were 9.3 ±1.2 and 9.7, and 8.6 ±1.4 and 8.6 yrs, respectively. The MN and MD age of pubertal SBs (n=48) and NCs (n=112) were 13.0 ±1.4 and 12.7, and 14.7 ±1.9 and 14.6 yrs, respectively. The difference in MN age between SBs and NCs was significant ($p<0.05$). For prepubertal subjects, sensitivity was 86.21% and specificity was 68.97%. The distance to corner was 0.3396, and the highest Youden index was 0.5517, corresponding to a PV of 215.02 mm3. The Area Under the Curve (AUC) was 0.8395 with a standard error of 0.0426 ($p<0.001$). For pubertal subjects, sensitivity was 81.25% and specificity was 79.46%. The distance to corner was 0.2781, and the highest Youden index was 0.6071, corresponding to a PV of 315.0 mm3. The AUC was 0.8460 with a standard error of 0.0337 ($p<0.001$).

Conclusion: To our knowledge, we present the first study on the sensitivity and specificity of PV in determining the etiology of SS. Our data suggest that prepubertal patients with a PV<215.02 mm3 and pubertal patients with a PV<315.00 mm3 have small pituitary glands. Statistically calculated cutoffs are necessary to accurately diagnose pituitary hypoplasia and should be utilized to determine the etiology of SS. Future studies should include children with Tanner staging and height SDs to generate more accurate PV cutoffs.

Reproductive Endocrinology

FEMALE REPRODUCTION: BASIC MECHANISMS

Effect of Poor Circumstance in Utero on Adiponectin Gene Expressions Through Epigenetic Changes in Offspring

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The links between obesity and metabolic syndrome in parents and their offspring and the role of genes and a shared environment are not completely understood. Adipocytokines play important roles in glucose and lipid metabolism. We have already developed the model mice for transgenerational effect of obesity and metabolic syndrome and demonstrated that exposure to a high fat diet in utero might cause a metabolic syndrome-like phenomenon through epigenetic modifications of adipocytokine, adiponectin and leptin gene expressions in offspring of the model mice. In this study, we examined whether poor circumstance in utero affected the adiponectin gene expression and epigenetic changes of this gene using samples from umbilical cord in patients with hypertensive disorder of pregnancy (HDP) and fetal growth restriction (FGR) or gestational diabetes mellitus (GDM) and heavy for date fetus (HFD) compared with normal pregnant women without HDP, GDM and abnormal fetal growth. We observed that the poor circumstance under HDP with HGR or GDM with HFD caused significantly lower adiponectin gene expression and higher methylation level of histone H3 at lysine 9 of the promoter of adiponectin gene compared with normal control. Thus, poor circumstance in utero affected adiponectin gene expressions through epigenetic modifications, which might result in the increased risk for metabolic syndrome of offspring.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Novel Use of Abaloparatide to Augment Spinal Fusion in Patient Undergoing Cervicothoracic Revision Surgery

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MON-365

Objective To present a case of using Abaloparatide (PTHrP 1–34 analogue) to promote spinal fusion in a patient with history of cervical instability s/p multiple cervical operations with non-union. **Case Presentation** 66 year-old female with a history of multiple sclerosis, obesity and hypothyroidism underwent neurosurgical evaluation of neck pain. She was found to have cervical spinal stenosis causing neck pain, radiculopathy, motor deficits and ataxia. Initially underwent anterior cervical discectomy and fusion which temporarily alleviated symptoms before suffering nonunion. Subsequently underwent two additional surgeries which also eventually failed. She presented to our facility for revision corpectomy and spinal fusion. Given her history of nonunion, endocrinology was consulted for evaluation of metabolic bone disease. No known personal or family history of metabolic bones disease. No history of chronic steroid use. Initial endocrine evaluation excluded common pathologies. A decision was made to pursue anabolic osteoporosis therapy to attempt to augment the spinal fusion process. Patient started on Abaloparatide 80mcg daily 2 weeks post procedure with planned 12-week therapy course. Cervical CT at 3 and 6 months showed post-surgical cervicothoracic fusion with no signs of nonunion. **Discussion** Abaloparatide is a 34 amino acid synthetic analogue of parathyroid hormone related peptide (PTHrP) which works by selectively activating PTH1 receptor found on osteoblasts. Currently anabolic therapies are only FDA approved for treatment of osteoporosis but there is reported off label use in cases of spinal fusions, arthroplasty and fracture healing. Studies have shown that presence of PTH and PTHrP are necessary for fracture healing. Animal studies have also shown that intermittent PTH promotes spinal fusion. This case represents a novel use for Abaloparatide to augment spinal fusion in a human clinical model. **Conclusion** Further studies are warranted to better understand mechanism of action, drug timing and duration for optimal treatment of anabolic therapies in bone fractures and healing. The use of anabolic therapies like Abaloparatide can be considered in patients undergoing spinal fusion surgery at high risk for non-union or undergoing revision for failed fusion.**References** *O'Loughlin PF, Cunningham ME, Bukata SV et al. Parathyroid Hormone Augments spinal fusion, fusion mass, and fusion mass quality in a rabbit spinal fusion model. Spine 2009 January; 34: 121–130*