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DISORDERS OF BODY COMPOSITION DURING CHRONIC OBSTRUCTIVE PULMONARY DISEASE DEVELOPMENT IN MALE PATIENTS

Summary

The article presents the results of study of body composition in men with chronic obstructive pulmonary disease (COPD). We revealed a reduction in lean (fat-free mass index, skeletal muscle index) and bone components (bone mineral component index) during COPD progression. Fat mass index initially rising fell on the 3rd stage of disease. The distribution of adipose tissue in male patients with COPD plays a role in the development of hypertension.

Keywords

Chronic obstructive pulmonary disease, body composition, fat mass, bone mass, leptin.

Systemic inflammation is very important in the physiopathology of chronic obstructive pulmonary disease (COPD) and its co-morbidities. This inflammation increases during COPD exacerbations [1, 2], suggesting that the latter can increase co-morbidities. Additionally, frequent COPD exacerbations may have an impact on malnutrition development in patients in advanced COPD stages.

Currently, it is accepted that the picture of systemic manifestations of COPD is not fully completed without the evaluation of patient's body composition parameters [3]. Thus, body mass index (BMI) is used for the prognostic BODE classification of COPD. The decrease in bone and muscular components of body composition in the case of normal patient's BMI level may occur against the background of fat mass (FM) component excess. However, excess body mass may be a consequence of increased muscular and bone tissues without raised body FM.

Body composition has not been enough investigated in COPD patients because currently scientists focus on the study of pathogenic mechanisms which lead to the disorders of body composition in COPD patients. Thus, an altered body composition (especially increased visceral FM) could be a significant contributor to low-grade systemic inflammation. Decreased BMI is often associated with the signs of protein-energy malnutrition in advanced stages of disease (in 25-50% of cases). Thereafter these disorders of body composition stimulate COPD development.

It was demonstrated [4] that acute exacerbation of COPD is accompanied by an impaired energy balance due to decreased food intake but increased resting energy expenditure. All this has a negative consequence on COPD patient's body composition particularly after repeated exacerbations [5]. Thus, malnutrition favors reducing the body lean mass (LM) in COPD patients. Visceral fat (constituting only about 10% of total FM) surrounds and infiltrates internal organs in the intrathoracic, intra-abdominal and intrapelvic compartments.

Fat free mass (FFM) can be subdivided into the intracellular compartment (body cell mass) which represents the energy-exchanging part, and the extracellular compartment, which represents substances outside the cells. Decrease in FFM is common in COPD (fluctuates from 20% in clinically stable out-patients to 35% in severe patients) and adversely affects respiratory and peripheral muscle function, exercise capacity and health status. Similarly, decreased LM (FFM=LM+BMC (bone mineral component)), particularly at the expense of muscular tissue is a significant problem in ≈1/3 of moderate to severe COPD patients [6, 7].

It was detected [8, 9] that BMI decrease has an unfavorable impact on COPD development, frequencies of exacerbations and decline of physical activity. Decreased BMI (particularly muscular tissue) is an independent predictor of COPD death. Thus, cases of muscular tissue decrease against the background of the normal or even increased level of BMI are frequently observed [10-12].

The reasons for malnutrition status and low BMI in COPD patients are not yet clear. According to some studies [13-16] they may include: primarily increased load on the respiratory muscles (enhanced

breathing work and its energy costs) and a high level of proinflammatory cytokines in the blood flow (that is closely associated with increased protein catabolism) as well as basic metabolism (increased by 25% compared with the healthy persons), to a smaller degree – expressed hypoxia; oxidative stress; the use of long active β2-agonists and teophylline; smoking, hypoandrogenism, decreased food intake and O2 saturation during eating; impaired regulation of both leptin and hormones that reduce food intake; disorders of gastro-intestinal tract (including the genetic aptitude to these disorders) as well as financial problems (low economic status). Additionally, an important part of «pulmonary cachexia» in severe COPD patients develops due to the disorders of protein metabolism attributed to increased protein degeneration [17]. TNF-α that accelerates the breakdown of protein and reduces the synthesis of albumin (as the main transport protein) also plays a certain role.

Thus, the development of progressive BMI decrease (by 10% in mild COPD and by 50% in severe COPD compared with the initial level) was a result of increased energy expenditure of respiratory muscles (by 15% during physical activity and rest) against the background decrease in the energy intake [6, 7, 18].

BMI is <20 kg/m^2 indicates the presence of malnutrition in the patients. But BMI does not often give complete information about the dynamics of body mass changes in COPD patients because BMI is often within the normal range (or increased to some extent) in some of them. Thus, only 17% of the patients with chronic respiratory failure had a BMI <20 kg/m^2 [19]. It was shown [20] that only ≈5% of patients with GOLD grade 0-2 of COPD had a BMI <18,5 kg/m^2 versus 15-30% of patients with grade 4 of disease. There is a distinct link between malnutrition of COPD patients and their mortality [20-22]. Thus, for the patients with an FEV1<50% the relative risk for death was a HR of 1,62 for a BMI<20 kg/m^2 and only 0,62 for the patients with a BMI ≥30 kg/m^2 . Three malnutrition profiles can be identified in advanced COPD: underweight patients with concomitant depletion of body LM (60%), underweight patients with a normal LM (20%) and patients of stable body weight with depletion of LM body [23].

