripheral tissues that express TRβ, whereas tissues that express TRα (e.g. heart) exhibit preserved sensitivity to thyroid hormones. Therefore, the clinical presentation is variable.

Case: We describe a 63-year-old woman who underwent radioiodine ablation in the 1980s for apparent "hyperthyroidism" (records unavailable), and was subsequently replaced with high doses of levothyroxine (1.75–2.4 mcg/kg/day) for iatrogenic hypothyroidism. Despite high-normal thyroid hormone levels, her TSH remained elevated anywhere between 31.68–86.7 mIU/L. An MRI sella was normal. Her picture was suspicious for RTH, and genetic testing confirmed a TRβ mutation: c.938T > C (p.Met313Thr).

Conclusion: Managing concomitant hypothyroidism in RTH can be challenging. Levothyroxine can be titrated to target the TSH back to baseline level (prior to onset of hypothyroidism), but in this case, the patient's baseline TSH was unknown. Biochemical markers of peripheral thyroid hormone activity (e.g. SHBG, ferritin) can be monitored, but these are nonspecific. Differing tissue-specific sensitivities to thyroid hormones can further complicate management, sometimes requiring β blockade to control tachycardia. Ultimately, this highlights the importance of careful clinical assessments in managing concomitant hypothyroidism in RTH, especially given her age and the risk of excess thyroid hormone action on the heart.

Diagnostic Challenges in the Diagnosis of Sex Cord Stromal Tumours: A Learning Experience

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Case: A 39-year-old woman with obesity (BMI 43) and diagnosed PCOS described oligo/amenorrhea since her late teenage years with severe hirsutism requiring daily shaving of face and chest. She had been treated with the oral contraceptive pill. Current investigations revealed total testosterone 7.4 nmol/L (N < 1.8), free testosterone 177 nmol/L (N < 30) with normal dehydroepiandrosterone, androstenedione, sex-hormone binding globulin, estradiol, and 250 mcg ACTH-stimulated 17-hydroxyprogesterone. Follicle stimulating hormone was 3.2 IU/L and luteinizing hormone 4.1 IU/L. She had no response to progesterone withdrawal. Her updated pelvic ultrasound showed a 5.1 cm simple cyst on the right ovary. Because the clinical picture did not fit with PCOS, an MRI of the pelvis was pursued and revealed an oval-shaped enhancing solid mass in the right ovary. The patient underwent a salpingo-oophorectomy, removing a 5 cm tumour pathologically classified as a sex cord stromal tumour with strong and diffuse positive staining for inhibin, calretinin and negative FOXL2 gene mutation. Her testosterone levels completely normalized post-operatively.

Conclusion: Ovarian sex-cord-stromal tumors are infrequent tumors with strong associations to hormone-mediated syndromes. The radiologic appearances of these tumors vary along with their morphologies. This case highlights the incorrect initial diagnosis of PCOS in a 39-year-old woman with amenorrhea, elevated testosterone, and a mass in the right ovary which was initially described as cystic on ultrasound. Imaging with MRI has increased sensitivity and more accurately described the mass leading to oophorectomy.

Disappearing Hemoglobin A1c in a Patient with a Rare Benign Variant

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A 64-year-old man with a BMI of 25.1 was referred to Endocrinology with a surprisingly low hemoglobin A1c (A1c). He used topical corticosteroids and ramipril to treat psoriasis and hypertension. He had no symptoms of hypoglycemia, a normal dietary pattern and physical exam. Investigations included a random glucose of 5.7 mmol/L, normal insulin, C-peptide, hemoglobin electrophoresis, liver enzymes and imaging. A1c values trended down from 5.7% in 2011 to $<$ 3.5% in 2018. The last A1c in 2018 identified an abnormal hemoglobin peak, prompting genetic testing. Sequencing identified a rare heterozygous variant in HBB (c.155C $>$ A, p.Pro51His). This benign missense mutation previously dubbed Hemoglobin North Manchester, was reported using the amino acid sequence, in one previous study of two blood-relatives in the UK. Notably, both this variant, as well as another benign variant at the same site, have only been identified in heterozygotes. While the underlying mechanism of the gradual A1c decline in our patient has yet to be determined, the lack of a homozygotic mutation at this site suggests the wildtype sequence is functionally conserved and that a homozygous variant may be pathogenic. This case demonstrates that, despite a variant being termed “benign”, whether genetic or a hemoglobin variant, terminology does not preclude clinical impact. Although the hemoglobin variant was not disease-causing, it did cause alarming HbA1c levels. In turn, this led to subsequent investigations, which, themselves, are not entirely benign. Thus, clinicians should consider hemoglobin variants when A1c is too high or low, or discordant with the clinical picture.

A Rare Case of Adrenocortical Carcinoma Presenting in Pregnancy

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Background: Cushing's syndrome (CS) and Adrenocortical Carcinoma (ACC) are exceedingly rare causes of hypertension in pregnancy associated with significant adverse outcomes. Here we present a case of Cushing's syndrome from ACC diagnosed in pregnancy.

