Prevalence of Osteoporosis and Osteopenia in Thai COPD Patients

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Objective: *To study the prevalence of osteoporosis and osteopenia in Thai COPD patients and the factors associated with osteoporosis.*
Material and Method: *A cross sectional study was used to evaluate 102 male stable COPD patients. Bone mineral density at lumbar spine (L2-4) and femoral neck were measured by dual energy x-ray absorptiometer scan. Demographic data including age, body mass index (BMI), inhaled corticosteroids use, tobacco smoke, force expiratory volume at 1 second (FEV1), and high sensitivity C-reactive protein (hs-CRP) were analyzed.*
Results: *The overall prevalence of osteoporosis and osteopenia according to the lowest T-score at either L2-4 or femoral neck were 31.4% and 32.4%, respectively. This prevalence of osteoporosis in COPD patients was higher than that in age-matched Thai males from historical data (31.4% vs. 12.6%, respectively). BMI and hs-CRP were significantly associated with osteoporosis. There was no association between osteoporosis and severity of COPD, age, smoking, and corticosteroid use. The predictive value of BMI < 20.5 kg/m^2 and hs-CRP > 2.3 mg/L demonstrated risk of osteoporosis in COPD patients (adjusted Odds ratio 7.2 and 4.1, respectively).*
Conclusion: *The prevalence of osteoporosis and osteopenia in Thai COPD patients was higher than that in normal age-matched Thai males. Osteoporosis was associated with low BMI and high level of hs-CRP when compared to COPD patients with normal bone mineral density.*

Keywords: *Bone mineral density, Chronic obstructive pulmonary disease, Osteoporosis, Prevalence, Systemic inflammation*

J Med Assoc Thai 2012; 95 (8): 1021-7
Full text. e-Journal: *http://jmat.mat.or.th*

Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality throughout the world. It will become the third leading cause of death worldwide by 2020[1]. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) has defined COPD as a preventable and treatable disease with some significant extra-pulmonary effects that may contribute to the severity in individual patients[2]. Its pulmonary component is characterized by airflow limitation that is not fully reversible and COPD itself also has co-morbid conditions includes weight loss, skeletal muscle dysfunction, coronary artery disease, and osteoporosis[3].

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Many studies have shown the evidence of systemic inflammation in COPD and this may explain the co-morbidity in COPD[4-6].

Osteoporosis is characterized by low bone mass and increased susceptibility to fracture[7]. From the aforementioned source, osteoporosis may be part of the extrapulmonary effects of COPD; if it occurs at the important area such as thoracic spine, the patients might develop breathing difficulty, decreased lung volume or restrictive ventilatory defects which then cause significant morbidity and impair quality of life[8]. However, this condition could be alleviated by early detection and treatment, especially in a high-risk group of patients, to prevent further complications. This will improve the quality of life in COPD patients.

Previous studies have shown that, the prevalence of osteoporosis and osteopenia in COPD patients is high, varying from 21 to 44%[9-11] and may have correlation with several factors such as

smoking, poor physical activity, vitamin D deficiency, low body mass index (BMI), glucocorticoids use, and severity of COPD[12-14]. However, all data came from Scandinavia, North America, and Europe. Here the authors aim to study the prevalence of osteoporosis and osteopenia in Thai COPD patients.

Material and Method

Study population and design

The cross sectional study was conducted between May 2009 and December 2010. COPD patients were recruited from the COPD clinic and outpatient department at Siriraj Hospital. Male stable COPD patients (last exacerbation > 4 weeks) with age > 40 years were included. Patients were excluded if they had asthma, any disease affecting bones and calcium homeostasis or were receiving drugs related to bone metabolism (*e.g.* hormonal replacement therapy, bisphosphonate, calcium or vitamin D supplement, oral corticosteroid). The present study protocol was approved by Siriraj Ethics Committee and all patients provided written informed consent before the enrollment.

Demographic data were collected including age, underlying disease, smoking history, inhaled corticosteroids use, body mass index (BMI), and co-morbidity. Blood was obtained and analyzed for complete blood count, renal function (blood urea nitrogen and creatinine), total calcium, phosphate, alkaline phosphatase, albumin, parathyroid hormone level, and high sensitivity C-reactive protein (hs-CRP).

Spirometry was performed according to the recommendation of American Thoracic Society[12] and reference values of Thai Thoracic Society[13]. Diagnosis of COPD and grading of severity were based on GOLD criteria[2].