Overweight and obesity are often observed (in ≈1/3 and ≈1/5 of cases respectively) in COPD patients [24]. The overweight/obesity did not worsen the respiratory function and symptoms in many cases of mild to moderate COPD [25] and even had a protective effect in relation to mortality [20, 26], while the patients with severe obesity (BMI>40 kg/m^2) had a significant increase in deaths due to respiratory disease [27]. Overweight/obesity favors the decrease in pulmonary function and appearance of cardiovascular pathology as well as the disorders of carbohydrate metabolism [10, 28].

The prevalence of metabolic syndrome fluctuates from ≈1/4 for all COPD grades [29] to 53%, 37% and 44% for GOLD grades 2, 3, 4 respectively [30]. Additionally, the presence of metabolic syndrome was associated with higher levels of the systemic inflammation markers and with negative impact in exacerbation. Thus, COPD patients with metabolic syndrome had more exacerbations over an 18-month period [31].

The FM level reflects an energetic adequacy of nutrition as well as correlates with physical activity and adaptive facilities of the body. A stable body mass during one year shows the presence of this balance. A shift in body composition towards a relative abundance of FM and reduced FFM (associated with worsening of lung function) in COPD was revealed. This shift was increased during COPD exacerbation due to: increased hypoxemia and sympathetic tone, more frequent use of β2-agonists and systemic glucocorticoids (GC) as well as reduced food intake. Decreased FFM is not always accompanied by a loss of BMI. Skeletal muscle accounts for 1/2 of the total body mass in a male with normal body weight. Skeletal muscle weakness in COPD is often accompanied by loss of FFM that is an indirect measure of muscle mass.

Thus, decreased BMI in COPD patients negatively influences the changes of the skeletal muscles including respiratory muscles [32-34]. All this indirectly promotes the development of muscle fatigue, decrease in physical activity and resistance to infection in COPD patients.

Hormonal changes are also closely linked to protein turnover. Thus, insulin suppresses the breakdown of protein, while growth hormone increases FFM and generates a positive nitrogen balance as well as a depletion of FM, insulin-like growth factors and anabolic hormones favor protein synthesis, whereas GC stimulates proteolysis. Thus, sex hormones take part in the formation and distribution of fat and muscular masses. Gradually decreasing levels of circulated androgens are revealed in males with age. It is related to increased levels of total and abdominal FM [35]. The serum level of testosterone has a negative correlation with body FM [36]. Trunk FM (disproportionately higher LM than FM) may have a direct mechanical effect on diaphragmatic position and its movement as well as on chest wall recoil that may increase airway responsiveness [37].

The role of adipokines (APN) – leptin and adiponectin – in the pathophysiology of COPD has recently been considered. Adipose tissue produces these APN which may also play a key role in inflammatory pulmonary diseases as well as in the relationship between lung pathology and adipose tissues [38]. Thus, APN significantly influence the inflammatory process, immune response, lipid metabolism, insulin susceptibility, angiogenesis and regulation of energy balance [39-40]. Pro-inflammatory effects

of APN (expressed by visceral fat components) can dominate under certain conditions.

Pro-inflammatory APN may have a direct access to portal and systemic circulation, influencing disproportionately a greater inflammatory effect of visceral fat relative to its small size. Furthermore, visceral fat (like intramuscular fat) may be associated with high systemic leptin and TNF-α concentrations and a low level of systemic adiponectin [41-42]. Additionally, appetite and nutrition intake were decreased due to leptin action whereas glucose and fat metabolism were increased [6, 43-44].

It was observed in normal subjects that TNF-α , IL-1, GC induce increased leptin level and decreased adiponectin level. But in COPD patients' false illusion of body positive energy balance may stimulate decreased appetite [15]. Leptin-resistance is common in overweight COPD patients. Decreased effect of leptin level in COPD may be associated with leptin binding with C-reactive protein.

Visceral obesity is associated [45, 46] with a high level of proinflammatory cytokines. Thus, a negative association between serum leptin level and the markers of systemic inflammation was revealed. The increased level of TNF-α and leptin in blood circulation favors decreased appetite and food intake in COPD patients, mostly with severe hypoxia [6]. Additionally TNF-α may decrease the body mass and all its components.

Leptin and other APN as well as their receptors are expressed in the human lung. The association between systemic or airway leptin and COPD in adults is currently controversial. Thus, hypoxia in COPD induces increasing leptin expression and decreasing appetite [6, 44]. Both leptin deficiency and resistance result in lung hypoventilation [47].