Case: A 27-year-old woman G1P0 presented at 32 weeks gestation with symptomatic hypertension. She had been diagnosed with diabetes and hypertension in late second trimester. Medical history was significant for depression and remote 2.3 cm right adrenal mass (functional workup not done). Examination revealed facial plethora, proximal weakness, and thick violaceous abdominal striae. Potassium was 2.9 mmol/L and ultrasound demonstrated a 6.6 cm right adrenal mass. Cushing's syndrome was diagnosed based on markedly elevated 24-hour urine cortisol of 2408 nmol/L and non-suppressed cortisol of 910 nmol/L after 1mg dexamethasone. ACTH was undetectable. Aldosterone-renin ratio, DHEAS, and metanephrines were normal. She underwent induction at 33 weeks for HELLP syndrome. Postpartum hypercortisolism testing was abnormal. CT demonstrated a 6.8 cm right adrenal mass (22 HU, relative washout 22.9%, absolute washout 42.3%, no metastasis). Pathology after right adrenalectomy revealed high-grade ACC.

Discussion: Fewer than 250 cases of CS and 20 cases of ACC in pregnancy have been reported. CS in pregnancy is difficult to diagnose and associated with significant morbidity and mortality. ACC is an aggressive malignancy with a median 5-year survival of less than 40%. Survival from ACC is lower in women diagnosed peri-partum compared to non-pregnant adults. Hormonal changes of pregnancy may contribute to proliferation of some adrenal masses.

Successful Off-Label Treatment of a Rare Case of Chromosome 6q24-related Neonatal Diabetes Mellitus

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Introduction: Methylation defect on chromosome 6q24 is the most common cause of transient neonatal diabetes mellitus (TNDM), affecting 1 in 200,000–400,000 births. Traditionally, patients are treated with insulin during the neonatal period until remission. We report a case of uniparental disomy at chromosome 6q24 successfully treated with off-label use of combined subcutaneous insulin infusion pump (CSII), continuous glucose monitoring (CGM), and sulfonylurea therapy.

Case: A one-day-old female, born at 41 weeks gestational age, was seen for persistent hyperglycemia. The infant was conceived through intracytoplasmic sperm injection with a sperm donor. Pregnancy was uncomplicated and she was delivered via caesarean section. Patient was small for gestational age (birth weight and length both 3rd percentile). No congenital anomalies were noted. Insulin infusion was initiated and transitioned to a CSII/CGM. Genetic testing revealed uniparental disomy at chromosome 6q24. Microarray testing determined a 1.1 Mb duplication encompassing PLAGL1 and HYAI genes (arr[GRCh37] 6q24.2(143785485_144895709)x3). At day 16, glyburide was initiated and titrated to 0.5 mg/kg PO BID, with a significant reduction in basal insulin rate. At day 28, insulin was discontinued and glyburide dose reduced. At day 33, glyburide was discontinued altogether. The patient has remained in remission and achieved appropriate growth and development.

Discussion: Overexpression of PLAGL1 may result in TNDM through decreased insulin beta cell mass, and/or through suppression of glucose-induced insulin secretion. In this case, off-label use of new technologies (CSII and CGM) and sulfonylurea were safe and effective therapies. Furthermore, use of sulfonylurea may have shorten the length of the TNDM.

Utility of Urinary Iodine Measurement for Optimizing Timing of RAI in a Challenging Case of Graves' Disease

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Case: We present a previously healthy 28-year-old female with thyrotoxicosis secondary to Graves' disease. Initial assessment revealed TSH of < 0.01 mIU/L(0.2–4.0 mIU/L), free T4 69.9 pmol/L(10–25 pmol/L) and free T3 26.2 pmol/L(3.5–6.5 pmol/L). On exam she had mild inactive Graves' ophthalmopathy. She was started on methimazole therapy at 10mg daily with a plan for close monitoring. Two months after treatment initiation she presented to the emergency department with fever and perianal pain and was found to have pancytopenia with neutrophils 0.1x10E9(2-9 x10E9), platelets 109x10E9(150–400x10E9) and hemoglobin 74 g/L(120–160 g/L). Methimazole was immediately discontinued and a bone marrow biopsy demonstrated features of aplastic anemia. Contrast enhanced CT scan was performed following methimazole discontinuation. Thyroid function tests two weeks later showed free T4 of > 100 pmol/L and free T3 23.1 pmol/L.

Discussion: Aplastic anemia is a rarely reported complication of methimazole therapy. Our case raises unique challenges in that the patient's neutropenia and severe thyrotoxicosis prohibited surgical management, and her recent exposure to iodinated contrast would render immediate administration of radioactive iodine ineffective. Although not formally validated for this indication, we were able to extrapolate from thyroid cancer literature the utility of spot urine iodine measurement to determine optimal timing of radioactive iodine administration. The patient was started on a low iodine diet, and spot urinary iodine levels were 3.13 umol/L at 2 weeks and 0.33 umol/L at 4 weeks post CT. Following review of her 4-week urinary iodine level, 12 mCi of radioactive iodine was administered. Post-treatment thyroid function tests are improving and the patient is clinically stable.

