to 1/2020). One month later, she presented to the ED with hypotension, anorexia, and fatigue. Biochemical evaluation showed severe DKA. She was diagnosed with ICI-mediated diabetes (low c-peptide, negative autoantibodies) and started on basal/bolus insulin. Her ICI-therapy was stopped. One month later, she was readmitted for hypotension and severe headaches and found to have hyponatremia (131 mmol/L), normokalemia, hyperprolactinemia (29 ng/ml), cortisol 4.5 µg/dl (previous random cortisol a month earlier was 33 µg/dl). Pituitary gland was normal on MRI. She was presumed to have secondary AI due to hypophysitis and started on steroid replacement. A CST performed after 3 months showed ACTH <5 pg/ml and cortisol response of 3.5 to 4.5 µg/dl. Her case was a usual presentation of ICI-mediated secondary AI as it occurred within 1-2 months of stopping ICI therapy.
Conclusion: As we continue to learn about ICI-mediated endocrinopathies, it is imperative to document the variation in timing of presentation. ICI-mediated hypophysitis can present at any time after the initiation of therapy. Given this variation, there is need for routine screening and early treatment of hypophysitis to reduce ED visits and readmission rates.

Neuroendocrinology and Pituitary

NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Interesting and Rare Case of Central Diabetes Insipidus in a Patient With Acute Myeloid Leukemia

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Introduction: Central Diabetes Insipidus (CDI) is the lack of antidiuretic hormone (ADH) leading to impaired urinary concentration and manifests with extreme thirst and excessive urination. Patients unable to drink fluids are at risk of severe dehydration and hypernatremia. CDI can be a rare complication of acute myeloid leukemia (AML). The occurrence of AML with CDI is extremely rare with only few case reports published. This combination of AML with CDI has been reported to be associated with monosomy 7 and inversion (3) (q21q26) and portends an overall a very poor treatment response resulting in poor outcomes.
Case Presentation: 66-year-old female with hypertension, polycythemia vera diagnosed in 2006 with transformation to AML in 2020. FISH studies revealed monosomy 7. Cytogenetic studies showed inv (3)(q21q26.2). Remission induction chemotherapy was initiated. Subsequently, neutropenic fever and sepsis secondary to Clostridium difficile colitis lead to hospitalization. Her sodium (Na) level gradually trended up and reached a peak of 157 mmol/l (range 136-145) and elevated serum osmolality at 311 mOsm/K with low urine osmolality 135 mOsm/K, low urine sodium at 13 and low urine specific gravity 1.006 concerning for CDI. She developed polyuria and received desmopressin (DDAVP) leading to improvements in urine osmolality to 267 mOsm/K, 30 minutes indicating CDI diagnosis. Her Na gradually normalized to 144 mmol/l, urine osmolality improved to 580 mOsm/K and urine specific gravity to 1.025. She is now on DDAVP 0.05 mg oral twice daily and her Na is in normal range. MRI pituitary did not show any evidence of metastatic lesion with intact pituitary bright spot. Her other pituitary hormonal workup was normal except for hypogonadotropic hypogonadism.
Discussion: The pathophysiology of AML and CDI is unclear. Leukemic cells infiltration of the neurohypophysis; thrombosis of small vessels in hypothalamic nuclei and the posterior pituitary; alterations of the neutrophil migration placed on the chromosome 7 leading to glycoprotein gp 130 production, a cell surface marker on granulocytes are some of hypothesis suggested. CDI has a variable onset in the course of myeloid malignancies. MRI pituitary can be normal in most of the cases.

Neuroendocrinology and Pituitary

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Intrasellar Cavernous Hemangioma, a Rare Condition Causing Both Radiological And Clinical Difficulties- a Case Report

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Introduction: Intrasellar cavernous hemangiomas constitute an extremely rare group of findings in endocrinological practice. Diagnosis remains challenging due to non-characteristic symptoms and neuroradiological features which may resemble those of pituitary adenomas.
Presentation of the Case: We present a case of female born in 1941, diagnosed with a hemangioma cavernosum located in the *Sella turcica*. In 2004 our patient presented with uncharacteristic symptoms: syncopes and chronic headaches. Computed tomography (CT) of the head revealed an intrasellar hyperdense tumor mass with radiological features suggesting a pituitary adenoma. In 2005 the patient underwent transcranial resection of the sellar mass, with subsequent oculomotor nerve palsy. In histopathological examination, diagnosis of cavernous hemangioma was determined. Between 2005 and 2020 patient was asymptomatic, with multiple follow-up head MRIs scans, showing gradual progression in size of the intrasellar tumor. The patient was consulted by a neurosurgeon, with no direct indications for surgical approach found. Furthermore, due to suprasellar expansion into the direct proximity of the

