Background

Infection with the human immunodeficiency virus (HIV) predisposes to endocrine disorders, manifesting as a metabolic phenotype that affects the entire adipose-musculoskeletal unit (AMS). The present cross-sectional study aimed to investigate differences in irisin and adiponectin concentrations between people living with HIV and healthy controls, as well as to explore potential correlations between the levels of the aforementioned adipokines and markers of calcium homeostasis.

Methods

46~HIV-infected individuals (all men), with a mean age of 52.85 ± 8.48 years and a body mass index (BMI) of $25.76\pm 2.65~kg/m^2$ and 39 healthy controls (all men) with a mean age of 45.44 ± 9.17 years and a BMI of $28.44\pm 6.29~kg/m^2$ were included in the study. Anthropometric data, adipokine levels, 25-hydroxyvitamin D [(25(OH)D)] status and parathyroid hormone (PTH) concentrations were evaluated in the two groups. The Mann Whitney U test was used to assess differences between the HIV group and the control group. Pearson's correlations and partial correlations for the relationship between adiponectin, irisin, and PTH levels were examined. The results were adjusted for several confounders, including 25(OH)D levels, body fat, muscle mass, and mean exposure to ultraviolet B radiation during the previous 45~days before blood sample collection. Results

The HIV group had a lower body fat mass $(20.76\pm6.18 \text{ vs } 33.55\pm7.60 \text{ kg}, P < 0.001)$ and a higher muscle mass $(59.09\pm8.13 \text{ vs } 50.71 \text{ vs } 12.08 \text{ kg}, P = 0.009)$ compared to the control group. Mean adiponectin concentrations were significantly lower in the HIV group compared to the control group: $5868\pm3668 \text{ vs } 9068\pm4277 \text{ ng/ml}, P = 0.011$. The same was applicable to irisin concentrations: $8.31\pm8.17 \text{ (HIV)} \text{ vs } 29.27\pm27.23 \text{ (controls) ng/ml}, P = 0.013$. A statistically significant and negative correlation was observed between irisin and PTH in the control group (r = -0.591; P = 0.033). In contrast, no significant correlation was observed between PTH and irisin in the HIV group (P = 0.898). Conclusion

Our results suggest a possible down-regulation of the inverse relationship between PTH and irisin in HIV patients and highlight that AMS dyshomeostasis could be involved in the development of skeletal and adipose HIV-related morbidities.

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EP137

Histomorphometric parameters of parathyroid glands after 60 days of sodium benzoate administration $\ _$

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Sodium benzoate (the food additive E211) is widely used as a preservative in the food and pharmaceutical industries. A direct correlation between sodium benzoate intake and the development of sensibilization, nephrotoxicity and hepatotoxicity, disorders of puberty, and genotoxicity has been established. However, the literature does not provide data on changes in morphometric parameters of parathyroid glands after long-term use of sodium benzoate. The aim of this work is to study the effect of 60-day sodium benzoate administration on the histomorphometric parameters of the parathyroid gland in rats. The experiment was performed on 18 white male mature rats divided into three groups. In group 1, animals were injected daily with 1 ml of sodium chloride through a feeding tube for 60 days, in groups 2 and 3, rats were injected with 1 ml of sodium benzoate solution at a dose of 500 mg/kg and 1000 mg/kg, respectively. Histological samples were prepared according to the standard procedure, sections were stained with hematoxylin-eosin and subjected to morphometry. Statistical analysis of the numerical data was performed using the Statistika 5.1 license program. In group 2, we observed a decrease by 1.47% (P > 0.05) in the largest size of parathyroid gland in comparison with group 1. The smallest size of the parathyroid gland decreased by 0.84% (P > 0.05), the number of nuclei of chief cells per unit section area decreased by 3.58% (P > 0.05), the mean diameter of chief cell nuclei decreased by 5.12% (P < 0.05), and functional index (number of nuclei of chief cells per unit section area*mean diameter of chief cell nuclei/20) decreased by 5.17% (P<0.05). In group 3, changes in morphometric parameters of the parathyroid gland became more intense. The largest size of parathyroid gland decreased by 2.45% (P > 0.05), the smallest size of the parathyroid gland decreased by 2.65% (p>0.05), the number of nuclei of chief cells per unit section area decreased by 5.27% (P < 0.05), the mean diameter of chief cell nuclei decreased by 10.12% (P < 0.05), and the functional index decreased by 8.11%(P < 0.05). 60-day intake of sodium benzoate causes dose-dependent changes in the histomorphometric parameters of the parathyroid glands in rats, which are indicative of their hypofunction.

