Results
Significant changes were found in BMI z-scores and the rates of OWOB from the time of surgery (34.2%), to 1, 2, 3, and 5 postoperative years (48.2%, 43.7%, 39.1%, and 36.3%, respectively). In OWOB group at the time of surgery, BMI z-scores decreased significantly over a 5-year period ($P < 0.001$). In a linear mixed model, significant difference was found in BMI trajectory over a 5-year period between age groups at surgery (below or above 8.0 years of age) and between HI groups ($P < 0.001$ for all). BMI z-scores over a 5-year period are greater in higher HI compared to lower HI group [Puget grade (2 vs. 0-1, $P < 0.01$) and SNUH grade (posterior vs. anterior and middle, $P < 0.01$)]. No significant differences were observed based on surgical approach, timing of GH initiation, or lifestyle education interventions. When multivariate-adjusted model was constructed including age at diagnosis, initial BMI category and each HI grade, not only age at surgery and initial OWOB (both $P < 0.01$) but also Puget grade ($P = 0.001$) and SNUH grade ($P = 0.007$) were significant factors for OWOB at 5 years postoperatively.
Conclusions
While HI was common in childhood-onset CRP patients, with one third of patients classified as OWOB at diagnosis, the rates of OWOB increased until the first postoperative year and then showed a decreasing trend over a 5-year period. Age at diagnosis, initial BMI category, and high HI grade were independent predictors for postoperative 5 year OWOB. Children who are older ($\geq$ 8.0 years of age), severely damaged hypothalamus, and obese at diagnosis are at high risk of being obese 5 years after surgery.

DOI: 10.1530/endoabs.110.EP1074

EP1075

JOINT2316

Cannulated prolactin test: a diagnostic approach to moderate hyperprolactinemia

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The aim was to determine the effectiveness of cannulated prolactin test in patients of reproductive age with moderate hyperprolactinemia (HP) and to analyze the frequency of specific and nonspecific complaints.
Materials and Methods
We examined 272 patients, 195w, 77m; age 30.2±9.9 yrs. After cannula insertion, prolactin was measured at 0(PRL0), 60(PRL1), and 120 minutes (PRL2). The results of the cannulated prolactin test (CPT) were considered positive- with HP remaining in all three samples (PRL0, PRL1, PRL2), negative -if HP was at PRL0 and/or PRL1 and without HP in all samples of the test. HP was diagnosed in accordance with the recommendations of the Endocrine Society: >20 ng/ml in men and >25 ng/ml in women. Mild HP is defined in men as a serum prolactin concentration between 19.5 and 100 ng/ml and in women 26.6 and 100 ng/ml.
Results
Median PRL0 was 30.1(21.2-41.2) ng/ml, median PRL1 24.8 (17.5-34.7) ng/ml and median PRL 2 ng/ml 19.5 (13.66-28.9). Approximately 1/3 (30.5%) of the patients reached a normal PRL0. Positive CPT was detected as pathological HP in 37.5% patients. Negative CPT was detected as stress-induced HP in 62.5 % patients. Determination of PRL0 with reference to pathological HP result in a high specificity and moderate sensitivity (sensitivity 54.1%, specificity 100.0%, positive predictive value (PPV) 100.0%, and negative predictive value (NPV) 51.8%, accuracy 69.3%). Determination of PRL1 with reference to pathological HP result in a high specificity and moderate sensitivity (sensitivity 67.1%, specificity 100.0%, positive predictive value (PPV) 100.0%, and negative predictive value (NPV) 71.76%, accuracy 82.1%). 216 patients (73.8 % of women and 79.2 % of men) had complaints, of them patients 161 (57.6 % women and 61 % men) had specific to HP (galactorrhea, menstrual disorders, reduced libido, infertility, gynecomastia, breast pain), 80 patients (25.3 % of women and 30 of men) had non-specific complaints (weight gain, acne, headache, dizziness, scrotum pain, fatigue). The main presenting symptom was gynecomastia in 40.3% of man, and menstrual irregularities in 36.2% of the women.
Conclusion
The cannulated prolactin test was useful in excluding pathological HP, stress-induced HP (negative CPT) was determine in 62.5% of patients. Patients with stress-induced HP (negative CPT) and patients with pathological HP (positive CPT) had no statistically significant differences in age and frequency of occurrence of specific and non-specific complaints and symptoms for HP. Determination of PRL in CPT with reference to pathological HP result in a moderate sensitivity and high specificity.

