uIU/mL (2.6-24.9), C-peptide 12.6 ng/mL (1.1-4.4), proinsulin 407.6 pmol/L (0. 0-10. 0), beta-hydroxybutyrate <0. 050 mmol/L (0. 02-0.27), insulin antibodies <5. 0 uU/mL, negative sulfonylurea screen, insulin-growth factor-1 (IGF-1) 77 ng/mL (90-278), and insulin-growth factor binding protein three (IGF-BP3) 2133 ug/L (2571-5982). Patient was treated with both diazoxide and hepatic artery embolization to reduce tumor burden. His dose of diazoxide was titrated to maintain normoglycemia. He had mild tumor lysis syndrome that did not require dialysis. His hypoglycemia resolved within a few days of treatment and was discharged from the hospital with a continuous glucose monitor (CGM), diazoxide and hydrocortisone. It is our belief that transformation of the liver lesions to insulinoma was the cause of his hypoglycemia. Conclusion: We recommend that patients with metastases from their neuroendocrine tumor who develop hypoglycemia be evaluated for insulinoma and if found should be treated with diazoxide and if not surgical candidates to consider hepatic artery embolization.

Presentation: No date and time listed

## Abstract citation ID: bvac150.1024

## Neuroendocrinology and Pituitary *ODP315*

## Hypoglycemia: Neuroendocrine Tumor of the Pancreas with Liver Metastasis and Transformation to Insulinoma

Basim Ali, MD, Ritodhi Chatterjee, MD, Rui Chen, MD, Betty La, MD, Son Nguyen, PGCME, Maryam Tetlay, MD, Madhuri Vasudevan, MD, and Dennis Villareal, MD

Background: Neuroendocrine tumors (NETs) are rare, can be found in any part of the body including the gastrointestinal tract, lung, and pancreas and have potential for metastasis. Clinical Case: A 39-year-old male with stage IV high grade neuroendocrine tumor of the pancreas with metastasis to the liver and lymph nodes whose disease progressed despite treatment with systemic therapy who presented with severe hypoglycemia. Diagnosis and management of his hypoglycemia was difficult. For the diagnosis, patient's serum blood glucose did not always correlate with his capillary blood glucose via finger sticks, which lead to a delay in his diagnosis. Also, his diagnosis was not straight forward as he did not respond adequately to the glucagon following a serum glucose of less than 55 mg/dL (normal 70-110 mg/dL) during a diagnostic fast. Once critical labs were obtained, it took days for the results. In the interim, his hypoglycemia was managed with dextrose infusions at 10%, 20% and as high as 50%, frequent meals, and high dose steroids, yet he still had daily symptomatic hypoglycemia. A diagnosis of insulinoma was made with the following critical lab results: insulin 70.4