gene expression for selected markers of beige adipocytes, including PAT2, CD137 and C/EBPβ, compared to control females. More crucially, thermogenically relevant UCP1 expression also did not differ between ERaKD females and controls. Taken together, these results suggest that knockdown of VMN ESR1 in adult female monkeys, while inducing modest weight gain after 1.5 years, may not markedly alter beige adipose gene expression of initially selected thermogenically relevant genes.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY

Expression of Programmed Death-Ligand 1 (PD-L1) in Human Pituitary Neuroendocrine Tumor

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Introduction

Some Pituitary NeuroEndocrine Tumors (PitNET) present an aggressive evolution and are resistant to standard management. Immunotherapy have shown durable efficacy in a variety of malignancies. The aim of this study was to explore the programmed death-ligand 1 (PD-L1) expression in varied subtypes of pituitary adenomas with assessment of their clinical behavior at diagnosis and follow-up.

Methods

We conducted a retrospective monocentric study, including all patients operated a PitNET between 2012 and 2018. PDL-1 immunostaining were performed using an European Conformity-In-Vitro-Diagnostic labeled anti-PDL1 antibody (Clone 22C3). PD-L1 immunostaining was evaluated as the percentage of tumor cell showing positive membrane staining, into four grades: grade 0 = <1%, grade 1 = 1 to 5%, grade 2 = 6 to 49% and grade 3 = ≥ 50%. PD-L1 expression was compared with tumor features (secretion, proliferation, invasion) and outcome.

Results

The study included one hundred and thirty-nine PitNET, including 84 (60%) nonfunctioning adenomas. Twenty-five PitNET were PD-L1 positive (18%), including 3 grade 3, 8 grade 2 and 14 grade 1. PD-L1 expression was not different between functioning and non-functioning adenomas (p=0.26). Among sixteen tumors with proliferative markers (Ki-67 ≥ 3% and p53 positive), only one was PD-L1 positive.

Conclusion

In our series, pituitary tumors rarely exhibit PD-L1 expression and this immune marker did not seem to be associated with any biological characteristic or behavior of the pituitary

tumors. Thus, PD-L1 staining is necessary before considering PD-L1 blockage in PitNET, in case of therapeutic impasse.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY

TSH Deficiency in Patients on Somatostatin Analog for TSH-PitNET

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Background: Somatostatin analogs (SSA) are efficiently used to control central hyperthyroidism in patients with thyrotropin-secreting pituitary neuroendocrine tumor (TSH-PitNET). The aim of this study was to describe the frequency of thyrotropin (TSH) deficiency under SSA in patients with TSH-PitNET. *Methods*: We retrospectively recruited patients presenting a central hyperthyroidism due to TSH-PitNET. Inclusion criteria were patients treated in first, second or third line by short or long-acting SSA, with central hyperthyroidism before SSA. Patients treated by radiotherapy or dopamine agonist were excluded. TSH deficiency was defined by either a low FT4 or low FT4 and FT3, associated with non-elevated TSH concentrations during SSA therapy. We analyzed the frequency of TSH deficiency and the characteristics of patients with or without TSH deficiency. *Results:* 46 patients were included in the study. SSA were used as the first-line therapy in 21 of 46 patients (46%). Central hyperthyroidism was controlled in 36 of 46 patients (78%). TSH deficiency appeared in 7 of 46 patients (15%), after a median time of 4 weeks (4–7) after the starting of SSA, and for a median duration of 3 months (2.5–3). The TSH deficiency occurred after 1 to 3 injections of long-acting SSA. There were no differences in terms of clinical and hormonal features and size of adenomas between patients with or without TSH deficiency. *Conclusions:* In patients with central hyperthyroidism due to TSH-PitNET, SSA can induce TSH deficiency. Thyrotropic function should be assessed before each injection of SSA in order to adapt the frequency of injection when control of thyrotoxicosis rather than tumor reduction is purpose of the treatment.

Reproductive Endocrinology

REPRODUCTIVE ENDOCRINOLOGY: REPRODUCTIVE FUNCTION AND DYSFUNCTION ON DEVELOPMENT

Does Androgen Exposure Result in Germline Transmission of PCOS-Like Phenotypes and Can It Be Reversed?

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