Decreased leptin level in COPD patients with low BMI compared with the patients with normal nutritional status as well as in patients with emphysematic phenotype compared with bronchitic ones has been shown in several studies [6, 7, 48]. Other researchers [49] detected the increase in the leptin level, TNF-α and IL-1 against the background of decreased adiponectin level in overweight COPD patients. Systemic and airway leptin concentrations may also be disproportionately higher in COPD patients and reflect a greater airway inflammation as well as COPD severity [50].

Currently, more attention is given to regional body distribution of FM and LM. Thus, FM deposited in the abdominal region in male subjects (due to genetic and hormonal factors, smoking, alcohol abuse and stress) results in android obesity («apple» type) with more frequent comorbid diseases. The share of FM in android and gynoid regions makes up ≈51% and ≈40% and their ratio is ≈1,3 in the norm [51].

It is well known, that accumulation of visceral FM is associated with the development of insulin-resistance [45]. The latter was often observed in COPD patients and was increased during exacerbation [52]. Not infrequently in COPD the control of lipolysis is altered by hyperinsulemia due to a greater insulin resistance (that is related to a systemic inflammation) even in normal weight COPD patients. It was detected [45, 53] that accumulation of FM in the upper abdominal region was often combined with the development of insulin-resistance, hyperinsulinemia and dyslipidemia compared with accumulation of FM in the lower abdominal region (gynoid type). Accumulation of intramuscular FM induces both insulin-resistance and muscular dystrophy [54].

Additionally, abdominal obesity negatively influences the pulmonary function [47]. Thus, the study of body composition is necessary for the complex evaluation of COPD patient's status and their following individualized treatment. Increase in the muscular and bone tissue is a significant part of the therapeutic target in the rehabilitation programs of COPD patients in advanced stages.

The main **purpose** of study was to evaluate various regional distribution of body FM and LM, the relationship between the latter and TNF-α and leptin as well as free testosterone level in relation to COPD development. This is the first study to evaluate various distributions of FM and LM in relation to COPD development in males.

Material and methods

The study was undertaken at the Pulmonology Department of the 10th Minsk Clinical Hospital in the years 2009 to 2013. All participants (from the general population of Minsk) gave their written informed consent. The study protocol was approved by the Human Studies Committee on Research Ethics at the Belarusian State Medical University. The inclusion criteria for the study were established before the trial and were strictly followed: males aged 40-69 years with different COPD grades and FEV1 increase <15% during bronchodilation tests were qualified for the study. Exclusion criteria included substantial uncontrolled co-morbidities. The study group consisted of 82 COPD patients (aged 40-67 years) with acute exacerbation of COPD (increased wheezing, dyspnea, sputum volume or sputum purulence one week prior to admission) of varying severity. These patients and the control group consisting of 16 healthy persons of comparable age, sex, smoking status and BMI were examined. Self-reported information was obtained from all patients using conventional questionnaires. Special emphasis was placed on the patient's history of chronic and progressive symptoms such as dyspnea, cough and wheezing as well as on smoking history. Thus, smoking status was divided into three variable categories: current, former and never-smokers. Most of the patients were current smokers or ex-smokers (who had stopped smoking at least 12

months before evaluation) but some of them were nonsmokers. The diagnosis of COPD and its severity was based on the GOLD guidelines (2010).

Additionally, current COPD was verified by patient self-report about diagnosed COPD plus either presence of current COPD symptoms during the previous 12 months or a patient report of current use of COPD medications. The expressiveness of dyspnea (as an indirect indicator of decreased exercise tolerance) was defined according to a modified Medical Research Council (MRC) questionnaire.

The patients were divided into three groups according to the severity of their COPD, which reflected the evolution of disease (Table 1). The first group ($COPD_1$) consisted of 19 patients with mild COPD (median age and duration of disease: 55 and 3 years respectively; FEV1: 82%; BMI: 27 kg/m^2; current smokers: 68%, ex-smokers: 17%, and nonsmokers: 15%). The second group, with moderate COPD ($COPD_2$) was composed of 43 patients (median age and duration of disease: 57 and 9 years respectively; FEV1: 56%; BMI: 30,3 kg/m^2; current, ex- and nonsmokers: 77%, 10% and 13% respectively). The third group, with severe COPD ($COPD_3$) was made up of 20 patients (median age and duration of disease: 63 and 13 years respectively; FEV1: 33%; BMI: 22,9 kg/m^2; current and ex-smokers: 96% and 4%).

