International Journal of Endocrinology

Огляд літератури
/Literature Review/

UDC 616.441-008.64:616.24 DOI: https://doi.org/10.22141/2224-0721.16.8.2020.222884

Mariia Sukholytka
Department of respiratory medicine, Thomayer hospital, Prague, The Czech Republic

Hypothyroidism and chronic obstructive pulmonary disease

For citation: Mìžnarodnij endokrinologìčnij žurnal. 2020;16(8):643-647. doi: 10.22141/2224-0721.16.8.2020.222884

Abstract. *The risk of chronic obstructive pulmonary disease (COPD), as well as thyroid diseases increases with age. COPD is a common systemic disease associated with chronic inflammation. Many endocrinological disorders, including thyroid gland diseases are related to systemic inflammation. Epidemiological studies suggest that patients with COPD are at higher risk of thyroid disorders. These associations are not well-studied and thyroid gland diseases are not included on the broadly acknowledged list of COPD comorbidities. They may seriously handicap quality of life of COPD patients. Unfortunately, the diagnosis may be difficult, as many signs are masked by the symptoms of the index disease. The comprehension of the correlation between thyroid gland disorders and COPD may contribute to better care of patients. In this review, we attempt to revise available literature describing existing links between COPD and thyroid diseases. The signs or symptoms of thyroid disorders may be non-specific, especially among the elderly, therefore the differential diagnosis between symptoms of COPD and symptoms related to thyroid disease can cause difficulties. Many data show higher risk of thyroid hormones alterations in COPD patients. Hypothyroidism may influence respiration by different mechanisms, even in subjects with intact respiratory system. Therefore, it is hard to distinguish whether hormonal changes are the reason or a consequence of different respiratory signs and symptoms. In some instances, the correction of hormonal alternations may improve the quality of life of COPD patients and other disease outcomes. The comprehension of an association between COPD, thyroid gland function and thyroid disorders may provide important information about the systemic nature of COPD.*

Keywords: *hypothyroidism; chronic obstructive pulmonary disease; comorbidity; thyroid gland*

Introduction

Chronic obstructive pulmonary disease (COPD) is a leading cause of morbidity and mortality worldwide [1]. The risk increases suddenly with age, and the peak incidence falls when patients are over 60 [1]. It has been acknowledged that chronic inflammation occurring in COPD is systemic in nature, but it occurs primarily in the lung, from where inflammatory cytokines "spill over" and inflammation spreads to other organs of the body [2].

Systemic inflammation may be driven by the main risk factor — cigarette smoking, and it persists after smoking cessation [3]. The inflammation has an impact on other systems, for example: cardiovascular, skeletal muscles, skeleton, brain etc. [4]. According to GOLD (Global Initiative for Obstructive Lung Disease), many comorbidities like coronary artery disease, diabetes and metabolic syndrome, depression, cachexia, osteoporosis are undoubtedly associated with COPD by their frequent co-occurrence [1]. The link between other chronic diseases and COPD may not be so obvious, but some data suggest many underrecognized associations [5]. COPD interferes with endocrinological homoeostasis not only by means of systemic inflammation [6]. Other factors like neurohormones, blood gas abnormalities, glucocorticoid administration also disturb hormonal balance [6]. On the other hand, hormones may affect regulation of breathing [7]. Some hormones act on the level of the central nervous system, some have an impact on peripheral chemoreceptors, others may contribute to this process by influencing the metabolism rate, and others exert their effect directly on receptors in the respiratory tract [7]. Drugs frequently used by COPD patients to treat comorbidities, such as amiodarone [8], lithium carbonate [9] or potassium iodine [10] may lead to hypothyroidism.

There is a growing evidence that thyroid gland function may be disturbed in COPD patients [2, 7, 11]. Some studies show that thyroid diseases are more frequent among patients

For correspondence: Mariia Sukholytka, Thomayerova nemocnice, Videnska 800, Praha 4 — Krc, 14059, Prague, The Czech Republic; e-mail: info@ftn.cz
Full list of author information is available at the end of the article.

with COPD. In a big population-based study performed in the city of Madrid, Spain, it was shown that the prevalence of a thyroid disease was higher in COPD patients (14.2 %) than the expected standardized prevalence of chronic diseases (11.16 %) [6]. The general occurrence of thyroid disorders is estimated at 14–20 % among stable COPD patients and at 70 % during an exacerbation [12].