Malignant Insulinoma: Octreotide Therapy in Diazoxide-Refractory Hypoglycemia

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Background: Insulinomas are rare insulin-secreting neuroendocrine tumours derived from pancreatic beta cells. Insulinomas do not typically metastasize. Surgical resection of benign tumours confers a favourable prognosis, but malignant disease typically necessitates medical therapy.

Case: Our patient is a 67-year-old male, with no history of diabetes or hypoglycemia, presenting to hospital with a spontaneous seizure. He was found to have severe hypoglycemia with serum glucose of 1.6 mmol/L. Further work-up during ongoing hypoglycemia revealed elevated serum insulin of 187 pmol/L and C-peptide of 8.18 nmol/L, with low beta-hydroxybutyrate of 0.2 mmol/L in keeping with endogenous hyperinsulinemia. An indium-111 pentetreotide SPECT-CT scan demonstrated a large octreotide-avid 6 cm pancreatic mass and a small hepatic mass concerning for metastatic insulinoma. Surgical resection was not an option due to significant comorbidities, including CHF and pulmonary hypertension. Euglycemia was maintained on 50% dextrose infusion, while Diazoxide was initiated. He had refractory hypoglycemia despite Diazoxide 300 mg/day. Subcutaneous Octreotide was initiated with immediate resolution of hypoglycemia, allowing for discontinuation of Diazoxide and dextrose. He reported no further episode of hypoglycemia upon discharge home and glucose was maintained at 5 to 7 mmol/L on Octreotide 6 mcg BID.

Discussion: Octreotide was extremely effective in the management of Diazoxide-refractory hypoglycemia in our patient. Conventional dosing of Octreotide in insulinoma is much higher at 100 mcg/day or more. Insulinomas are typically small at presentation (< 2 cm). The presentation in our patient is unique because his large tumour size, renal disease, and hepatic congestion allowed for minimal octreotide dosing.

A Rare Case of Ectopic ACTH Syndrome Arising One Year after the Diagnosis of Non-Functioning Pancreatic Neuroendocrine Tumor

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Case Report: A 69-year-old man was referred for suspicion of a pancreatic neuroendocrine tumor (pNET) seen incidentally on a thoracic CT-scan. MRCP demonstrated a 2.6×3.2×2.7 cm mass in the junction of the body and tail of the pancreas. FNA was performed and confirmed the diagnosis of grade 1 well-differentiated pNET. Almost one year later, he developed rapidly proximal weakness, fatigue, severe hypokalemia, refractory hypertension and new-onset diabetes. Morning basal serum cortisol was 1128 nmol/L with an ACTH 45.1 pmol/L. Late-night salivary cortisol was markedly increased at 227.7 nmol/L ($N < 7$) and 24-hour urinary free cortisol (UFC) was elevated more than 100 times the ULN at 13 471 and 13 374 nmol/d ($N < 120$). 4-mg intravenous dexamethasone suppression test confirmed nonsuppressed cortisol level, with a nadir of 1128 nmol/L ($N < 83$). Moreover, there was no cortisol response to the desmopressine stimulation test. Pituitary MRI showed a cystic intrasellar lesion of 3 mm, suggesting most probably a Rathke's cleft cyst. A diagnosis of ectopic ACTH-dependant Cushing's syndrome was made. The patient underwent distal pancreatosplenectomy and the final pathology revealed a 5.0 cm-sized grade 2 well-differentiated pNET pT3N0. Immunohistochemical positivity for ACTH was found in 50% of the tumor. Postoperatively, the patient showed clinical, biochemical and radiologic remission. In conclusion, we report a case of EAS from transformed non-functioning pNET, which is extremely rare. It highlights the importance of early detection and that surgical resection is the treatment of choice for this condition when possible.

Hyperandrogenism in NGLY1-Related Congenital Disorder of Deglycosylation (NGLY-CDDG): A Rare and Novel Association?

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NGLY-CDDG is a rare congenital disorder of deglycosylation, characterized by complex neurological features, hypolacrimia, and elevated liver transaminases. Less than 30 patients have been described. Endocrine abnormalities have not been consistently found, although adrenal insufficiency has been reported.

Objective: to present a patient with NGLY-CDDG and ovarian hyperandrogenism.

Case: 17-year-old girl with NGLY-CDDG, refractory seizures, dystonia, was seen in Endocrinology for amenorrhea. Puberty onset was around age 12 years, and she had experienced vaginal spotting 8 months before the visit. Family history was unremarkable. On physical exam, hirsutism was noted on the chin, chest and abdomen. Genital exam showed signs of estrogenization, no clitoromegaly, Tanner 4 breast development. She had achieved midparental height, BMI was normal. Investigations revealed normal gonadotropin and estradiol levels (within adult range), testosterone 9.15 nmol/L (confirmed by repeat Liquid Chromatography and Tandem Mass Spectrometry), bioavailable testosterone 1.81 nmol/L, 17OHP 3.1 nmol/L, cortisol 472 nmol/L, dehydroepiandrosterone sulfate 0.4 umol/L. Tumor markers were negative. Abdominal ultrasound and pelvic MRI demonstrated normal ovaries without any masses. After 6 months of follow-up, without clinical changes, cyclical progesterone was recommended.

