healthy controls. However, whether CHL1 gene regulates islet function remained to be explored.
Methods: PCR and Western Blot were applied to investigate the tissue distribution of CHL1 in wild-type C57BL/6J mice. The islet expression of CHL1 gene was observed in pancreatic islets of NOD mice and high-fat-diet C57BL/6J mice of different ages. MIN6 cells with siRNA to silence CHL1 or with lentivirus to overexpress CHL1 were constructed. Effects of the gene on proliferation, apoptosis, cell cycle and insulin secretion were determined by using CCK8, EdU, TUNEL, AV/PI, GSIS, electron microscopy and flow cytometry.
Results: CHL1 was localized on the cell membrane and expressed in the nervous system, islet of pancreas and gastrointestinal tract. CHL1 was hypoexpressed in the pancreatic islets of obese mice, hyperexpressed in the pancreatic islets of NOD mice and in vitro after treated with cytokines. After silencing CHL1 in MIN6 cells, insulin secretion decreased in 20 mM glucose with down-regulation of INS1, SLC2A2 gene, and transmission electron microscope showed the number of insulin secretary granules <50nm from the cell membrane was significantly
reduced. Silencing of CHL1 in MIN6 cells induced cell proliferation, reduced apoptosis rate, prolonged the S phase of cell cycle and shortened the G1 phase with downregulated expression of p21, p53 and up-regulated expression of cyclin D1, opposite results were found in CHL1 over-expressing MIN6 cells. Proliferation induced by silencing of CHL1 was inhibited by ERK inhibitor (PD98059), which indicates that ERK pathway is essential for signaling by these molecules in pancreatic β cell.
Conclusion: The expression of CHL1 gene was significantly decreased in the pancreatic islets of obese mice induced by high-fat diet. The low expression of CHL1 gene promotes the proliferation of MIN6 cells through the ERK pathway and affect cell cycle through the p53 pathway. This may be one of the mechanisms that pancreatic β cells compensatory hyperplasia in the stage of obesity-induced pre-diabetes.
Key words: CHL1, pancreatic islets, β cells, cell proliferation

Neuroendocrinology and Pituitary

CASE REPORTS IN UNUSUAL PATHOLOGIES IN THE PITUITARY II

Sinonasal Papilloma Masquerading as a Pituitary Macroadenoma

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MON-260
Background: Sinonasal tumors are rare, with annual worldwide incidence of approximately 1 in 100,000, and are not commonly considered in the differential diagnosis of pituitary tumors (1). Sinonasal tumors are well known for their invasiveness, tendency to recur and association with malignancy. We present a case of sinonasal papilloma presenting as a large suprasellar mass.
Clinical Case: A 61 year-old male with a past medical history including type 2 diabetes mellitus presented with chief complaints of headaches and visual disturbances over the past 6 months. Prior to admission he experienced episodes of left eye midline deviation associated with diplopia. New onset dysphagia associated with leftward tongue deviation prompted him to seek medical attention. The social history was notable for chemical exposures in his work at a hair salon; he is sexually active with his husband. He has had no sexually transmitted infections and has been vaccinated against human papilloma virus (HPV). CT of the brain showed a large sellar mass.
A subsequent MRI of the pituitary demonstrated a large destructive mass centered on the clivus elevating the pituitary gland into the suprasellar cistern. The mass measured 6 cm x 4.5 cm in the axial plane with displacement without invasion of the cavernous sinuses. The mass extended anteriorly into the ethmoid sinuses and extended posteriorly into the prepontine cistern displacing the basilar artery.
Pituitary hormonal analysis included a 250 mcg Cosyntropin stimulation test resulting with a random cortisol of <1.0 ug/dl rising to 17.7 ug/dl following Cosyntropin administration. Additional anterior pituitary results included FSH of 3.8 mIU/ml (1.5-14 mIU/ml), LH of 1.3 mIU/ml (1.4-7.7 mIU/ml), total testosterone of 230 ng/dl (300-700 ng/dl), and prolactin 11.1 ng/ml (2.6-13 ng/ml).
Ophthalmology was consulted for visual field testing which proved normal, however a partial left cranial nerve VI palsy was noted likely secondary to cavernous sinus involvement. A biopsy of the sellar mass was obtained by bedside nasal endoscopy. The initial biopsy was consistent with a non-dysplastic, inverted sinonasal papilloma with negative HPV and P16 serologies. The patient underwent resection of the pituitary mass, with surgical pathology showing superficially invasive squamous cell carcinoma arising from sinonasal papilloma.
Conclusion: This is one of the very few cases reported in the literature of a sinonasal papilloma masquerading as a pituitary mass. Sinonasal papilloma should be considered when evaluating large destructive suprasellar tumors. Although a benign tumor, the local aggressiveness of sinonasal papilloma and the potential to give rise to squamous cell carcinoma highlights the significance of identifying this lesion.

Adrenal

ADRENAL - CORTISOL EXCESS AND DEFICIENCIES

Peri-Operative Glucocorticoids in Patients Having Total Joint Replacements: Help or Harm?