The thyroid diseases occur more frequently among women than men with COPD [6], the same as in the general population [13].

The purpose of this review is to discuss the available data on the coexistence of hypothyroidism and COPD.

Materials and methods

The initial search was conducted using PubMed with the subject headings "pulmonary disease, chronic obstructive pulmonary disease" and "hypothyroidism". All abstracts were assessed for relevance, and articles of the relevant studies were retrieved. Subsequent searches utilized the following combinations of subject headings on PubMed: "CODP & thyroid hormones" or "COPD & thyroid disorders" or "COPD & hypothyrodism" or "COPD & nonthyroidal illness syndrome".

Chronic inflammation as a possible link between COPD and hypothyroidism

Chronic inflammation in COPD is associated with production of interleukin (IL)-1β, tumor necrosis factor (TNF)-α, IL-8, IL-6, and fibrinogen by alveolar macrophages and neutrophils [14]. In humans, intravenous administration of recombinant IL-6 resulted in acute reduction of triiodothyronine (T_3) and thyroid stimulating hormone (TSH) levels [15]. Tobacco smoke contains considerable amounts of free radicals that may damage the structure of the respiratory tract and promote inflammation [1, 16]. Cigarette smoke attracts activated inflammatory cells to the lungs — another source of free radicals and oxidants contributing to sustained inflammation [17]. Also the endocrine system is not inert to the components of cigarette smoke. Higher levels of serum total triiodothyronine (tT_3) were found in young healthy smokers comparing to nonsmoking control subjects [16], what may suggest that smoking acts independently of coexistent diagnosis of COPD.

Hypothyroidism

The prevalence of hypothyroidism in the general population is 0.9 % among men and 4.8 % among women [19]. Its frequency increases with age [20]. Some studies have shown the relationship between thyroid hormone levels and blood gas parameters [2]. C. Terzano et al. [2] described lower blood oxygen pressure (pO_2) in patients with overt hypothyroidism, compared to other groups consisting of patients with subclinical hypothyroidism, normal subjects and patients with hyperthyroidism. Moreover, in the same research, the authors observed significant increase of the blood carbon dioxide pressure (pCO_2) levels in patients with hypothyroidism, although a correlation between TSH and pCO_2 was not present [2]. Dimopoulou et al. reported strong positive correlation between serum total triiodothyronine/total thyroxine (tT_3/tT_4) ratio and arterial oxygen pressure (PaO_2), but only in COPD patients with FEV1 < 50 % predicted value, and not in those above this threshold [21]. This observation suggests the relationship between low conversion rate of T_4 to T_3 in peripheral tissues and hypoxemia in most severe COPD patients [22]. In another study, it was demonstrated that obstruction severity is associated with reduced basal and stimulated TSH [23]. However, the exact mechanism of the above connection is not known.

Muscle weakness is one of the symptoms of hypothyroidism [24, 25]. The deterioration of main respiratory muscle function [26] aggravates already weakened ventilation in patients with COPD [2]. Some adapting mechanisms in COPD patients have been described, like shortening of the length of sarcomeres and an increase in the concentration of mitochondria [26]. The muscle weakness may manifest in worse spirometry results [2]. In hypothyroidism mean inspiratory pressure (MIP) and mean expiratory pressure (MEP) are decreased [2, 27], supposedly due to decreased respiratory muscle strength [28].

The association of free T_3 (fT_3) level with arterial blood gases and pulmonary function parameters (vital capacity (VC) or forced vital capacity (FVC), FEV1, peak expiratory flow (PEF)) has been demonstrated [29]. Another study also confirmed lower values of MEP in COPD patients with hypothyroidism than in those without this condition [30]. The authors observed significantly lower values of FVC, FEV1/FVC, forced expiratory flow at 25 and 75 % (FEF 25–75 %) in this group of patients [31]. Others reported on correlation between MEP and MIP values and thyroid function [2]. Interestingly, there is also a positive correlation between TSH values and acute exacerbations frequency [31]. Among the patients with hypothyroidism, the exacerbations occurred more frequently than in patients without hypothyroidism, and TSH value turned out to be the only significant determinant of exacerbation frequency [31].