Discussion: Abnormal protein glycosylation impacts the endocrine system; gonadal failure, and less frequently hyperandrogenism have been reported in these disorders. To the present, this has not been found in NGLY-CDDG. Our patient presented with secondary amenorrhea, hirsutism, elevated testosterone, normal adrenal androgens and normal imaging studies. Ovarian hyperandrogenism in NGLY-CDDG may be a novel association.

Diagnosis and Management of Hyperosmolar Hyperglycemic State: A Quality Improvement Study

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Background/Objective: Hyperosmolar Hyperglycemic State (HHS) is a life-threatening hyperglycemic emergency. Despite high mortality and morbidity, there is a paucity of evidence for HHS management and patients are often treated with diabetic ketoacidosis protocols. This study describes the diagnosis and management of HHS at a teaching hospital.

Methods: A retrospective chart review was conducted of patients 18 years and older from June 1, 2014–June 30, 2019 who were diagnosed with "HHS," "hypernatremia," "hyperglycemia," or "hyperosmolality." Patients diagnosed with and/or who met the Diabetes Canada Clinical Practice Guidelines HHS diagnostic criteria were included.

Results: Forty-nine charts met the inclusion criteria. These included 29 (59.2%) males and 20 (40.8%) females, mean age 65 years (+/-15.3). Forty (81.6%) patients were admitted with an average hospital admission of 7.7 (+/-8.8) days, 9 (18.4%) were discharged from the emergency department, and 2 (4.1%) patients died. Twenty-eight patients (57.1%) had pre-existing type 2 diabetes. The average HbA1C was 11.8% (+/- 2.7). Seventeen (34.7%) patients were diagnosed with HHS without obtaining a serum osmolality, 14 (28.6%) were diagnosed despite serum osmolality of $<$ 320 mOsm/kg, and 4 (8.2%) met diagnostic criteria but were not diagnosed with HHS. Intravenous (IV) normal saline, ringer's lactate and a combination of fluid types were used in 34 (69.4%), 3 (6.1%), and 10 (20.4%) patients respectively. The majority of patients received IV (n = 26, 53%) or subcutaneous (n = 20, 40.8%) insulin.

Conclusion: This study demonstrates variability in HHS diagnosis and management. The heterogeneity of local practices suggests that a standardized protocol may enable a more consistent approach to patient care.

Surgical Outcomes Among Primary Aldosteronism Patients Without Visible Adrenal Lesions

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Background: Primary aldosteronism (PA) is the most common form of remediable hypertension. Many patients with unilateral PA have normal adrenal imaging, but little is known about their outcomes following adrenalectomy. The aim of this study was to evaluate outcomes after adrenalectomy in patients with unilateral PA and normal-appearing adrenal imaging.

Methods: We retrospectively evaluated consecutive individuals seen at a Canadian referral center between January 2006 and May 2018 with PA, normal cross-sectional adrenal imaging, and lateralizing adrenal vein sampling who underwent adrenalectomy. Baseline characteristics were recorded and rates of clinical and biochemical adrenalectomy outcome graded according to the Primary Aldosteronism Surgical Outcome criteria were determined.

Results: A total of 40 patients were included. Clinical outcomes were available for 36 people (mean age, 54.6 years; 86% male; mean body mass index [BMI], 31.0 kg/m2; median follow-up, 9.8 months), with 31 (86%) demonstrating a complete or partial response and 5 (14%) with no response. Biochemical outcomes were available for 33 people (mean age, 54.7 years; 91% male; mean BMI, 31.4 kg/m2; median follow-up, 2.7 months), with 28 (85%) showing a complete or partial response and 5 (15%) with no response.

Conclusions: Response rates in this cohort were comparable to those observed in larger cohorts of patients with distinct adenomas. Thus, the prognosis after adrenalectomy is highly favourable for patients with unilateral PA and normal-appearing adrenal imaging, even among those with poor prognostic factors (older age, male sex, obesity). Patients with lateralizing disease should be considered for surgery despite apparently normal adrenal imaging.

Insulin Pumps Versus Multiple Daily Injections in Type 1 Diabetic Pregnancies

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Background: Pregnancy is associated with physiological increased resistance to insulin, leading to increased insulin requirements for women with type 1 diabetes. Poor glycemic control in pregnancy is associated with neonatal and maternal complications. Insulin has been traditionally administered subcutaneously through multiple daily injections (MDIs) however, use of insulin pumps or continuous subcutaneous insulin infusion (CSII) has become more widespread. As glycemic control has direct influence on perinatal and maternal outcomes, comparing MDIs and CSII in pregnant women with Type 1 Diabetes Mellitus (DM1) is of interest.

Aim: To investigate whether there is a difference in birth weights in infants born from DM1 mothers using CSII compared to MDIs.

