November 2018, used Diasorin’s chemiluminescent assays for 25OHD and 1,25(OH)_2D, and a second group (group B), comprising 88 samples, used the same 25(OH)D assay and LC-MS/MS method for 1,25(OH)_2D.

1,25(OH)_2D measurement in the group A used a chemiluminescent competitive assay (Liaison XL, Diasorin). The 1,25(OH)D LC-MS/MS assay includes a previous sample prep, extraction, derivatization and chromatography. APCI+ is followed by SRM (Selected Reaction Monitoring) and CAD (Collision Activated Dissociation) fragmentation. Precision studies showed, between run CVs of 6.8% to 7.4% and within run of 2.9% to 5.5%.

In vitro investigations testing standards and spiked samples with 25(OH)D, 25(OH)D_2, 3-epimer-25(OH)D, and 24R,25(OH)D were also used to verify possible analytical interferences in the 1,25(OH)D LC-MS/MS.

Results: In group A, 25(OH)D median was 371 ng/mL (928 nmol/L), range 154 ng/mL to 856 ng/mL; 1,25(OH)_2D median of 350 pg/mL (875 pmol/L), range 41 pg/mL to 1280 pg/mL. Correlation (Spearman) between 25(OH)D and 1,25(OH)_2D was r = 0.8649 (P < 0.001).

In group B, 25(OH)D showed a median of 349 ng/mL (872 nmol/L), range 171 to 756 ng/mL; 1,25(OH)_2D median of 54 pg/mL (135 pmol/L), range 24 pg/mL to 108 pg/mL. Correlation between 25(OH)D and 1,25(OH)_2D was r = 0.185 (P = 0.08).

In group A 189 samples had calcium measurement (median 9.7 mg/dL, range of 8.7 to 13.6 mg/dL), 182 creatinine (median of 1.0 mg/dL, range 0.6 to 2.0 mg/dL) and 179 PTH (median 19 pg/mL, range 5 to 68 pg/mL). In group B 75 cases had measurements of calcium (median 9.7, range 8.6 to 16.6 mg/dL), 75 of creatinine (median 0.8, range 0.3 to 2.5 mg/dL) and 75 of PTH (median 20, range 9 to 49 pg/mL). The in vitro tests showed a slight interference from 25(OH) D_3, 3-epimer-25(OH)D_3 and 24R,25(OH)D_3 molecules in the LC-MS/MS method.

Conclusion: our results confirm data already published showing interference of high levels of 25(OH)D in 1,25(OH)_2D measured by immunoassay and, in a milder way, by LC-MS/MS (1). V. Care should be taken in the interpretation of 1,25(OH)_2D values in samples with high 25(OH)D values.

1. Hawkes CP, Schnellbacher S, Singh RJ, Levine MA. 25-Hydroxyvitamin D can interfere with a common assay for 1,25-dihydroxyvitamin D in vitamin D intoxication. J Clin Endocrinol Metab. 2015; 100:2883-2889.

Cardiovascular Endocrinology

HYPERTRIGLYCERIDEMIA; INFLAMMATION AND MUSCLE METABOLISM IN OBESITY AND WEIGHT LOSS I

Sex Hormones Therapy Differentially Modulates HDL Function in Transgender Individuals

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Background/aim: The main proposed atheroprotective function of high-density lipoproteins (HDL) lays on their role to promote macrophage cholesterol efflux. An insightful way to learn more about the effects of sex hormones on HDL function is to study changes during hormone therapy. The present study was aimed at evaluating the effects of exogenous sex hormones administration on HDL cholesterol efflux capacity (CEC) within transgender individuals. CEC estimates the ability of HDL to remove cholesterol from cells, i.e. the initial step in reverse cholesterol transport.

Subjects/Methods: Transmen were treated with testosterone gel, a mix of testosterone esters once every three weeks) or testosterone undecanoate once every twelve weeks, whereas transwomen were treated with either oral estradiol valerate or a transdermal application of estradiol (patches). Cyproterone acetate was prescribed as a testosterone-blocking agent to all transwomen. HDL function was evaluated by a radioisotopic technique. Hormone levels, lipids and HDL function were evaluated after one year of follow-up.

Results: In transmen (n= 15), testosterone markedly increased (+ 97%; p < 0.0001), whereas luteinizing hormone (LH) decreased significantly (- 64%; p = 0.049). Total cholesterol and low-density lipoprotein cholesterol (LDL-C) were not affected by testosterone treatment, whilst triglycerides (TG) were raised (+ 11.76%; p = 0.0078) and HDL-C reduced (- 19.6%, p=0.0103). Concerning HDL CEC, only the aqueous diffusion process was lowered (- 9.8%; p = 0.0032), an effect directly correlated with HDL-C changes (r = 0.6242, p = 0.0002). Total-, ATP-binding cassette transporter (ABCA1), and ABCG1-mediated CEC were not affected by testosterone treatment. In transwomen (n= 15), estradiol levels were raised (+200%, p=0.013) whereas LH and testosterone significantly reduced, i.e. - 97% for both. Relative to lipids, estradiol supplementation reduced total cholesterol (− 10.7%, p = 0.0017), LDL-C (− 14.3%, p = 0.0024) and HDL-C (− 10.9%, p = 0.0058). Total HDL CEC decreased (− 11%, p=0.0001) with a specific decrement in CEC mediated by the ATP-binding cassette transporter (ABCA1) (−24%, p = 0.0003) and aqueous diffusion (−4.7%, p = 0.0014). This last was associated to a reduction in LDL-C (r = 0.4084, p = 0.0251). Conversely, the drop in ABCA1 and total CEC did not associate to reductions in HDL-C levels.

Conclusions: In transmen, testosterone supplementation was associated with a reduction in aqueous diffusion-mediated CEC, an effect potentially dependent to HDL-C changes. In transwomen, estrogen significantly decreased HDL function (CEC), independent of HDL-C levels changes.

Bone and Mineral Metabolism

CLINICAL ASPECTS OF OSTEOPOROSIS AND VITAMIN D ACTION

Incidence Rate of Post-Thyroidectomy Immediate Hypocalcemia and Its Relation to Pre-Operative Vitamin D and Parathyroid Hormone Levels

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