this is the first case reported of metastatic MCC within a functional adrenal adenoma.

References:
Baek, SH, et al. "Merkel cell carcinoma of the Axilla and Adrenal Gland: A Case Report with Imaging and Pathologic Findings." *Case Reports in Medicine*. Volume 2015, Article ID 931238.
Dongyan, L., S. Kumar. "An exceedingly rare adrenal collision tumor: adrenal adenoma-metastatic breast cancer-myelolipoma." *Journal of Community Hospital Internal Medicine Perspectives*, 2017. Vol. 7, No. 4, 241-244.
Martin, JT, et al. "Metastatic adenocarcinoma within a functioning adrenal adenoma: a case report." *Cases Journal* 2009, 2:7965.

Tumor Biology

TUMOR BIOLOGY: GENERAL, TUMORIGENESIS, PROGRESSION, AND METASTASIS

Glycoprotein NMB (GPNMB) Is Pro-Tumorigenic in TSC2-Null Cancer Cells and Is a Potential Drug Target and Biomarker for Lymphangioleiomyomatosis (LAM).

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SAT-141

Glycoprotein NMB (GPNMB) is Pro-Tumorigenic in TSC2-Null Cancer Cells and is a Potential Drug Target and Biomarker for Lymphangioleiomyomatosis (LAM).

Lymphangioleiomyomatosis (LAM) is an estrogen-sensitive lung disease found almost exclusively in women that is characterized by hyperproliferation of smooth muscle cells forming small tumors, or LAM lesions throughout the lungs of patients. Growth of these tumors leads to progressive loss of pulmonary function, and sometimes subsequent lung transplantation. LAM tumor cells contain mutations in either the *TSC1* or *TSC2* genes, leading to activation of the mTORC1 pathway. In fact, mTOR inhibitors such as sirolimus are commonly used to treat LAM; however, these drugs are not always effective and have significant side effects, suggesting the need for new therapeutic targets. Interestingly, another important feature of LAM cells is that they express melanocytic markers that are normally found in melanocytes or melanoma cells. From RNASeq analysis of a mouse model for LAM that we designed, we discovered significant upregulation of the melanocytic marker Glycoprotein Non-Metastatic Melanoma Protein B (GPNMB), a type I transmembrane protein. GPNMB was not only highly expressed in our mouse model (a uterine specific TSC2-null mouse), it was also expressed in TSC2-null cell lines, and human LAM patient lung samples. In our hands, knocking down GPNMB expression by siRNA directed against *GPNMB* mRNA decreased migration and proliferation in TSC2-null cells. Additionally, we found that GPNMB's large ectodomain is shed by TSC2-null cells and can be detected in the blood of human patients with LAM. Finally, MMP 2 and 9 can be secreted as a result of ectodomain shedding and its interaction with integrins. Accordingly, we did indeed see a decrease in MMP 2/9 expression in TSC2-null cells with reduced GPNMB expression from treatment with siRNA directed against *GPNMB* mRNA. Overall, our results demonstrate the potential importance of GPNMB in LAM tumor progression, and suggest that GPNMB may be a possible LAM biomarker and target for its treatment.

Diabetes Mellitus and Glucose Metabolism

DIABETES TECHNOLOGY

Comparison of the Accuracy and Concordance of 3 CGM Devices vs SMBG During Aerobic Exercise

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Introduction: Real-time continuous glucose monitoring (rt-CGM) and flash glucose monitoring (FGM) devices have become important tools for managing type 1 diabetes. These devices are approved for management decisions in steady-state conditions, however there is a decline in accuracy during aerobic exercise with respect to MARD and lag time.[1] It is possible that newer technologies may be superior to previous devices.

Question: With the newest rtCGM, FGM, and long-term CGM devices, do we continue to see an increase in MARD during continuous aerobic exercise? Is there a difference between glucose readings of the 3 devices when worn simultaneously during exercise?

Design: A single subject with T1DM, experienced in glucose management during exercise, wore 3 devices simultaneously - the DEXCOM G6 (San Diego, CA; rt-CGM1, worn on the abdomen), the Eversense (Germantown, DM; long-term CGM or rt-CGM2, implanted in the left arm), and the Abbott Freestyle Libre 14-day (Chicago, IL; FGM, worn on the right arm). The rt-CGM2 was calibrated using a blood glucose meter (Ascensia Contour Next) which was also used for comparator SMBG. Glucose was recorded 10 minutes before and after exercise and every 10 minutes during a 60 minute run at moderate intensity. 6 exercise sessions were averaged for data analysis. Subject wore an insulin pump and reduced the basal rate by 50% 90 minutes prior to exercise and resumed the basal immediately post-exercise. Carbohydrates were not used within 3 hours prior to exercise but could be consumed during exercise if needed to avoid hypoglycemia.

Results: Glucose value during 60 minutes of exercise dropped from mean of 167 to 114 mg/dL with SMBG, 174 to 115 mg/dL with rt-CGM, 175 to 115 with rt-CGM2, and 150 to 106 mg/dL with FGM. Average measured glucose was 140.0, 145.8, 145.6, and 129.3 mg/dL for SMBG, rt-CGM1, rt-CGM2, and FGM respectively. P-value <0.05 for FGM. MARD (calculated compared to SMBG) for 10 minutes pre-exercise, during exercise, and post-exercise for rt-CGM1 was 5.1%, 11.7%, and 8.6% respectively. For rt-CGM2 MARD was 7.7%, 11.4%, and 10.0% respectively. For FGM, MARD was 12.7%, 5.3%, and 21.3% respectively. Overall MARD was 9.8% for rt-CGM1, 10% for rt-CGM2, and 8.0% for FGM.

Conclusions: Blood glucose values dropped with aerobic exercise with observed lag between CGM and SMBG.