recurrence or regrowth and clinical variables in meningioma. Eighty nine patients with no corticosteroid therapy were included. Blood tests and tumor characteristics were collected from medical records. Recurrence-free survival was evaluated using Cox regression and Kaplan-Meier curves. Of the 89 cases, 73 (82%) were grade I and 16 (18%) grade II. The mean age was 53±13.9 years, with higher frequency in women, 2:1 proportion. The most frequent subtypes were meningothelial (40.4%), transitional (23.5%) and atypical (17.9%), 64% with peripheral location and 64% had a size greater than 3 cm. Regarding tumor resection, 49 (55.1%) underwent complete surgery (40 remained with tumor (81.6%) and 9 relapse (18.3%)) and 40 (44.9%) submitted to partial resection surgery (29 remained with persistent lesion (72.5%) and 11 regrowth (25%)). In total, 20 (22.4%) cases of recurrence or regrowth were observed. The median recurrence-regrowth free survival (RFS) was 62 months, 96.1% at 1 year, 67.4% at 3 years and 51.2% at 5 years. In univariate analysis, anemia (p=0.04), neutrophilia (p=0.02) and neutrophilis/lymphocyts ratio (NLR) (p=0.03) were associated with an increased risk of recurrence or regrowth and poor RFS. In multivariate, the interaction between anemia and NLR >4 represented a higher risk of recurrence or regrowth (p=0.003). The preoperative presence of anemia, neutrophilia, and NLR was associated with an increased risk of recurrence or regrowth in meningiomas, emphasizing the importance of preoperative evaluation of these parameters.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL GLUCOSE METABOLISM AND DIABETES

The Expression of TBC1 Domain Family, Member 4 (TBC1D4) in Skeletal Muscles of Insulin-Resistant Mice in Response to Sulforaphane.

Nasser M. Rizk, MD, PHD[1], Amina Saleh, BSC[1], Abdelrahman ElGamal, MD[1], Dina Elsayegh, BSC[1], Isin Cakir, PhD[2], Masoud Ghamari-Langroudi, phD[3].
[1]Qatar University, Doha, Qatar, [2]University of Michigan, Ann Arbor, MI, USA, [3]Vandrbilt University, Nashivelle, TN, USA.

MON-613

The Expression of TBC1 Domain Family, member 4 (TBC1D4) in Skeletal Muscles of Insulin-Resistant Mice in Response to Sulforaphane.

Background: Obesity is commonly accompanied by impaired glucose homeostasis. Decreased glucose transport to the peripheral tissues, mainly skeletal muscle, leads to reduced total glucose disposal and hyperglycemia. TBC1D4 gene is involved in the trafficking of GLUT4 to the outer cell membrane in skeletal muscle. Sulforaphane (SFN) has been suggested as a new potential anti-diabetic compound acting by reducing blood glucose levels through mechanisms not fully understood (1). The aim of this study is to investigate the effects SFN on TBC1D4 and GLUT4 gene expression in skeletal muscles of DIO mice, in order to elucidate the mechanism(s) through which SFN improves glucose homeostasis.

Methodology: C57BL/6 mice (n=20) were fed with a high fat diet (60%) for 16 weeks to generate diet induced obese (DIO) mice with body weights between 45–50 gm. Thereafter, DIO mice received either SFN (5mg/kg BW) (n=10) or vehicle (n=10) as controls daily by intraperitoneal injections for four weeks. Glucose tolerance test (1g/kg BW, IP) and insulin sensitivity test (ITT) were conducted (1 IU insulin/ g BW, IP route) at the beginning and end of the third week of the injection.

At the end of 4 weeks of the injection, samples of blood and skeletal muscles of both hindlimbs were collected. The expression levels of GLUT4 and TBC1D4 genes were analyzed by qRT-PCR. Blood was also used for glucose, adiponectin and insulin measurements.

Results: SFN-treated DIO mice had significantly lower non-fasting blood glucose levels than vehicle-treated mice (194.16 ± 14.12 vs. 147.44 ± 20.31 mg/dL, vehicle vs. SFN, p value=0.0003). Furthermore, GTT results indicate that the blood glucose levels at 120 minutes after glucose infusion in was (199.83±34.53 mg/dl vs. 138.55±221.78 mg/dl) for vehicle vs. SFN with p=0.0011 respectively. ITT showed that SFN treatment did not enhance insulin sensitivity in DIO mice. Additionally, SFN treatment did not significantly change the expression of TBC1D4, and GLUT4 genes in skeletal muscles compared to vehicle treatment (p values >0.05).

Furthermore, SFN treatment did not significantly affect the systemic insulin (1.84±0.74 vs 1.54±0.55 ng/ml, p=0.436), or adiponectin (11.96 ±2.29 vs 14.4±3.33 ug/ml, p=0.551) levels in SFN vs. vehicle-treated DIO mice, respectively.

Conclusion: SFN treatment improves glucose disposal in DIO mice, which is not linked to the gene expression of GLUT4 and TBC1D4 and its mechanism of glucose disposal in skeletal muscles. Furthermore, SFN treatment did not improve insulin level, and the insulin sensitizer hormone adiponectin as potential players for enhancing insulin sensitivity.

1.
Axelsson AS, Tubbs E, Mecham B, Chacko S, Nenonen HA, Tang Y, et al. Sci Transl Med. 2017;9(394).

Thyroid

THYROID DISORDERS CASE REPORTS II

A Rare Case of Lithium-Induced Thyroiditis

Vladimir Lokshin, MD[1], Lina Soni, MD[2], Sujata Shrestha, MBBS[3], Helena Abby Guber, MD[4].
[1]State University of New York Downstate Medical Center, Brooklyn, NY, USA, [2]SUNY Downstate Medical Center, New York, NY, USA, [3]SUNY Downstate Medical Center, Brooklyn, NY, USA, [4]NY Harbor-HCS, Brooklyn Campus, Brooklyn, NY, USA.

SAT-517

Background: Lithium is known to cause both hypo- and rarely hyper- thyroidism. It inhibits release of thyroid hormone and reduces the intrathyroidal iodothyronine/iodothyrosine ratio. Due to direct toxic or immunostimulatory effect, lithium can also cause thyroiditis. Lithium-induced thyroiditis is a rare entity with an incidence rate of about 1.3 cases per 1000 person-years. Given its generally painless and transient nature, symptoms of thyrotoxicosis may erroneously be attributed to an exacerbation of mania.

Clinical Case: We report the case of a 29 y/o man with bipolar disorder on lithium therapy who presented with a 2