время беременности: недоношенностью, декомпенсированной фетоплацентарной недостаточностью, прогрессирующей гипоксией плода. Во второй группе ГИЭ I и ГИЭ II выявлена у 8,6%, что также можно связать с осложнениями во время беременности: декомпенсированной фетоплацентарной недостаточностью, прогрессирующей гипоксией плода.

Выводы

Человечество решило проблему лечения бесплодия с помощью внедрения методов вспомогательных репродуктивных технологий. В настоящее время появились проблемы вынашивания беременных после экстракорпорального оплодотворения и не всегда мы можем получить рождение здорового ребенка без ухудшения состояния здоровья будущей мамы. Так, по нашему ретроспективному анализу мы получили, что беременность после ВРТ может осложниться фетоплацентарной недостаточностью, развитием преэклампсии, рождением детей в асфиксии, что является мировой проблемой. Необходимо оценить факторы, в результате которых развиваются осложнения беременности после ВРТ и профилактировать данные осложнения.

Литература

2. Балтович А.В., Ермаков Н. В., Ульянов и др. Проблемы беременности после ЭКО. Научный вестник Тюменской медицинской академии, 1999, № 1.
3. Брауде П. Оплодотворение ооцитов человека и культивирование преимплантационных эмбрионов человека. Биология развития млекопитающих. Редактор М. Манн. М.: Мир, 1990.
4. Кулаков В.И., Бахтиаров О.В., Леонов Б.В. Оценка здоровья детей, рожденных в результате ЭКО и искусственной инсеминации. Акушерство и гинекология, 1995, № 3.
5. Курцер М.А., Краснопольская К.В., Ароян Р.Н. Перинатальные исходы пациентов после ЭКО. Акушерство и гинекология № 2, 2003.
6. Леонов Б.В., Кулаков В.И. Общая характеристика ЭКО и переноса эмбрионов. Кн.: ЭКО и новые направления в лечении женского и мужского бесплодия (теоретические и практические подходы). Под редакцией В.И. Кулаков. М.: Мед. Информ. Агентство; 2000.
7. Новицкая Н.И. Диссертация: Курс беременности и перинатальных исходов после оплодотворения в пробирке, 2008.

References

1. Baltovich A. V., Ermakov N. In., Ulyanov, etc the Problems of pregnancy after IVF. Scientific Bulletin of the Tyumen Medical Academy, 1999, No. 1.
2. Braude P. Fertilization of human oocytes and culturing human preimplantation embryos. Developmental biology of mammals. Ed. By M. Mann. M.: Mir, 1990.
3. Kulakov V. I., Bakhtiarov O. V., Leonov B. V. Assessment of the health of children born resulting from IVF and artificial insemination. Obstetrics and gynecology, 1995, No. 3.
4. Kurtser M. A., Krasnopolskaya K. V., Aroian P. H. Perinatal outcomes of patients after IVF. Obstetrics and gynecology No. 2, 2003.
5. Leonov B. V., Kulakov V. I. General characteristics of the IVF and embryo transfer. KN.: ECO and new directions in the treatment of female and male infertility (theoretical and practical approaches). Edited by V.I. Kulakov. M.: Med. Inform. Agency; 2000.
6. Novitskaya N.I., Dissertation: the Course of pregnancy and perinatal outcomes after in vitro fertilization, 2008.

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Исследование накопления висцерального жира у пациентов с хронической обструктивной болезнью легких

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Целью данного исследования было изучение брюшного накопления висцерального жира и связь между висцеральным жиром и тяжестью болезни ХОБЛ. Мы провели клинические и лабораторные тесты, в том числе тест легочной функции у пациентов с ХОБЛ (n = 188) и контрольной группы, в который вошли больные без обструкции (n = 48). Мы использовали массовый характер состава тела, чтобы оценить индекс массы тела (ИМТ), массы скелетных мышц (СММ), брюшного висцерального жира (VF) и подкожного жира (СФ). Группа ХОБЛ имела большую VF площадь, чем в контрольной группе. Распространенность нетучных субъектов с повышенным В.Ф. было больше в Глобальной Перво-TIVE при хронических стадиях обструктивной болезни легких I и II, чем в других стадиях ХОБЛ.
Ключевые слова: абдоминальное ожирение, хроническая обструктивная болезнь легких, висцеральный жир.

