corticosteroid prophylaxis (p=0.025), postnatal steroids use (p=0.000), mechanical ventilation (p=0.000), pulmonary bronchodysplasia (p= 0.000), leukomalacia (p=0.06), patent ductus arteriosus (p=0.000), retinopathy of prematurity (p= 0.008), late onset sepsis (p= 0.09). In 197 patients post-discharge clinical follow up at 1, 3 and 24 months of correct age (CA) was performed. Around 88% of all our sample showed normal neurological development. 12% at 1 and 3 months had abnormal general movements (both writhing and fidgety movements) or absent (p = 0.001). At 24 months CA patients with abnormal/absent fidgety movements had neurological disabilities and 83% were EUGR. At 24 months, 17% had weight <10th centile and all were EUGR. 25% showed an overgrowth (weight >75th centile) with a probably increased risk of metabolic disease later in life. The incidence of EUGR increased over the years due to the augmentation in preterm births with lower GA. The first 14 days of life were a critical period and nutrition is known to be mandatory to promote newborns' growth (Asbury 2019). The EUGR condition negatively affected the neurological (Chien 2018) and auxological (Takayanagi 2018, Wood 2018) outcome of preterm infants and the early recognition of this condition is extremely important in order to implement a careful and prolonged follow-up.

Neuroendocrinology and Pituitary

CASE REPORTS IN UNUSUAL PATHOLOGIES IN THE PITUITARY

Presence of Aberrant Adrenocorticotropic Hormone Precursors in Two Cases of McCune- Albright Syndrome

Satoshi Yamagata, MD, PhD[1], Shinobu Takayasu, MD, PhD[1], Koshi Makita, MD[1], Kazunori Kageyama, MD, PhD[1], Yuta Okawa, MD[2], Yutaka Oki, MD,PhD[2], Yuko Asari, MD[1], Ken Terui, MD[1], Makoto Daimon, MD, PhD[1].

[1]HIROSAKI UNIV SCHL OF MED AND HOSP, Hirosaki, Japan, [2]HAMAMATSU UNIV SCHL OF MED AND HOSP, Hamamatsu, Japan.

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Background: McCune-Albright syndrome (MAS) is a rare disorder. MAS is caused by an activating postzygotic somatic mutation in the *GNAS*, and, is classically defined by the occurrence of fibrous dysplasia (FD), café-au-lait skin macules, and precocious puberty. Autonomous GH and/or PRL production in MAS has been reported. However, there have been no reports of ACTH excess in MAS. **Method:** Plasma ACTH and serum cortisol (F) levels were assessed using electrochemiluminescence immunoassays (Eclusys ACTH^{TM} and Eclusys Cortisol II^{TM}, respectively; Roche Diagnostics K.K., Tokyo, Japan).**Clinical Cases:** Case1; 42-year-old man showed craniofacial deformities and suffered from multiple bone fractures. He was diagnosed with FD at the age of 23 years. Café-au-lait macules were found on his back. He had slightly acromegaloid features. He showed no cushingoid features. Pituitary adenoma or hyperplasia was not detected by MRI. The diagnosis of GH excess was confirmed by no suppression of serum GH levels by a 75-g oral glucose tolerance test (nadir GH: 2.34 ng/mL) and an elevated serum IGF-I level (307 ng/mL; normal range: 92-257 ng/mL). The patient was treated with monthly subcutaneous lanreotide injection and then GH excess was well controlled. Basal ACTH and F levels in blood were 40.6-63.4 pg/mL and 8.0-10.5 μg/dL, respectively. The urinary free cortisol (UFC) level was 53μg/day. Autonomous F excess was excluded by the level of midnight F (1.2 μg/dL) and the level of F (0.2 μg/dL) after a low-dose (1 mg) dexamethasone suppression test (DST). Case2; A 32-year-old man was diagnosed with MAS and gigantism at the Pediatrics Department at the age of 5 years. Treatment of GH excess was well controlled by monthly octreotide depot. He had no acromegaloid features and no cushingoid features. Café-au-lait macules were observed from the left flank to the back. Pituitary adenoma or hyperplasia was not detected by MRI. Basal ACTH and F levels in blood were 35.5-73.1 pg/mL and 7.0-11.7 μg/dL, respectively. The UFC level was 61μg/day. Autonomous F excess was excluded by the level of F (<0.2 μg/dL) after a low-dose (0.5 mg) DST.Possibility of primary adrenal insufficiency was excluded by ACTH stimulation test and/or insulin tolerance test in both cases. The involvement of 11β-HSD1 by GH excess and PC1/3 deficiency were also excluded. Gel exclusion chromatography was then performed. POMC and pro-ACTH were detected and the aberrant ACTH/normal ACTH ratio was 42% in both cases. **Conclusion:** This is the first report of the presence of aberrant ACTH precursors, particularly POMC, in MAS. A high ratio of circulating ACTH to F may suggest secretion of inactive ACTH precursors in MAS. Further investigations are required to determine whether *GNAS* mutations or other mechanisms are involved in the presence of aberrant ACTH precursors in MAS.

Genetics and Development (including Gene Regulation)

GENETICS AND DEVELOPMENT AND NON-STEROID HORMONE SIGNALING II

Crude Protein of Pyropia Yezoensis Protects Against Tumor Necrosis Factor-á-Induced Myotube Atrophy by Regulating the Mitogen-Activated Protein Kinase and Nuclear Factor-Kappab Signaling Pathways in C2C12 Myotubes

Min-Kyeong Lee, PHD, Nam Taek-Jeong, PHD, Youn Hee Choi, PhD.

Pukyong National University, Busan, Korea, Republic of.

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Proinflammatory cytokines induce ubiquitin-proteasome-dependent proteolysis by activating intracellular factors in skeletal muscle, leading to muscle atrophy. Therefore, we investigated the protective effect of *Pyropia yezoensis* crude protein (PYCP) on tumor necrosis factor (TNF)-α-induced muscle atrophy *in vitro*. Mouse skeletal muscle C2C12 myotubes were treated for 48 h with TNF-α (20 ng/mL) in the presence or absence of PYCP (25, 50, and 100 μg/mL). PYCP at concentrations up to 100 μg/mL did not affect cell viability. Exposure to TNF-α for 48 h significantly decreased the diameter of myotubes, which was increased by treatment with 25, 50, and 100 μg/mL PYCP. PYCP inhibited TNF-α-induced intracellular reactive oxygen species accumulation in C2C12 myotubes. In addition,