Table 1. Baseline characteristics of the control group and COPD patients groups according to different degrees of airflow limitation severity (GOLD spirometry level, 2010) on admission (Me; 25;75)

Parameters	Control (n=16)	COPD1 (n=19)	COPD2 (n=43)	COPD3 (n=20)
Median age (years): < 60/ > 60 years (%)	51 (47; 55) 73/27	55 (51; 58) 79/21	57 (53; 60)* 66/34	63(56; 67)*,**,*** 50/50
Body mass index (kg/m^2)	28,3 (27,5; 29,1)	29,0 (27; 32,5)	30,3 * (26,4; 33,2)	22,9*, **,*** (20,7; 27,7)
Present smokers (%)	60	68	77	96*,**
Smoking history, packs/years index	17 (10; 30)	20 (12; 30)	21 (8; 30)	30 *,**,*** (25; 40)
Median duration of COPD (years)	–	3 (1; 6)	9 (4; 12)	13**,*** (10; 19)
Number of exacerbations in the last year	–	1 (1; 2)	2 (1; 3)	3**,*** (2; 4)
MRC Dyspnea Index (score)	–	0 (0; 1)	2 (1; 3)	3**,*** (2; 3)
FVC (% pred.)	100 98; 112)	86* (75; 90)	57 *,** (48; 66)	39 *,**,*** (32; 46)
FEV1 (% pred.)	88 (77; 94)	82 * (78; 87)	56 *,** (50; 65)	33 *,**,*** (22; 44)
FEV1/FVC (%)	98 (89; 104)	70 * (62; 72)	67 *,** (57; 75)	51 *,**,*** (39; 58)
SpO_2 (%)	97 (96; 97)	97 (96; 97)	96 (95; 97)	93 *,**,*** (90; 95)
C-reactive protein (mg/dL)	0,4 (0,1; 2,0)	0,3 (0,1; 1,2)	4,3*,** (1,9; 8,3)	3,3 *,** (1,3; 5,3)

** – p<0,05 vs. the control group; ** – p<0,05 vs. COPD1; *** – p<0,05 vs. COPD2 group.*

As shown in Table 1, the control group (with normal pulmonary function) did not differ from $COPD_1$ patients in terms of median age, the percent of patients under 60 years, BMI and intensity of smoking. Compared with the controls, $COPD_1$ patients had only decreased ventilation parameters, while $COPD_3$ patients had a significantly higher median age, disease duration, MRC breathlessness index and the number of current smokers than the control group and $COPD_{1,2}$ patients, whereas mean BMI of $COPD_3$ group was significantly lower than in the latter.

As Table 1 shows, a significant increase in the number of exacerbations for the past year was found in severe COPD compared with mild to moderate COPD. A progressive decrease in ventilation parameters was noted as the disease developed: FVC dropped from 86% in $COPD_1$ to 39% in $COPD_3$; FEV1 and blood oxygen saturation dropped from 82% to 33% and from 97% to 93% respectively.

The patients (mostly $COPD_{2–3}$) were treated in the hospital with a combination of inhaled long-acting anticholinergic (or β_2-agonists) plus inhaled and sometimes systemic (intermittent/continuous) corticosteroid (5 days of dexametasone intravenously, 4 mg a day) as well as antibiotics if there was clinical evidence of Type 1 or 2 exacerbation according to Anthoninsen criteria. A short acting inhaled bronchodilatator was provided for rescue case.

The detailed clinical investigation of these patients included: smoking status, self-reported co-morbidities and previous treatment, respiratory spirometry, physical activities of daily living, physical examination, day-time pulse oximetry, chest radiography and blood laboratory analyses. TNF-α, leptin and free testosterone levels were detected by immynoenzyme (ELISA) methods.

Distribution of body FM for the whole body (total) and its regions (i.e. trunk, arms, legs) were measured by DEXA. We calculated: fat mass index (FMI=FM/growth^2); lean mass index (LMI=LM/growth^2) that reflects nonfat and nonbone soft tissue content; skeletal muscle index (SMI=fat free mass of arms+legs/growth^2), fat-free mass index (FFMI=free fat mass of body/growth^2) and bone mineral component index (BMCI=bone mineral component of body/growth^2). BMI=FMI+LMI+BMCI. DEXA assessed not only skeletal muscle and visceral mass but also fat deposits present within and around muscle and viscera.

The statistical analysis was performed using Statistica 8.0 software (2007, Statsoft Inc., USA). The preliminary analysis of the variables under consideration was a Shapiro-Wilk test of correspondence to normal distribution. The results of the analysis were shown as median and interquartile range (25-75%) because all the parameters differed from normal distribution. The comparison of non-parametric parameters in two independent groups was carried out using a Mann-Whitney test, while in three or more independ-

ent groups it was performed using the Kruskal-Wallis rank sum test. Fisher's exact test was used to detect statistically significant differences between independent groups according to the frequency characteristic of the investigated parameter. Spearman's rank correlation coefficient (rs) was used to describe the relationship between the two quantitative variables that differed from normal distribution. The level of statistical significance was set at p <0,05.