Bone mineral density (BMD; g/cm^2) was conducted by using dual energy x-ray absorptiometry scan (DXA scan; Lunar DF + 15974 densitometer, Lunar (GE), Madison, WI, USA) at second to fourth lumbar spine (L2-4) and femoral neck. The BMD measurement by DXA scan is a gold standard and highly accurate technique according to World Health Organization (WHO) recommendation[7]. The BMD values were compared with a young normal control population of the same gender and race, and were expressed as a T-score. T-score values above -1.0 were normal, between -1.0 to -2.5 were osteopenia, and below -2.5 were osteoporosis. The lowest T-score at either L2-4 or femoral neck determined the diagnosis.

Outcomes

The primary outcome was the prevalence of osteoporosis and osteopenia in Thai COPD patients. Secondary outcome was the association between BMD and factors including age, BMI, tobacco smoking, inhaled corticosteroids use, post-bronchodilator FEV_1% predicted, and hs-CRP.

Statistical analyses

Sample size was 93 patients based on the prevalence of osteoporosis from a previous study[10] with power 90% at level of significance 0.05. All statistical analyses were performed using the SPSS software package, version 13 (Chicago, IL, USA). Continuous variables were presented as mean ± standard deviation (SD) and discrete variables were presented as percentage. Analysis of variance (ANOVA) was used to compare mean and Chi-square test was used to compare groups of non-discrete data. Regression analysis was used to assess whether any of the background variables were predictive of BMD. P-values < 0.05 were considered to be of statistical significance.

Results

One hundred two male COPD patients were enrolled. Patient characteristics are presented in Table 1. Mean age was 71.2 years and most of them were older than 70 years (60.8%). Average tobacco smoking was 41.8 pack years. Two-thirds of patients used inhaled corticosteroids for which the fluticasone equivalent dose was 667.8 mcg/day. Mean post-bronchodilator FEV_1 was 62.4%predicted and severity of COPD grading by GOLD criteria was 18.7% in stage I, 48.0% in stage II, 28.4% in stage III, and 4.9% in stage IV. Laboratory values included total white blood cell count, total calcium, phosphate, parathyroid hormone level were in normal range, excepted for hs-CRP, which was higher than normal value (5.9 ± 10.4 mg/L).

Prevalence of osteoporosis and osteopenia

BMD values of all COPD patients at L2-4 and femoral neck were 1.04 ± 0.20 and 0.83 ± 0.14 g/cm^2, respectively. T-score at L2-4 and femoral neck were -1.26 ± 1.59 and -0.83 ± 1.10, respectively.

Out of 102 patients, the prevalence of osteoporosis and osteopenia at L2-4 were 30.4% and 25.5%, respectively and the prevalence of osteoporosis and osteopenia at femoral neck were 7.0% and 35.0%, respectively. According to the lowest T-score at either

Table 1. Demographic and baseline patient characteristics

	n = 102
Age, years	71.2 ± 8.1
Age group, n (%)	
50-59	7 (6.9)
60-69	33 (32.3)
≥ 70	62 (60.8)
BMI, kg/m^2	21.4 ± 3.6
Tobacco, pack-years	41.8 ± 28.5
Inhaled Corticosteroid use, n (%)	73 (71.6)
Inhaled fluticasone equivalent dose, mcg/day	667.8 ± 253.6
Spirometric value	
Post-bronchodilator FEV_1, liter	1.3 ± 0.5
Post-bronchodilator FEV_1, % predicted	62.4 ± 24.1
% reversibility	9.4 ± 9.6
COPD staging, n (%)	
Stage I	19 (18.7)
Stage II	49 (48.0)
Stage III	29 (28.4)
Stage IV	5 (4.9)
hs-CRP, mg/L	5.9 ± 10.4

Values are given as mean ± SD and other values as number (%)
BMI = body mass index; FEV_1 = force expiratory volume at 1 second; hs-CRP = high sensitivity C-reactive protein

L2-4 or femoral neck determined the diagnosis, the overall prevalence of osteoporosis was 31.4% and osteopenia was 32.4%.

Factors associated with osteoporosis and osteopenia

The factors associated with osteoporosis were evaluated and the present study population was compared between osteoporosis and normal BMD groups (Table 2). BMI and hs-CRP were significantly associated with osteoporosis. No difference between age, post bronchodilator FEV_1, smoking, and inhaled corticosteroid use were found when compared in both groups.