Further studies are needed to verify if impaired thyroid function really increases the risk of COPD exacerbation and if proper hormonal treatment would impact the clinical outcome. Authors of other studies reported on lower spirometry parameters in patients without clinically diagnosed hypothyroidism, but with low values of thyroid hormones, that still remained within normal limits [29]. It is of note that the muscle weakness, caused by hypothyroidism, is reversible after treatment [32]. Thyroid dysfunction, defined by the use of thyroid function tests (TFT), was demonstrated in more than half of COPD patients with respiratory failure (52.3 %) and in slightly lower percentage of patients without this condition (44.4 %) [33], however, the difference between the groups was not statistically significant. Moreover, patients with both low levels of fT_3 and free T_4 (fT_4) needed invasive mechanical ventilation more often than those from the group with normal TFT scores [34]. The authors also observed the higher rate of in-hospital mortality among patients with low levels of either fT_3 or fT_4 [34]. Significant difference of TSH level between groups of patients with COPD exacerbation and healthy subjects was demonstrated [34]. Lower fT_3 in the course of COPD exacerbation was also confirmed by other authors [29]. The authors also found that fT_3 negatively correlated with bicarbonate levels and fT_4, also negatively, with hemoglobin [35].

Non-thyroidal illness syndrome

Non-thyroidal illness syndrome (NTIS) may be defined as reduced conversion of T_4 to T_3 in different acute and chronic systemic disorders. NTIS occurs more frequently than hypothyroidism and subclinical hypothyroidism [36]. This clinical entity is characterized by a decreased tT_3 and fT_3, normal or decreased tT_4 and fT_4, and unchanged TSH levels [37]. The mechanisms leading to NTIS are largely unknown and further studies in this field are required [38].

The hormonal changes during follow-up after COPD exacerbation followed normalization of PaO_2 and $PaCO_2$ during recovery. The increase of TSH levels following improvement of hypoxia and stabilization of clinical condition denotes delayed pituitary response to TRH, which was earlier impaired by hypoxia [39]. Serum tT_3, fT_3 and tT_3/tT_4, which decrease with age and in COPD exacerbations as shown in the cited study, indicate decreased metabolic clearance of T_4 and decreased peripheral conversion to T_3. This inverse correlation suggests that aging may also play a role in thyroid dysfunction in addition to hypoxemia [40]. According to foregoing findings, thyroid disease should not be recognized during COPD exacerbation because of alternations in thyroid hormones related to severe clinical condition and not to true thyroid disease [41]. In another study, it was shown, that NTIS may be regarded as independent predictor of prolonged weaning in intubated COPD patients [42]. Importantly, prolonged weaning is associated with increased mortality and morbidity in the intensive care unit [43].

Presumably, NTIS (especially low fT_3) may reflect severity of inflammation, hypoxia or other pathological processes associated with COPD exacerbation [44]. The authors also reported on successful weaning from mechanical ventilation (MV) after proper hormonal supplementation in patients with newly diagnosed hypothyroidism [45]. Although the authors noticed that further studies are necessary to assess the significance of thyroid hormones supplementation in patients with NTIS [45]. It should be remembered that various factors can affect thyroid function by stimulating or suppressing it. More frequent occurrence of pulmonary infections among patients with NTIS has been demonstrated [46]. Some cytokines and immune system cells restrain thyroid gland activity. Therefore, treatment of pulmonary infections or any infection, becomes an important aim to reach.

Conclusions

The signs or symptoms of thyroid disorders may be nonspecific, especially among the elderly, therefore the differential diagnosis between symptoms of COPD and symptoms related to thyroid disease can cause difficulties. Many data show higher risk of thyroid hormones alterations in COPD patients. Hypothyroidism may influence respiration by different mechanisms, even in subjects with intact respiratory system. Therefore, it is hard to distinguish whether hormonal changes are the reason or a consequence of different respiratory signs and symptoms. In some instances, the correction of hormonal alternations may improve the quality of life of COPD patients and other disease outcomes. The comprehension of an association between COPD, thyroid gland function and thyroid disorders may provide important information about the systemic nature of COPD.

Conflicts of interests. Author declares the absence of any conflicts of interests and their own financial interest that might be construed to influence the results or interpretation of their manuscript.

References

1. Singh D, Agusti A, Anzueto A, et al. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Lung Disease: the GOLD science committee report 2019. Eur Respir J. 2019 May 18;53(5):1900164. doi:10.1183/13993003.00164-2019.

