Endocrine Abstracts

May 2024 Volume 99
ISSN 1479-6848 (online)

26th European Congress of Endocrinology 2024

11–14 May 2024, Stockholm, Sweden

ECE 2024
26th European Congress of Endocrinology

published by
bioscientifica

Online version available at
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Volume 99
May 2024

26th European Congress of Endocrinology 2024

European Society of Endocrinology

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Objective
This study aimed to determine possible covariates regulating serum CD5L and to test its potential suitability as additional TH biomarker during pregnancy.
Subjects and Methods
A sandwich ELISA for serum CD5L was established using newly raised antibodies. Circadian effects and the impact of liver disease on serum CD5L concentrations were assessed. Serum samples from pregnant women with well-characterized TH and trace element status were analyzed, and CD5L concentrations were correlated with other indicators of TH status including TSH, fT4, fT3, copper and selenium concentrations.
Results
The new quantitative assay for CD5L showed high accuracy. Serum CD5L was stable in dilution and refreezing experiments and did not show strong circadian variance or dependency on liver disease. In serum of pregnant women, CD5L correlated positively to fT3, but not to fT4 or TSH. Significant positive correlations of CD5L were observed with serum levels of the TH-responsive trace elements selenium and copper.
Conclusion
The data support the potential suitability of serum CD5L as an additional marker of TH status, with potential value for pregnancy and thyroid disease. This notion needs to be tested in sufficiently large clinical studies.
References
1 Nock S *et al*. 2020. CD5L Constitutes a Novel Biomarker for Integrated Hepatic Thyroid Hormone Action. Thyroid. 30: 908-923.
2 Osinga JAJ *et al*. 2022. TSH and FT4 Reference Intervals in Pregnancy: A Systematic Review and Individual Participant. J Clin Endocrinol Metab. 107(10): 2925-2933.
3 M Astiz, I Heyde, and H Oster. 2019. Mechanisms of Communication in the Mammalian Circadian Timing System. Int J Mol Sci. 20(2): 343.
DOI: 10.1530/endoabs.99.P381

P382

Antithrombotic/anticoagulant drugs did not increased the risck of bleeding during thyroid fna (nor its whithdrawal up to one month reduced its risk)

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Introduction
Thyroid fine-needle aspiration cytology (TFNA) is a widely used diagnostic method that is generally safe. Aiming to reduce haemorrhage doctors frequently advise patients to withdrawal antithrombotic/anticoagulant (AT/AC) drugs before TFNA. There are no guidelines recommending so and the so called 'novel anticoagulants' have been poorly studied in this context. Some patients are likely being exposed to increased thrombotic risk unnecessarily. Evidence is little but increasing indicating no extra risk of haemorrhagic complications and possibly no need of antithrombotic/anticoagulant (AT/AC) drug withdrawal.
Objective
To compare the incidence of haemorrhage in patients with and without AT/AC drugs during FNA in our centre.
Method
Retrospective observational study of the FNAs performed between the 1st of may 2019 and the 31st of december 2021. Records of haemorrhage and drug treatment were made prospectively during FNA. All FNAs were performed by the same operator, using the same technique and needle gauge. In warfine treated patients FNA was performed if INR was 2-3.
Results
We evaluated 491 FNAs, patients average age was 61y.o. The general incidence of haemorrhage was 3.6%. Of the patients with this complication only 2 were taking AT/AC drugs (11%), one had stopped aspirin 3days, the other stopped dabigatran 1week and the rest was off any AT/AC drugs. The total of patients treated with AT/AC drugs was 78 (16%). In this group the incidence of haemorrhage was 2.5%, represents 2 patients both treated with antithrombotic drugs (one aspirin and the other clopidogrel). There were no haemorrhages with enoxaparin nor with any oral anticoagulant. There was no statistically significant difference (SSD) (p 0, 63) in the incidence of haemorrhages in the treated vs untreated group ($n=348$). There was no SSD (p 0, 30) in the incidence of haemorrhages in the treated group vs the group that stopped therapy 24h to 3days before the procedure ($n=16$). There was no SSD (p 0, 46) in the incidence of haemorrhages between the group that stopped therapy 24h to 3days before the procedure and the group that stopped therapy 4days to 1month before the procedure ($n=13$).
Conclusion
There was no significant association of haemorrhage with drug treatment. The incidence of complications was lower in the treated group. There were no complications in patients treated with anticoagulants, haemorrhage occurred in patients taking antithrombotic drugs only (the incidence was lower than in the untreated group). To withdrawal AT/AC drugs before the procedure did not provide a statistically significant decrease in haemorrhagic complications.
DOI: 10.1530/endoabs.99.P382

P383

Simultaneous presentation of thyrotoxicosis and diabetic ketoacidosis in two previously healthy men

