Diabetes Mellitus and Glucose Metabolism

DIABETES COMPLICATIONS II

Diabetic Macular Edema Thickness in Asians and Caucasians: Is There a Difference?

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Background: Diabetic macular edema (DME) is a manifestation of diabetic retinopathy (DR) which can result in vision loss. DME affects over 750,000 US adults over the age of 40, costing 33% more in Medicare dollars than those patients without DME. Ethnicity plays a role in the development of DR but there are no studies about DME and HbAlc in Asians versus Caucasians. As Asians are the fastest growing ethnic group in the US and are at an increased risk of developing Type 2 Diabetes Mellitus (T2DM), there is a need for more knowledge about DME compared to Caucasians.

Purpose: Evaluation of diabetic macular edema thickness between Asian and Caucasian Diabetic patients.

Methods: A retrospective chart review (2017-2019) of consecutive diabetic patients were performed at California Retina Clinic. Inclusion criteria: DME thickness was measured by Optical Coherence Tomography (OCT) (Heidelberg Spectralis, Heidelberg, Germany), OCT +/-3 months of their HbA1c lab test, control (C) non-diabetic patients=HbA1c <6.0%; For T2DM patients diagnosed by primary care physician: HbA1c ≥6.0%. Exclusion criteria: Age under 18 or over 90 years. Data analysis: R Project version 3.6.1 (R Foundation for Statistical Computing, Vienna, Austria). Two-way ANOVA was used to determine the difference between the four groups. The post-hoc test, Tukey’s HSD was used for p-values (<0.05).

Results: Of 500 consecutive patients: 80 patients met inclusion criteria: T2DM Asian (ADM), T2DM Caucasian (CDM), C Asian (CA), C Caucasian (CC). n=80 pts, avg age=59.187 years (sd=15.42, range 23-89 years), Female=44, Male=36;

ADM: n=20, avg age=60.90 years (sd=11.22, range 37-79 years), Female=10, Male=10, HbA1c range = 6.7-11.9%, Avg HbA1c = 7.59%, Avg DME=276.28 um (sd=25.93 um, range = 238.5-342 um);

CDM: n=20, avg age=68.05 years (sd=10.54, range 46-87 years), Female=9, Male=11, HbA1c range = 6.0-11.5%, Avg HbA1c = 7.52%, Avg DME=298.68 um (sd=33.84 um, range = 253.5-397.5 um); HbA1c in ADM v CDM: p=0.8(ns)

CA: n=20, avg age=52.85 years (sd=16.81 years, range 34-84 years), Female=10, Male=10, HbA1c range = 4.8-5.5%; Avg DME=267.05 um (sd=21.09 um, range = 230-310 um).

CC: n=20, avg age=62.95 years (sd=13.47 years, range 40-89 years), Female=14, Male=6, HbA1c range = 4.8-5.6%, Avg DME=285.68 um (sd=16.58 um, range = 248.5-312.0 um).

ADM v CDM and DME [F(1, 76)=13.267, p = 0.00049]. Tukey’s HSD post hoc tests were carried out. CDM v ADM
had a significantly higher avg DME: 22.4 um (p=0.031). Body Mass Index (BMI): ADM patients (avg= 25.6) compared to CDM patients (28.5):(p=0.05, t-test).

Conclusion: In this small study, T2DM Asians had less macular edema thickness than T2DM Caucasians with similar HbA1c. T2DM Asian patients had lower Body Mass Index than T2DM Caucasians. Body Mass Index may be an additional factor to consider in the evaluation of diabetic macular edema in Asians and Caucasian Diabetic patients.

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CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES

Hypothalamic Gliosis in Adolescents with Type 1 Diabetes and Disordered Eating Behaviors
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Title: Hypothalamic gliosis in adolescents with type 1 diabetes and disordered eating behaviors

Background:
The hypothalamus and brainstem are thought to be the principal homeostatic brain areas responsible for regulating appetite and weight. Research suggests that inflammation plays a role in the onset and maintenance of eating-related maladaptive behaviors. Hypothalamic inflammation and reactive gliosis mediate disruptions in energy homeostasis, especially with obesity. Data from SEARCH in Youth for Diabetes has demonstrated high prevalence of disordered eating behaviors (DEB) including insulin omission and binge eating, in individuals with type 1 diabetes (T1D). Limited studies have used neuroimaging techniques for investigation of hypothalamic gliosis in individuals with T1D and DEB.

