the patient's compressive symptoms. Cytology showed inflammatory cells (mostly neutrophils) and numerous bacteria. The patient was emergently taken to the operating room for neck exploration, hemithyroidectomy, and incision/drainage of a suspected thyroid abscess. A drain was placed and removed POD 2 after minimal output.
The patient was discharged on oral antibiotics. 1-week post-operatively, the patient returned to the ED due to reaccumulation of the abscess. This was successfully treated with IR placement of a drain. The drain was removed 2-weeks post-operatively, and the patient is doing well.
Conclusion: Thyroid abscesses are rare but possible in young and immune-competent patients. While the imaging findings can point towards a more common diagnosis, such as thyroid carcinoma, avoiding anchoring bias is important. Imaging data should be considered in the context of the clinical picture to avoid the possibility of misdiagnosis.

Pediatric Endocrinology

PEDIATRIC SEXUAL DIFFERENTIATION, PUBERTY, AND BONE BIOLOGY

Mice Lacking Paternally Expressed DLK1 Reach Puberty at a Lower Body Weight Than Littermate Controls

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SUN-100
Body fat content along with a variety of genetic, environmental and psychosocial factors are responsible for the development and maintenance of reproductive function, especially in females. Epidemiologic studies indicate a relationship between increased body mass index and earlier puberty in girls. In contrast, a significant delay in puberty and menarche is seen in girls who are very physically active and have markedly diminished body fat. This link between reproduction and metabolism was reinforced with the recent report of loss-of-function mutations in the *Delta-like homolog 1* (*DLK1*) gene in girls with central precocious puberty (CPP) and increased body fat. *DLK1* is a paternally expressed gene located on chromosome 14q32.2 in a locus associated with Temple syndrome (TS), an imprinting disorder caused mainly by maternal parental disomy (mUPD). *Dlk1* knockout mice display pre- and postnatal growth retardation, a phenotype that overlaps with human mUPD14. However, precocious puberty, a common finding associated with TS, was not carefully characterized in these mice. We used a *Dlk1* deficient mouse model to determine the effects of Dlk1 on pubertal maturation. We confirmed by RT-qPCR that *Dlk1* mRNA was undetectable in the mediobasal hypothalamus, where kisspeptin and other regulators of puberty are expressed, of $Dlk^{+/p-}$ mice (which inherited the mutant allele from their father) whereas it was present in $Dlk^{+/+}$ mice. As reported previously, body weight was significantly lower in juvenile male and female $Dlk^{+/p-}$ mice, compared to wild-type littermate controls. Interestingly, mutant and control female mice achieved vaginal opening, a marker of puberty onset, at a similar age ($Dlk^{+/p-}$: 29.8 ± 1.5 days, n=11 *vs.* $Dlk^{+/+}$: 29.1 ± 0.7 days, n=15, p=0.6) despite a considerably lower body weight in the *Dlk1* deficient mice at the time of vaginal opening ($Dlk^{+/p-}$: 10.1 ± 0.8 g *vs.* $Dlk^{+/+}$: 14.3 ± 0.3 g, p<0.0001). Similarly, in the $Dlk^{+/p-}$ males, preputial separation occurred at a lower body weight than in controls ($Dlk^{+/p-}$: 12.4 ± 0.3 g, n=9 *vs.* $Dlk^{+/+}$: 14.1 ± 0.2 g, n=19, p<0.0001). We hypothesize that the lack of *Dlk1* at the hypothalamic level may be attenuating the effect of the low body weight on determining pubertal onset. These findings suggest that DLK1 is an important link between body weight and pubertal development in mice, as has been shown in humans.

Steroid Hormones and Receptors

STEROID AND NUCLEAR RECEPTORS

Roles of Progesterone Receptor Isoform B in Non-Small Cell Lung Cancer Tumor Progression

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Lung cancer is a leading cause of cancer mortality worldwide. Premenopausal women often has worse survival with advanced stages of the disease compared to postmenopausal women, suggesting an involvement of sex steroids and their receptors in the progression of non-small cell lung cancer (NSCLC). Progesterone receptor (PR) was reported to be involved in an inhibition of NSCLC cell proliferation and correlated with better clinical outcome. In addition, PRB suppressed epidermal growth factor (EGF)-induced NSCLC cell proliferation and activation of ERK1/2, in the absence of progestin. However, clinical and biological significance of PRB in NSCLC patients has remained virtually unknown. Therefore, we performed immunohistochemistry using monoclonal antibody specific to the N-terminus of PRB (250H11 mAb) and 1294mAb which could detect both PRA and PRB in 124 NSCLC cases: 94 adenocarcinoma and 30 squamous cell carcinoma (SCC). Overall survival (OS) was analyzed using the Kaplan-Meier plotter (KM plotter) database, examining the correlation between the status of PRs and survival rate of the patients.
19 cases were immunohistochemically positive for PRB and 23 PRA/B positive NSCLC cases, and all of four cases harboring abundant PRs were also positive for PRB. Therefore, PRB positivity was considered to be significantly correlated with the whole PR (<0.01). Of particular interest, the abundance of PR or PRB was significantly correlated with lower tumor size in total NSCLC (p=0.0395) and SCC (p=0.023), and tended to be correlated with pleural invasion in adenocarcinoma cases (p=0.051). In addition, PRB positive cases tend to have lower tumor size than those