2. Terzano C, Romani S, Paone G, Conti V, Oriolo F. COPD and thyroid dysfunctions. Lung. 2014 Feb;192(1):103-9. doi:10.1007/s00408-013-9537-6.

3. Oudijk EJ, Lammers JW, Koenderman L. Systemic inflammation in chronic obstructive pulmonary disease. Eur Respir J Suppl. 2003 Nov;46:5s-13s. doi:10.1183/09031936.03.00004603a.

4. Agustí A, Edwards LD, Rennard SI, et al; Evaluation of COPD Longitudinally to Identify Predictive Surrogate Endpoints (ECLIPSE) Investigators. Persistent systemic inflammation is associated with poor clinical outcomes in COPD: a novel phenotype. PLoS One. 2012;7(5):e37483. doi:10.1371/journal.pone.0037483.

5. van der Molen T. Co-morbidities of COPD in primary care: frequency, relation to COPD, and treatment consequences. Prim Care Respir J. 2010 Dec;19(4):326-34. doi:10.4104/pcrj.2010.00053.

6. García-Olmos L, Alberquilla A, Ayala V, et al. Comorbidity in patients with chronic obstructive pulmonary disease in family practice: a cross sectional study. BMC Fam Pract. 2013 Jan 16;14:11. doi:10.1186/1471-2296-14-11.

7. Doehner W, Haeusler KG, Endres M, Anker SD, MacNee W, Lainscak M. Neurological and endocrinological disorders: orphans in chronic obstructive pulmonary disease. Respir Med. 2011 Oct;105 Suppl 1:S12-9. doi:10.1016/S0954-6111(11)70005-1.

8. Padmanabhan H. Amiodarone and thyroid dysfunction. South Med J. 2010 Sep;103(9):922-30. doi:10.1097/SMJ.0b013e3181e90500.

9. Bocchetta A, Loviselli A. Lithium treatment and thyroid abnormalities. Clin Pract Epidemiol Ment Health. 2006 Sep 12;2:23. doi:10.1186/1745-0179-2-23.

10. Markou K, Georgopoulos N, Kyriazopoulou V, Vagenakis AG. Iodine-Induced hypothyroidism. Thyroid. 2001 May;11(5):501-10. doi:10.1089/105072501300176462.

11. Laghi F, Adiguzel N, Tobin MJ. Endocrinological derangements in COPD. Eur Respir J. 2009 Oct;34(4):975-96. doi:10.1183/09031936.00103708.

12. Chaudhary SC, Ahmad T, Usman K, et al. Prevalence of thyroid dysfunction in chronic obstructive pulmonary disease patients in a tertiary care center in North India. J Family Med Prim Care. 2018 May-Jun;7(3):584-588. doi:10.4103/jfmpc.jfmpc_84_17.

13. Prakash R, Harish M, Suryanarayana V, Padma Priya M. Evaluation of thyroid functions in chronic obstructive pulmonary disease. World J Med Sci. 2014;11:57-8. doi:10.5829/idosi.wjms.2014.11.1.82110.

14. Korytina GF, Akhmadishina LZ, Kochetova OV, Aznabae-

va YG, Zagidullin SZ, Victorova TV. Inflammatory and Immune Response Genes Polymorphisms are Associated with Susceptibility to Chronic Obstructive Pulmonary Disease in Tatars Population from Russia. Biochem Genet. 2016 Aug;54(4):388-412. doi:10.1007/s10528-016-9726-0.

15. Sanguinetti CM. N-acetylcysteine in COPD: why, how, and when? Multidiscip Respir Med. 2016 Feb 3;11:8. doi:10.1186/s40248-016-0039-2.

16. Agin Kh, Namavary J. A Survey on Thyroid Hormonal Status among Moderate to Severe Stable Chronic Obstructive Pulmonary Disease. Int J Med Toxicol Forensic Med. 2013;3(4):106-113. doi:10.22037/ijmtfm.v3i4(Autumn).3643.

17. Meral I, Arslan A, Him A, Arslan H. Smoking-related Alterations in Serum Levels of Thyroid Hormones and Insulin in Female and Male Students. Altern Ther Health Med. 2015 Sep-Oct;21(5):24-9.

18. Leach PT, Gould TJ. Thyroid hormone signaling: Contribution to neural function, cognition, and relationship to nicotine. Neurosci Biobehav Rev. 2015 Oct;57:252-63. doi:10.1016/j.neubiorev.2015.09.001.

