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Background: Hodgkin's and non-Hodgkin's lymphomas represent the most common hematological malignancies (HE) affecting men in reproductive age. Several protocols of cancer therapies have significantly improved survival rates of young patients, despite having a negative effect on testicular function, including damage or alterations to the sperm genome. The consequences of these treatments on sperm DNA represent a major concern since damage to the paternal genome may have detrimental effects on offspring. Data on post-therapy sperm DNA alterations are still controversial and limited to 2 years, thus the right timing for natural conception after treatment remains uncertain.

Objective: To evaluate the effect of cytotoxic therapies on the integrity of the sperm genome (Sperm DNA Fragmentation, SDF) after 2 (T2) and 3 years (T3) from the end of treatment in HM patients.

Methods: We analyzed the SDF of 2 HM patients groups, one evaluated 2 years post-therapy (T2 group, $n = 26$) and one evaluated 3 years post-therapy (T3 group, $n = 25$). The analysis, based on terminal-uridine nick and labelling end assay (TUNEL), was performed on 10×10^6 sperm cells. We evaluated total and brighter % SDF (SDFtot and SDFbr, respectively). The SDFbr is more strictly associated with sperm fertilizing potential and fertility outcome. Data of each group were compared with those of 58 healthy fertile men (control group) in a cross-sectional analysis.

Results: At each time points (T2 and T3) patients were divided according to the type of treatment: ABVD ($n = 11$ and 9, respectively), R-CHOP ($n = 2$ and 3, respectively) and mixed therapies (two or more different chemotherapies or chemo+radiotherapy) ($n = 13$ both for T2 and for T3). The mean %SDFtot and %SDFbr of the control group were 29.11 ± 11.11% and 19.53 ± 9.48%, respectively. (i) T2 group: both %SDFtot and %SDFbr in subjects treated with ABVD (39.06 ± 15.08%, 28.54 ± 14.28%) or with mixed therapies (43 16 ± 20.64%, 35.45 ± 19.62%) were significantly higher than those of fertile men ($p < 0.05$, $p \leq 0.01$ and $p = 0.001$, $p < 0.001$, respectively). (ii) T3 group: no significant differences were observed at T3 between patients and controls, with the exception of % SDFtot in the group that underwent mixed therapies (37.29 ± 15.16%, $p < 0.05$). (iii) Severe DNA damage expressed as % SDFbr >75th percentile of "normality" (>25%) was observed in 55% (6/11) and 62% (8/13) of patients treated with ABVD and mixed therapies, respectively, in the T2 group. In the T3 group, SDFbr >25% was observed in 33% of patients treated with either ABVD (3/9) or R-CHOP (1/3), and in 46% (6/13) of those treated with mixed therapies.

Conclusion: Our study indicates a long-term effect of cytotoxic therapies on DNA integrity in a relatively large proportion of patients. Pathological SDF values (above the 75th percentile of fertile controls) 3 years after treatment, were observed after all type of treatment with the highest percentage in patients treated with most aggressive therapies such as combined chemotherapies or chemo+ radiotherapy. DNA fragmentation analysis, particularly % SDFbr, should be proposed both to monitor the long-term effect of cytotoxic therapies and to help in decision making on the timing of natural pregnancy.

P061

Mutations in the ADGRG2 gene as a cause of CBAVD

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Background: Congenital Bilateral Absence of the Vas Deference (CBAVD) is a common cause of obstructive azoospermia. Around 80% of these men have mutations in the CFTR gene. Part of the remaining 20% have a combined uro-genital problem. However, for the majority of the patients without CFTR alterations, the underlying (genetic) cause remains unknown. In 2016 Patat et al. for the first time described mutations in the X-linked ADGRG2 gene as causal of CBAVD. They analysed 26 CBAVD patients without kidney problems. In three patients, a truncating mutation was detected. Subsequently, Yang et al. (2017) described 2 more patients (out of 18) with missense mutations in the ADGRG2.

Methods: In the present study, we have investigated 20 CBAVD patients without mutations in the CFTR gene. Echography had already excluded kidney problems for the majority of these patients before their ADGRG2 gene (NM_001079858.2) was analysed by Sanger sequencing.

Results: We have identified a single patient with a truncating mutation in the ADGRG2 gene: c.920C>A, p.Ser307*. This nonsense variant has not been described before, and is located in exon 16 (from the 29 exons). Consequently, the majority of the protein (711 amino acids) is missing. Altogether, 64 patients (reported or included in our study) have been analysed for the presence of mutations in the ADGRG2 gene. PRESENTLY, around 9% of CFTR negative patients are hemizygous for an ADGRG2 gene mutation with nonsense mutations frequently seen.

Conclusion: It is worthwhile to include the analysis of the AGRGR2 gene in a routine diagnostic setting, after full investigation of the CFTR gene. Furthermore, since for a large group of patients, the underlying molecular mechanism IS STILL unknown, it is worthwhile to (re-)analyse THEM by genome-wide molecular analyses.