Methods and Results: This is a retrospective cohort study of women diagnosed with DM1 on either MDIs or CSII whose pregnancies were followed at our institution. The following data was collected on chart review: age, years since DM1 diagnosis, BMI and weight gain at each trimester, total daily insulin dose at delivery, presence of retinopathy or nephropathy, gestational age at delivery, delivery method, presence of hypertension, pre-eclampsia or eclampsia, birth weight, sex of infant and neonatal complications. Preliminary analysis of our currently available data demonstrates that in age-matched groups of women on MDI (N = 44) compared to CSII (N = 8), there was no significant difference in infant birth weights (3410g $\pm$ 764 vs. 3522g $\pm$ 630, respectively). There was also no significant difference in neonatal complications (jaundice, hypoglycemia, shoulder dystocia, macrosomia, respiratory distress or prematurity). Women on CSII had been diagnosed with DMI for a significantly longer period of time (20 vs. 14 years). Women on MDI were on higher total daily doses of insulin per body weight at time of delivery ($p < 0.05$). Despite this, there was no significant difference in glycemic control or weight gain during pregnancy between the groups. There was also no significant difference in the presence of hypertension, pre-eclampsia, eclampsia or in C-section rates.

Discussion: Given the paucity of studies and increasing use of pump technology among diabetic women, there remains a need for further research comparing the effects of MDIs and pumps on perinatal outcomes.

Improving Care Delivery for Young Adults with Type 1 Diabetes via a Multi-Faceted Interdisciplinary Intervention

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Background: Young adults (YA) with type 1 diabetes (T1D) represent a vulnerable population at risk for loss to follow-up and complications. To address this, a multi-faceted quality improvement (QI) intervention that included a transition navigator, an interdisciplinary flowsheet, and virtual care was implemented at a YA T1D program using a stepwise approach beginning in 2019.

Objective: To determine whether this QI intervention improves attendance and care delivery amongst YA with T1D.

Methods: The attendance of patients seen between January 2017 and August 2020 were tracked monthly on a run chart. A pre-post analysis was performed to compare secondary outcomes including A1c reduction, incidence of diabetes-related ED visit/hospitalizations and psychosocial counselling rates in new patients followed for 12 months.

Results: There were 2240 scheduled appointments. Attendance improved from 59% to 79% ($p < 0.01$) with virtual care, demonstrated by a shift sustained over 6 months after its implementation. Forty-two patients were included in the pre-post analysis (n = 27 in the pre-intervention and n = 15 in the post-intervention period with no significant difference in baseline characteristics). Mean patient age was 20.2+/-2.9 years with average diabetes duration of 11.1+/-5.3 years, 28.5% were male and baseline average A1c of 8.6+/-1.7%. Preliminary analysis demonstrates there was significant improvement in preconception counselling rate (76% *vs.* 100%, p = 0.048) following the intervention with no difference noted in A1c reduction or incidence of diabetes-related ED visits/hospitalizations.

Conclusions: Virtual care was effective in improving attendance for YA with T1D. Data analysis for patients assessed in September to December 2020 is underway.

Optimizing Antidiabetic Medication Re-Initiation Upon Discharge of Patients Who Transitioned Through the Intensive Care Unit

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Background: Medication discrepancies are frequent when a patient transitions through the intensive care unit (ICU). Oral antidiabetic agents (OADAs) are often discontinued or replaced by insulin therapy in patients admitted to the ICU. However, there is little data on whether OADAs are re-initiated on hospital discharge.

Methods: We undertook a retrospective cohort study looking at the prevalence of OADAs taken on admission that were not re-prescribed at hospital discharge of patients who had transitioned through the ICU. We collected data through chart review of patients admitted from April 2016 to April 2017. A univariate analysis was performed to identify variables associated with the discontinuation of OADAs, with a focus on Metformin.

Results: Data from 120 critically ill diabetic patients who survived until hospital discharge were analyzed. There were 102 patients on at least one OADA before hospitalization: 87 on Metformin, 38 on a sulfonylurea, 37 on a DPP-4 inhibitor, 2 on a SGLT2 inhibitor. 28 patients (27.5%) were discharged without any OADA, while Metformin was discontinued in 31 patients (35.6%). Pre-existing CKD (OR 5.318; p = 0.010), creatinine level on hospital admission (OR 1.033; p = 0.002), need for vasopressor support during ICU stay (OR 2.850; p = 0.023), as well as creatinine level on hospital discharge (OR 1.025; p = 0.002) were significantly associated with the discontinuation of Metformin.

Conclusion: A significant number of diabetic patients who survive their ICU stay are discharged from the hospital without re-prescription of their OADAs. Further studies are needed to better understand the long-term impact of this phenomenon.

Neonatal Impacts of a Subcutaneous Insulin Protocol for Intrapartum Glucose Control Compared to Intravenous Insulin in Women with Gestational Diabetes

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Background: Glycemic control during labour is necessary to decrease both maternal and fetal complications in women with gestational diabetes (GDM). Canadian guidelines recommend intravenous (IV) although few studies compared the safety between IV and SC insulin protocols.

Objective: We compared the incidence of neonatal hypoglycemia and neonatal intensive care unit (NICU) admission between SC and IV insulin protocols for intrapartum glucose control.

Methods: Before June 2018, at Hôpital Maisonneuve-Rosemont, mothers with GDM were managed with IV insulin during labor. A decision was made to introduce a SC insulin protocol. We performed a retrospective cohort study to determine rates of neonatal hypoglycemia 6 months before the introduction of the new protocol (from December 1, 2017 to May 30, 2018) and 6 months after (from July 1, 2018 to December 31, 2018). We performed stratified analysis and regression models controlling for prematurity and birth weight.