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MON-168
Introduction: There is limited evidence to guide perioperative steroid regimen in patients receiving chronic glucocorticoid (GC) therapy. Many patients who undergo minor surgical procedures receive stress-dose steroids. While hemodynamic instability and hypotension are well-recognized

risks of inadequate peri-operative GCs, there may be harms associated with using doses higher than necessary. Whether surgical outcomes differ according to peri-operative steroid dose is not known. We hypothesized that patients who had greater GC exposure have less hypotension, but higher rates of hyperglycemia and post-operative complications.
Methods: This retrospective study investigated the relationships between peri-operative GC use and post-operative complications following total hip/knee joint replacement (arthroplasty) in patients with rheumatoid arthritis (RA). All GCs were converted to prednisone equivalents; GC exposure was assessed by number of doses and total cumulative dose during the hospitalization. Complications (infection, thromboembolism and cardiovascular events) were determined by chart review.
Results: Of 432 patients with RA included, half (54%) underwent knee arthroplasty. Mean age was 64±12 years, 78% were women. Thirty percent of patients were on home GCs (mean dose 7±4mg/day). Median cumulative GC dose during hospitalization was 37mg [IQR 27, 57]. Compared to patients who only received one peri-operative dose of steroids, those who received multiple doses had a greater risk of post-operative complications (OR 3.319 (95% CI 1.03, 12.62; p<0.05] and hyperglycemia, glucose >180 mg/dl, [OR 1.812(0.99, 3.32; p<0.05]. They did not have an increase in hypotension or need for pressors. Among patients who received steroids while in the hospital (90%), there was a small but significant dose response relationship with hyperglycemia (r=0.16; p<0.01). Higher cumulative dose was also associated with higher risk of complications; for every 10 mg increase in cumulative dose, the risk of complications increased by 15% (p<0.01).
Conclusions: Among RA patients undergoing arthroplasty, we did not find that lower doses of GCs were related to more hypotension. However, patients with higher GC exposure were more likely to have hyperglycemia and post-operative complications. Our results suggest that use of peri-operative GC is not without risk, and the lowest doses possible should be considered. Further studies are needed to confirm these findings and to define the optimal dosing strategies for patients receiving peri-operative GCs.

Neuroendocrinology and Pituitary

ADVANCES IN NEUROENDOCRINOLOGY

Activation of GFRAL Neurons Decreases Food Intake via Aversive Pathways

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SUN-269

Growth and differentiation factor 15 (GDF15), an anorexigenic peptide that represents a promising candidate for anti-obesity treatment, acts via GDNF Family Receptor Alpha Like (GFRAL), which is expressed almost exclusively on a subset of neurons in the area postrema (AP). To determine the function and mechanisms of action for GFRAL neurons, we generated *Gfral*^{cre} and conditional *Gfral*^{CreERT} mice. Although their chemogenetic (DREADD-mediated) activation promoted FOS in a variety of brainstem, hypothalamic, and limbic nuclei, GFRAL neurons projected only to the nucleus of the solitary tract (NTS) and the parabrachial nucleus (PBN), where they innervated and activated aversive/anorexigenic GCRP-expressing cells. Tetanus-toxin-mediated silencing of PBN CGRP neurons abrogated the aversive and anorexic effects of GDF15. Furthermore, while non-gastrointestinal (GI) stimuli (e.g., GDF15 and LPS, but not feeding or gut peptide mimetics) activated GFRAL neurons, chemogenetically activating these cells decreased gastric emptying, suppressed feeding, and promoted a conditioned taste aversion. These findings suggest that GFRAL neurons link non-GI anorexigenic signals to the control of gut physiology and to the aversive suppression of food intake. Additionally, because the chemogenetic activation of GFRAL neurons suppressed food intake more strongly than GDF15 in lean mice, additional modes of activating GFRAL neurons may augment the anorectic potential of GDF15.

Neuroendocrinology and Pituitary

CASE REPORTS IN CLASSICAL AND UNUSUAL CAUSES OF HYPOPITUITARISM II

Brain Fog: An Important Cue to Neurosarcoidosis and Its Flare-Up?

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MON-245

Introduction:
Sarcoidosis is a multisystem inflammatory disorder characterized by noncaseating granulomas in various organ systems, mainly the lung and lymphatic system. Neurosarcoidosis (NS) involving central or peripheral nervous system is uncommon and Hypothalamic-pituitary (HP) NS is rarer.
Case:
A 45-years-old African American man presented with a few days' history of cognitive slowness (brain fog) and a sense of loss of direction. He was discharged a few months ago from another hospital after being admitted for shortness of breath and hypercalcemia of 13 mg/dl, which improved after hydration. CXR showed mediastinal and bilateral hilar enlargement. He was discharged home with outpatient pulmonary appointment but was readmitted to our hospital with altered mental status. Calcium level on this admission was 11.5 mg/dl. CT brain showed a sellar/suprasellar lesion, which was better visualized on MRI as enlargement and enhancement of pituitary gland, pituitary stalk, optic chiasm, left and right optic tract and nerves, and hypothalamus. He had left hemi-temporal field defect. Work up revealed inappropriately high normal 1,25 vitamin D, low PTH, PTrp and vitamin D. He has anterior hypopituitarism and mildly elevated prolactin. EBUS with mediastinal lymph node biopsy was non-diagnostic, however, excisional biopsy of mediastinal node showed non-caseating granuloma. ACE level, flow cytometry, infectious work up and serum IgG4 were normal.
Discussion:
NS may present as cranial nerve palsy, chronic headache with incidental HP mass and endocrine dysfunction such