optic nerve compression. Rarely, it can present with diplopia from cranial nerves III, IV and VI (3.7%) and cavernous sinus involvement (1).
Clinical Case:
A 40 year old woman presented with left eye pain, blurry vision, ptosis and diplopia for 2 days, preceded by headache for 2 weeks. Exam was remarkable for left eye ptosis, mild proptosis, downward and outward gaze and inability to adduct her left eye. Endocrinological exam revealed free T4 0.67 ng/dL (Nl 0.70 - 1.48), TSH 0.67 ng/dL (Nl 0.70–1.48), estradiol <10 pg/mL, LH 1.0 mIU/mL, FSH 6.9 mIU/mL, prolactin 23.3 ng/ml (Nl 5.2–26.5) and IGF-1 95 ng/mL (Nl 52–328). Cortisol was not assessed as patient was already on steroids. Work-up revealed atypical ANCA (1:320) but normal C-ANA (<1:20), P-ANCA (<1:20), and the rest of immune work-up was negative including ACE, ESR, CRP, ANA, serine protease and myeloperoxidase. No systemic manifestations were present concerning for systemic autoimmune disease. CSF exam was unrevealing including a normal ACE level. MRI revealed an enlarged pituitary gland with suprasellar extension containing a focal area of T2 hyperintensity and slight T2 hypointensity at the posterior aspect of the gland. There was a midline, thickened infundibulum, enhancement of both cavernous sinuses and narrowing of right internal carotid artery without occlusion. Endoscopic endonasal transsphenoidal biopsy of pituitary lesion confirmed diagnosis of lymphocytic hypophysitis and did not meet criteria for IgG4 hypophysitis. After 4 weeks of prednisone, she had significant symptomatic improvement and repeat MRI showed decreased pituitary size but persistent abnormal enhancement of the pituitary gland and cavernous sinuses.
Conclusion:
The atypical and variable clinical and radiological findings of lymphocytic hypophysitis can mimic other inflammatory, infiltrative lesions, pituitary tumor with apoplexy and Tolosa Hunt Syndrome. Tolosa Hunt syndrome is an idiopathic granulomatous inflammation of the cavernous sinus involving cranial nerves II to VI and often presenting with painful ophthalmoplegia. Pituitary involvement and carotid artery narrowing have been observed (2). Our case highlights a patient with cranial nerve III palsy and significant cavernous sinus involvement, clinically concerning for Tolosa Hunt syndrome, but confirmed by biopsy to be lymphocytic hypophysitis. There are no specific serum markers to distinguish lymphocytic hypophysitis from other entities and when uncertain, diagnosis is best established by biopsy.
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Bone and Mineral Metabolism

OSTEOPOROSIS: DIAGNOSIS AND CLINICAL ASPECTS

Validation of a Deep Learning Based Algorithm to Diagnose Vertebral Compression Fractures

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SUN-370
Vertebral Compression Fractures are common in patients above age 50, but are often undiagnosed. Patients with one VCF are at higher risk of other osteoporotic fractures. Zebra Medical Imaging developed a VCF detection algorithm, utilizing a combination of traditional machine vision segmentation and convolutional neural network (CNN) technology, to detect VCFs from evaluating CT images of the chest, and/or abdomen/pelvis.
We conducted an independent and blinded validation study to estimate the operating characteristics of the Zebra VCF detection algorithm in identifying VCFs on de-identified data from previously completed CT scans of chest and/or abdomen/pelvis from 1200 women and men aged 50 or older who had those scans (for multiple reasons) at the clinics and hospitals affiliated with the Cedars Sinai Medical Center. Each set of scans were independently read by two of three board certified, practicing neuroradiologists to identify and grade VCF at each evaluable vertebra (using the semiquantitative scale of Genant and colleagues). When there was disagreement between radiologists, the respective scans were reviewed by a senior neuroradiologist who provided a final evaluation. The final determination of presence and severity of VCF by the neuroradiologists was used as the reference standard. The Zebra VCF detection algorithm evaluated the CT scans in a separate workstream from that used by the neuroradiologists (The algorithm and neuroradiologists were blind to each other's evaluations).
The Zebra VCF algorithm was not able to evaluate CT scans for 113 patients. Of the remaining 1087 CT patients, 588 (54%) were women. Median age was 73 (range 51, 102; interquartile range 66, 81). The four neuroradiologists who evaluated the CT scans each had over 10 years of experience in neuroradiology. For the 1087 Zebra evaluated patients, 227 had at least one VCF (90 with mild VCF, 81 with moderate VCF, and 56
with severe VCF; 115 of the 1087 Zebra evaluated patients (10.6%) presented with two or more VCFs). The sensitivity and specificity of the Zebra VCF algorithm in diagnosing any VCF were 0.66 (95% confidence interval 0.59, 0.72) and 0.90 (95% confidence interval 0.88, 0.92) respectively; and for diagnosing moderate/severe VCF were 0.78 (95% confidence interval 0.70, 0.85) and 0.87 (95% confidence interval 0.85, 0.89) respectively.
The Zebra VCF algorithm works to identify approximately three-quarters of moderate to severe VCF in patients, aged 50 and above, who receive CT scans for other reasons. Implementing the Zebra VCF algorithm within radiology systems may help to identify patients at increased fracture risk and could support the diagnosis of osteoporosis, and thus be a valuable adjunct for population health.

Cardiovascular Endocrinology

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

Hypertriglyceridemia-Induced Pancreatitis in a Pregnant Female Treated with Plasmapheresis

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SAT-568

Hypertriglyceridemia-Induced Pancreatitis in a Pregnant Female Treated with Plasmapheresis

Background: Gestational hypertriglyceridemia can lead to critical and even life-threatening consequences to both mother and fetus. A well-known consequence is hypertriglyceridemia-induced acute pancreatitis. Few case reports described the successful management of triglyceride (TG) induced pancreatitis in pregnant women using plasmapheresis.