Results: Of the 451 neonates, 188 (41.7%) were in the IV group and 263 (58.3%) in the SC group. Maternal characteristics were similar in both groups, except for prior history of GDM (17.2% *vs.* 31.4%) p = 0.006. There was no difference in any pregnancy characteristics. Rate of hypoglycemia was (14.1% *vs.* 13.2%) p = 0.80 and rate of recurrent hypoglycemia was (34.6% *vs.* 32.4%) p = 0.85. Insulin management was not associated with hypoglycemia in the multivariate model (p = 0.53).

Conclusion: Our data support intrapartum glycemic control with SC insulin. Rate of neonatal hypoglycemia are comparable between our cohorts.

The Use of Genetic Testing Panels in Dyslipidemia: A Quality Improvement Project

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Background: Genetic testing panels are used to identify the most common genetic causes of dyslipidemia, and the results of these panels can guide treatment and management. The objective of this quality improvement project was to assess the appropriateness of genetic testing panels requested by our institution.

Methods: Genetic testing panels sent for analysis from January 2018 to December 2019 were identified. Ordering physician specialty, patient personal and family medical history, lipid panel results, and genetic testing results were collected. Then, validated Familial Hyperlipidemia (FH) scores (Simon-Broome Registry Criteria, Dutch Lipid Clinic Network Criteria, FH Canada criteria) were calculated for patients who underwent genetic testing for suspected FH.

Results: There were 36 genetic test panels sent out for analysis during the study period, of which 24 were accessible for data analysis. Pathogenic mutations were identified in 7/24 (29%) of the analyzed panels. The 19/24 (79%) of the panels were requested by lipid specialists, and all of the panels positive for pathogenic mutations were requested by lipid specialists. Interestingly, 23/24 (94%) of the patients met the Canadian criteria for at least considering genetic testing, suggesting that most panels were appropriately requested. Only 3/24 (12%) of patients had insufficient criteria for FH by the Simon-Broome criteria, but all of these carried pathogenic mutations.

Conclusion: These results suggest that at our institution, using the Canadian criteria identifies a greater number of patients for genetic testing and for appropriate diagnosis and treatment.

Characterizing Types of Diabetes Clinical Questions and Answers Provided via eConsults

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Background: A regional eConsultation service enables primary care providers to directly ask a specialist a clinical question, avoiding a face-to-face referral in over 45% of cases. Endocrinology is the third most popular specialty on the service. In order to fully understand the potential of eConsult services to reduce face-to-face visits, further characterization of the types of questions asked and answers provided is needed. The objective of this study was to describe the diabetes-related clinical questions and advice provided.

Methods: Two coders (G.G. and W.B.) categorized 128 of 891 Endocrinology eConsults (received from 2017–2018) as diabetes-related questions. One hundred diabetes consults were coded based on preset taxonomies to capture type of clinical question(s) and answer(s). Descriptive statistics were used to describe the findings.

Results: The most commonly asked diabetes clinical question was "what drug to choose next" (44%), while 15% of questions asked how to adjust insulin. Seven percent of questions asked about "diagnosing type of diabetes" and 10% of questions were on "addressing co-morbidities/complications". Forty-eight percent of all specialist answers gave advice on a specific name/class of drug with dose, while 12% gave advice on specific name/class without a dose. The indication/rationale for drug choice was outlined in a quarter of answers. Referral to Endocrinologist was recommended in 15% of cases.

Conclusion: Endocrinologists are most commonly asked about medication adjustments in diabetes-related eConsult questions. Although often explicit in name and dose of drug, providing rationale for the recommendation may help build capacity in primary care.

Retrospective Cohort Study Using Electronic Health Data to Create an Edmonton Obesity Staging System Dashboard

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Background: The Edmonton Obesity Staging System (EOSS) is a better predictor of mortality than body mass index (BMI). We conducted a retrospective cohort study to determine the feasibility of establishing EOSS using health administrative data, and creating a clinical dashboard.

Methods: Data were extracted from a health administrative database between July 2016 and July 2019 and EOSS scores were assigned. Individuals ≥ 18 years, with a BMI ≥ 30 kg/m2 who had at least one visit with a physician contributing data to the database, were included. Descriptive statistics and ordinary least squares (OLS) regressions were produced using Oracle SQL Developer, Python 3.4 and Stata 16.

Results: Of 23,460 patients included, majority had obesity class I (54%), and an EOSS score of 2 (53%), indicating established obesity-related comorbidities. When BMI was compared to EOSS scores, EOSS was a better risk stratification tool. Of the variables included, age was the main factor that explained EOSS variation (31%). We were able to assign EOSS scores to 97.7% of patients. Missing data ranged from 11–18% for comorbidities. An obesity dashboard was created using the Canadian Primary Care Sentinel Surveillance Network Data Presentation Tool.