To assess independent predictive values of BMI and hs-CRP by using ROC curve, cut-off values for BMI at below 20.5 kg/m^2 (AUC = 0.714, p = 0.002) and hs-CRP at above 2.3 mg/L (AUC = 0.642, p = 0.046) were used to determine risk of osteoporosis in COPD patients. From linear regression, BMI of lower than 20.5 kg/m^2 and hs-CRP of more than 2.3 mg/L have a crude odds ratio 5.6 (95% CI; 1.9, 16.3; p = 0.001) and 2.9 (95% CI; 1.1, 8.0; p = 0.033), respectively and the adjusted odds ratio for BMI < 20.5 kg/m^2 was 7.2 (95% CI; 2.2, 23.3; p = 0.001) and hs-CRP > 2.3 was 4.1 (95% CI; 1.3, 13.1; p = 0.019) (Table 3).

Discussion

The present study showed the high prevalence of osteoporosis in Thai male COPD patients (31.4%; 95% CI: 23.2, 40.9), and this was higher than the age-matched Thai male population from a previous study (12.6%)[14]. The prevalence of osteoporosis in COPD patients was also higher than that in the general Thai male population when the authors stratified by age group (50-59 years; 28.6% vs. 8.4%, 60-69 years; 24.2% vs. 10.6%, ≥ 70 years; 35.5% vs. 21%, respectively).

The prevalence of osteoporosis and osteopenia in the present study corresponded with three previous studies in COPD patients. The first study, Graat-Verboom L et al[11] showed the prevalence of osteoporosis was 21% and osteopenia was 41% in COPD patients and mean FEV_1 was 42.1% predicted. The second, Ferguson TG and colleagues from the

Table 2. Factors associated between osteoporosis and normal BMD groups

	Osteoporosis (n = 32) mean ± SD	Normal (n = 37) mean ± SD	p-value
Age, years	74.70 ± 8.20	71.00 ± 7.40	0.057
BMI, kg/m^2	20.00 ± 3.40	22.80 ± 3.10	0.005*
Post bronchodilator FEV1,% predicted	60.30 ± 27.10	65.00 ± 23.10	0.433
Smoking, pack years	51.00 ± 30.10	39.30 ± 29.50	0.109
Corticosteroid use, n (%)	26 (81.3)	27 (73.0)	0.599
hs-CRP, mg/L	8.81 ± 13.78	5.28 ± 8.88	0.046*

Values are given as mean ± SD and other values as number (%)
* p-value < 0.05

Table 3. Risk of osteoporosis according to BMI and hs-CRP

	Risk of osteoporosis			
	Crude OR	p-value	Adjusted OR+	p-value
BMI < 20.5, kg/m^2	5.6 (1.9, 16.3)	0.001*	7.2 (2.2, 23.3)	0.001*
hs-CRP > 2.3, mg/L	2.9 (1.1, 8.0)	0.033*	4.1 (1.3, 13.1)	0.019*

Values are odds ratio and 95% confidence intervals
+ Adjusted for BMI, and hs-CRP
* p-value < 0.05

TORCH study[15] found the prevalence of osteoporosis and osteopenia in moderate to severe COPD patients (mean FEV_1 44%predicted) were 23% and 42%, respectively. The last study, Jorgensen NR, et al[10] reported the prevalence of osteoporosis and osteopenia in severe COPD patients (mean FEV_1 32.6% predicted) were 44.8% and 22.3%, respectively. However, these studies included subjects from both genders, so that the prevalence of osteoporosis and osteopenia may be affected by gender because the prevalence of osteoporosis and osteopenia in females is high[16,17]. Indeed the most of COPD patients are male and the prevalence of osteoporosis in general male population is lower than in female. The present study, therefore, included only males because it wished to avoid a bias from high prevalence of osteoporosis in female. Interestingly, the present study population in the present study had a lesser degree of COPD (mean FEV_1% predicted was 64.6%) than three previous studies but the high prevalence of osteoporosis was similar to these studies (31.4% vs. 21 to 44.8%, respectively).

The present study demonstrated low BMI was associated with osteoporosis and corresponded with the previous studies by Vrieze A et al[18] which showed low BMI was a risk factor for developing osteoporosis (odd ratio 4.7), but they measured BMD by quantitative ultrasound at calcaneus which is not the gold standard technique. Graat-Verboom L et al[11,19] reported 12-fold increased risk of osteoporosis in cathetic COPD patients compared with normal BMD patients, whereas being overweight and obesity had a substantial protective effect. Furthermore, Bolton and colleagues[20] found the highest prevalence of osteoporosis (50%) and osteopenia (50%) was in cathetic COPD patients.

