

for later further thyroid ablation. In his follow-up visit, three months later, he reported no pain on ambulation. **Discussion:** For each type of thyroid malignancy, several genes have been identified. However, to date, no common gene mutation responsible for the pathogenesis of the different tumor types has been determined. For instance, point mutations of the *RAS* oncogene are found in about 40% of thyroid neoplasms (*N-RAS*, *H-RAS*, and *K-RAS*, in order of decreasing frequency) including both PTC and FTC. No single theory can completely explain the pathogenesis of these tumors in all cases, and so, with the present level of understanding of the disease, a combination of theories must be accepted. Management of collision tumors of the thyroid gland is usually complex owing to the presence of dual pathology in the tumor tissues and given the fact that literature on this condition is scarce. Generally, the treatment needs to be individualized. **Conclusion:** Most likely, a rare phenomenon of simultaneous mutation of different genes can give birth to contemporary different thyroidal neoplasms. **References:** Zhu Z, Gandhi M, Nikiforova MN, et al. Molecular profile and clinical-pathologic features of the follicular variant of papillary thyroid carcinoma. An unusually high prevalence of ras mutations. *Am J Clin Pathol* 2003; 120:71.

Reproductive Endocrinology

CLINICAL STUDIES IN FEMALE REPRODUCTION I

Screening for Gestational Diabetes Mellitus:

Universal or Selective Screening? Screening for Gestational Diabetes Mellitus: Universal or Selective Screening?

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SAT-012

Screening for gestational diabetes mellitus: universal or selective screening?

Introduction:

The presence or absence of risk factors is often employed in screening for Gestational Diabetes Mellitus (GDM). The risk factors for GDM includes previous delivery of macrosomic babies, family history of type 2 diabetes mellitus, previous GDM among others. The impact of selective screening is yet to be fully evaluated in our environment.

Objective

To determine the impact of selective screening on diagnosis of gestational diabetes mellitus

Methods

The study was a prospective open cohort study carried out from 1st March to 30th November 2017 at the Lagos University Teaching Hospital (LUTH), Lagos, Nigeria. Ethical approval was obtained from the Health Research Ethics Committee of Lagos University Teaching Hospital (LUTH) before commencement of the study

All the pregnant women were categorized into either risk group or control group based on the presence or absence of clinical risk factors for GDM. All participant had 75g Oral

Glucose Tolerance test (OGTT) done at 24 to 28 weeks gestation and follow up till delivery.

The data obtained were age, risk factors for GDM, fasting plasma glucose, one-hour post glucose load plasma glucose & two-hour post glucose load plasma glucose. The data were presented as mean, standard deviation, percentages & chi square. The p value ≤ 0.05 was considered significant

Results

Ninety pregnant women were screened for GDM. Forty-four women had risk factors for GDM while 46 were non risk group. Their mean age was 32.6 ± 5 years. The mean age for the risk & non-risk group were similar.

The overall prevalence of GDM using the IADPSG criteria was 23.3%. The percentage of women in the risk group with GDM was 38.6% while those women in the non risk group with GDM was 8.7% which was statistically significant (p value 0.004).

Discussion

The most commonly identified risk factors for GDM in this study were family history of type 2 diabetes mellitus, history of unexplained miscarriage & previous history of delivery of macrosomic babies.

Some women in the non-risk were diagnosed, even though the prevalence was lower than that observed among women with risk factors for GDM. Approximately one in ten women would have been missed if selective screening was employed in this study.

Most of the women in the non-risk group who were diagnosed with GDM were managed with medical nutritional therapy while majority of women in the risk group had insulin therapy.

Conclusion

The findings in our study further supports the idea of universal screening for GDM in order to avoid missed diagnosis.

Keywords: gestational diabetes mellitus, Screening, oral glucose tolerance test

Thyroid

BENIGN THYROID DISEASE AND HEALTH DISPARITIES IN THYROID I

Personalized Treatment Planning for Radioiodine Therapy of Graves' Disease; The Collar Therapy Indicator (CoTI)

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SAT-417

Introduction Since its introduction 80 years ago, the therapeutic I-131 dosage has usually been tailored to individual patient requirements based on the uptake of a tracer radioiodine (RAI) dose. Estimated exposure has typically been extrapolated from the results of activity measurements at one or two time points, e.g., at 4 and 24 hours. We now know that treatment of hyperthyroid Graves disease with these methods lead to a 13–25% rate of failure to cure hyperthyroidism and a 46–80% rate of long-term hypothyroidism in cured patients. There is a need for a much more personalized