Conclusion: The EOSS comorbidity-driven approach of risk stratification provides a more nuanced assessment than BMI alone. An obesity dashboard makes this information easily accessible for quality improvement and individual clinical care. A high proportion of patients in our region are in the EOSS 2 category, providing an opportunity to intervene and improve clinical outcomes for people living with obesity.

Co-morbidity Assessment in Acromegaly: Colonoscopy Versus Osteoarthropathy

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Pituitary growth hormone excess is associated with co-morbidities due to the trophic effects of growth hormone. Recommendations for screening colonoscopies is well established, while bone mineral densities (BMD) and spinal x-rays are new recommendations in a 2020 Consensus on the Diagnosis and Treatment of Acromegaly (JCEM 105(4):e937, 2020). The objective of this study is to assess the prevalence of screening colonoscopy versus the detection of osteoarthropathy in patients with acromegaly. This is a retrospective chart analysis of 108 patients with confirmed acromegaly, from two pituitary clinics from 2000 to 2018 in a single centre. Deceased and out-of-province patients were excluded. Records of colonoscopies, BMDs, joint imaging and surgeries were reviewed. Descriptive analysis and chi-squared analysis was performed. The cohort was 54.6% male (55/108) with 74% of patients having age-controlled IGF-1 levels. 71.2% (77/108) had one colonoscopy, and 36.1% had at least two and one case of colon cancer was detected. BMD was performed in 51.8% of patients (56/108) which detected four patients with osteoporosis. Spinal x-rays occurred in 63.8% (69/108) which detected 2 patients with vertebral fractures. A total of 23% joint replacements occurred including 12 hip, 9 knee, spinal surgery in 5, and wrist/ankle surgery in 4. There was no statistical difference between frequencies of colonoscopies, BMD or spinal x-rays. Osteoarthropathy is common in acromegaly but is not detected through traditional screening tools. Given the impaired quality of life secondary to osteoarthropathy, better screening tools and early intervention are important.

Better Understanding Hypoglycemia in People Living with Type 1 Diabetes (T1D)

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Introduction: For patients living with T1D, hypoglycemia is a major obstacle in achieving optimal glycemic control with significant adverse implications on individuals' quality of life. To improve hypoglycemia prevention and management, the BETTER registry for people living with T1D was launched in April 2019 in the province of Quebec, Canada.

Methods: Participants self-register online in the BETTER registry and are asked to answer a series of 3 questionnaires. We report the results of phase I among participants of ≥14 years old, which aims to provide a comprehensive portrait of the population with T1D.

Results: Mid-August 2020, 1014 individuals (61.7% female, age 41.0 ± 15.6 years old, duration of diabetes 22.7 ± 15.6 years, 92.8% with T1D and 7.2% with LADA, 76.5% with HbA1c < 8%) have registered. Of the participants, 99.5% were on insulin therapy (43.0% on insulin pumps, 55.0% on insulin injections, 1.5% using both), and 76.1% were using continuous or flash glucose monitoring. They reported, on average, 6.47 ± 7.67 episodes of hypoglycemia (< 3 mmol/L) in the last month. Since their diagnosis, 39.3% of participants had at least one episode of severe hypoglycemia (SH: requiring help from another person or use of glucagon or hospitalization or loss of consciousness). Among those who reported SH in the last 12 months (n = 117), the average number of episodes was 3.18 ± 5.73 during this period.

Conclusion: In a group of patients widely using technology for T1D management, hypoglycemia remains frequent. The BETTER registry will allow further understanding of this challenging reality and enable future research aiming to reduce hypoglycemia episodes.

High Sensitivity of 18F-FDG PET/CT in a Large Cohort of Pheochromocytomas and Paragangliomas: Correlation of Genetic Mutation Status and Imaging Uptake Levels

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Background: The role of 18F-FDG PET/CT in the investigation of pheochromocytomas (PHEOs) and paragangliomas (PGLs) (PPGL) is emerging and is currently mainly focussed on metastatic tumors.

Objective: To assess the sensitivity of 18F-FDG PET/CT in a large cohort of PPGLs that was characterized genetically.

Method: We retrospectively studied patients with PPGLs that had 18F-FDG PET/CT and germline genetic characterization at Centre hospitalier de l'Université de Montréal (CHUM) from 2005–2020.

Results: The cohort included 115 patients (57 PHEOs, 58 PGLs), the mean age was of 47.7+/-15.9 yo and, 62.9% were female. Germline mutations were identified in 22.8% of PHEOs and 41.4% of PGLs. The overall 18F-FDG PET/CT sensitivity was of 94.8% (109/115); 94.7% (54/57) for PHEO, 93.1% (27/29) for head and neck PGLs and 96.6% (28/29) for thoraco-abdominal PGLs. Among the 23 (20%) patients with malignant PPGLs, the mean SUVmax was of 11.9 which did not differ from the benign group ($p = 0.54$). Interestingly, patients carrying a germline mutation in cluster 1 genes (SDHx, FH, VHL) (n:27) had a higher mean SUVmax compared to patients carrying a mutation in cluster 2 genes (n:10) (RET, NF1, MAX) (21.2 *vs.* 5.2 $p = 0.0006$) as in non-mutated patients (n:78) (21.2 *vs.* 8.12 $p < 0.0001$).