Systemic inflammation plays an important role in co-morbidity of COPD, and osteoporosis may be in part of the extrapulmonary effects. Mean value of hs-CRP in COPD patients from the present study was high, which can be explained from systemic inflammation, and supports the previous data that indicates COPD patients had a low grade of systemic inflammation by increasing of several inflammatory markers including CRP[3,4,21,22]. The authors used hs-CRP to determine the systemic inflammation in the present study because in several studies were shown level of CRP increased in COPD patients[5,23-26]. In the present study, COPD patients with osteoporosis had significantly higher hs-CRP level than normal BMD patients. This finding may support the concept of systemic inflammation contributed to pathogenesis of osteoporosis[27], and corresponds with a previous study[28] showing that higher level of hs-CRP was associated with lower BMD and higher levels of bone turnover markers.

No association was found between BMD and post-bronchodilator FEV_1 or severity of COPD. This result is different from the two previous studies[18,29] that showed BMD was significantly decreased when there is an increasing severity of COPD. In the present study, the number of subjects in each severity group was small, especially in GOLD stage III and IV, and that may explain why severity of COPD was not associated with BMD.

Osteoporosis has been a recognized side effect of long-term systemic corticosteroid therapy, but shows little or no effect from inhaled corticosteroids[30]. In the present study, no association between BMD and inhaled corticosteroids use was found; that corresponds with both the TORCH study and EUROSCOP study. In TORCH study[15], changes in BMD at hip and lumbar spine over 3 years were small and no significant differences between treatment arms of 658 patients with moderate to severe COPD, who received a placebo, salmeterol, fluticasone, or salmeterol/fluticasone twice daily. In EUROSCOP study[31], they did not find a significant change in BMD at L2-4 and femoral neck, in 912 mild COPD patients who received 800 mcg/day of budesonide or placebo for period of 3 years. Thus, long term inhaled corticosteroid therapy may have little or no effect on BMD in COPD patients.

In summary, the prevalence of osteoporosis and osteopenia in Thai COPD patients was higher than that in a normal Thai male population. Osteoporosis was associated with low BMI when compared COPD patients with normal BMD. Screening of BMD should be considered in COPD patients with BMI < 20.5 kg/m^2 and hs-CRP > 2.3 mg/L.

Acknowledgement

The present study was supported grant by Faculty of Medicine Siriraj Hospital, Mahidol University. The authors wish to thank Mr. Suthipol Udompanthurak for his contribution to the statistical analysis. The authors wish to thank Mrs. Tasneeya Suthamsamai, Ms. Kanokwan Ratanasaenglert, and staffs in the COPD clinic, Division of Respiratory Diseases and Tuberculosis, Department of Medicine Siriraj Hospital who greatly contributed to this study.

Potential conflicts of interest

None.