Investigation of visceral fat accumulation in patients with chronic obstructive pulmonary disease

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The aim of this study was to investigate the abdominal visceral fat accumulation and the association between visceral fat and the severity in COPD patients. We performed clinical and laboratory tests, including pulmonary function test in COPD patients (n = 188) and control group, which included subjects without airflow obstruction (n = 48). We used body mass composition scale to evaluate the body mass index (BMI), skeletal muscle mass (SMM), abdominal visceral fat (VF) and subcutaneous fat (SF). The COPD group had a larger VF area than the control group. The prevalence of non-obese subjects with an increased VF was greater in the Global Initiative for Chronic Obstructive Lung Disease Stages I and II than in the other stages of COPD.
Keywords: abdominal obesity, chronic obstructive pulmonary disease, visceral fat

Introduction

Chronic obstructive pulmonary disease (COPD) is a disease with systemic comorbid conditions including hypertension, diabetes mellitus, and cardiovascular diseases due to systemic inflammation [1,5]. Cardiovascular disease, which is often related to obesity, accounts for approximately 30% of mortality in more advanced COPD patients who tend to have muscle loss [10].

The accumulation of visceral fat causes endocrine disturbances such as adipokine dysregulation [9]. Features of acute-phase activation and low-grade inflammation, including elevated levels of fibrinogen, C-reactive protein, and interleukin (IL)-6, are particularly associated with central or visceral obesity. High levels of IL-6 or tumor necrosis factor-α and low levels of adiponectin are observed when there is an excess accumulation of visceral fat [14].

The aim of this study was to investigate the abdominal visceral fat accumulation and the association between visceral fat and the severity in COPD patients.

Methods

Patients with COPD were consecutively recruited to the study in 2008, 2010 and 2012, at the Pulmonology Department of First National Central Hospital (Ulaanbaatar, Mongolia). All patients classified into four stages according to the American Thoracic Society/European Respiratory Society guidelines [9].

Exclusion criteria were respiratory disorders other than the COPD, malignancy, overt cardiac failure, recent surgery, severe endocrine, hepatic or renal diseases. The control group included 48 healthy persons with similar ages, having normal pulmonary function tests. Pulmonary functional tests were evaluated by using of spirometer ST-320 (Mitsubishi, Japan). All pulmonary function tests were performed at the 10-15 minute after inhaling short-term β2-agonist Salbutamol in dosage 250 mcg. Forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) were expressed as a percentage of the predicted value for age, sex, and height. Three technically acceptable measurements were performed in each patient, and the highest value was included in the analyses. PaO2 was correlated with an oxygen saturation (SaO2), what was measured by finger pulse-oxymeter and expressed as a percentage

Body height and weight were measured (BWB-800 and Rainbow scale, respectively; Tanita Co, Tokyo, Japan). Body mass index (BMI) was calculated as the ratio of body weight to height (m2). The criteria for BMI classification of the World Health Organization (WHO) for the Study of Obesity were applied as follows: BMI , 18.5 kg/m2, underweight; 18.5 kg/m2 # BMI , 25 kg/m2, normal weight; 25 kg/m2 # BMI , 30 kg/m2, obese; and BMI $ 30 kg/m2, severely obese. Fat-free mass (FFM), subcutaneous fat (SF) and visceral fat (VF) were measured by bioelectrical impedance analysis at frequencies of 5, 50, 250, and 500

kHz (HBF-361 KaradaScan, Omron, Tokio, Japan). The FFM index (FFMI) and FM index (FMI) were calculated as the ratio of FFM or FM to height (m2), respectively.

Blood samples were collected from an antecubital vein after overnight fasting; serum triglyceride, high-density lipoprotein (HDL)-cholesterol, and fasting blood glucose levels were measured.

Statistical analysis was carried out using SPSS 20. Continuous variables are shown as means ± S.E.M. To assess the relationships between selected variables, linear regression analyses was used. A p – value less than 0.05 ($P<0.05$) was considered as significant.

Results

The COPD group (n = 188) had a higher smoking index than the control group (n = 48). No significant differences in BMI or FFMI were observed between the two groups. The VF was significantly greater in the COPD group than in the control group ($P = 0.023$). No difference in SF was observed between the two groups ($P = 0.460$) (Table 1).