Objectives: To determine the feasibility of assessing hypothalamic gliosis with structural MRI in adolescents with T1D with and without DEB.

Research design and methods:
Adolescents with T1D, aged 13-19, were invited to participate. Participants with current use of medications known to alter appetite were excluded. Participants completed the Diabetes Eating Problem Survey – Revised (DEPS-R). A score ≥ 20 was considered indicative of DEB. Height, weight and waist circumference were obtained, and BMI was calculated. HbA1c was obtained from their prior clinic visit, within 2 months of the study visit. Participants came in fasting for the MRI study visit. Basal insulin (glargine) was administered the night before, and participants on insulin infusion continued with their basal insulin infusion. Participants received rapid-acting insulin prior to the MRI study, and blood glucose was measured before and after the MRI. Medial basal hypothalamic (MBH) gliosis was measured by T2 relaxation time.

Results:
Eight subjects (50% female, mean age 17.8±2.3 years) have completed the study without adverse outcomes. Mean HbA1c was 8.5% (range 7.3-10%). Five subjects screened positive for DEB. There was no significant difference in BMI between DEB and non-DEB groups. In this cohort, females had longer T2 relaxation times in left MBH than males (p=0.035). Compared to non-DEB group, participants with DEB had longer T2 relaxation time in left MBH, adjusted for sex and age (p=0.001). In this initial sample, relationships between MBH T2 relaxation times and glycemic control, BMI or waist circumference did not emerge.

Conclusion:
The study protocol with insulin injection and MRI to study the hypothalamic gliosis in individuals with T1D is feasible. Structural MRI indicated increased T2 relaxation times as a marker of hypothalamic gliosis in participants with DEB. Further studies with larger sample size are crucial to validate these findings and to study specific eating behaviors and their associations with MBH gliosis in individuals with T1D.

Thyroid

THYROID DISORDERS CASE REPORTS III

Discrepant Thyroid Function Tests in Adenocarcinoma-Is This a True Thyroid Disorder?
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Background: Many causes of abnormal thyroid function tests (TFTs) occur that may or may not reflect a true thyroid disorder. The most common include: immune check point inhibitors therapies (ICI) used to treat various types of cancers; biotin supplements, which may interfere with thyroid function test assays; euthyroid sick syndrome; as well as amiodorone therapy for cardiac disorders. Clinical Case: A 67-year old female patient with type 2 diabetes mellitus, taking insulin and oral antihepyglicemic agents, with hyperlipidemia, hypertension and coronary artery disease, who had abnormal TFTs (TSH was 3.7 to 4.9 uIU/ml; ref range 0.27-4.2 uU/mL), and Free T4 was 0.92 to 1.06 ng/dL; ref range 0.55-1.6 ng/dl) prior to the diagnosis of metastatic adenocarcinoma of the lungs. She was initially treated with radiation. TFTs were unchanged. Her CEA was noted to be 129.5 (0-3.0 ng/mL). However, following chemotherapy with Tarceva (Erlotinib) 50 mg po daily, the TSH increased to 7.6 uIU/ml with Free T4 of 3.19 ng/dL. She remained clinically euthyroid. A thyroid ultrasound showed 1 small sub centimeter nodule in each thyroid lobe. The patient later admitted to also taking biotin for an unknown period of time. TSH antibodies and TSI were both negative. Free T4 by dialysis was normal. While still taking Tarceva her TSH was noted to be 2.5 to 3.8 uIU/ml and both Free T4 and Free T3 were elevated and was 6.57 pg/mL;ref range=2.52-4.34 pg/mL). Six months later, the Free T4 decreased to 1.08 ng/dL. Thyroid antibodies and thyroglobulin remain normal. The patient remained clinically euthyroid. Conclusion: It is important to note that several factors can cause abnormal thyroid function tests, such as Immune check point inhibitors therapy, with the exact mechanism for abnormal TFTs unknown, and can also be associated with either Grave’s hyperthyroidism or Hashimoto’s hypothyroidism,as well as other autoimmune endocrine disorders. Biotin, a common supplement, has