Results

The distribution of COPD patients with different severity of disease including their BMI level is presented in Fig. As seen from it, the part of BMI with the normal range was increased in the course of COPD development. So, it was detected in 10%, 20% and 58% of $COPD_{1,2,3}$ patients respectively. We revealed significant difference between $COPD_1$ and $COPD_2$ as well as between $COPD_2$ and $COPD_3$. Overweight and obesity were naturally decreased as COPD became worse. Thus, overweight was detected in 50%, 28% and 16% of $COPD_{1,2,3}$ patients respectively against the background of significant difference between $COPD_1$ and $COPD_2$ as well as between $COPD_2$ and $COPD_3$.

We revealed that the number of the patients with the decreased LMI level (Table 2) was higher among the patients with normal BMI in the course of COPD progression. We did not detect these patients in $COPD_1$ group, whereas 1 and 6 such patients in $COPD_{2,3}$ were revealed respectively. A significant difference between $COPD_2$ and $COPD_3$ was shown.

Meanwhile decreasing LMI was detected in 30% of $COPD_3$ patients against the background of normal values of BMI. The latter (Table 1) was decreased during COPD development but meanwhile it was in the normal range (or increased to some extent) in most of the observed patients. This phenomenon was detected against the background of significant increase in the share of the patients with deficiency of LM (represented substantially by muscular tissues).

Significantly decreased FMI, LMI, BMCI and SMI (Table 3) were revealed only in moderate to severe COPD patients. Significant difference between $COPD_1$ and $COPD_2$ according to levels of FMI and BMCI was

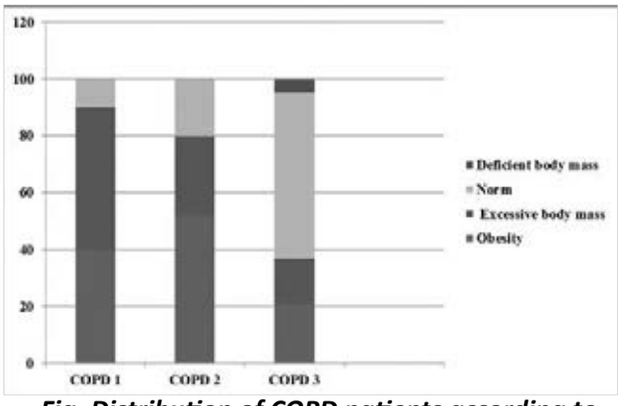

Fig. Distribution of COPD patients according to their BMI

Table 2. Distribution of COPD patients with different severity according to degree of BMI and LMI (%)

BMI (kg/м²)	$COPD_1$		$COPD_2$		$COPD_3$	
	LMI (kg/m²)					
	<16,6	>16,6	<16,6	>16,6	<16,6	>16,6
Deficiency (BMI <18,5)	0	0	0	0	5	0
Normal(BMI ≥18,5 – 24,9)	0	10	2	17	30**	26
Overweight (BMI = 25,0 – 29,9)	0	50	2	22*	0	16*
Obesity (BMI ≥30,0)	0	40	0	56	0	21*,**

** – p<0,05 vs. COPD1; ** – p<0,05 vs. COPD2 group.*

Table 3. Dynamics of FMI, LMI and BMCI levels (kg/m²) among males according to COPD severity (Me; 25%,75%)

Parameter	Control (n=16)	$COPD_1$ (n=19)	$COPD_2$ (n=43)	$COPD_3$ (n=20)
FMI	7,0 (6,2; 7,7)	8,3* (6,2; 10,1)	9,7* (6,8; 11,0)	4,7*,**,*** (3,1; 9,3)
LMI	20,6 (19,9; 22,0)	20,8 (20,1; 21,4)	20,1** (18,8; 21,2)	17,7*,**,*** (16,5; 18,9)
BMCI	1,09 (0,99; 1,12)	1,04 (0,97; 1,08)	1,01 (0,92; 1,08)	0,89*,**,*** (0,83; 0,94)
SMI	9,37 (8,76; 9,70)	9,41 (8,81; 9,74)	8,92*,** (8,05; 9,51)	7,61*,**,*** (7,13; 8,38)

** – p<0,05 vs. the control group; ** – p<0,05 vs. COPD1; *** – p<0,05 vs. COPD2 group.*

Table 4. The share of FM in different body parts in COPD patients with various disease severity (Me; 25%,75%)

Region	Control (n=16)	$COPD_1$ (n=19)	$COPD_2$ (n=43)	$COPD_3$ (n=20)
Arms	19,8 (15,7; 22,0)	21,3* (17,6; 25,5)	25,9*,** (21,4; 29,0)	18,2*** (13,2; 30,0)
Legs	19,9 (17,2; 23,8)	24,9* (22,3; 29,2)	26,1* (22,9; 31,0)	18,7**,*** (16,6; 28,6)
Trunk	29,5 (27,1; 34,2)	34,6 (31,8; 37,6)	36,7* (31,0; 39,2)	29,2**,*** (16,3; 35,9)
Android	35,3 (32,1; 38,9)	40,1 (31,5; 42,1)	41,7* (36,2; 46,1)	32,6**,*** (19,1; 41,2)
Gynoid	26,1 (23,2; 32,6)	28,9 (26,4; 33,1)	31,2* (27,3; 35,8)	25,9*** (20,7; 33,2)

