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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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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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