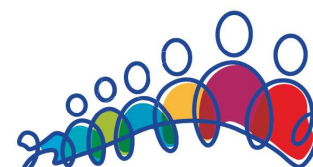


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Endocrinology Across the Life Course

10–13 May 2025, Copenhagen, Denmark



Connecting Endocrinology
Across the Life Course

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ESPE

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Adrenal and Cardiovascular Endocrinology

EP1

JOINT86

A case of mistaken identity: hypoadrenalism and the cirrhotic paradox

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Introduction

Persistent hypotension and hyponatremia are common clinical features in patients with cirrhosis, for which an endocrine consult is not uncommon. Additionally, hypoalbuminemia is a common finding in cirrhosis, with reductions in total hormone assay levels, including cortisol, which leads to a false interpretation of hypocortisolemia. Whether or not there is truly an increased risk (or diagnosis) of hypoadrenalism in patients with underlying cirrhosis remains unknown.

Objectives

The primary objective of this study is to evaluate if cirrhosis is associated with a greater risk for adrenal insufficiency. Secondary objectives include comparing serum levels of albumin, total cortisol, and free cortisol between patients with (and without) cirrhosis, to assess for significant differences.

Methods

The study was performed as a retrospective cohort study, with data collated from TriNetX Global Collaborative Network, providing de-identified patient information from 143 healthcare organizations worldwide. Two cohorts were assessed in this study: Group A (patients with cirrhosis who have had a total cortisol measurement, $n = 43,786$) and Group B (patients without cirrhosis who have had total cortisol measurements, $n = 1,249,256$). Propensity score matching was employed to allow for balancing between the cohorts with $n = 53,220$; this was achieved by controlling for age, race, gender, body mass index, A1c, alcohol use, systemic corticosteroid use, and underlying inflammatory diseases of the liver.

Results

The results of this study noted no greater risk for primary, secondary, tertiary or drug-induced hypoadrenalism in patients with cirrhosis compared to those without, but rather a statistically significant (but likely clinically negligible) reduction in risk (Relative Risk 0.929, 95% CI: 0.872-0.99, $P = 0.0239$). Secondary outcomes noted a significantly lower mean albumin level in Group A (3.171 vs 3.58, $P < 0.0001$) but higher mean total cortisol (14.06 vs 12.188, $P < 0.0001$). There was no significant difference regarding free cortisol between both cohorts ($P = 0.9822$).

Conclusion

This study suggests that cirrhosis does not confer a greater risk for adrenal insufficiency (of any cause). As expected, patients with cirrhosis did demonstrate hypoalbuminemia, however, they appeared to overall exhibit higher mean total cortisol levels (without a significant difference in free cortisol). These findings support a cautious approach to adrenal function testing in patients with cirrhosis, and for reserving the diagnosis of adrenal insufficiency when there is true clinical suspicion.

DOI: 10.1530/endoabs.110.EP1

EP2

JOINT1339

Effects of switching from conventional glucocorticoid therapy to modified-release hydrocortisone (MR-HC) on the steroid metabolome in patients with 21-hydroxylase deficiency

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Background

New hydrocortisone formulations with modified release are increasingly used in treating patients with congenital adrenal hyperplasia (CAH) due to 21-hydroxylase deficiency (21OHD). The goal is better androgen control while reducing overall glucocorticoid exposure through more physiological hydrocortisone release and replacement. However, the effects of switching from conventional glucocorticoid therapy to modified-release hydrocortisone (MR-HC) on steroid metabolism remain uninvestigated

Methods

The steroid metabolome in 24-h urine samples was analyzed in 22 patients with classic CAH due to 21OHD (10 women, 12 men). Before the switch, 10 patients received conventional hydrocortisone, 9 prednisolone, and 3 combination therapy. The switch to MR-HC was performed using equivalent dose conversion (prednisolone conversion factor = 5). Urine samples were collected before and six months after the switch and analyzed using gas chromatography-mass spectrometry (GC-MS).

Results

Based on serum androstenedione and 17-OHP levels, three patients were classified as not well controlled at baseline. After switching, one patient transitioned from well-controlled to not well-controlled, and another showed the opposite trend. Median hydrocortisone-equivalent dosages were 25 mg (IQR 24.375–35.625) before and 27.5 mg (IQR 20–35) after the switch to MR-HC ($P = 0.109$). The switch led to a trend toward increased major cortisol metabolites (5 α -THF + THF + THE, $P = 0.093$) and overall urinary cortisol metabolites (5 α -THF + THF + THE + a-C + b-C + a-Cl + b-Cl; $P = 0.059$) in those previously on HC. No absolute change in 17-OHP metabolites (Po-5 β 3 α , Po-5 α 3 α , PT, 11-OH-P) or significant change in overall 11-oxygenated androgens was observed. In women, 11-deoxygenated androgens showed a declining trend ($P = 0.071$), leading to a significant increase in the 11-oxygenated (11OH-An + 11O-An + 11OH-Et) to 11-deoxygenated androgen ratio (An + Et + A5-3 β , 17 α + A5-3 β , 17 β + DHEA + 16 α -OH-DHEA + A5T-16 α) from 0.64 (IQR 0.24–2.10) to 2.75 (IQR 1.67–5.75, $P = 0.007$), indicating increased adrenal suppression. In men, no significant change in 11-oxygenated or deoxygenated androgens was found. However, individually, significant PT reductions (6790 vs. 2982 μ g/d) were seen in 14/22 patients, 11OH-An reductions (828 vs. 426 μ g/d) in 12/22, and An reductions (823 vs. 433 μ g/d) in 12/22.

Conclusion

While overall glucocorticoid exposure remained comparable, trends toward increased urinary cortisol metabolites suggest a shift in cortisol metabolism, possibly reflecting optimised cortisol availability resulting in increased adrenal androgen suppression. Individual patients of both sexes may therefore benefit from a switch to modified-release hydrocortisone.

DOI: 10.1530/endoabs.110.EP2

EP3

JOINT3739

Echocardiographic evaluation of functional and morphological alterations in obesity: a comparison between obese individuals with and without Metabolic Syndrome

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Background

Obesity has been identified as an independent risk factor for heart failure. The risk of heart failure is dependent on body mass index (BMI).

Aim

This study aimed to examine the effects of obesity on myocardial function and morphology, and to compare these alterations in obese individuals with and without metabolic syndrome (MetS +/- group).

Methods

A total 125 subjects with a BMI more than 25 kg/m² underwent metabolic and clinical evaluation. An evaluation of conventional echocardiographic parameters and cardiac deformation by 2D speckle tracking echocardiography was conducted. The mean age was 45.0 \pm 9.6 years (female: 58.7%), and the average BMI was 35.01 \pm 6.53. In 74% of the subjects, the duration of overweight/obesity was over 10 years. Metabolic syndrome was diagnosed in 54 patients (70%). Two dimensional echocardiographic evaluation showed that the MetS⁺ group had a larger LA maximal volume and a LA volume indexed for body height than the MetS⁻ group, but the differences were not statistically significant ($P = 0.068$, $P = 0.098$, respectively). The MetS⁺ group had significantly lower LA ejection fraction compared to the MetS⁻ group (LAEF% = 46.89 vs 50.13, $P = 0.03$). Regarding the strain analysis, it was also found that the MetS⁺ group had significantly lower values for the peak longitudinal deformation of LA in the reservoir phase (PALS) ($P = 0.008$). The LV mass