** – p<0,05 vs. the control group; ** – p<0,05 vs. COPD1; *** – p<0,05 vs. COPD2 group.*

not detected. FMI was increased in $COPD_{1,2}$ whereas in $COPD_3$ it was significantly decreased compared with $COPD_{1,2}$. Thus, the levels of LMI and BMCI were decreased in $COPD_3$ compared with the control.

More changes of FM were detected in the trunk and android regions and fever – in legs and arms (Table 4). It was shown that the changes of FM in the abovementioned body regions were similar and had the phased character. Thus, we detected increased FM share of all body regions in $COPD_{1,2}$ groups, while in $COPD_3$ there was decreased FM share below the control group level. The significant differences according to these parameters were revealed between $COPD_{1,2}$ and $COPD_3$ as well as between $COPD_2$ and the control.

Deposition of FM in definite body regions (par-

ticularly in android and trunk) in COPD patients may be associated with certain co-morbidities, hypertension and coronary heart disease (CHD) being the most prevalent ones in COPD. Therefore we additionally assessed the influence of the body FM distribution upon the presence of these two frequent COPD co-morbidities.

Thus, we detected an increased number of COPD patients with concomitant hypertension: 41%, 81% and 74% in $COPD_{1,2,3}$ groups respectively in the course of COPD development. There were significant differences between COPD1 and $COPD_{2,3}$ groups according to the share of patients with concomitant hypertension (χ^2=12,52, p<0,05 and χ2=3,68, p<0,05 respectively). We revealed (Table 5) significant difference of all the parameters that characterized distribution of body FM in COPD patients with hypertension compared with COPD isolated patients.

These two groups did not differ according to age (58 and 57,5 years), while BMI was higher in COPD plus hypertension group compared with COPD isolated group (30 and 25,3 kg/m^2 respectively; p<0,05). A similar picture was observed concerning the levels of triglycerides (1,59 and 1,16 mmol/l respectively; p<0,05) as well as glucose (5,7 and 5,2 mmol/l respectively; p<0,05). Table 5 shows that the central type of FM deposition (with excess FM accumulation in android region) prevails in patients with COPD plus hypertension.

We revealed a significantly increased share of COPD patients with concomitant CHD during COPD development: from 18% in COPD1 to 61% in $COPD_2$ and to 75% in $COPD_3$. There were significant differences according to CHD prevalence between COPD1 and $COPD_{2,3}$ (χ^2=10,47, p<0,05 and χ^2=13,65, p<0,05 respectively). However, we did not detect significant differences of the parameters that characterized distribution of body FM between these two COPD groups (with CHD and without it).

The leptin level was increased during COPD development (Table 6): by 18%, 75% and 79% in $COPD_{1,2,3}$ groups respectively compared with the control (p<0,05 for $COPD_{2,3}$). Similar dynamics was detected concerning TNF-α level. It was increased by 21%, 52% and 64% in $COPD_{1,2,3}$ groups respectively compared with the control (p<0,05 for $COPD_{2,3}$). The level of free testosterone was significantly decreased in the course of disease development: from 7,6 and 7,4 pg/ml in $COPD_{1,2}$ groups to 5,2 pg/ml in $COPD_3$.

Table 5. Distribution of body FM in different body regions and their relations in COPD patients with/without hypertension (Me; 25%,75%)

Region	COPD isolated, (n=26)	COPD with hypertension, (n=59)
Arms (%)	18,7 (13,9; 24,6)	24,9* (20,0; 29,0)
Legs (%)	21,5 (16,6; 25,9)	26,1* (22,5; 31,0)
Trunk (%)	28,8 (16,9; 35,0)	36,2* (32,1; 39,2)
Android (%)	31,4 (22,3; 40,6)	41,4* (36,2; 45,2)
Gynoid (%)	26,8 (21,1; 31,9)	30,2 (26,8; 34,4)
Total level of FM (%)	25,8 (17,3; 30,7)	31,1* (26,9; 34,5)
Total level of LM (%)	74,2 (69,3; 82,7)	69,3* (65,5; 72,5)
Android/gynoid regions	1,12 (1,04; 1,27)	1,28* (1,18; 1,42)
Trunk/ total FM	0,56 (0,54; 0,60)	0,61* (0,58; 0,65)
Legs/total FM	0,30 (0,26; 0,33)	0,26* (0,24; 0,29)
Arms+legs/trunk	0,71 (0,60; 0,80)	0,59* (0,51; 0,67)

** – p<0,05 between these groups*

Discussion

Analyses showed significant changes of body composition during COPD development. Thus, significant decrease in BMI value as well as the proportion of patients with overweight and obesity against the background of increased patients share with normal BMI in the course of disease development was detected.