References

1. Lopez AD, Shibuya K, Rao C, Mathers CD, Hansell AL, Held LS, et al. Chronic obstructive pulmonary disease: current burden and future projections. Eur Respir J 2006; 27: 397-412.
2. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease [Internet]. Revised 2011 [cited 2011 Sep 15]. Available from: http://www.goldcopd.org
3. Barnes PJ, Celli BR. Systemic manifestations and comorbidities of COPD. Eur Respir J 2009; 33: 1165-85.
4. Gan WQ, Man SF, Senthilselvan A, Sin DD. Association between chronic obstructive pulmonary disease and systemic inflammation: a systematic review and a meta-analysis. Thorax 2004; 59: 574-80.
5. van Eeden SF, Sin DD. Chronic obstructive pulmonary disease: a chronic systemic inflammatory disease. Respiration 2008; 75: 224-38.
6. Karadag F, Karul AB, Cildag O, Yilmaz M, Ozcan H. Biomarkers of systemic inflammation in stable and exacerbation phases of COPD. Lung 2008; 186: 403-9.
7. World Health Organization. Prevention and management of osteoporosis: report of a WHO scientific group [Internet]. Geneva: WHO; 2003 [cited 2009 Jan 11]. Available from: http://libdoc.who.int/trs/WHO_TRS_921.pdf
8. Jorgensen NR, Schwarz P. Osteoporosis in chronic obstructive pulmonary disease patients. Curr Opin Pulm Med 2008; 14: 122-7.
9. Biskobing DM. COPD and osteoporosis. Chest 2002; 121: 609-20.
10. Jorgensen NR, Schwarz P, Holme I, Henriksen BM, Petersen LJ, Backer V. The prevalence of osteoporosis in patients with chronic obstructive pulmonary disease: a cross sectional study. Respir Med 2007; 101: 177-85.
11. Graat-Verboom L, Spruit MA, van den Borne BE, Smeenk FW, Martens EJ, Lunde R, et al. Correlates of osteoporosis in chronic obstructive pulmonary disease: An underestimated systemic component. Respir Med 2009; 103: 1143-51.
12. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, et al. Standardisation of spirometry. Eur Respir J 2005; 26: 319-38.
13. Dejsomritrutai W, Nana A, Maranetra KN, Chuaychoo B, Maneechotesuwan K, Wongsurakiat P, et al. Reference spirometric values for healthy lifetime nonsmokers in Thailand. J Med Assoc Thai 2000; 83: 457-66.
14. Pongchaiyakul C, Apinyanurag C, Soontrapa S, Soontrapa S, Pongchaiyakul C, Nguyen TV, et al. Prevalence of osteoporosis in Thai men. J Med Assoc Thai 2006; 89: 160-9.
15. Ferguson GT, Calverley PM, Anderson JA, Jenkins CR, Jones PW, Willits LR, et al. Prevalence and progression of osteoporosis in patients with COPD: results from the TOwards a Revolution in COPD Health study. Chest 2009; 136: 1456-65.
16. Vestergaard P, Rejnmark L, Mosekilde L. Osteoporosis is markedly underdiagnosed: a nationwide study from Denmark. Osteoporos Int 2005; 16: 134-41.
17. Limpaphayom KK, Taechakraichana N, Jaisamrarn U, Bunyavejchevin S, Chaikittisilpa S, Poshyachinda M, et al. Prevalence of osteopenia and osteoporosis in Thai women. Menopause 2001; 8: 65-9.
18. Vrieze A, de Greef MH, Wijkstra PJ, Wempe JB. Low bone mineral density in COPD patients related to worse lung function, low weight and decreased fat-free mass. Osteoporos Int 2007; 18: 1197-202.
19. Graat-Verboom L, van den Borne BE, Smeenk FW, Spruit MA, Wouters EF. Osteoporosis in COPD outpatients based on bone mineral density and vertebral fractures. J Bone Miner Res 2011; 26: 561-8.

20. Bolton CE, Ionescu AA, Shiels KM, Pettit RJ, Edwards PH, Stone MD, et al. Associated loss of fat-free mass and bone mineral density in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2004; 170: 1286-93.
21. Agusti A. Systemic effects of chronic obstructive pulmonary disease: what we know and what we don't know (but should). Proc Am Thorac Soc 2007; 4: 522-5.
22. Tkac J, Man SF, Sin DD. Systemic consequences of COPD. Ther Adv Respir Dis 2007; 1: 47-59.
23. Sin DD, Lacy P, York E, Man SF. Effects of fluticasone on systemic markers of inflammation in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2004; 170: 760-5.
24. Man SF, Connett JE, Anthonisen NR, Wise RA, Tashkin DP, Sin DD. C-reactive protein and mortality in mild to moderate chronic obstructive pulmonary disease. Thorax 2006; 61: 849-53.
25. de Torres JP, Pinto-Plata V, Casanova C, Mullerova H, Cordoba-Lanus E, Muros dF, et al. C-reactive protein levels and survival in patients with moderate to very severe COPD. Chest 2008; 133: 1336-43.
26. Dahl M, Vestbo J, Lange P, Bojesen SE, Tybjaerg-Hansen A, Nordestgaard BG. C-reactive protein as a predictor of prognosis in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2007; 175: 250-5.
27. Lehouck A, Boonen S, Decramer M, Janssens W. COPD, bone metabolism, and osteoporosis. Chest 2011; 139: 648-57.
28. Ding C, Parameswaran V, Udayan R, Burgess J, Jones G. Circulating levels of inflammatory markers predict change in bone mineral density and resorption in older adults: a longitudinal study. J Clin Endocrinol Metab 2008; 93: 1952-8.
29. Kjensli A, Mowinckel P, Ryg MS, Falch JA. Low bone mineral density is related to severity of chronic obstructive pulmonary disease. Bone 2007; 40: 493-7.
30. Goldstein MF, Fallon JJ Jr, Harning R. Chronic glucocorticoid therapy-induced osteoporosis in patients with obstructive lung disease. Chest 1999; 116: 1733-49.
31. Johnell O, Pauwels R, Lofdahl CG, Laitinen LA, Postma DS, Pride NB, et al. Bone mineral density in patients with chronic obstructive pulmonary disease treated with budesonide Turbuhaler. Eur Respir J 2002; 19: 1058-63.

