Treatment aimed at normalizing cortisol levels was initiated, and glycemic control was reassessed post-treatment.
Results
A strong positive correlation was found between baseline cortisol and fasting blood glucose ($r=0.68$, $P<0.01$), as well as HbA1c ($r=0.72$, $P<0.01$). After treatment, patients who achieved cortisol suppression showed significant improvements in both fasting blood glucose (mean decrease of 23%) and HbA1c (mean decrease of 0.9%).
Discussion
This study confirms that cortisol excess significantly impairs glycemic control in CS patients. The correlation between cortisol levels and both fasting blood glucose and HbA1c suggests that controlling cortisol levels is a key strategy for managing diabetes in this population.
Conclusion
The findings highlight the importance of cortisol suppression in improving glycemic control in CS patients. Monitoring cortisol levels may provide valuable insights into the management of diabetes in this context.

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EP144

JOINT1513

Adrenal ganglioneuroma: a rare mass and diagnostic challenge

Mohamed Khairi Arous[1], Kouloud Boujelben[1], Mouna Elleuch[1], Yassmine Abdelkafi[1], Faten Haj Kacem Akid[1], Fatma Mnif[1], Nadia Charfi[1], Mouna Mnif[1], Mohamed Abid[1], Dhoha Ben Salah[1] & Nabila Rekik Majdoub[1]
[1]Hedi Chaker University Hospital, Department of Endocrinology, Sfax, Tunisia

Introduction
Adrenal ganglioneuroma (AG) is a rare tumor comprising less than 5% of all adrenal masses. Its morphological and radiological characteristics are nonspecific, which can complicate the diagnostic process. We report the case of an AG in a 16-year-old girl.
Case Report
A patient with no medical history was referred for evaluation of an adrenal mass. She reported paroxysmal lumbar pain over the past 4 years. Clinical examination was unremarkable, laboratory tests were notable only for iron deficiency anemia. Computed tomography revealed a right adrenal mass extending across the midline, measuring 83×39×64mm, with a density of 29HU and negative absolute and relative washout. Hormonal exploration indicated that the mass was non-functioning. Magnetic resonance imaging demonstrated close conncections with the inferior vena cava, the portal trunk, the aorta, and the liver. FDG-PET scan showed a mildly hypermetabolic adrenal mass with no other pathological uptake. The pathological study concluded an AG. The patient underwent surgical resection of the mass with a straightforward postoperative course.
Discussion and conclusion
Ganglioneuroma is a benign neuroblastic tumor, with adrenal localization accounting for approximately 21% of cases. The radiological characteristics are nonspecific and can be confused with adrenal cortical carcinoma and pheochromocytoma, thus contraindicating biopsy in this context. It is often sporadic, although associations with ROHHAD syndrome, MEN2A, and Turner syndrome have been reported. Management is based on surgical resection; AG is characterized by close vascular conncetions, which complicates its surgical approach. The prognosis after surgery is favorable, and no adjuvant treatment is required.

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EP145

JOINT742

Familial form of adrenoleukodystrophy with late diagnosis of chronic adrenal insufficiency (clinical case)

Daria Danilenko[1], Natalia Likharad[1], Tatsiana Mokhort[2] & Alena Mokhort[2]
[1]1 City Clinical Hospital, Minsk, Belarus; [2]Belarusian State medical University, Endocrinology, Minsk, Belarus

Introduction
Adrenoleukodystrophy (ADL) is a rare genetic disease characterized by abnormal metabolism of very long-chain fatty acids due to mutations in the ABCD1 gene, which leads to their accumulation in the central and peripheral nervous system, adrenal cortex and gonads. The disease has a variable clinical spectrum and prognosis.
Results
Patient U., 41 y.o., with complaints of severe general weakness, dizziness, decreased blood pressure, dyspepsia, had chronic adrenal insufficiency for the first time. ADL was established at the age of 6 (family history: my brother has ADL with severe myeloneuropathy, my cousin has cerebral ADL). Since the age of 15, he had not received special treatment, he was constantly worried about weakness, and he added salt to his food. The deterioration of the condition, provoked by errors in the diet, was accompanied by vomiting, hypotension, and progressive weakness. Seeking medical help led to the diagnosis of pancreatitis and relief from infusion therapy during the day. The condition worsened after a day - BMI - 16 kg/ m^2, skin with slight hyperpigmentation, muscles are hypotrophic, blood pressure 80-50 mm Hg. Facial hair is sparse. Laboratory data: hyponatremia (125.68 mmol/l), hyperkalemia (6.71 mmol/l), daily cortisol profile (after administration of glucocorticoids for emergency indications) 185 - 107,8 - 64,1 nmol/l (79-536 nmol/l), ACTH from 01/15/2025: at 8.00 - 1312 pg/ml (7.2 - 63.3 pg/ml), testosterone -2.4 nmol/l (8.64 – 29 nmol/l) with an increase in LH and FSH levels. The diagnosis: primary adrenal insufficiency, hopogonadism on the background of adrenoleukodystrophy with minimal manifestations of myodoneuropathy. Parenteral glucocorticoid therapy was initiated from 300 mg/day with infusion therapy with dose reduction and transfer to oral (hydrocortisone 20 mg /day in the morning, fludrocortisone 0.1 mg in the afternoon). The patient's condition improved, weakness was relieved, blood pressure was 110/70 mmHg, hyperpigmentation decreased, and electrolyte levels returned to normal.
Conclusion
In addition to neurological symptoms, ADL is accompanied by the development of adrenal insufficiency and hypogonadism, which can develop at any age.