The level of FMI was increased in $COPD_{1,2}$ and decreased in $COPD_3$ group. Similar changes were revealed according to the analysis of FM distribution in all the studied body regions. Thus, no peculiarities of body FM distribution in separate regions were detected. The levels of LMI, BMCI and SMI were decreased in the course of COPD progression. The depletion of FFM seems largely related to the increased level of systemic inflammation in severe COPD. The decrease in SMI was similar to LMI dynamics during COPD development. Thus, there was a positive correlation between LMI and SMI (ρ=0,87, p<0,05). These data show that SMI and LMI are interrelated and reflect the decrease in skeletal muscles component during COPD development.

The loss of muscle mass was aggravated by FM accumulation. Fatty muscle infiltration leads to decreased muscle strength and inadequate functioning. We revealed that the level of BMI did not give complete information about disorders of nutritional status in male COPD patients because BMI remained within the normal range (or slightly increased) in most of them. Thus, increased FMI in mild-to-moderate COPD patients ($COPD_{1,2}$ groups) could mask the loss of muscular and bone tissues against the background of the normal values of BMI.

We cannot detect characteristic changes of LMI and BMCI in mild-to-moderate COPD. Probably, DEXA method assessed not entirely fat-free LM but included smaller and highly metabolically active ectopic

Table 6. Dynamics of leptin, TNF-α and free testosterone levels during COPD development (Me; 25%,75%)

Parameter	Control (n=11)	$COPD_1$ (n=22)	$COPD_2$ (n=29)	$COPD_3$ (n=18)
Leptin, ng/ml	1,54 (1,53; 1,60)	1,82* (1,62; 2,20)	2,74*,** (2,32; 3,37)	2,80 *, **,*** (2,24; 3,39)
TNF-α, ng/ml	8,6 (6,3; 12,0)	10,1 (4,97; 21,8)	13,6*,** (10,4; 23,6)	14,7*,** (10,0; 71,8)
Free testosterone, pg/ml	10,5 (6,8; 12,3)	7,6* (3,7; 9,1)	7,4* (3,4; 10,0)	5,2*,** (2,8; 8,7)

** – p<0,05 vs. the control group; ** – p<0,05 vs. $COPD_1$; *** – p<0,05 vs. $COPD_2$ group*

FM within the skeletal muscle and viscera. Therefore, our findings in respect to body composition in overweight COPD men could be to a certain extent explained by the excess of ectopic FM. Additionally, intramuscular fat infiltration was more pronounced in moderate-to-severe COPD patients [55].

The absence of significant differences between the control group and mild to moderate COPD according to the distribution of FM in arms, legs and trunk as well as the presence of these differences in severe COPD could be due to the fact that skeletal muscle in different body regions may have different metabolic and inflammatory effects in advanced grade of disease. Another possible explanation of this phenomenon is that smaller ectopic fat deposits (the components assessed by DEXA as LM) became more important in severe COPD (due to higher inflammatory status) than larger and physiological fat deposits.

Additionally, we performed the correlation analysis of relative contributions of FM and LM for better understanding of their possible role in the development of the disorders of pulmonary function in COPD. The presence of the interrelationship between LMI, BMCI and certain parameters of ventilation was found. Thus, we detected moderate positive significant associations between BMCI and FEV1 and FVC (ρ=0,39; 0,33 respectively) as well as between LMI and FVC, FEV1 and their ratio (ρ=0,30; 0,34 and 0,30 respectively). Additionally, we revealed significant moderate negative associations between LMI, BMCI and dyspnea expressiveness (ρ=-0,44 and -0,36 respectively). Thus, these associations indicated that decreased muscular and bone tissue components of patient's body were combined with the disorders of lung function and lowered tolerance to physical activity. Surprisingly, but SCI did not correlate with the parameters of ventilation.

The revealed associations between some indices of body composition and definite laboratory tests (TNF-α, leptin, free testosterone) verified the systemic character of pathologic processes in COPD. The presence of positive correlation between FMI and leptin level could be explained by the association of intramuscular fat with high leptin and low adiponectin concentrations [56-57] as well as increased expression of TNF-α [58]. Thus, we detected a negative moderate correlation (ρ=-0,38; p<0,05) between free testosterone level and FMI as well as a positive correlation between leptin and FMI (ρ=0,32; p<0,05), while LMI was correlated with MRC score (ρ=-0,44; p<0,05). BMCI had a significant association with SpO2 and MRC score (ρ=0,46 and ρ=-0,36 respectively). These data emphasize a negative influence of severe hypoxia on muscular and bone tissue components of COPD male body.