right optic nerve, the patient was disqualified for Gamma-Knife radiotherapy.
The last MRI of the hypothalamic-pituitary area in 2020 revealed a polycyclic, homogeneous, 33x31x29 mm mass, filling in the space of the Sella turcica, with strong enhancement after contrast administration. Invasion of the surrounding structures, including the clivus, right cavernous sinus and right trigeminal cave were described. Bilaterally, internal carotid arteries and right optic nerve adhered directly to the lesion. Pituitary gland was compressed by the tumor mass. In July 2020, in order to verify the ambiguous radiological and clinical characteristics of the lesion including tumor regrowth and its invasiveness, a transsphenoidal partial resection was performed. Tissue samples were collected for the histopathological examination, which confirmed the initial diagnosis of cavernous hemangioma originating from the cavernous sinus. During a multidisciplinary tumor board, having taken into consideration relatively stable clinical condition and high risk possible surgical complications, the patient was currently disqualified from neurosurgical re-operation nor radiotherapy. Surprisingly, during whole follow up, patients pituitary function remained unimpaired. A watchful waiting approach, with radiological and endocrinological follow up were scheduled. **Conclusion:** To date, only few cases of intrasellar cavernous hemangiomas have been reported. Intrasellar hemangiomas may originate from the vascular tissue of the cavernous sinus. Surgical removal remains the recommended treatment modality, but radiosurgery could be a therapeutic option as well. Stable patients with no clinical symptoms may remain in observation.

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NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Is It the COVID-19 and Untreated Panhypopituitarism a Deadly Combination: A Case Report?

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Background: The possible long-term consequences of coronavirus disease (COVID-19) on the endocrine system are still not known. With the number of cases rising we have reported the first possible implications of COVID-19 on panhypopituitarism in Bosnia and Herzegovina. **Clinical Case:** A 59-year-old man was referred to the Endocrinology Counseling Center on 12/10/2020. For 15 years he did not take thyroid and adrenal replacement therapy. As a child, he was treated for short stature, as well as his two sisters. He had signs and symptoms of myxedema. He was urgently hospitalized to the Department of Endocrinology. His condition has worsened in the last month, more intensely in the last 10 days. He complained of general weakness, malaise, drowsiness, shortness of breath, fatigue, constipation, swelling of the lower legs. The patient was enormously adipose and moved harder with help of a walking stick. The thyroid gland appeared smaller on palpation. Laboratory investigations showed low and borderline levels of the following hormones TSH 0.590 (0.3-4.2 mU/L), FT4 1.44 (12.0-22.0 pmol/L), FT3 0.858 (3.1-6.8 pmol/L), ACTH at 8 am 2.59, ACTH at 16 pm 8.44 (7.20-63.3 pg/ml), GH 0.090 (0.0-14.0), FSH <0.7 (1.6-9.7 IU/L), LH <0.7 (0.7-7.8 IU/L), prolactin <30.8 (78-380 uIU/ml), testosterone 1.01 (above 50 years 2.5-21.6 nmol/l), cortisol at 8 am 557, cortisol at 16 pm 674, cortisol at 11 pm 674 (morning: 123-626 nmol/L, afternoon 46-389 nmol/L), DHS 1.87 (2.17-11.7 nmol/L). On 12/10/2020 he tested negative for SARS-CoV-2 infection. On 14/10/2020 his condition worsened. He was somnolent, and even though he was admitted to diuretic therapy, he had decreased urine output. His D-dimer was 3.93 (0-0.055 mcg/mL). CT findings of thoracic organs described cardiomegaly, atherosclerotic altered thoracic aorta, and changes in the pulmonary parenchyma on both sides. On 16/10/2020 the patient was transferred to the cardiology department. SARS-CoV-2 test was repeated, and it was positive. The patient exited due to pulmonary arrest on 17/10/2020. **Conclusion:** There are several possible mechanisms that may describe that COVID-19 infection exacerbates the symptoms of myxedema and panhypopituitarism, and consequently leads the patient to severe acute respiratory failure.

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NEUROENDOCRINOLOGY AND PITUITARY CASE REPORTS

Isolated Central Adrenal Insufficiency Due to Nivolumab: A Case Report

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Background: Immune checkpoint inhibitors are immunomodulatory antibodies directed against programmed cell death 1 (PD-1), Nivolumab, or cytotoxic T-lymphocyte antigen-4 (CTLA-4) Ipilimumab. They have improved outcomes in patients with advanced cancers including renal cell carcinoma, non-small cell lung cancer and melanoma. Several immune related adverse events (irAEs) have been recognized with use of immune checkpoint inhibitors, including those involving the endocrine system. We present a case of a patient presenting with isolated central adrenal insufficiency in the context of Nivolumab use. **Clinical Case:** Our patient is a 54-year old man with pre-existing primary hypothyroidism and metastatic renal cell carcinoma treated with Nivolumab. After receiving a total of 14 cycles of Nivolumab, he presented to the Emergency room with his sister, who found him confused and lethargic. On presentation, he was found to be hypoglycemic (random glucose was 2.2 mmol/L). On physical examination, his vital signs were stable and he appeared euvolemic. He was disoriented without focal neurological deficits. Initial blood work revealed sodium 134 mmol/L, (Normal 135-145mmol/L), potassium 4.5 mmol/L (Normal 3.5-5 mmol/L), and TSH being 12.6 mIU/L (Normal 0.4-4 mIU/L). He was resuscitated with IV Dextrose 50% bolus then admitted to hospital and kept on an IV dextrose infusion. While his glucose improved, he was found to have hyponatremia, and confusion persisted. His nadir sodium was 116 mmol/L without seizures or loss of consciousness and required treatment with hypertonic saline. Giving