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Graves' disease and type 1 DM (T1DM) both have an autoimmune aetiology. Also, Thyrotoxicosis (TT) has previously been described as a possible precipitant of diabetic ketoacidosis (DKA) in patients with T1DM. Due to the similarities in their clinical presentation, DKA can mask the diagnosis of TT and vice versa. We report two cases of the simultenous presentation of thyrotoxicosis and diabetic keto-acidosis in two previously healthy men. Case 1: A 27-year-old man with no history of any disease presented to the emergency department (ED) with unintentional weight loss of 15 kg in 1 month, excessive sweating, abdominal pain, anxiety and tremors of the extremities. On examination, the patient appeared anxious, dehydrated. Blood pression was 107/68 mmHg and heart rate at 98 beats/min. He had palpable goitre. Investigations revealed that HbA1c was 10.7%, blood glucose was 19 mmol/l with 3+ ketonuria and compensated mild acidosis. Thyroid function test revealed TSH (0.01 mIU/L), free T4 (35 pmol/L). Thyroid scintigraphy revealed a diffuse hypercaptating goiter. Both anti-GAD and anti-TSH receptor antibody were positives. The patient was managed with carefully administered intravenous fluids, intravenous insulin and electrolyte replacement. After resolution of DKA, the patient was transitioned to subcutaneously administered insulin and Carbimazole 20 mg was added. Case 2: A 24-year-old man with familial history hashimoto's disease presented to the emergency department (ED) with unintentional weight loss of 14 kg in 6 months, excessive sweating and palpitations. On examination, Blood pression was 121/64 mmHg and heart rate at 104 beats/min. He had palpable goitre. Investigations revealed that HbA1c was 14.9 %, blood glucose was 19 mmol/l with 2+ ketonuria and compensated mild acidosis. Thyroid function test revealed TSH (0.006 mIU/L), free T4 (57 pmol/L). Thyroid scintigraphy revealed a diffuse hypercaptating goiter. The patient was managed with carefully administered intravenous fluids, intravenous insulin and electrolyte replacement. After resolution of DKA, the patient was transitioned to subcutaneously administered insulin and Carbimazole 30 mg was added.
Discussion
Thyroid hormones affect glucose metabolism at the cellular level by causing insulin resistance, upregulating glucose production by glycolysis and gluconeogenesis pathways and increasing gut absorption of glucose. Thyroxine also decreases serum insulin levels by increasing renal excretion. The resulting state of insulinopenia and insulin resistance causes disinhibition of hormone-sensitive lipase. This leads to unchecked lipolysis and fatty acid oxidation with increased ketones production.
DOI: 10.1530/endoabs.99.P383

P384

Echocardiographic differences between the mild form of subclinical hypothyroidism and healthy subjects

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Background
Treatment of subclinical hypothyroidism (ScH) when TSH is between the upper reference value and 7mU/L, especially in patients younger than 65 years is controversial.
Objectives
To compare the risk factors for atherosclerosis and echocardiographic parameters in patients with ScH1 (4, 2≤TSH≤7mU/L) to euthyroid subjects and patients with ScH2 (TSH>7mU/L).
Material and Methods
Prospectively 54 consecutive patients with newly diagnosed ScH (19 with TSH≤7mU/L (ScH1) and 35 with TSH>7mU/L (ScH2)) started for the first time with levothyroxine therapy, and 30 healthy subjects were recruited from the outpatient department of the University Clinic of Endocrinology in Skopje, R. of N. Macedonia. Laboratory analyses and an echocardiography study were done at the first visit and after 5 months in a euthyroid stage in patients with ScH.
Results
The mean age and TSH value in ScH group were 43.1±12.4y., and 8.71± 1, 9mU/L. Compared to healthy controls, patients with ScH1 had a higher mean triglycerides and non-HDL-C ratio (1.52±0.9 vs 1.1±0.6, and 4.3±1.1 vs 3.79±0.9, $P<0.05$), lower E/A ratio (1.05 ± 0.25 vs 1.26 ± 0.36, $P<0.05$), higher E/e' sep. ratio (8.56 ± 2.63 vs 6.04 ± 1.64, $P< 0.01$), higher myocardial performance index (MPI) (0.47 ± 0.09 vs 0.43 ± 0.07, $P< 0.05$), lower global longitudinal strain (GLS) (-19.34 ± 2.0 vs -20.9 ± 1.7%, $P< 0.05$), and lower S wave derived by tissue Doppler imaging (0.074 ± 0.01 vs 0.092 ± 0.01 m/s, $P< 0.01$). Compared to ScH2, patients with ScH1 have lower GLS but without statistical significance. Levothyroxine treatment (L-T4T) in patients with ScH1 contributed to higher EF (61.9 ± 5.2 vs 63.1 ± 4.6%, $P< 0.05$), lower E/e' sep. ratio (8.56 ± 2.63 vs 7.21 ± 2.23, $P< 0.05$), and lower MPI (0.47 ± 0.09 vs 0.43 ± 0.05%, $P< 0.05$), compared to values in ScH1 patients at baseline. The same parameters were improved in the ScH2 group after L-T4T.
Conclusions
In a small study, patients with ScH1 vs healthy individuals had subtle changes in certain parameters that indicate involvement of diastolic function of the left ventricle in ScH, and these parameters improved after L-T4T.