DOI: 10.1530/endoabs.110.EP1075

EP1076

JOINT3034

Celebral salt wasting syndrome in a patient with known central diabetes insipidus on the background of mcap overgrowth syndrome

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Introduction
Salt-wasting syndrome constitutes a significant disorder of sodium and water homeostasis, commonly observed in individuals with intracranial pathology, such as central nervous system infections, cerebral ischemia, elevated intracranial pressure, or following neurosurgical procedures. Central diabetes insipidus is another disorder affecting sodium homeostasis and is associated with these conditions. The coexistence of these two disorders poses a substantial challenge for pediatric endocrinologists, particularly in critically ill patients.
Aim/Methods
We present the case of a patient with MCAP overgrowth syndrome who developed salt-wasting syndrome on the background of pre-existing central diabetes insipidus.
Results
The patient had a known history of MCAP syndrome (megalencephaly, macrocephaly, polymicrogyria), with genetic testing revealing a mutation in the PIK3CA gene. At 13 months of age, the patient underwent ventriculoperitoneal shunt placement. Postoperatively, he developed central adrenal insufficiency, central hypothyroidism, and central diabetes insipidus, and treatment with hydrocortisone, levothyroxine, and desmopressin was initiated. Six months later, due to Chiari type 1 malformation, a decompressive laminectomy at the A1 level was performed. At 20 months of age, the child presented with fever of central origin, and brain CT revealed the presence of subdural hygromas. The patient was treated with prednisolone. Laboratory findings demonstrated hyponatremia, mildly elevated urea, low urine specific gravity (SG = 1001), and natriuresis (urinary sodium = 49 mmol/L). The diagnosis pointed towards salt-wasting syndrome, and treatment included oral sodium supplementation and fludrocortisone.
Conclusions
Salt-wasting syndrome is an important cause of hyponatremia and clinical dehydration, particularly in the setting of structural and functional abnormalities of the central nervous system. It must be differentiated from syndrome of inappropriate antidiuretic hormone secretion (SIADH). The pathophysiology of salt-wasting syndrome involves both sympathetic nervous system dysfunction and the secretion of natriuretic peptides in response to brain pathology. The coexistence of salt-wasting syndrome with central diabetes insipidus is rarely reported in pediatric populations. Nevertheless, progressive central nervous system damage or repeated neurosurgical procedures are risk factors that warrant heightened vigilance by treating physicians.

DOI: 10.1530/endoabs.110.EP1076

EP1077

JOINT2458

Challenging management of electrolyte imbalances in neurosurgical patient: cerebral salt wasting case report

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Introduction
Electrolyte disorders are common in central nervous system (CNS) diseases, including subarachnoid hemorrhage, traumatic brain injury, cerebral tumors, infections and postoperative neurosurgical setting. These conditions can be associated with both the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and to cerebral salt wasting (CSW). Hypothalamic-neurohypophyseal system affections instead, can lead to central diabetes insipidus (CDI). CSW is characterized by hyponatremia and hypovolemia due to excessive sodium loss in urine. SIADH presents as euvolemic hyponatremia due to inappropriate ADH secretion. CDI is primarily characterized by polyuria due to insufficient ADH. The table below summarizes the key differences among these

Endocrine Abstracts

May 2025 Volume 110
ISSN 1479-6848 (online)

Joint Congress of the European Society for Paediatric Endocrinology (ESPE) and the European Society of Endocrinology (ESE) 2025: Connecting Endocrinology Across the Life Course

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