conflict about youths' gender identity was reported by 40.1%, but was not associated with age of accessing care, types of providers seen, length of time accessing care, or age at first appointment. This research will help fill gaps in knowledge for health care providers about youth accessing gender affirming medical care, enhancing gender-affirming care and support for these youth and their parents/families.

Tumor Biology

TUMOR BIOLOGY: GENERAL, TUMORIGENESIS, PROGRESSION, AND METASTASIS

A Multi-Omics Analysis of Advanced Papillary Thyroid Cancer

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SAT-LB25

Molecular profiling of papillary thyroid carcinoma has largely been confined to exome sequencing of non-aggressive cancer.[1,2] Canonical mutations in BRAF and RAS are significantly represented in thyroid tumors, but these mutations have not resulted in diagnostics and therapeutics for advanced disease. To broadly examine the molecular landscape of advanced disease, we conducted a multi-omic analysis of 34 cases of advanced papillary thyroid carcinoma, including patient-matched lymph node metastases, primary tumor, adjacent-normal thyroid and germline. Our genome-wide multi-omic analysis links the regions of activated chromatin with expressed transcripts and proteins, identifying regulatory elements at primary tumor and nodal metastases stages of thyroid cancer progression. Distal regulatory elements putatively upregulate expression of MAPK-pathway genes in both tumors and metastases (36 genes ($p = 0.0057$) in tumors and 76 genes ($p = 0.0011$) in metastases). Furthermore, tumors and metastases harbor accessible chromatin regions that appear to be bound by MAPK transcription factors, FOS and JUN (p-value $<10^{-150}$ for tumors and metastases). This study identifies regulatory elements that mediate MAPK activity in tumors and metastases of advanced papillary thyroid carcinoma and may ultimately lead to diagnostics and therapeutics that utilize advanced-thyroid-cancer-specific epigenetic targets. *References*

• Cancer Genome Atlas Research Network. Integrated genomic characterization of papillary thyroid carcinoma. Cell. 2014
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Diabetes Mellitus and Glucose Metabolism

DIABETES COMPLICATIONS II

Euglycemic Diabetic Ketoacidosis in a Patient on Canagliflozin Presenting With Hypoglycemia

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MON-LB119

*Background:*Sodium glucose co-transporter 2 (SGLT-2) inhibitors are newer class of antihyperglycemics that cause reversible inhibition of the sodium-glucose cotransporters in the renal proximal tubules resulting in increased urinary glucose. Common side effects include yeast and urinary tract infections. The US Food and Drug Administration issued a safety warning pertaining to the development of diabetic ketoacidosis (DKA) with the use of SGLT2 inhibitors. The mechanisms by which SGLT2 inhibitors cause euglycemic DKA are unclear. SGLT2 inhibitors may lead to a decrease in either endogenous or exogenous insulin and an increase in glucagon production.[1] This insulin deficiency or resistance may be mild in Type 2 diabetics, however, preventing the profound spike in blood glucose seen in traditional DKA. Here, we report a case of euglycemic DKA in a patient on Canagliflozin who presented initially with hypoglycemia.
*Clinical case:*A 70 year old female presented with altered mental status for 1 day duration. Her past medical history is significant for type 2 Diabetes Mellitus, being managed on Canagliflozin, Glimepiride and Janumet. One week prior to admission she had lumbar spinal fusion surgery. Since then she has been feeling weak and tired with poor oral intake, but continued to use her medications. Initial laboratory findings showed blood glucose of 68 (70-100 mg/dl) without any acidosis. Her altered mental status was attributed to higher opioid doses which she received prior. Oral hypoglycemic agents have been held. On 2nd day of hospitalization, patient became more lethargic and complained of nausea. Laboratory testing revealed a serum glucose of 250 mg/dL, serum bicarbonate of 13 (21–31 mmol/L), and Anion gap of 25 (3.6–11.0 mmol/L). With the suspicion of DKA, a beta-hydroxy butyrate level was obtained which was elevated at 90.10 (0 – 4.16 mg/dL). Venous blood gas analysis was significant for pH 7.23 (7.31-7.41) and pCO2 – 28 (41-51 mmHg). Urinalysis showed ketosis and glucosuria. Patient was diagnosed as euglycemic diabetic ketoacidosis from Canagliflozin in presence of precipitating factors - stress and poor intake. Patient was treated with insulin drip and intravenous fluids with reduction in anion gap and correction of acidosis within 24hrs. There was a gradual improvement in her mental status. She was discharged on subcutaneous insulin, and all other diabetic medications were stopped.
*Conclusion:*Our case highlights the importance of being vigilant in a patient on Canagliflozin, euglycemic DKA can occur even if they present initially with hypoglycemia and no acidosis.
Reference: 1. Ogawa W, Sakaguchi K. Euglycemic diabetic ketoacidosis induced by SGLT2 inhibitors: possible mechanism and contributing factors. *J Diabetes Investig*. 2016;7(2):135-138.

Thyroid

THYROID NEOPLASIA AND CANCER

Validation of TI-RADS (Thyroid Imaging, Reporting and Data System) Follow-Up Recommendations

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