Conclusion: In our study, 18F-FDG PET/CT had a high sensitivity of 94.8%. SUVmax values did not correlate with malignancy but seemed to be higher in cluster 1 associated with SDHx, VHL or FH mutations. Further studies are needed to demonstrate the mechanisms that explain this distinct 18F-FDG uptake pattern.

Romosozumab: The Newest Agent Approved for Osteoporosis in Canada; A Review of the Pharmacology, Efficacy and Safety

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Background: The World Health Organization has declared osteoporosis a public health crisis. The International Osteoporosis Foundation recommends treating patients at highest risk of fracture directly with an osteoanabolic agent followed by an antiresorptive agent. Romosozumab is a novel monoclonal antibody approved in Canada in October 2019. It is currently the only agent on the market that both promotes bone formation and inhibits bone resorption.

Objective: This review summarizes the pharmacology, clinical efficacy, and safety profile of romosozumab.

Methods: A literature search using MedLine and Embase from 1996–April 2020, using the keywords “romosozumab” and “randomized control trial”, yielded 144 results.

Results: Eight randomized control trials involving 12,625 patients were included. Phase III studies in postmenopausal women and men showed that romosozumab improved bone mineral density (BMD) and decreased fracture risk when compared to placebo. In active-control studies performed in post-menopausal women, 12 months of romosozumab had greater efficacy than alendronate and teriparatide with respect to risk of fracture and BMD, respectively. One phase III trial showed that serious cardiovascular events (i.e. cardiac ischemic events and cerebrovascular events) were observed more often with romosozumab than alendronate.

Conclusion: Clinical trials have proven romosozumab to be a promising new osteoporosis therapy, particularly for those at highest risk of fracture. One year of treatment with romosozumab is superior to all other standard therapies with respect to increasing BMD and reducing the risk of fracture. Further studies are required to determine if romosozumab increases the risk for cardiovascular and/or cerebrovascular events as suggested by one trial.

Improving Access to Osteoporosis Specialists Through Electronic Consultation

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Background: The Champlain BASE (Building Access to Specialists through eConsultation) electronic consultation (eConsult) service is a secure online platform where primary care physicians (PCPs) can pose clinical questions to specialists. The objective of this study is to identify the types of osteoporosis questions being asked and the impact of the advice provided by eConsultation specialists.

Methods: All endocrinology eConsults submitted between 2017–2018 through the Champlain BASE service were evaluated, identifying 114 (12.8%) which were specific to osteoporosis. Two independent reviewers classified each osteoporosis eConsult by question types and recommendations provided, based on preset taxonomies. Plans for referral following eConsultation, impact on care provided and overall satisfaction rates were measured via PCP surveys.

Results: Within the 114 osteoporosis consultations, 203 questions were asked and 256 recommendations were provided. The most common questions were regarding treatment initiation (15.3%), bone mineral density (BMD) frequency (12.8%) and drug holidays (10.8%). The most common recommendations pertained to BMDs (16.4%), starting specific pharmacotherapy (12.5%) and starting calcium/vitamin D (9.7%). The PCP received their reply in a median of 4.4 days. In 60.1% of cases, the PCP received new information they would be applying to the care of their patient. Only 12.3% of cases needed an in-person referral after the eConsult. Overall, 96.5% of PCPs found the eConsult responses to be valuable.

Conclusion: eConsultation improves access to specialist care in a timely manner. It is an effective way to answer osteoporosis questions, impact the care PCPs provide to their patients and reduce the need for face-to-face visits.

Optimizing the Treatment of Inpatient Hypoglycemia at a Quaternary Care Hospital: A Quality Improvement Initiative

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Background: Prompt treatment of hypoglycemia is important to minimize length of stay and mortality in hospitalized patients with diabetes. This study aims to increase the percentage of inpatients with hypoglycemia achieving rapid correction with implementation of a hypoglycemia management order set.

Methods: The Model for Improvement QI framework was applied to minimize the delay in hypoglycemia treatment at a quaternary care hospital of 455 beds. Root causes were identified at a multidisciplinary committee meeting and a hypoglycemia management order set was subsequently implemented hospital-wide. To evaluate its effectiveness, blood glucose measurements and times are being obtained from the Electronic Medical Record from 24 months pre-implementation to 12 months post-implementation of the order set to calculate the percentage of inpatients with hypoglycemia achieving rapid correction ($>$ 4 mmol/L in less than or equal to 20 minutes). Chart review of nursing documentation for patients with treatment delays and surveys of nurses, pharmacists, and residents will be conducted to identify additional root causes. Plan-Do-Study-Act (PDSA) cycles will be used to develop further improvement strategies.

Results: Preliminary root cause analysis revealed lack of education in using the order set, inadequate ward supplies for hypoglycemia treatment, and infrequent glucose checks while treating hypoglycemia. Data analysis to assess the order set's impact on hypoglycemia treatment is underway to conduct future PDSA cycles.

Conclusions: Future QI interventions to address the delay in inpatient hypoglycemia treatment may include automated order reminders, optimizing ward supply management, and increased education. Ongoing data analysis will be conducted to implement future PDSA cycles.

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