Clinical Case:

A 28-year-old primigravida patient, in the 29th week of gestation, was admitted with acute onset of epigastric pain and nausea for 24 hours. Laboratory findings were remarkable for an elevated serum lipase of 505 U/L (ref 23–300) and an abnormal lipid profile. Her total cholesterol was 1651 mg/dl and triglycerides (TG) from an undiluted sample was 1361 mg/dl. When a 1:5 dilution was performed the result was higher at >4000 mg/dl. She was transferred to the ICU for treatment of acute pancreatitis. She has no family history of hypertriglyceridemia. No MRI was obtained. Gemfibrozil, Lovaza™ (omega-3-acid ethyl esters), and an insulin infusion were started but serum TG levels did not improve. On hospital day 2 she developed worsening tachycardia, tachypnea with laboratory findings of metabolic acidosis and hypocalcemia. As there was no reduction in triglyceride levels with medical therapy and her clinical status was deteriorating, the treating multidisciplinary team decided to initiate plasmapheresis. After one session, TG levels decreased from >4000 mg/dl to 1829 mg/dl and continued to decline to 721 mg/dl. Hospital day 6 her TG level rose to 1245 mg/dl prompting a second plasmapheresis. TG levels decreased to 770 mg/dl shortly after but rose the next day to 1365 mg/dl. She underwent a 3rd plasmapheresis after which her TG ranged from 400–700 mg/dl for the remainder of her hospitalization. On the day of discharge, her TG level was 733 mg/dl. She was advised to restrict fat intake and continue both gemfibrozil and Lovaza™ but despite this her TGs again increased to 1693 mg/dl. From that point she started weekly sessions of plasmapheresis for a total of 8 sessions prior to an uneventful vaginal delivery at 36 weeks of gestation. One month later her lipid profile dramatically improved. Total cholesterol was 233 mg/dl and triglycerides were 304 mg/dl while on lipid lowering therapy.

Conclusion: Pancreatitis during pregnancy is associated with a high maternal and fetal death rate. Early treatment is important for the survival of the mother and fetus. Plasmapheresis is an alternative and safe treatment for cases that are not responsive to medical therapy. It can be administered safely to reduce triglyceride levels and diminish the systemic inflammatory response leading to a shortened hospital stay and better outcomes.

Adrenal

ADRENAL - TUMORS

Dehydroepiandrosterone Sulfate (DHEAS) Levels Predict Weight Gain in Women with Anorexia Nervosa

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SAT-167

Introduction: Anorexia nervosa (AN) and atypical AN (defined as weight loss and all the psychological features of AN but BMI>18.5 kg/m^2) are serious disorders characterized by undernutrition and complicated by endocrine dysregulation. Predictors of recovery, including serum biomarkers, are lacking. Prior studies have suggested that higher urinary free cortisol (UFC) may predict weight gain in women with AN, but 24-hour urine collections are not feasible in a real-world setting. Like cortisol, the adrenal androgen dehydroepiandrosterone (DHEA) and its sulfated form DHEAS, which has a longer half-life, are stimulated by ACTH. We hypothesized that DHEAS levels would correlate with UFC and be a predictor of weight gain in women with AN.

Methods: We prospectively studied 34 women with AN and atypical AN, mean age 27.4 ± 7.7 years (mean ± SD), who received placebo in a randomized trial. AN and atypical AN were diagnosed by SCID. Baseline DHEAS and 24-hour UFC were measured by LC-MS/MS (Endocrine Sciences, Calabasas Hills, CA). Weight and body composition were assessed at baseline and 6 months later by DXA and cross-sectional abdominal CT at L4.

Results: At baseline, mean weight was 51.3 ± 4.9 kg. Of the 18 subjects who gained weight (range 0.1–10.3 kg), 28% were eumenorrheic, 39% amenorrheic, and 33% on oral contraceptives at baseline; baseline reproductive status was similar for subjects who did not subsequently gain weight. In the group as a whole, mean baseline DHEAS level was 173 ± 70 μg/dL (0.7 ± 0.3 times the mean normal range for age) and mean baseline UFC for subjects who completed testing (n=15) was 20 ± 18 μg/24h (normal range 0–50 μg/24h). Higher DHEAS levels at baseline predicted weight gain over 6 months ($r=0.61$, $p<0.001$), which remained significant after controlling for age, baseline BMI, OCP use, and SSRI/SNRI use ($p<0.001$); none of these covariates were predictors of weight gain. Baseline DHEAS levels predicted an increase in fat mass ($r=0.40$, $p=0.03$) and appendicular lean mass ($r=0.38$, $p=0.04$) by DXA, and abdominal fat by CT ($r=0.60$, $p<0.001$); the associations remained significant after controlling for the above factors. UFC did not predict change in weight ($r=0.37$, $p=0.17$) or body composition. DHEAS levels were positively associated with UFC ($r=0.61$, $p=0.02$).

Conclusion: In women with AN, higher DHEAS levels are a predictor of weight gain and increases in fat mass, skeletal muscle mass, and abdominal fat. Serum DHEAS correlates with UFC, a predictor of weight gain in prior studies. DHEAS may be a more practical biomarker of recovery, as 24-hour urine collections are challenging. Further studies are needed to determine whether higher DHEAS levels are a marker of global adrenal stress response and a reflection of higher cortisol levels, which may stimulate weight gain, or an independent predictor of weight gain in AN and atypical AN, perhaps through neuromodulation.