

Endocrine Abstracts

May 2015 Volume 37
ISSN 1479-6848 (online)

17th European Congress of
Endocrinology 2015

16–20 May 2015, Dublin, Ireland



published by
bioscientifica

Online version available at
www.endocrine-abstracts.org



17th European Congress of Endocrinology

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ESE Office

Euro House
22 Apex Court
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Bristol BS32 4JT, UK

Contact:
Tel:
Fax:
E-mail:
Web site:

Andrea Davis
+44 (0)1454 642247
+44 (0)1454 642222
info@euro-endo.org
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Bioscientifica Ltd
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Contact:
Tel:
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Website:

Claire Arrigoni
+44 (0)1454 642240
+44 (0)1454 642222
conferences@bioscientifica.com
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EP210**Serum gonadotropins secretion is not reduced with advancing age in HIV-infected females: results of a case-control study in menopausal women**Chiara Diazzi^{1,2}, Giulia Brigante^{1,2}, Giovanni Guaraldi³, Manuela Simoni^{1,2} & Vincenzo Rochira^{1,2}¹Unit of Endocrinology, Department of Biomedical, Metabolic and Neural Sciences, University of Modena and Reggio Emilia, Modena, Italy;²Azienda USL of Modena, Modena, Italy; ³Metabolic Clinic, Infectious and Tropical Diseases Unit, Department of Medical and Surgical Sciences for Children and Adults, University of Modena and Reggio Emilia, Modena, Italy.**Introduction**

HIV infection treated with highly active antiretroviral therapy (HAART) seems to be associated with hypogonadism in men. Less is known in HIV-infected women gonadal status. The aim of this study is to investigate gonadal function, in menopausal HIV-infected women compared sex- and age-matched healthy subjects (HS).

Methods

We retrospectively compared 188 HIV-infected women with 192 HS selected reviewing record charts and laboratory database respectively. We considered only women older than 50 years and we grouped them according to their age (50–54; 55–59; and >60 years). Basal serum LH, FSH, estradiol, and testosterone were measured. The FSH cut-off of 40 UI/l for establishing menopausal status.

Results

The percentage of subjects with FSH levels >40 UI/l was higher in HIV-infected women (67.5%) than in healthy controls (59.4%). This difference was found also in the younger subgroup (38% vs 27%). FSH serum levels in HIV-infected women (54.08 ± 31.47 mUI/ml) did not differ ($P=0.27$) from HS (50.87 ± 31 mUI/ml). Accordingly, no significant differences were found in LH, estradiol, and testosterone levels.

Conclusions

Menopause seems to occur at a younger age than HS in HIV-infected women. Moreover, differently from what was documented in HIV-infected male counterpart, HIV-infected women seem to not develop hypogonadotropic hypogonadism, but have a tendency to higher serum FSH at a younger age (<54 years) suggesting premature hypergonadotropic hypogonadism. With this in view menopause may be considered an element of the process of premature aging associated with HIV infection and its comorbidities.

DOI: 10.1530/endoabs.37.EP210

EP211**Association between Apa-I polymorphism in the vitamin D receptor gene and metabolic syndrome in polycystic ovary syndrome**Betânia R Santos^{1,2} & Poli Mara Spritzer^{1,2}¹Gynecological Endocrinology Unit, Division of Endocrinology, Hospital de Clínicas de Porto Alegre, Porto Alegre/RS, Brazil; ²Federal University of Rio Grande do sul, Porto Alegre/RS, Brazil.

Women with polycystic ovary syndrome (PCOS) have higher prevalence of metabolic disturbances such as changes in lipid profile, diabetes, hypertension and metabolic syndrome. Variants on vitamin D receptor (VDR) gene have also been related to metabolic comorbidities in general population. Therefore, the aim of the present study was to investigate whether Apa-I polymorphism (rs7974232) in the VDR gene is associated with metabolic syndrome and endocrine profile in PCOS. In this cross-sectional study 190 PCOS (Rotterdam criteria) and 100 non-hirsute and ovulatory control women were enrolled. Endocrine and clinical measurements were assessed and genotypic analyses were evaluated by real time PCR. PCOS women were younger (22.9 ± 6.7 vs 25.2 ± 7.7 years; $P=0.013$) and had significantly higher BMI (29.7 ± 6.4 vs 27.0 ± 6.1 kg/m²; $P=0.001$), total testosterone (0.90 ± 0.40 vs 0.54 ± 0.17 ng/ml; $P<0.001$) and fasting insulin (16.87 (9.81–26.97) vs 11.09 (7.34–15.44); $P<0.001$). Metabolic syndrome was