19. Unnikrishnan AG, Kalra S, Sahay RK, Bantwal G, John M, Tewari N. Prevalence of hypothyroidism in adults: An epidemiological study in eight cities of India. Indian J Endocrinol Metab. 2013 Jul;17(4):647-52. doi:10.4103/2230-8210.113755.

20. Morganti S, Ceda GP, Saccani M, et al. Thyroid disease in the elderly: sex-related differences in clinical expression. J Endocrinol Invest. 2005;28(11 Suppl Proceedings):101-4.

21. Dimopoulou I, Ilias I, Mastorakos G, Mantzos E, Roussos C, Koutras DA. Effects of severity of chronic obstructive pulmonary disease on thyroid function. Metabolism. 2001 Dec;50(12):1397-401. doi:10.1053/meta.2001.28157.

22. Mortoglou A, Candiloros H. The serum triiodothyronine to thyroxine (T3/T4) ratio in various thyroid disorders and after Levothyroxine replacement therapy. Hormones (Athens). 2004 Apr-Jun;3(2):120-6. doi:10.14310/horm.2002.11120.

23. Bratel T, Wennlund A, Carlström K. Impact of hypoxaemia on neuroendocrine function and catecholamine secretion in chronic obstructive pulmonary disease (COPD). Effects of long-term oxygen treatment. Respir Med. 2000 Dec;94(12):1221-8. doi:10.1053/rmed.2000.0953.

24. Sarinc Ulasli S, Bozbas SS, Ozen ZE, Ozyurek BA, Ulubay G. Effect of thyroid function on COPD exacerbation frequency: a preliminary study. Multidiscip Respir Med. 2013 Oct 1;8(1):64. doi:10.1186/2049-6958-8-64.

25. Kostoglou-Athanassiou I, Ntalles K. Hypothyroidism - new aspects of an old disease. Hippokratia. 2010 Apr;14(2):82-7.

26. Singh D, Agusti A, Anzueto A, et al. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Lung Disease: the GOLD science committee report 2019. Eur Respir J. 2019 May 18;53(5):1900164. doi:10.1183/13993003.00164-2019.

27. Bacakoğlu F, Başoğlu OK, Gürgün A, Bayraktar F, Kiran B, Ozhan MH. Can impairments of thyroid function test affect prognosis in patients with respiratory failure? Tuberk Toraks. 2007;55(4):329-35.

28. Sadek SH, Khalifa WA, Azoz AM. Pulmonary consequences of hypothyroidism. Ann Thorac Med. 2017 Jul-Sep;12(3):204-208. doi: 10.4103/atm.ATM_364_16.

29. van Tuyl M, Blommaart PE, de Boer PA, et al. Prenatal exposure to thyroid hormone is necessary for normal postnatal development of murine heart and lungs. Dev Biol. 2004 Aug 1;272(1):104-17. doi:10.1016/j.ydbio.2004.03.042.

30. Ittermann T, Gläser S, Ewert R, Felix S, Völzke H, Dörr M. Serum thyroid-stimulating hormone levels are not associated with exercise capacity and lung function parameters in two population-based studies. BMC Pulm Med. 2014 Sep 2;14:145. doi:10.1186/1471-2466-14-145.

31. Simonides WS, van Hardeveld C. Thyroid hormone as a determinant of metabolic and contractile phenotype of skeletal muscle. Thyroid. 2008 Feb;18(2):205-16. doi:10.1089/thy.2007.0256.

32. Khushu S, Rana P, Sekhri T, Sripathy G, Tripathi RP. Bioenergetic impairment in human calf muscle in thyroid disorders: a 31P MRS study. Magn Reson Imaging. 2010 Jun;28(5):683-9. doi:10.1016/j.mri.2010.01.006.

33. Okamoto T, Kanazawa H, Hirata K, Yoshikawa J. Evaluation of oxygen uptake kinetics and oxygen kinetics of peripheral skeletal muscle during recovery from exercise in patients with chronic obstructive pulmonary disease. Clin Physiol Funct Imaging. 2003 Sep;23(5):257-62. doi:10.1046/j.1475-097x.2003.00500.x.

34. Iyer SK, Menon SK, Bahuleyan B. An Analysis of Dynamic Pulmonary Functions of Hypothyroid Patients. J Clin Diagn Res. 2017 Mar;11(3):CC10-CC12. doi:10.7860/JCDR/2017/24653.9579.