Table 1 Subject characteristics

	Control	COPD	*P* value
Subjects (n)	48	188	–
Age (years)	59.9±5.6	59.32±5.5	0.126
Smoking index (pack-years)	11.3±2.2	21.7±16.5	0.004
FEV1/FVC (%)	0.83±0.11	0.62±0.08	0.0001
FEV1% predicted (%)	83.98±11.77	57.82±17.07	0.0001
PaO2 (mmHg)	89.9±3.4	69.4±14.2	0.0001
BMI (kg/m2)	27.2±4.4	26.9±6.1	0.451
FFMI (kg/m2)	32.7±5.2	30.1±8.1	0.382
VF (%)	10.6±0.65	13.03±0.84	0.023
SF (%)	23.8±0.64	22.5±0.81	0.460

Note: Data are presented as the mean ± standard deviation, median (interquartile range), or numbers.

Abbreviations: BMI, body mass index; FEV1, forced expiratory volume in 1 second; FFMI, fat-free mass index; FVC, forced vital capacity; PaO2, partial pressure of oxygen; SF, subcutaneous fat; VF, visceral fat.

Regarding metabolic parameters, the COPD group had significantly lower fasting glucose levels than the control group. No significant differences were observed between the two groups with respect to triglycerides, or HDL-cholesterol levels. (Table 2).

Table 2 Comparison of metabolic profiles between the COPD and control groups

	Control	COPD	*P* value
Subjects (n)	48	188	–
Glucose (mg/dL)	102.5 (88.5–116.5)	95.0 (87.0–103.0)	0.005
TG (mg/dL)	104.0 (62.0–146.0)	112.0 (75.5–148.5)	0.498
HDL(mg/dL)	56.0 (45.0–67.0)	56.5 (44.5–68.5)	0.832

Note: Data are presented as the mean ± standard deviation, median (interquartile range), or numbers (%).

Abbreviations: HDL, high-density lipoprotein, TG-triglycerides

Data on body composition and abdominal fat percentage at various COPD stages are shown in Table 3. Stages III and IV were combined into one group designated "Stages III+IV", because the number of subjects in Stage IV was so small that these subjects could not be examined separately. The FFMI in Stages III+IV was significantly lower than that in both Stage II and Stage I ($P <0.05$). This suggests that FFMI decreases with the progression of COPD. In contrast, VF tended to increase in COPD stages, although not all differences were statistically significant. The percentages of non-obese patients with VF more than 10% in Stages I, II, and III+IV were 37.0%, 48.9%, and 58.9%, respectively. The prevalence of non-obese patients with VF more than 10% in Stages III+IV was significantly higher than that in the other stages ($P <0.05$).

Table 3 Differences in variables related to muscle and fat between the COPD stages

	Control	Stage I	Stage II	Stages III+IV
Subjects (n)	48	45	90	53
FFMI (kg/m2)	32.7±0.52	31.9±1.2	30.0±0.5	$28.7\pm0.21^{*\#\&}$
BMI (kg/m2)	27.2±1.4	28.3±1.01	26.9±1.7	$25.9\pm0.07^{\#}$
VF (%)	10.6±0.65	$15.2\pm1.8^{*}$	13.1±2.1	$11.0\pm1.2^{*\#\&}$
SF (%)	23.8±0.64	23.4±0.8	22.8±0.6	$21.2\pm0.9^{*\#\&}$

Note: Data are presented as the mean ± standard deviation; (P <0.05):*- compared to control; #-between Stage I and III+IV; &- between Stage II and III+IV

Abbreviations: BMI, body mass index; FFMI, fat-free mass index; SF, subcutaneous fat; VF, visceral fat.