present in 26.5% of PCOS and in 4.8% of controls. The genotypic distribution for Apa-I SNP in PCOS (AA: 32.1%, AC: 46.3%, CC: 21.6%) and controls (AA: 36.0%, AC: 48.0%, CC: 16.0%) was similar. PCOS participants with the CC genotype (CC vs CA+AA) of Apa-I had higher risk for metabolic syndrome (OR: 2.133; 95% CI 1.020–4.464, $P=0.042$). While the analyses among control participants showed that metabolic syndrome is more frequent in CC than CA+AA genotype (13.3% vs 2.9%), no significance was found, (OR: 5.154; 95% CI 0.665–39.954, $P=0.145$), maybe because of the low prevalence of metabolic syndrome in this group. The CC genotype was also associated with higher systolic blood pressure ($P=0.009$), total cholesterol ($P=0.040$) and LDL ($P=0.038$) in both PCOS and control groups (ANOVA two-way). In conclusion, the present results suggest that variant Apa-I in VDR gene may be associated with metabolic syndrome in women with PCOS.

Disclosure

This work was supported by grant from CNPq INCT 573747/2008-3, Brazil.

DOI: 10.1530/endoabs.37.EP211

EP212**XbaI and PvuII oestrogen receptor alpha gene polymorphism and Y chromosome deletions in infertile vs fertile men**Suzana Vladoiu¹, Dana Dinu¹, Gabriela Anton², Anca Botezatu², Dana Manda¹, Sabina Oros¹, Olga Ianas¹, Diana Paun¹, Corin Badiu¹ & Roxana Rosca¹¹C.I.Parhon National Institute of Endocrinology, Bucharest, Romania;²Stefan S. Nicolau Institute of Virology, Bucharest, Romania.

Only a few genes involved in spermatogenesis have clinical importance: Y chromosome micro deletions in the region called azoospermia factor (AZF) and oestrogen receptor alpha gene polymorphisms (ESR1).

Objective

The study aim to evaluate ESR1 and Y chromosome deletion in infertile men.

Subjects and methods

43 infertile men, and 34 fertile men aged 20–50 years 50, were enrolled after signing the informed consent. Screening for microdeletions in the azoospermia factor (AZF) region of Y chromosome was performed by multiplex PCR and oestrogen receptor alpha (ESR1) gene polymorphisms XbaI and PvuII was performed by RFLP.

Results

In infertile patients ESR XbaI polymorphism 15 cases were wild type homozygote (XX), 23 heterozygote (Xx) and four mutant homozygote (xx). The frequency of x allele was 0.37, and 0.63 for X allele, $\chi^2=1.299$. In normal patients ESR XbaI polymorphism 14 cases were homozygote for normal allele (XX), 17 were heterozygote (Xx) and three were mutant homozygote (xx). The frequency of x allele in population was 0.34, for X allele the frequency was 0.66, $\chi^2=0.464$. In infertile patients ESR PvuII polymorphism 12 cases were homozygote (PP), 22 were heterozygote (Pp) and eight were mutant homozygote (pp). The frequency of p allele was 0.45, and 0.55 for P allele, $\chi^2=0.137$. In normal patients ESR PvuII polymorphism ten cases were homozygote for normal allele (PP), 18 were heterozygote (Pp) and six were mutant homozygote (pp). The frequency of p allele was 0.44, for P allele the frequency was 0.56, $\chi^2=0.184$. 6.97% of all patients presented microdeletions in AZFc region and 2.32% in AZFb region, 6.97% in AZFb and AZFc regions.

Conclusion

For the two ESR1 studied polymorphism the investigated group is in HW equilibrium. No significant differences were found between the mutant allele's frequency between the infertile patients and the control group.

Disclosure

Acknowledgement: This work received financial support through the project entitled 'CERO – Career profile: Romanian Researcher', grant number POSDRU/159/1.5/S/135760, co-financed by the European Social Fund for Sectorial Operational.

DOI: 10.1530/endoabs.37.EP212

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