35. Koral L, Hekimsoy Z, Yildirim C, Ozmen B, Yorgancioglu A, Girgin A. Does thyroid replacement therapy affect pulmonary function tests in patients with subclinical hypothyroidism? Saudi Med J. 2006 Mar;27(3):329-32.

36. Warner MH, Beckett GJ. Mechanisms behind the non-thyroidal illness syndrome: an update. J Endocrinol. 2010 Apr;205(1):1-13. doi:10.1677/JOE-09-0412.

37. De Groot LJ. Non-thyroidal illness syndrome is a manifestation of hypothalamic-pituitary dysfunction, and in view of current evidence, should be treated with appropriate replacement therapies. Crit Care Clin. 2006 Jan;22(1):57-86, vi. doi:10.1016/j.ccc.2005.10.001.

38. Van den Berghe G. Non-thyroidal illness in the ICU: a syndrome with different faces. Thyroid. 2014 Oct;24(10):1456-65. doi:10.1089/thy.2014.0201.

39. Langouche L, Jacobs A, Van den Berghe G. Nonthyroidal Illness Syndrome Across the Ages. J Endocr Soc. 2019 Oct 16;3(12):2313-2325. doi:10.1210/js.2019-00325.

40. Maiden MJ, Torpy DJ. Thyroid Hormones in Critical Illness. Crit Care Clin. 2019 Apr;35(2):375-388. doi:10.1016/j.ccc.2018.11.012.

41. El-Yazed HA, El-Bassiony MR, Eldaboosy SA, Gendi AE, Hashim M. Assessment of thyroid functions in patients with chronic obstructive pulmonary disease. Egyptian Journal of Chest Diseases and Tuberculosis. 2013;62(3):387-391. doi:10.1016/j.ejcdt.2013.07.009.

42. Gow SM, Seth J, Beckett GJ, Douglas G. Thyroid function and endocrine abnormalities in elderly patients with severe chronic obstructive lung disease. Thorax. 1987 Jul;42(7):520-5. doi:10.1136/thx.42.7.520.

43. Creutzberg EC, Casaburi R. Endocrinological disturbances in chronic obstructive pulmonary disease. Eur Respir J Suppl. 2003 Nov;46:76s-80s. doi:10.1183/09031936.03.00004610.

44. Mancini A, Corbo GM, Gaballo A, et al. Relationships between plasma CoQ10 levels and thyroid hormones in chronic obstructive pulmonary disease. Biofactors. 2005;25(1-4):201-4. doi:10.1002/biof.5520250124.

45. Fliers E, Bianco AC, Langouche L, Boelen A. Thyroid function in critically ill patients. Lancet Diabetes Endocrinol. 2015 Oct;3(10):816-25. doi:10.1016/S2213-8587(15)00225-9.

46. van der Spek AH, Surovtseva OV, Jim KK, et al. Regulation of Intracellular Triiodothyronine Is Essential for Optimal Macrophage Function. Endocrinology. 2018 May 1;159(5):2241-2252. doi:10.1210/en.2018-00053.

47. Pashkovska NV. Pseudothyroid dysfunction in clinical practice: how to avoid diagnostic errors. Mìžnarodnij endokrinologìčnij žurnal. 2018;14(4):344-353. doi:10.22141/2224-0721.14.4.2018.140188. (in Ukrainian).

48. Yasar Z, Kirakli C, Cimen P, Ucar ZZ, Talay F, Tibet G. Is non-thyroidal illness syndrome a predictor for prolonged weaning in intubated chronic obstructive pulmonary disease patients? Int J Clin Exp Med. 2015 Jun 15;8(6):10114-21.

49. Montesinos MDM, Pellizas CG. Thyroid Hormone Action on Innate Immunity. Front Endocrinol (Lausanne). 2019 Jun 4;10:350. doi:10.3389/fendo.2019.00350.