ความชุกของโรคกระดูกพรุนและภาวะมวลกระดูกพร่องในผู้ป่วยโรคปอดอุดกั้นเรื้อรังในประเทศไทย

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วัตถุประสงค์: *เพื่อศึกษาความชุกของโรคกระดูกพรุนและภาวะมวลกระดูกพร่อง รวมถึงปัจจัยที่มีส่วนเกี่ยวข้องในผู้ป่วยโรคปอดอุดกั้นเรื้อรังในประเทศไทย*

วัสดุและวิธีการ: *เป็นการศึกษาแบบตัดขวางในผู้ป่วยโรคปอดอุดกั้นเรื้อรังเพศชายจำนวน 102 ราย วัดความหนาแน่นมวลกระดูกที่ตำแหน่งกระดูกสันหลังส่วนบั้นเอวที่ 2 ถึง 4 และที่ส่วนคอของกระดูกต้นขาโดยวิธี dual energy x-ray absorptiometer scan เก็บข้อมูลและวิเคราะห์ปัจจัยต่าง ๆ ได้แก่ อายุ เพศ ดัชนีมวลกาย การใช้ยาคอร์ติโคสเตอรอยด์ ชนิดสูดพ่น การสูบบุหรี่ ค่า force expiratory volume ที่ 1 วินาที และระดับ high sensitivity C-reactive protein (hs-CRP)*

ผลการศึกษา: *ความชุกรวมของโรคกระดูกพรุนและภาวะมวลกระดูกพร่องเมื่อประเมินโดยอาศัยค่ามวลกระดูกต่ำที่สุดที่กระดูกสันส่วนบั้นเอวหรือที่คอของกระดูกต้นขาตำแหน่งใดตำแหน่งหนึ่งเท่ากับร้อยละ 31.4 และ 32.4 ตามลำดับ ความชุกโดยรวมของโรคกระดูกพรุน ในผู้ป่วยโรคปอดอุดกั้นเรื้อรังมากกว่าประชากรไทยเพศชายในช่วงอายุเดียวกันจากข้อมูลการศึกษาในอดีต (ร้อยละ 31.4 เปรียบเทียบกับร้อยละ 12.6 ตามลำดับ) ค่าดัชนีมวลกาย และระดับ hs-CRP มีความสัมพันธ์กับการเกิดโรคกระดูกพรุนอย่างมีนัยสำคัญทางสถิติ ส่วนระดับความรุนแรงของโรคปอดอุดกั้นเรื้อรัง อายุ การสูบบุหรี่ การใช้ยาคอร์ติโคสเตอรอยด์ชนิดสูดพ่น ไม่สัมพันธ์กับการเกิดโรคกระดูกพรุน ดัชนีมวลกายที่น้อยกว่า 20.5 กิโลกรัมต่อตารางเมตร และระดับ hs-CRP ที่มากกว่า 2.3 มิลลิกรัมต่อลิตร พบเป็นปัจจัยเสี่ยงของการเกิดโรคกระดูกพรุนในผู้ป่วยโรคปอดอุดกั้นเรื้อรัง (ค่า adjusted Odds ratio เท่ากับ 7.2 และ 4.1 ตามลำดับ)*

สรุป: *ความชุกของโรคกระดูกพรุนและมวลกระดูกพร่องในผู้ป่วยโรคปอดอุดกั้นเรื้อรังในประเทศไทยสูงกว่าประชากรเพศชายปกติในช่วงอายุเดียวกัน โรคกระดูกพรุนมีความสัมพันธ์กับดัชนีมวลกายที่ต่ำและระดับ hs-CRP ที่สูง เมื่อเปรียบเทียบกับผู้ป่วยโรคปอดอุดกั้นเรื้อรังที่มีค่าความหนาแน่นมวลกระดูกปกติ*