P062

Testosterone (T) and estradiol (E2) are poorly associated to the reduction of bone mineral density (BMD) in Young/Middle Aged Men with Human immunodeficiency virus (HIV)

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Background: Osteopenia and osteoporosis, as well as hypogonadism, are common findings in men with HIV-infection and they occur at a younger age than healthy subjects. The reduction of BMD is due to both HIV-related and HIV-unrelated factors. Previous studies suggest that T deficiency is not or poorly associated with reduced BMD in HIV context. On the other hand, estrogens are considered more important than androgens for bone health in general population, but data about their role in HIV-infected men are still scanty.

Objective: To investigate the relationship between BMD and circulating sex steroids assessed by Liquid Chromatography tandem Mass Spectrometry (LC-MS/MS) in a cohort of young/middle aged HIV-infected men.

Methods: Prospective, cross-sectional, observational study on 233 consecutive HIV-infected male patients with ongoing Highly Active Antiretroviral Therapy (HAART), attending the Multidisciplinary Metabolic Clinic of Modena. Body composition and BMD at total body, lumbar spine (L1 to L4) and total hip were measured using a Hologic QDR-2000 densitometer (DXA). LC-MS/MS was used for hormonal assays. Statistical analysis: The nonparametric Mann–Whitney *U* test was used for group comparisons because variables were not normally distributed at the Kolmogorov-Smirnov test. Correlations were performed using linear regression models.

Results: Two hundred and thirty-three HIV-infected patients were enrolled (mean age 45.29 $\pm$ 5.33 years) with average duration of HIV-infection of 190.8 $\pm$ 102.8 months. Eight patients (3.4%) had hypogonadism, defined as total T serum levels below 300 ng/dL. Considering results at DXA examination, BMD was normal in 36.5% and reduced in 63.5% (55.8% osteopenia, 7.7% osteoporosis). Both total T and E2 did not significantly differ comparing patients with normal BMD to patients with reduced BMD. Body and lumbar BMD did not show any significant difference between eugonadal patients and patients with low T and/or low E2, while both femoral BMD and femoral T-score were significantly higher in patients with E2 above 20 pg/mL than in those with E2 below 20 pg/mL ($p = 0.043$ and $p = 0.033$, respectively). At linear and stepwise multiple regression analyses, BMD was positively associated with total lean mass ($R^2 = 0.154$, $p < 0.0001$); apart from it, neither T nor E2 correlated with BMD and T-score at any site.

Conclusion: Classical factors associated to BMD as E2 and T seem to be less relevant in this model of male osteoporosis. Other specific HIV-related factors, such as changes in body composition and consequent lipodystrophy, could be more deeply involved than sex steroids as potential mechanisms in bone loss in this setting. Finally, we confirm the high prevalence of reduced BMD in young/middle aged HIV-infected men, representing one of the clinical hallmarks of the premature aging process related to HIV infection.

P063

Endogenous testosterone supports spermatogenesis even in the absence of gonadotrophins: evidence from a case report

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Background: In patients with testicular dysgenesis syndrome, reduced semen quality and testicular cancer are common. We report a case of a testicular tumour in a patient with a history of cryptorchidism and oligoasthenospermia. He had an unusual hormonal profile, which was not fully explained by the pathological findings.

Case report: A 31-year-old man was referred to our tertiary care andrology unit for primary infertility with a history of bilateral orchidopexy during childhood. Testes were small (12 cc). Gynaecomastia was absent. Semen analysis repeatedly showed oligoasthenospermia (2.4–7.1 million/mL, 85–92% immotile). Gonadotropins (LH and FSH < 0.1 U/L) were undetectable, but testosterone and estradiol were normal (850.7 ng/dL and 38.6 ng/L). Prolactin, other pituitary hormones, DHEAS, AFP, HCG and inhibin B were also normal. He denied using anabolic steroids. Suppressed gonadotrophins suggested a sex steroid producing testicular tumour. However, scrotal ultrasound only showed diffuse microcalcifications and three millimetric hypolucent lesions in the left testis, but no intratesticular mass. There were no suspicious lesions nor microcalcifications in the right testis. To further investigate the possibility of increased testicular sex steroid production, selective testicular venous sampling was performed. In the left spermatic vein, testosterone and estradiol levels were very high (3744 ng/dL and 378 ng/L), with a testis-to-periphery gradient of 4.4 and 9.0 respectively. There was no gradient in the right spermatic vein. These results confirmed increased sex steroid producing in the left testis. However, histopathological examination after orchidectomy revealed a multifocal seminoma (largest diameter 3 mm) and profuse germ cell neoplasia in situ. There were neither isolated syncytiotrophoblastic cells, nor choriocarcinoma. Leydig cell hyperplasia was present without Leydig cell tumour. HCG was remeasured with three different methods, all showing very low HCG between 0.6 and 1.1 IU/L. After orchidectomy gonadotrophin levels increased (LH 24.3 U/L, FSH 10.3 U/L), with normal total testosterone and estradiol, indicating recovery of suppression of the hypothalamic-pituitary-testis axis. Sperm concentration increased (10 million/mL.)

Conclusion: (i) Our case shows that endogenous testosterone may support spermatogenesis even without gonadotropins. (ii) In patients with suppressed gonadotropins, normal sex steroid levels and no testicular mass, selective testicular venous sampling can be useful in identifying the site of hormonal overproduction. (iii) Thus far, the

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