DOI: 10.1530/endoabs.99.P384

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hCG-TSHR cross-interaction: a rationale for in hyperemesis gravidarum?

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About 50% of women have nausea and vomiting during pregnancy. In about 35% of women who have these symptoms, nausea and vomiting are clinically significant, worsening their living conditions and consisting in a case of gender gap. The severe form, hyperemesis gravidarum, ranges from 0.3 to 1.0% of cases and is characterized by persistent vomiting, gestational thyrotoxicosis, weight loss of more than 5%, ketonuria, hypokalemia and dehydration, although the pathophysiological mechanism is unknown. Some studies found that increasing human chorionic gonadotropin (hCG) levels overlap the fall of thyroid stimulating hormone (TSH) levels, the increase of thyroid hormone (T3 and T4) levels, and the appearance of hyperemesis gravidarum. Thus, it was hypothesized that hCG binds to TSH receptor (TSHR), perturbing thyroid functions and triggering hyperemesis gravidarum. The aim of this study is to characterize hCG-TSHR cross-interactions *in vitro*, finding a rationale to support the clinical hints. Mechanistic experiments evaluating TSHR-dependent cell signaling pathways were performed in COS7 cell line. Cells were transfected with TSHR-coding plasmid and treated with pM-nM hCG doses before Gs and Gq protein-mediated pathway analysis. Intracellular levels of cyclic adenosine monophosphate (cAMP) and calcium ions (Ca^{2+}) increase were measured by bioluminescence resonance energy transfer (BRET), while inositol monophosphate (IP1) was evaluated by homogeneous time resolved fluorescence (HTRF). Results were compared by Kruskall-Wallis test ($n=5$; $P<0.05$) and corrected by Dunn's post-hoc test. Results demonstrated that 50 nM hCG activates the TSHR/Gαq pathway, resulting in intracellular IP1 and Ca^{2+} increase, while no cAMP activation occurred. Results were compared to those obtained from transfected cells treated with the vehicle, in the absence of hCG, which did not result in any Ca^{2+}/IP1 increase. These data support the clinical relationship between hCG and thyroid functions in hyperemesis gravidarum.

DOI: 10.1530/endoabs.99.P385

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Pathogenic variants of CHEK2 gene in thyroid cancer (TC) patients with a personal and/or familial history of other malignancies

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CHEK2, located on chromosome 22q, is a tumor suppressor gene. Its' pathogenic variants are often associated with a tumor predisposition syndrome 4, with an increased risk of breast, prostate and colorectal cancers. There are some reports of an increased risk of papillary thyroid cancer (PTC) in carriers of the *CHEK2* pathogenic variants. Current guidelines, however, do not recommend general screening of TC patients. The study assessed the prevalence of pathogenic *CHEK2* variants in patients diagnosed with TC with a personal and/or familial history of other malignancies. The study was a retrospective analysis of a group of 163 patients (138 females and 25 males, mean age of 48.9 years) diagnosed with TC and with a positive personal and/or familial history of other mutation-related malignancies. *CHEK2* (exons 4, 5, 12) were analyzed by Sanger sequencing. If negative – deletions analysis was performed with MLPA. Pathogenic or likely pathogenic *CHEK2* variants were found in 25 patients (21 females and four males; 15, 3% of the study group). There was no significant difference in the mean age at diagnosis in patients positive and negative for *CHEK2* pathogenic variants (46 vs 49 years, respectively; $P=0.39$). The average number of malignancies in the family members was higher in patients harboring *CHEK2* pathogenic variants; the difference was not statistically significant (1.88 vs 1.63; $P= 0.36$). There was no difference in the personal history (12% vs 29.7%, respectively) or family history (84% vs 83%, respectively) of other malignancies in *CHEK2*- positive and negative PT cancers. There was no significant difference between *CHEK2* positive and negative patients in number of breast, prostate and colon cancers combined in family members. Thyroid cancer was significantly more common in family members of patients with *CHEK2* pathogenic variants (9 cases vs 12 cases in *CHEK2* negative group, $P= 0.006$).
Conclusions
The frequency of *CHEK2* pathogenic variants in a preselected group of PT patients with a personal and/or familial history of other malignancies is similar to that previously reported in the unselected group of Polish patients with PTC. There is currently no convincing data justifying the selective screening for *CHEK2* in such a group. It seems reasonable to advise genetic testing for *CHEK2* pathogenic variants in TC patients with positive family history of thyroid cancer.

DOI: 10.1530/endoabs.99.P564

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Molecular characterization of circulating tumor cells (CTCs) in sporadic medullary thyroid carcinoma (spMTC) patients

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Objectives
Distant metastases (DM) and/or biochemical persistent disease (BPD) in MTC, adversely affect disease prognosis. Calcitonin and CEA doubling-times (DTs) are