Discussion

The present study yields several interesting findings. First, the COPD group had a larger VF than the control group, although there was no difference in BMI between the two groups. Furthermore, the median VF in the COPD group was decreasing with increasing of disease stage. This finding may be related to the significantly higher prevalence of subjects receiving treatment for hypertension or dyslipidemia in the COPD group than in the control group. A previous investigation by the Japan Society for the Study of Obesity also suggests that the risk of having at least one obesity related disorder such as dyslipidemia, hypertension, or hyperglycemia increases at VF area $ 100 cm2 [4]. Second, the prevalence of non-obese patients with excessive visceral fat was highest in Stages III+IV of COPD. Steuten et al (2006) have reported that patients malnourished with progressing of COPD [13]. Indeed, decreased skeletal muscle mass was observed in the more severe COPD stages. Furthermore, BMI, FFMI and SF are decreased with increasing severity in the present study. Similarly, Ogawa et al (2009) have reported that BMI and subcutaneous fat are lower in predominantly emphysematous COPD patients, although thoracic subcutaneous fat was measured rather than abdominal fat. There are several possible explanations for this observation. It has been reported that skeletal muscle mass in advanced stages of COPD decreases with increased energy expenditure, physical inactivity owing to dyspnea, decreased exercise capacity, inadequate diet or systemic inflammation [10,11]. Decreased skeletal muscle mass may result in further physical inactivity, leading to excess visceral fat accumulation, especially in the more advanced stages of COPD.

Our findings may partially explain the increased risk of cardiovascular diseases in COPD patients, especially in those with more advanced stages. COPD patients, even those with a normal BMI, may have an increased risk of cardiovascular disease owing to excessive accumulation of visceral fat. In this regard, careful assessment of abdominal obesity is needed for non-obese COPD patients, especially those at more advanced stages, because they tend to show abdominal subcutaneous fat loss. In a previous study, adipocyte dysfunction was found to be associated with adipose tissue hypoxia [7]. Therefore, we hypothesize that excessive visceral fat is another source of systemic inflammation in COPD and contributes toward altered body composition. In particular, patients with reduced pulmonary function, who also tend to be hypoxic, may be more susceptible to this effect, because systemic hypoxia is also associated with systemic inflammation [8]. However, the role of adipose tissue in the pathogenesis of systemic inflammation in COPD has not yet been fully examined.

Although in the present study we have not registered received doses of oral corticosteroids, it should be noted that systemic corticosteroid therapy may induce visceral obesity as a result of glucocorticoid-mediated redistribution of stored energy and the stimulatory effect on intake [3]. When it comes to a possible strategy for disease management, pulmonary rehabilitation is suggested in anticipation of not only increased muscle strength but also visceral fat loss.

In conclusion, COPD patients have excessive visceral fat, despite the absence of obesity.

References

1. Бурцева Е.В. Исследование нутритивного статуса пациентов ХОБЛ с помощью методов антропометрии и биоимпендансометрии // Современные проблемы науки и образования. – 2012. – №2; URL: www.science-education.ru/102-5912.

[Burceva E.V. Issledovanie nutritivnogo statusa pacientov HOBL s pomoshu metodov antropometrii i bioimpendansometrii // Sovremennue problemu nauki I obrasovaniy.- 2012.- №2 URL: www.science-education.ru/102-5912].

2. Agustí AG, Noguera A, Sauleda J, Sala E, Pons J, Busquets X. Systemic effects of chronic obstructive pulmonary disease. *Eur Respir J*. 2003;21(2):347–360.

3. Dallman MF, la Fleur SE, Pecoraro NC, Gomez F, Houshyar H, Akana SF. Minireview: glucocorticoids-food intake, abdominal obesity, and wealthy nations in 2004. Endocrinology. 2004;145(6):2633–2638.

4. Examination Committee of Criteria for 'Obesity Disease' in Japan, Japan Society for the Study of Obesity. New criteria for 'obesity disease' in Japan. Circ J. 2002;66(1):987–992.

5. Furutate R, Ishii T, Wakabayashi R, Motegi T, Yamada K, Gemma A, Kida K. Excessive visceral fat accumulation in advanced chronic obstructive pulmonary disease. International Journal of COPD. 2011:6 423-430

6. Global Initiative for Chronic Obstructive Lung Disease. Executive summary. Global strategy for the diagnosis, management and prevention of chronic obstructive pulmonary disease. http://www.goldcopd.com. Date last updated: March 2009. Accessed September 7, 2010.

7. Hosogai N, Fukuhara A, Oshima K, et al. Adipose tissue hypoxia in obesity and its impact on adipocytokine dysregulation. Diabetes. 2007;56(4):901–911.