Additionally, in acute exacerbation of COPD we detected a negative correlation between free testosterone level and total FM (ρ=-0,34, p<0,05) as well as FM of arms (ρ=-0,35, p<0,05). These results correspond to other reports which show decreased free testosterone level against the background of increased body FM in COPD men [35, 36, 59]. Possible explanations of our data may also include the effects of sex and gonadotrophic hormones, sex-specific effects of adipokines and intramuscular lipid deposition.

The intermittent treatment with systemic steroids was used only in one moderate COPD patient and in three severe COPD patients. Therefore, we do not think that our findings can be partially explained by the effect of systemic steroids on the repartition of FM and LM.

We expected to find more changes of FM in the upper part of the body taking into account higher mobility of this region in moderate to severe COPD patients. However we did not detect that FM of arms (which constitutes ≈13% of total FM) is a better indicator for COPD severity (among the patients with $COPD_{1,2}$) evaluation than other larger fat compartments of the body. It is known, that FM of arms may have a different metabolic profile compared with trunk FM or FM of legs. Thus, FM of legs (also increased in $COPD_{1,2}$) may enhance serum adiponectin concentrations and be «metabolically benign and protective» relative to other fat compartments.

We revealed increased concentrations of leptin and TNF-α against the background of free testosterone level decrease during COPD development that corresponds with the results of other scientists [35, 60]. Similar changes of leptin and TNF-α levels in total COPD group were noted (n=91, p=0,32, p<0,05), which may be due to the production of these factors by fat tissue.

A high value of BMI is a collection of various phenotypes, some (but not all) relating to adiposity. Various distributions of FM and skeletal muscle may have varying mechanical and inflammatory properties and consequent COPD-related effects. For instance, trunk FM (that constitutes the metabolic syndrome) may carry a higher risk than arm or leg fat for COPD patients. Further, trunk FM may have a greater mechanical impact on FVC than FM of arms or legs and this effect may be important in COPD pathogenesis.

Trunk FM includes two subcomponents: superficial abdominal subcutaneous fat (that is metabolically benign) and deep abdominal subcutaneous fat that is metabolically active (similarly to visceral fat). DEXA cannot separate these two subcomponents and inaccurately assesses ectopic fat as LM. Trunk FM is a collection of fat compartments with varying metabolic activity, including visceral, deep abdominal wall subcutaneous and superficial abdominal wall subcutaneous fat. Among these trunk FM components, visceral fat most strongly correlates with characteristic features of the metabolic syndrome. Abdominal subcutaneous and visceral FM may be a relatively more important source of leptin and adiponectin respectively. Intramuscular FM is strongly

associated with insulin resistance and low serum concentrations of adiponectin, although its relationships in COPD are not known.

The accumulation of FM in android region is associated with visceral obesity. The increase in android/gynoid ratio shows the prevalence of FM accumulation in the abdominal region. In some cases we detected central (in the trunk region) and peripheral types (less) of obesity in these COPD patients. The presence of central type of obesity is confirmed by the increase of FM ratio in the trunk region to total FM and decrease level FM of legs to total FM as well as the ratio of FM level in arms plus legs to trunk FM.

Conclusions

The changes in the body composition can be clinically recognized by weight loss in general and particularly by loss in LM during the development of COPD. BMI does not give complete information about disorders of the body composition in male COPD patients. Thus, BMI was within the normal range (or was even increased) in many of mild-to moderate COPD patients. The levels of LMI and BMCI were decreased only in severe COPD, while FMI was increased in mild-to-moderate COPD and then it was decreased in severe COPD. Severe COPD was associated with decreased share of FM in the arms, legs, trunk as well as in android and gynoid regions.

The central type a distribution of FM in the male COPD patients is associated with hypertension.

The levels of leptin and TNF-α were increased during COPD development, while the level of free testosterone was decreased. The presence of associations between these parameters and indices of the body composition indicated the systemic character of pathologic processes taking place during COPD development.

Conflict of interests

The authors no conflicts of interests.

Financial disclosure

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НАРУШЕНИЯ КОМПОНЕНТНОГО СОСТАВА ТЕЛА У МУЖЧИН С ХРОНИЧЕСКОЙ ОБСТРУКТИВНОЙ БОЛЕЗНЬЮ ЛЕГКИХ

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Резюме

В статье приведены результаты исследования компонентного состава тела у мужчин с хронической обструктивной болезнью легких (ХОБЛ). В ходе прогрессирования ХОБЛ выявлено снижение следующих индексов: безжировой массы тела, костного минерального и скелетного мышечного. Индекс жировой массы, первоначально нарастая, снижался на третьей стадии заболевания. Определено, что характер распределения жировой ткани при ХОБЛ у мужчин играет роль в развитии артериальной гипертензии.

Ключевые слова: хроническая обструктивная болезнь легких, компонентный состав тела, жировая масса, костная масса, лептин.