approach to RAI dosing based on individual RAI tissue uptake, kinetics. This can be achieved only after including multiple data points during the evaluation of tissue uptake. The Collar Therapy Indicator (CoTI), a device placed in cloth collar around the neck resembling a turtle neck sweater collar with a connecting wire and recording box, has been shown in small feasibility studies to provide data regarding radioiodine exposure that correlates with conventional methods of measuring I-123 and I-131 uptakes after diagnostic dose administration and/or therapy for thyroid disorders. **Methods;** We hypothesized that the device's continuous measurement capability will permit more accurate estimates of radiation exposure to thyroid tissue than conventionally employed methods assessing fractional uptake at one or a few time points. It may also provide information about the extent of variability in the absorbed radiation dose among patients with hyperthyroidism. We performed a feasibility study in a patient with graves' disease to see the difference between tradition methods of I-123 uptake and the CoTI; (1) We compared the conventional quantitative uptake-derived thyroid time activity curve (TAC) as well as the Area Under the Curve (AUC)(based on percent uptake at 6 hour and 24 hour time points) to that obtained using the CoTI.(2) We evaluated the uptake and clearance kinetics of diagnostic I-123 administered.(3) We also evaluated patient experience in using the CoTI device with a survey instrument. **Results;** The CoTI plotted TAC and AUC offered a different approach from the conventional methods of calculation (6 hr and 24 hr % uptake) of I-123 TAC and AUC. The patient reported no difficulty in using the device and the device itself was not inconvenient. **Conclusions;** The calculation of % uptake as well as rate of uptake within the thyroid by CoTI might help us, in achieving a more personalized approach to I-131 RAI dose calculation for treatment of Graves' disease. The preliminary research findings that we have generated will help us investigate different aspects of RAI uptake within the thyroid and will hopefully lead to solutions, for some of the common issues and problems arising out of random dosing of RAI.

Adipose Tissue, Appetite, and Obesity ADIPOSE TISSUE BIOLOGY AND OBESITY

Lack of Adipose-Specific Hexose-6-Phosphate Dehydrogenase Improves Fat Metabolic Phenotype and Increases Insulin Sensitivity in Mice

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SAT-591

Lack of adipose-specific hexose-6-phosphate dehydrogenase improves fat metabolic phenotype and increases insulin sensitivity in mice.

ABSTRACT

Increased glucocorticoids (GCs) production in adipose tissue promotes the development of visceral obesity and the MS. The action of GCs in adipose tissue is tightly regulated by 11 β -hydroxysteroid dehydrogenase type 1 (11 β -HSD1) coupled with hexose-6-phosphate dehydrogenase (H6PDH). However, the phenotypic consequences of reduction of fat-specific GC action caused by inactivation of H6PDH specifically in adipose tissue have not been explored. To better understand the physiological effects of adipose H6PDH, we generated a tissue-specific animal model of adipocyte-specific H6PDH deletion under the control of the murine adipocyte-specific adiponectin promoter (H6PDH^{AcKO} mice). H6PDH^{AcKO} mice exhibited complete absence of H6PDH expression and decreased GC production with reduction of 11 β -HSD1 in adipose tissue. These mice had decreased abdominal fat mass with decreased adipose lipogenic gene expression associated with reduction of their transcription factor C/EBPs mRNA levels. Moreover, H6PDH^{AcKO} mice also decreased adipose lipolysis and reduced plasma FFA levels. In addition, H6PDH^{AcKO} mice decreased fasting glucose levels and increase tolerance to glucose and insulin. These data suggest that H6PDH^{AcKO} mice may provide a good model for study the potential contribution of fat-specific H6PDH inhibition to improve the metabolic phenotype in vivo.

Neuroendocrinology and Pituitary HYPOTHALAMIC-PITUITARY DEVELOPMENT AND FUNCTION

Hypothalamic Pituitary Adrenal Axis Hyperactivity in Db/db Mice

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SAT-282

Background: Patients with diabetes present hypercortisolemia, which may exacerbate not only glycemic control but also diabetic complications and fatty liver (1). Blood steroid hormone levels including corticosterone and aldosterone are high in db/db mice (2), although the mechanism is still unclear. **Objective:** To reveal the mechanism underlying the hypothalamic-pituitary-adrenal (HPA) axis hyperactivity in obese and diabetic condition. **Method:** Total RNA from hypothalamus, pituitary, and adrenal glands in ten-week-old db/db and db/+ mice were extracted and analyzed with DNA microarray and real-time PCR. Experiments were approved by the Institutional Animal Care and Use Committee. **Results:** Hypothalamic Crh mRNA expression was not changed in db/db mice, although Avp expression was 2.8-fold higher than db/+ mice. Pituitary Crhr1 and Pomc expression were higher (3.0- and 7.0-fold, respectively), and Nr3c1 expression was lower (0.7-fold) in db/db mice than db/+ mice. Adrenal Mc2r, Nr5a1, Cyp11b1, and Cyp11b2 were higher