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EP146

JOINT1581

A challenging diagnosis in a patient with primary adrenal insufficiency

Livia Celia Lasca[1], Aurora Alina Să?ianu[2] & Alina Cristina Silaghi[3]
[1]Cuore Medical Clinic, Cluj-Napoca, Romania; [2]Medena Clinic, Oradea, Romania; [3]Endocrinology Department, County Clinic Emergency Hospital, Cluj-Napoca, Romania, Cluj-Napoca, Romania

X-linked adrenoleukodystrophy (X-ALD) is a rare, genetically determined metabolic disease with strong clinical heterogeneity. It affects approximately 1/20,000 Caucasian men. Adrenomyeloneuropathy (AMN) is a variant type of X-ALD that usually occurs in adult males and primarily affects the spinal cord, often presenting as progressive spastic paralysis. We present the case of a 43-year-old man, with no family history, diagnosed in adolescence with primary adrenal insufficiency and treated with Prednisone for a relatively short duration of time (six years). Years later, he was presented to the emergency room with nausea and intractable vomiting. Upon examination, his blood pressure was 90/54 mmHg, heart rate 100 beats/min, weight 40 kg, height 165 cm. He had dry mucous membranes and hyperpigmented skin. The neurological examination was unremarkable. Cardiopulmonary examination showed no abnormalities; the abdomen was without alterations; the meningeal tests were negative. Further testing revealed hyponatremia 130 mM/l (135-145), hyperkalemia 5.6 mM/l (3.6-5.5), while routine blood tests for liver function, kidney function, lipids, blood glucose, CRP, urine, and stool routine tests demonstrated no significant abnormalities. Normal thyroid function, baseline cortisol 12 µg/dl (5-25), baseline adrenocorticotropin >2.000 pg/ml (7.2–63.4), renin >630 µUI/ml (2.8-39.9), total testosterone was 2.6 nM/l (4.16-35.36), luteinizing hormone 24.9 UI/l (2.6-6.8), follicle-stimulating hormone 9.79 UI/l (1.5-19.4). After the diagnosis of primary adrenal insufficiency, he started treatment with corticosteroids and mineralocorticoids. Three months later, he presented with rapid, severe neurological deterioration, including apathy, dysarthria, spastic paraplegia, extensive muscle atrophy, urinary, and fecal incontinence. A brain MRI scan revealed active demyelination, high bilateral symmetrical signal intensity of the white matter in T2 and Flair within the corpus callosum, corticospinal tracts at the level of the brainstem, and middle cerebellar peduncles. Spectrographic recordings showed neuronal destruction. No cervical medullary atrophy on MRI imaging of the cervical cord. Additional laboratory findings: Hepatitis B, syphilis, Borelli, and HIV tests were all negative. The determination of very long-chain fatty acids showed a high level in serum. Molecular analysis of the ABCD1 gene reported hemizygous missense pathogenic mutation c.1252C>T, p.(Arg418Trp). After the diagnosis of AMN, he continued with supplementation for his adrenal insufficiency, developed sepsis two months later due to a urinary infection, and died due to complications. This case underscores the importance of considering AMN in patients with primary adrenal insufficiency, particularly when there is neurological decline. Early identification in such cases could facilitate timely interventions that may prevent or delay disease progression.

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EP147

JOINT2589

Case of spontaneous adrenal tumour infarction resembling pheochromocytoma

Olga Wajtryt[1], Elwira Bakuła-Zalewska[2], Dorota Łącka-Lipińska[1], Aleksandra Budrewicz[1], Piotr Góralski[1] & Marek Dedecjus[1]
[1]Maria Sklodowska-Curie National Research Institute of Oncology, Department of Endocrine Oncology and Nuclear and Nuclear Medicine,

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