8. Johnston AK, Mannino DM, Hagan GW, Davis KJ, Kiri VA. Relationship between lung function impairment and incidence or recurrence of cardiovascular events in a middle-aged cohort. *Thorax*. 2008;63(7):599–605.

9. Matuzawa Y. Adiponectin: identification, physiology and clinical relevance in metabolic and vascular disease. Atheroscler Suppl. 2005;6(2):7–14.
10. McGarvey LP, John M, Anderson JA, Zvarich M, Wise RA. Ascertainment of cause-specific mortality in COPD: operations of the TORCH Clinical Endpoint Committee. *Thorax*. 2007;62(5):411–415.
11. Molennar EA, Massaro JM, Jacques PF, et al. Association of lifestyle factors with abdominal subcutaneous and visceral adiposity: the Framingham Heart Study. Diabetes Care. 2009;32(3):505–510.
12. Pitta F, Troosters T, Spruit MA, Probst VS, Decramer M, Gosselink R. Characteristics of physical activities in daily life in chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2005;171(9):972–977.
13. Steuten LM, Creutzberg EC, Vrijhoef HJ, Wouters EF. COPD as a multicomponent disease: inventory of dyspnoea, underweight, obesity and fat free mass depletion in primary care. Prim Care Respir J. 2006;15(2):84–91.
14. Yudkin JS. Adipose tissue, insulin action vascular disease: inflammatory signals. *Int J Obes Relat Metab Disord*. 2003;27(Suppl 3):S25–S28.

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Вентилятор-индуцированное повреждение легких у новорожденных детей. Пути снижения

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В данном обзоре представлены современные тенденции протективной вентиляции у новорожденных детей. Рассмотрены варианты вентилятор-индуцированного повреждения легких, среди которых наиболее частым является волюмотравма. Сохраняется актуальность экспериментальных и клинических исследований биологической травмы легких, концепция которой есть описание биохимических процессов высвобождения воспалительных медиаторов вследствие механической вентиляции. Наблюдается связь между вентилятор-индуцированным повреждением легких и развитием бронхолегочной дисплазии. У глубоко недоношенных детей в настоящее время имеет место “новая” форма бронхолегочной дисплазии – паренхиматозное легочное заболевание, характеризующееся нарушением роста и развития альвеол и сосудов малого круга кровообращения. Ряд авторов считает, что применение неинвазивных методов стартовой вентиляции у недоношенных новорожденных является профилактической мерой по снижению риска развития бронхолегочной дисплазии. Современная протективная искусственная вентиляция легких предусматривает два основных направления снижения вентилятор-индуцированного повреждения легких: уменьшение дыхательного объема (Vt) и принцип допустимой (пермиссивной) гиперкапнии. Применение методики пермиссивной гиперкапнии и режимов с целевым объемом позволяет снизить вероятность вентилятор-индуцированного повреждения легких у новорожденных детей. Несмотря на ограничение показаний к искусственной вентиляции легких в современной неонатологии и широкому применению неинвазивной вентиляции, для пациентов, действительно нуждающихся в ИВЛ, применение режимов с целевым объемом дает лучшие шансы на уменьшение осложнений вентиляции.

Ключевые слова: бронхолегочная дисплазия, протективная вентиляция, пермиссивная гиперкапния, режимы с целевым объемом.

Ventilator-induced lung injury in newborn infants. Ways to reduce

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This review presents the current trends protective ventilation in newborn infants. Volumotrauma – the most common variant of ventilator-induced lung injury. The modern research is devoted to the study of biotrauma, which is the release of inflammatory mediators in response to mechanical ventilation. There is a correlation between the ventilator-induced lung injury and the development of chronic lung diseases in infants. Now we have the “new” form of bronchopulmonary dysplasia – parenchymal lung disease characterized by impaired growth and development of the alveoli and blood vessels of the pulmonary circulation. Some authors believe that the use of noninvasive ventilation as a starting method of respiratory support reduce the risk of bronchopulmonary dysplasia. The modern protective ventilation involves two main principles to reduce ventilator-induced lung injury: a decrease in tidal volume (Vt) and the principle of permissive hypercapnia. Application of the method of permissive hypercapnia and modes of the target volume can reduce the likelihood of ventilator-induced lung injury in newborn infants. Despite