Received 06.11.2020
Revised 24.11.2020
Accepted 01.12.2020

Information about author
Mariia Sukholytka, Department of respiratory medicine, Thomayer hospital, Prague, The Czech Republic; e-mail: info@ftn.cz

Mariia Sukholytka
Department of respiratory medicine, Thomayer hospital, Prague, The Czech Republic

Гіпотиреоз та хронічна обструктивна хвороба легень

Резюме. Ризик хронічної обструктивної хвороби легень (ХОХЛ), а також захворювань щитоподібної залози зростає з віком. ХОХЛ — загальне системне захворювання, пов'язане з хронічним запаленням. Багато ендокринологічних розладів, включаючи захворювання щитоподібної залози, пов'язані із системним запаленням. Епідеміологічні дослідження показують, що в пацієнтів із ХОХЛ підвищений ризик розладів щитоподібної залози. Ці асоціації недостатньо вивчені, і тиреоїдні захворювання не включені до загальновизнаного списку супутніх захворювань ХОХЛ. Водночас вони можуть значно погіршити якість життя хворих на ХОХЛ. На жаль, діагностика доволі складна, оскільки багато ознак маскуються симптомами основного захворювання. З'ясування кореляції між захворюваннями щитоподібної залози та ХОХЛ сприятиме кращій допомозі особам із такою патологією. У цьому огляді проаналізовано доступну літературу, що описує існуючі зв'язки між ХОХЛ та захворюваннями щитоподібної залози. Ознаки або симптоми зниженої функції щитоподібної залози можуть бути неспецифічними, особливо серед осіб похилого віку, тому диференціальна діагностика між симптомами ХОХЛ та симптомами, пов'язаними із захворюваннями щитоподібної залози, може викликати труднощі. Багато даних свідчать про більш високий ризик зміни функціонального стану щитоподібної залози у хворих на ХОХЛ. Гіпотиреоз може впливати на дихальну функцію різними механізмами, навіть в осіб з інтактною дихальною системою. Тому складно розрізнити, чи є гормональні зміни причиною або наслідком різних ознак та симптомів порушення дихання. У деяких випадках корекція гормональних змін може покращити якість життя хворих на ХОХЛ та інші наслідки захворювання. Установлення взаємозв'язків між ХОХЛ, функцією щитоподібної залози та тиреоїдними розладами може надати важливу інформацію про системну природу ХОХЛ.

Ключові слова: гіпотиреоз; хронічна обструктивна хвороба легень; коморбідність; щитоподібна залоза; огляд

Mariia Sukholytka
Department of respiratory medicine, Thomayer hospital, Prague, The Czech Republic

Гипотиреоз и хроническая обструктивная болезнь легких

Резюме. Риск хронической обструктивной болезни легких (ХОБЛ), а также заболеваний щитовидной железы увеличивается с возрастом. ХОБЛ — общее системное заболевание, связанное с хроническим воспалением. Много эндокринологических расстройств, включая заболевания щитовидной железы, связаны с системным воспалением. Эпидемиологические исследования показывают, что у пациентов с ХОБЛ повышенный риск расстройств щитовидной железы. Эти ассоциации недостаточно изучены, и тиреоидные заболевания не включены в общепризнанный список сопутствующих заболеваний ХОБЛ. В то же время они могут значительно ухудшить качество жизни больных ХОБЛ. К сожалению, диагностика довольно тяжелая, поскольку многие признаки маскируются симптомами основного заболевания. Выяснение корреляции между заболеваниями щитовидной железы и ХОБЛ будет способствовать лучшей помощи лицам с такой патологией. В этом обзоре проанализирована доступная литература, описывающая существующие связи между ХОБЛ и заболеваниями щитовидной железы. Признаки или симптомы пониженной функции щитовидной железы могут быть неспецифическими, особенно среди пожилых людей, поэтому дифференциальная диагностика между симптомами ХОБЛ и симптомами, связанными с заболеваниями щитовидной железы, может вызвать трудности. Многие данные свидетельствуют о более высоком риске изменения функционального состояния щитовидной железы у больных ХОБЛ. Гипотиреоз может влиять на дыхательную функцию разными механизмами, даже у лиц с интактной дыхательной системой. Поэтому трудно различить, являются гормональные изменения причиной или следствием различных признаков и симптомов нарушения дыхания. В некоторых случаях коррекция гормональных изменений может улучшить качество жизни больных ХОБЛ и другие последствия заболевания. Установление взаимосвязей между ХОБЛ, функцией щитовидной железы и тиреоидными расстройствами может предоставить важную информацию о системной природе ХОБЛ.

Ключевые слова: гипотиреоз; хроническая обструктивная болезнь легких; коморбидность; щитовидная железа; обзор