SUN-284
Background:
Adipsic diabetes insipidus (ADI) is a rare disorder triggered by injury to the hypothalamus impairing thirst osmoreceptors and the antidiuretic hormone syntheses leading to absence of thirst.

We are reporting a rare case of ADI Post astrocytoma resection who presented with a severe hypernatremia postoperatively.

Clinical Case:
A 16-year-old female known to have hypothalamic pilocystic astrocytoma post incomplete resection in 2014.

She presented in 2018 with one week history of sudden onset headache, progressively worsen over time, worst in the morning, there were no relieving factors. On examination she was conscious alert oriented GCS 13/15, Vital signs were normal, fundus exam within normal, pupil equal bilateral reactive to light, there was no focal neurological deficit. Systemic examination was unremarkable. Radiological imaging revealed interval size progression of previously seen suprasella region with extension into the floor of the third ventricle. Investigations: Renal profile & pituitary function showed elevated Prolactin, TSH, normal thyroxine. She was instructed to maintain her fluid intake 1–1.5 L/Day. Systemic examination was unremarkable. BP: 100/65, no postural drop, Pulse: 95, RR: 16, O2 Sat: 98%.

Postoperatively, she developed asymptomatic hypernatremia with inability to sense thirst. On examination she was conscious alert oriented GCS 13/15, Vital signs were normal, fundus exam within normal, pupil equal bilateral reactive to light, there was no focal neurological deficit. Systemic examination was unremarkable. Radiological imaging revealed interval size progression of previously seen suprasella region with extension into the floor of the third ventricle. Investigations: Renal profile & pituitary function showed elevated Prolactin, TSH, normal thyroxine. She was instructed to maintain her fluid intake 1–1.5 L/Day. Systemic examination was unremarkable. BP: 100/65, no postural drop, Pulse: 95, RR: 16, O2 Sat: 98%.

Conclusion:
Astrocytoma rarely linked with development of central diabetes insipidus and even fewer with adipsia. The absence of polydipsia and polyuria can be misleading.

Long-term therapy should consider the benefits of vasoressin and fluid therapy against the risk of brain edema/herniation. Body weight and electrolytes monitoring essential & should be maintained a long with strict fluid intake.

Tumor Biology
ENDOCRINE NEOPLASIA CASE REPORTS I

Malignant Transformation of a Benign Glucagonoma After 13 Years
Mohamed K.M. Shakir, MD, Aqueel Usman, MD, Elizabeth M. Bauer, MD, Thanh Duc Hoang, DO.
Walter Reed National Military Medical Center, Bethesda, MD, USA.

SUN-926
Glucagonoma are rare neuroendocrine tumors arising solely in the pancreas and approximately 25 % of glucagonoma cases start in a benign form. We hereby report a 76-year-old male who was initially diagnosed with asymptomatic benign glucagonoma. Follow-up surveillance 13 years later shows that the tumor has undergone differentiation into a malignant form. Case Report: A 63-year-old man was referred for evaluation of an enhancing lesion (2.7 cm) at the pancreatic tail. He was completely asymptomatic with normal physical exam. Initial labs: normal except for elevated serum glucagon level (206 pg/mL, ref 0–60). A 2-hour oral GTT confirmed the autonomy of glucagon secretion by the tumor. Somatostatin and other tumor markers were normal. PET scan showed abnormal uptake at the distal pancreatic tail, correlating with the CT scan findings. The patient underwent laparoscopic distal pancreatectomy, with removal of a 2.8cm mass which predominantly expressed glucagon. Plasma glucagon level in the peripheral venous blood and intraoperative splenic vein dropped to <50 pg/mL immediately after the surgical resection. Following surgery patient remained completely asymptomatic for the next 13 years with normal blood glucose, glucagon, insulin, chromogranin levels, complete blood count and liver functions. Additionally patient continued to have normal surveillance MRIs of the abdomen. 13 years following removal of pancreatic mass, an elevated glucagon serum level (230 pg/mL) was noted on routine surveillance screening. At this time patient also remained asymptomatic. Abdominal MRI and PET scan revealed a 4-cm pancreatic mass with hepatic metastases. Biopsy of the hepatic lesion confirmed glucagonoma. Patient was treated with Lanreotide which has normalized the serum glucagon levels and the tumor size remained stable for the 12 months of follow up. Discussion: In our patient the glucagon secreting tumor without any classic presenting symptoms was found incidentally and the asymptomatic glucagonoma treated surgically presumably at an earlier stage. It is known that some glucagonomas are associated with serum levels of the peptide in the “physiologically elevated” range, even in the presence of necrolytic migratory erythema. The complete remission without any treatment lasting for more than 13 years confirmed the benign nature of the tumor. It is also reported that glucagonomas less than 2 cm in size has less potential for metastasis. The usual recommendation is to monitor these patients post-resection to a maximum of 10 years although in our patient the malignant nature of the tumor was expressed 13 years after initial resection. The reason for malignant transformation after this prolonged period remains unknown. This case highlights the importance of continuous monitoring neuroendocrine tumors even beyond 10 years after surgery.