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EP138

Clinical characteristics of sporadic and MEN1-associated primary hyperparathyroidism in young patients

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Introduction

Primary hyperparathyroidism (PHPT) is commonly the most early manifestation of multiple endocrine neoplasia syndrome type 1 (MEN1), but it can also occur in young patients without any inherited disorders. The aim of our study was to compare the clinical characteristics of PHPT in young patients with MEN1 syndrome and with sporadic disease.

Patients and Methods

The study included 72 patients with MEN-1 associated PHPT and 168 patients with sporadic disease. All patients were tested for MEN1 mutations. Electronic medical records were used to analyze clinical data of each patient. Patients who had sufficient data at the time of manifestation of PHPT (before the first surgery) were included. Results

Both groups consisted mostly of young patients and there was no significant difference in the age of manifestation: 31 [25; 39] in the MEN1-group vs 34 [29; 37] in sporadic PHPT (P = 0.308). We did not receive a difference in preoperational levels of parathormone (145.2 pg/ml [98.7; 227.0] in MEN1 group and 152.07 pg/ml [113.90; 241.38] in sporadic PHPT, P = 0.248, as well as in albumin-corrected total blood calcium, phosphorus, and daily calciuria (p > 0.05 for all)). Also, no statistical difference was found in postoperative levels of PTH (P = 0.605) and total blood calcium (P=0.466). Our groups did not differ in the prevalence of nephrocalcinosis/nephrolithiasis (59% in the MEN1group and 63% in the sporadic PHPT, P=0.615). Bone mineral density less than -2.0 SD (Z-score) was found more often in MEN1group: 54 vs 34% in sporadic patients, P=0.031. There were significant differences in the preoperative number of the parathyroid (PT) tumors at the time of the primary surgery (95% of patients with sporadic PHPT and 41% of patients in the MEN1 group had only one PT tumor preoperatively, P < 0.001). Differences were also obtained by histological characteristics of the PT tumors (the frequency of adenoma, hyperplasia, and carcinoma occurrence (P < 0.001)), recurrence of PTHT after the surgery (36% of patients in MEN1 group and 1% in sporadic PHPT, P< 0.001), and family history of other endocrine neoplasia (69% of patients in MEN1 group and 18% in the sporadic PHPT, P < 0.001).

Conclusion

Number of PT tumors, their histological characteristics, recurrence rate and family history should be taken into consideration when evaluating young patients with PHPT.

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EP139

Differential diagnosis between classic primary-, normocal cemic- and secondary hyperparathyroidism based on PFI and ${\bf WI}$

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Introduction

Patients with primary hyperparathyroidism (PHPT) present high calcium and PTH levels while some patients present also high PTH levels but normal calcium levels (NHPT) and such patients be considered as having normocalcemic hyperparathyroidism. On the other hand vitamin D deficient patients develop secondary hyperparathyroidism (SHPT). Sometimes normocalcemicand secondary hyperparathyroidism coexist. Therefore in clinical settings it is very difficult to distinguish primary hyperparathyroidism and normocalcemic hyperparathyroidism from secondary hyperparathyroidism. Aim

The aim of our case control study is to differentiate these entities using the parathyroid functional index (PFI=PTH \times Ca devided by P) and the Wisconsin Index (WI=PTH \times Ca).